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ANNUAL MEETING ABSTRACTS

2026 ACMT Annual Scientific Meeting Abstracts – Boston, Massachusetts

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DAY 1: PLATFORMS, ABSTRACTS 001-004

001. Association of Myocardial Systolic Function With In-Hospital Mortality in Patients With Drug Overdose-Associated Shock

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Background: While echocardiographic assessment of systolic function can guide resuscitation of patients with shock after acute drug overdose, the prognostic importance of systolic function in this setting is not well described.
Hypothesis or Research Question: We hypothesized that markers of systolic function including left ventricular ejection fraction (LVEF), left ventricular outflow tract velocity time integral (LVOT VTI), stroke volume index (SVI), and cardiac index (CI) would associate with excess mortality risk.
Methods: We conducted a retrospective chart review of adults (>18 years old) who received hemodynamic support (vasopressors, inotropes, or mechanical circulatory support) and transthoracic echocardiography (TTE) after suspected acute drug overdose, 2013—2023, at our institution. Patients with missing systolic function data or alternative causes of shock (e.g., sepsis) were excluded. In-hospital mortality was ascertained via chart review. Systolic function was categorized as normal (LVEF 50-70%), reduced (<50%), or hyperdynamic (>70%). Multivariable logistic regression adjusted for age, sex, prior heart failure or coronary artery disease, cardiac arrest pre-TTE, and drug of overdose to estimate associations between TTE variables and in-hospital mortality. Optimal cutoffs for maximizing sensitivity and specificity for in-hospital mortality were determined using receiver operating characteristic curves.
Results: After exclusions, 106 patients were included (median age 47.0 [IQR 31.0—55.8] years, 61 [57.6%] male, 25 [23.6%] with cardiovascular drug overdose). Forty-one (38.7%) patients died during hospitalization. The median LVEF was higher in survivors vs. non-survivors (59.0% vs. 50.0%, p=0.008). While individual LVEF values were not independently associated with in-hospital mortality (p=NS), having a reduced LVEF was independently associated with increased odds of in-hospital mortality (Reduced LVEF vs. normal LVEF, aOR 4.32, 95% confidence interval 1.16—18.5, p=0.04). Similarly, LVOT VTI was independently and inversely associated with in-hospital mortality (aOR per 1-cm increase in LVOT VTI: 0.82, 95% confidence interval 0.67—0.94, p=0.01). The optimal LVOT VTI and LVEF cutoffs for identifying those at risk for in-hospital mortality were 13 cm and 47%, respectively. SVI and CI were not independently associated with in-hospital mortality (p=NS).
Conclusion: In overdose-associated shock, systolic function as determined by LVEF phenotype and LVOT VTI on TTE, were independently associated with risk for in-hospital mortality. Further research is warranted to characterize the use of TTE for risk stratification in overdose. Limitations include retrospective design, varied echocardiogram timing and hemodynamic support, and absence of routine confirmatory drug testing.

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002. Hemodynamic Effects of Xylazine and Medetomidine in ED Patients with Opioid Overdose

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Background: Alpha2-adrenergic agonists (A2A), including xylazine and medetomidine, have emerged as novel synthetic opioid supply adulterants. Prior studies have linked qualitative A2A detection to clinical outcomes in emergency department (ED) overdose patients, but few have examined relationships between blood concentrations and clinical findings.
Hypothesis or Research Question: Among ED patients with opioid overdose, (1) what are the median blood concentrations of fentanyl, xylazine and medetomidine; (2) what demographic characteristics are associated with xylazine/medetomidine detection; and (3) what clinical parameters and outcomes are associated with xylazine/medetomidine detection?
Methods: We conducted a prospective observational cohort study at two New York City EDs from April-October 2025. Patients with suspected opioid overdose who had blood collected during clinical care were enrolled. Waste serum/plasma was de-identified and analyzed at the Center for Forensic Science Research and Education (CFSRE) using liquid chromatography quadrupole time-of-flight mass spectrometry (LC-QTOF-MS) to quantify fentanyl, xylazine, medetomidine, and their metabolites. Data from the index visit and 30-days pre/post visits were extracted from the electronic health record. LC-QTOF-MS results were linked to clinical data. Statistical analyses were performed using SAS.
Results: Of 912 patients screened, 134 (14.6%) met inclusion criteria. 78 (58.2%) had blood samples available and 53 samples from one site were analyzed. Xylazine was detected in 60.4% and medetomidine in 39.6% of samples. 64.2% had ≥ 1 A2A detected and 35.8% had both. Median xylazine concentration was 0.1 ng/ml (IQR 1.2). Median medetomidine concentration was 0.0 ng/ml (IQR 1.5). No metabolites were detected. A2A positive patients were predominantly 60+ years old (58.8%), male (85.3%), and had Medicare (35%) or Medicaid (28.6%). Patients with any A2A detected had lower initial heart rate [HR] (91 vs 66 bpm, p=0.004) than those without A2As. Patients with both A2As also had lower initial HR (91 vs 66 bpm, p=0.0006). After stratifying into high and low concentration groups, patients with high xylazine concentrations had lower HR (66 vs 80 bpm, p=0.0096), higher systolic blood pressure [SBP] (141 vs 127 mmHg, p=0.022), and higher diastolic blood pressure [DBP] (93 vs 75 mmHg, p=0.0035) compared to low concentrations. Patients with high medetomidine concentrations also had lower HR (79.5 vs 59.5 bpm, p=0.0014), higher SBP (128.5 vs 142.5 mmHg, p=0.049) and higher DBP (75 vs 91.5, p=0.0051).
Conclusion: In this first-of-its-kind study of ED opioid overdose patients, confirmatory A2A concentrations were low. A2A overdoses were common in elderly males and associated with lower initial HR and higher initial SBP and DBP.

003. Detecting Cannabis Intoxication and Impairment Using Wearable Sensors

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Background: Cannabis decriminalization/legalization has contributed to dramatic increases in the availability and recreational use of cannabis products. Unlike ethanol, there are no standard validated assessments to identify cannabis induced intoxication and impairment. Wearable sensors could offer a way to objectively identify cannabis intoxication.
Hypothesis or Research Question: Wearable sensors are a feasible and acceptable method of assessing cannabis intoxication in healthy participants.
Methods: This observational pilot study evaluated participants who smoked their personal cannabis products purchased from licensed dispensaries in Colorado. Participants recruited through Reddit advertisements presented to the Colorado State University campus for consenting, baseline testing, and distribution of a wrist-worn sensor (Empatica EmbracePlus). This device collected heart rate (HR), skin temperature, electrodermal activity (EDA), and accelerometry data continuously throughout the study

period. Participants then went home and were monitored by study staff using a Zoom videoconference. Participants were assessed at timepoints T0, T1, and T2 (roughly 25 minutes between timepoints) using the DRUID impairment application, 2-minute sit/stand/walk exercises, and Subjective High Assessment Scale (SHAS) surveys. Participants smoked their cannabis products ad libitum during two sessions 10-minutes before time points T1 and T2. The biometric data, DRUID scores, and subjective intoxication scores were analyzed using descriptive statistics, repeated measures analysis of variance (RMANOVA), repeated measures correlation, and paired one-sample t-tests to determine potential associations and correlations. Acceptability was measured using a 2-question survey.

Results: 154 participants were screened, 20 consented and completed the study protocol. Eleven were males, mean age 32.1 (range 22-58) years. Twelve participants smoked before T1 and T2 and eight participants only smoked before T1. DRUID scores and cumulative SHAS demonstrated significant increases across timepoints ($p=0.003$ and $p<0.001$ respectively). There was a significant increase in HR from baseline during free activity periods prior to all timepoints ($p<0.001$), with strong correlations with changes in DRUID scores ($r=0.75$, $p<0.001$) and SHAS ($r=0.57$, $p<0.001$). 2-minute sit/stand/walk testing also showed significant HR increases at T1 (p's<0.001) and T2 (p's<0.01). EDA significantly decreased during all activities between T1 and T2 (p's<0.05). There were no significant changes in temperature or aggregate accelerometry data between activity timepoints. Use of this technology was found acceptable by >75% of participants.

Conclusion: This study demonstrates it is feasible and acceptable to use wearable sensors to detect signs of cannabis intoxication. HR and EDA exhibited significant changes following cannabis use. Changes in HR correlated with significant increases in subjective and objective assessment of impairment.

Medical Toxicology Foundation: This research was supported by a 2024 MTF Innovative Research, Teaching, & Practice Grant.

004. Human Pharmacokinetics of Fentanyl, Xylazine, and Medetomidine in Patients Presenting to the Emergency Department After Non-Fatal Opioid Overdose

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Background: Xylazine and medetomidine are veterinary anesthetics and alpha-2 agonists commonly found as adulterants in the illicit fentanyl supply in the United States (US). The pharmacology of xylazine is well-understood in animals, but its pharmacokinetics are poorly characterized in people. Understanding xylazine and medetomidine pharmacokinetics in humans, especially in the context of fentanyl overdose, could assist in resuscitation efforts for individuals exposed to these drugs.

Hypothesis or Research Question: Our objective was to elucidate the pharmacokinetics of these substances present among adults who received care at an emergency department (ED) after an acute, non-fatal, opioid overdose.

Methods: Adults (≥ 18 years old) presenting to an urban, academic US ED were recruited following a suspected opioid overdose with concomitant xylazine exposure. The presence of xylazine was verified using a point-of-care urine test in the ED. Serial blood samples were obtained at 30-min to 1-hour intervals and collected in gray top collection tubes containing sodium fluoride and potassium oxalate. Blood samples were analyzed for fentanyl and its metabolite norfentanyl; xylazine and its metabolites 2,6-xylidine, 1-(2,6-xylyl)-2-thiourea, 3-hydroxy xylazine, and 4-hydroxy xylazine; and medetomidine and its metabolite 3-hydroxy medetomidine. Analyte concentrations were quantified using a Waters® Acquity UPLC coupled with a Waters Xevo® TQ-S micro liquid chromatograph tandem quadrupole mass spectrometer (LC-QQQ-MS). Pharmacokinetic profiles for fentanyl, norfentanyl, and xylazine were determined.

Results: Thirteen participants were included in the study. Initial blood concentration (mean±SD) was 15±14 ng/mL for fentanyl, 23±19 ng/mL for norfentanyl, and 46±53 ng/mL for xylazine. 3-Hydroxy xylazine and 1-(2,6-xylyl)-2-thiourea were the only xylazine metabolites detected in blood, with concentrations ranging from <1-5 ng/mL and 5-20 ng/mL, respectively. Medetomidine was detected in four patients (48±29 ng/mL). Among the eight participants who provided five or more consecutive blood samples, the respective half-lives of fentanyl, norfentanyl and xylazine were: fentanyl (537±451 mins), norfentanyl (472±188 mins), and xylazine (345±145 mins). Our findings show that initial blood xylazine and medetomidine concentrations were about three times higher than those of fentanyl in a cohort of patients exposed to these substances. Xylazine half-life was determined to be about 6 hours, whereas fentanyl half-life was somewhat longer at about 9 hours.

Conclusion: Overall, these findings provide key information about the pharmacokinetics of xylazine, medetomidine, and fentanyl in people who use opioids and can inform post-overdose resuscitation protocols.

DAY 1: MODERATED POSTERS, ABSTRACTS 005-011

005. Number of Vials of Anti-Venom to Achieve Control in Rattlesnake Envenomations

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Background: In recent years, both Crotalidae polyvalent immune Fab (ovine) (CroFab) and Crotalidae Immune F(ab')2 equine (Anavip) is approved for treatment of North American pit vipers. There is limited post-marketing data on the number of vials of anti-venom needed to achieve clinical control after rattlesnake envenomation.
Hypothesis or Research Question: Both Fab and F(ab')2 will be equally effective in achieving control.
Methods: The North American Snakebite Registry, a prospective sub-registry of the ToxIC Core Registry, collects detailed clinical information on snake envenomations that receive a medical toxicology consultation. This analysis evaluated the number of anti-venom vials required to achieve clinical control following a rattlesnake envenomation. An additional question on clinical control after each anti-venom administration was added in January 2021. Therefore, cases entered before 2021, as well as those cases who did not achieve control, those where the number of vials administered before control achieved was not documented, and those who received both anti-venoms were excluded. Clinical control was defined based on the treating toxicologist's recorded assessment. Multivariable regression was performed. The number if vials were converted in a 2:1 ratio to facilitate comparison (2 vials Anavip equivalent to one vial CroFab).
Results: A total of 250 cases met eligibility, including 60 patients (24%) who received Crofab alone and 190 (76%) who received Anavip alone. The median and interquartile range (IQR) adjusted number of vials of Anavip and CroFab were 8 (IQR 5-12) and 6 (IQR 6-10). After adjusting for age, sex, bite location, and pre-envenomation use of anticoagulants and anti-platelet agents, CroFab was associated with less number of vials to achieve clinical control (-2.46; 95% CI: -3.78, -1.14).
Conclusion: CroFab required significantly fewer vials to achieve clinical control than Anavip, suggesting potential differences in dosing requirements between the two antivenoms.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

006. Comparing Patient Outcomes in Crotalidae Polyvalent Immune Fab Dosing Strategies in Eastern Copperhead (Agkistrodon Contortrix) Envenomation: A Regional Poison Center Review

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Background: It is unclear if the use of maintenance doses of Crotalidae polyvalent immune fab antivenom (FabAV) as directed in the package insert leads to better patient outcomes compared to using loading doses only in eastern copperhead (*Agkistrodon contortrix*) envenomation.
Hypothesis or Research Question: What are the differences in outcomes of FabAV dosing strategies based on poison center recommendations?
Methods: This is a retrospective review of copperhead envenomations reported to the regional poison center from 1/1/04 to 10/22/24. Patients were seen in a healthcare facility, given FabAV, and had documentation of receiving loading dose(s) (LD) with or without maintenance dose(s) (MD). LD was defined as a bolus dose of FabAV used to gain control of symptoms. MD was defined as a dose given after envenomation control was established. Cases were excluded if no envenomation occurred, Crotalidae immune $F(ab')_2$ was administered, the patient did not receive an initial LD of four vials, incomplete documentation, or the case was lost to follow up. The primary objective was to compare length of stay (LOS) and snake severity score (SSS) between patients who received LD and MD (LD+MD Group) of FabAV and patients who only received LD (LD Group) of antivenom. The SSS was categorized as mild (≤ three), moderate (four - seven), or severe (≥ eight). Descriptive statistics were used to summarize the primary objective. Association between the groups and LOS was determined with a Mann-Whitney U test. Association between groups and SSS severity was

determined with a Fisher's exact test. Other categorical data was analyzed using a Fisher's exact test.

Results: 251 cases met study criteria. 143 patients were in the LD Group while 108 patients were in the LD+MD Group. Median LOS was 1.60 days (IQR 1.00 - 2.05 days) in LD Group, and 2.05 days (IQR 1.57 - 2.75 days) in LD+MD Group ($p<0.001$). 14.0% in LD Group and 16.7% LD+MD Group had moderate severity (NS). No patient had severe severity. 10.5% in LD Group and 13.0% in LD+MD Group received multiple LDs (NS). 71.3% in LD Group and 74.1% LD+MD Group received opioids (NS).

Conclusion: This study demonstrates that the omission of FabAV maintenance doses may result in shorter LOS. Major limitations are cases were limited to those that were reported to the regional poison center and thus does not reflect dosing strategies of other regions, and majority of cases were mild severity.

007. False-Positive and False-Negative SEFRIA Fentanyl Immunoassay: A Retrospective Single-Center Review

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Background: Fentanyl immunoassays are frequently used for urine drug screening but may yield false-positive and false-negative results. Understanding the rate and causes of false-positive and false-negative screens may ameliorate diagnostic confusion, unnecessary investigations, and psychosocial consequences.

Hypothesis or Research Question: What is the frequency of false-positive and false-negative fentanyl immunoassay results at our institution, and which substances are most associated with these false positives?

Methods: We performed a retrospective chart review at a pediatric tertiary care center between January 1 and Jun 30, 2024. We included all patients who tested fentanyl or norfentanyl positive in either the immunoassay or confirmatory drug testing. Qualitative urine fentanyl screen was tested by the SEFRIA Fentanyl assay on Abbott Alinity c analyzer with limit of detection (LOD) at 1.0 ng/mL. Paired confirmatory toxicology testing for many agents was done on Sciex 4600 Triple Quad-Time of Flight (QTOF) mass spectrometer (Sciex) with LOD at 2.5 ng/mL for fentanyl and 5.0 ng/mL for norfentanyl. Descriptive analysis was performed of fentanyl, norfentanyl, and all QTOF positive agents. Primary outcomes were the percentages of fentanyl immunoassay false-positives and false-negatives. Secondary outcome was the frequency of agents associated with false-positives.

Results: 74 patients met inclusion criteria. 41 had positive fentanyl immunoassay results. Of those, 15 (36.6%) were negative on confirmatory testing. 61 patients tested positive for fentanyl or norfentanyl on QTOF, and of those, 26 (42.6%) also had a positive immunoassay (thus, 57.4% were false negatives). Sensitivity was 62%, and positive predictive value was 41%. Among the 41 patients with positive fentanyl immunoassay and negative QTOF, the most frequently detected substances included: trazodone (7), cannabinoids (6), hydroxyzine (6), quetiapine (5), venlafaxine (5), levetiracetam (4), and midazolam/metabolites (4). 24 other agents were detected (Figure 1).

Conclusion: Our study demonstrates high false-positive and false-negative rates of fentanyl urine immunoassays at our institution. Trazodone, the most common drug detected in patients with a presumed false-positive immunoassay, is noted in the SEFRIA package insert to have cross-reactivity. Various antipsychotic and antidepressant medications are also noted by the manufacturer to have cross-reactivity, which is reflected in our study. A limitation of this study is that the fentanyl immunoassay has a lower LOD than the QTOF, leading to potential overestimation of false-positivity. Due to the commonality of high-false positive and high-false negative tests, results should be confirmed when making important clinical decisions, and further investigation is warranted.

008. Efficacy of Fab vs. Fab2 for Patient-Centered Outcomes After Rattlesnake Envenomation in the North American Snakebite Registry

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Background: Superior efficacy of Fab vs Fab_2 antivenom for prevention of tissue toxicity after North American snakebite is debated. 'Time to control', often used to assess antivenom efficacy, may not reflect ultimate outcomes. Patient-centered outcome data is needed.

Hypothesis or Research Question: There is no difference in functional deficit or permanent tissue loss after Fab vs. Fab_2 for treatment of rattlesnake envenomation in the North American Snakebite Registry (NASBR).

Methods: This analysis is based on Toxicology Investigators Consortium's NASBR data 2013-2024 comparing

patient-centered outcomes after Fab vs Fab_2 following rattlesnake envenomations. Cases with delayed antivenom >12 hours or administration of both antivenoms were excluded. Primary outcomes were functional deficits or tissue loss on follow-up. Secondary outcomes included necrosis, surgical procedures, or suspicion of compartment syndrome. Fab and Fab_2 groups were compared using Chi square/Fisher's exact test for categorical variables and Mann-Whitney U test for continuous variables. Logistic regression was used to compute adjusted associations between Fab vs. Fab_2 for outcomes with sufficient sample sizes (functional deficit and necrosis).

Results: 785 cases were included for analysis. 559 (71.2%) received Fab and 226 (28.8%) Fab_2. Baseline patient and envenomation characteristics were similar between groups. Time to antivenom was 2.0 vs. 2.2 hours for Fab and Fab_2, respectively. Median follow-up time was 9 days (IQR 5-14) for Fab and 14 days (IQR 8-19) for Fab_2. There was no difference in functional deficit between groups (Fab 59/479, 12.3% vs. Fab2 19/217, 8.8%; p=0.168). Permanent tissue loss was reported in 5/437 (1.1%) Fab cases and 4/213 (1.9%) Fab_2 cases (p=0.4845). Tissue loss included three skin grafts and one fingertip deformity after Fab and two skin grafts and two-digit amputations after Fab_2. Necrosis was more common after Fab_2 (50/559, 8.9% Fab vs. 34/226, 15.0% Fab_2; p=0.0123). Procedures including dermotomy, fasciotomy, and debridement were similar in both groups (Fab 6.98%, Fab_2 4.87%; p=0.2732). See Table 1. After adjusting for bite location, time to antivenom, use of tourniquet or suction, and antivenom type, presence of a digit bite was significant for functional deficit (Odds Ratio [OR]: 2.51; 95% CI: 1.55, 4.11). Digit bite (OR: 6.98; 95% CI: 4.11, 12.42) was associated with increased odds of necrosis; Fab was associated with reduced odds of necrosis (OR: 0.56; 95% CI: 0.34, 0.93).

Conclusion: There was no difference in functional deficit or permanent tissue loss after Fab vs Fab_2 for rattlesnake envenomations in NASBR. Fab compared to Fab_2 was associated with a reduced odd of necrosis.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

009. Electroencephalogram Findings in Patients With Suspected Toxicologic Exposures

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Background: Seizures associated with toxicologic exposures pose diagnostic and therapeutic challenges. Electroencephalography (EEG) is frequently used in this setting despite uncertain impact on clinical decision-making and resource utilization, such as anticonvulsant strategy, ICU admission, and clinical course prognostication. This study aims to determine the clinical utility of EEG in patients with toxic exposures, the first of its kind encompassing multiple hospital systems.

Hypothesis or Research Question: Epileptiform activity is largely confined to patients with witnessed clinical seizures or a preexisting seizure disorder.

Methods: This was a retrospective chart review at two urban teaching hospitals, from 10/24/2015 to 12/29/2022, with IRB approval. Inclusion criteria included adults (≥18 years) who had toxicology consultation and underwent EEG during the same clinical encounter identified via procedure codes. EEG findings of interest were defined as discharges consistent with epileptiform activity (EA). Descriptive statistics summarized the cohort. Chi-square test with Yates correction was used for differences in proportions across demographics.

Results: A total of 214 patients (median age=41, 50.9% male) met inclusion criteria and were classified into four subgroups: clinical seizure with EA (n=14, 6.5%), clinical seizure without EA (n=71, 33%), no clinical seizure with EA (n=7, 3.3%), and no clinical seizure without EA (n=122, 57%). Eighty-five patients (40%) experienced a witnessed clinical seizure prior to or at presentation; among these, 14 (16%) exhibited EA on EEG, while 71 (84%) did not. Of patients with clinical seizures who had EA on EEG, 5 (36%) died. All patients who experienced clinical seizures with EA and survived their hospital stay (n=9, 4.2%) were discharged on antiepileptic drugs (AEDs). The majority of these (n=7, 78%) had a previously reported underlying seizure disorder. EA was significantly more likely among patients with a prior history of seizures (p=0.029). EA was significantly less likely for GTC seizure types than focal or myoclonic types (p=0.001).

Conclusion: EEG can aid in detecting epileptiform activity after toxicologic exposures; 21 patients (9.8%) included in this study had EA. A notable proportion of patients without clinically observed seizures nonetheless exhibited EA on EEG, highlighting a potential gap between EEG findings and clinical presentation. The association between EA, prior seizure history, and seizure subtype supports consideration of individualized EEG deployment. Furthermore, routine use of EEG may have limited impact on long-term management decisions in those with established seizure disorders, prompting a reassessment of its clinical utility in this context.

010. Therapeutic Approaches in Beta-Blocker and Calcium Channel Antagonist Poisonings: Findings From the Toxicology Investigators Consortium Core Registry

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Background: Beta-blockers (BB) and calcium channel antagonists (CCA) are potentially lethal in overdose. Multiple therapies are used to treat these poisonings and there is considerable clinical heterogeneity in their management.
Hypothesis or Research Question: What therapies do medical toxicologists use to treat patients with BB and CCA poisoning?
Methods: This analysis consists of data from the Toxicology Investigators Consortium (ToxIC) Core Registry. The ToxIC Core Registry was queried for cases involving acute BB or CCA exposures reported from January 2017 to September 2025. Cases were excluded if they involved chronic exposures, if signs or symptoms were not present, if the signs/symptoms were reported by the toxicologist as unlikely to be related to the toxic exposure, or if the relation between signs/symptoms and the toxic exposure was unknown. We used Chi-Square Tests of independence to compare receipt of toxicologic treatments across the three exposure groups (BB, CCA, BB/CCA mix). When results were significant, we then conducted post-hoc pairwise comparisons between groups using chi-square tests with Bonferroni correction in order to avoid type I error.
Results: Nine hundred sixty-one cases were included. Five hundred eighty-three were BB exposures, 255 were CCA exposures, and 123 were combined BB and CCA exposures. Coingestants were common (N=679, 70.7%). Toxicological treatment was administered to 842 patients (87.6%). The most common treatment was intravenous (IV) fluid resuscitation (N=637, 66.3%), followed by vasopressors (N=382, 39.8%) and glucagon (N=263, 27.4%). Hyperinsulinemic-euglycemic therapy (HIET) was administered to 140 patients (14.6%) and intravenous lipid emulsion (ILE) to 54 patients (5.6%). Extracorporeal membrane oxygenation (ECMO) was initiated for 33 patients (3.4%). BB poisoning was more frequently treated with glucagon than CCA poisoning (31.9% vs. 13.3%, p< 0.001), while CCA poisoning was more frequently treated with HIET (24.7% vs. 7.7%, p< 0.001), ILE (9.4% vs. 3.6%, p=0.003), and/or vasopressors (47.8% vs. 31.7%, p< 0.001).There was no significant difference in IV fluid resuscitation across groups (p=0.106). ECMO was used more frequently for CCA (6.7%) and CCA-BB (8.1%) poisoning than BB poisoning (6.7% vs. 1% and 8.1% vs. 1%, respectively, p<0.001 for both), but there was no statistically significant difference in ECMO usage between CCA and CCA-BB poisoning.
Conclusion: In the ToxIC Core Registry, BB and CCA poisoning are most often treated with IV fluid resuscitation and vasopressors. Glucagon is used more frequently in BB poisoning, while HIET, ILE, vasopressors, and ECMO are used more frequently in CCA poisoning.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

011. What Best Predicts the Need for Liver Transplantation in Children Following an Acetaminophen Overdose?

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Background: Acetaminophen is the most common cause of xenobiotic-induced liver failure in the Western world and may necessitate liver transplant. Historically, the King's College Criteria are utilized in adult patients to predict the need for liver transplantation, although a single episode of hypoglycemia may be a better predictor. This study was conducted to determine which is the best predictor of the need for a liver transplant in pediatric patients following an acetaminophen overdose.
Hypothesis or Research Question: Both the King's College Criteria and hypoglycemia can adequately predict the need for liver transplantation in pediatric patients.
Methods: We conducted a retrospective chart review of all pediatric patients (age <18) admitted to one of six tertiary care medical centers with active toxicology services with a diagnosis of either liver failure or acetaminophen toxicity. All records were reviewed to determine which patients had liver injury (AST or ALT > 1000 IU/L), which was determined by the treating clinicians to be due to acetaminophen toxicity. Patients with liver failure from other etiologies as well as children with acetaminophen exposure without liver

injury were excluded. Sensitivity analysis and likelihood ratios were determined for both the King's College Criteria and hypoglycemia (defined as glucose < 60 mg/dL at any time, or < 65 mg/dL while on a 10% dextrose containing solutions). A composite endpoint of death or liver transplant was employed.

Results: A total of 202 patients were identified. Seven patients met the composite endpoint. Twenty-six patients met the King's College Criteria for transplant. The King's College Criteria were 85% sensitive (95% CI [42.1-99.6%]) and 89% specific (95% CI [84.6-93.6]). The negative predictive value was 99.4 (95% CI [96.9-100%]). Sixteen patients had hypoglycemia. The sensitivity of hypoglycemia for predicting the composite endpoint was 83.3 (95% CI [35.9-99.6%]) with a specificity of 93.6 (95% CI [88.9-96.8%]). The negative predictive value was 99.4% (95% CI [96.6-100%]).

Conclusion: Both hypoglycemia and the King's College Criteria had similar test characteristics for predicting the composite endpoint, and either may be used to predict the need for liver transplantation in pediatric patients.

DAY 1: POSTERS, ABSTRACTS 012-076

012. Not Your Typical "Kratom": 7-Hydroxy Mitragynine, Mitragynine Pseudoindoxyl, and Related Mitragynine Alkaloids Appearing in Smoke Shop Products and Intoxication Events

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Background: Mitragynine (MG) is a plant alkaloid and the primary psychoactive component of Kratom (Mitragyna speciosa). 7-Hydroxy mitragynine (7OHMG) is a structurally similar alkaloid found naturally in Kratom but in small amounts. MG is metabolized to 7OHMG and further to mitragynine pseudoindoxyl (MP). In 2016, the U.S. DEA decided not to schedule MG and 7OHMG, leaving Kratom alkaloids uncontrolled. In recent years, concurrent with the rise of "smoke shops", commercial sale of Kratom products, high dose MG preparations, and now marketed 7OHMG and MP products have surged. These products include tablets, concentrates, extracts, edibles (e.g., gummies, ice cream cones), and beverages.

Hypothesis or Research Question: What MG alkaloids are appearing in this newly diverse "Kratom" market and what impacts are they having on consumers? This presentation will showcase results from drug products and forensic investigations involving MG alkaloid labeled products.

Methods: Drug products (e.g., powder, pills) were analyzed by GC-MS and LC-QTOF-MS. Biological specimens underwent comprehensive toxicological analysis by LC-QTOF-MS, including quantitation of Kratom alkaloids. Data processing included targeted, concurrent spectral identification of MG, 7OHMG, MP, and dihydro 7-hydroxy mitragynine (aka MGM-15), as well as isomers (e.g., speciogynine, speciociliatine, mitraciliatine) and additional alkaloids (e.g., paynantheine, ajmalicine, mitraphylline, corynantheidine).

Results: Drug products marketed as containing 7OHMG and/or MP are continuing to be tested. Most products contained 7OHMG as the primary component; one product contained MP as the primary component. All products contained at least detectable amounts of MG, as well as other alkaloids; however, ratios of substances varied.

In early 2025, our laboratory began receiving inquiries about cases suspected of being 7OHMG intoxications. To date, more than 30 cases with history of 7OHMG product ingestion were tested. All cases tested positive for 7OHMG, in addition to varying levels of MG and MP (similar to drug product results). In one case, a decedent was found in bed alongside a product labeled "Ohms". The decadent had no history of illicit drug use. Autopsy showed significant pulmonary congestion, foamy fluid, and no other major abnormalities. Toxicology testing reported 7OHMG (20 ng/mL), MP (140 ng/mL), and MG (<5 ng/mL). The manner of death was accident, and the cause of death was probable 7OHMG toxicity.

Conclusion: 7OHMG and MP are reportedly 10x and 100x more potent than MG, respectively. Testing for these two alkaloids is extremely limited based on published reports, and 7OHMG exhibits poor stability, especially in biological matrix. Intoxication events involving these kratom alkaloids are increasing yet under-investigated.

013. Diagnosis and Management of Alcoholic Ketoacidosis

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Background: Alcoholic ketoacidosis (AKA) is characterized by metabolic derangements amidst or after cessation of excessive alcohol use. Recommended treatments include dextrose containing intravenous fluids and thiamine.

Hypothesis or Research Question: Diagnosis and use of recommended treatments for alcoholic ketoacidosis will occur in < 50 % of patients.

Methods: We performed a three-month (1/12023-3/31/23) retrospective chart review of adults (age 18 and older) presenting to the ED with chief complaints or diagnoses related to "alcohol withdrawal" or "detoxification" using the SlicerDicer feature of our electronic medical record (EMR),

Epic (Epic Systems Corporation; Verona, WI). We excluded patients with a confirmed acute medical condition including diabetic ketoacidosis, seizure, pancreatitis, sepsis, pulmonary embolism, medication overdose, or intracranial hemorrhage. We excluded patients with withdrawal or overdose of other substance. Patients with an alcohol-related visit to an ED in the past month were excluded. AKA was defined as the presence of an elevated anion gap and the presence or ketonuria in these patients with known alcohol use. We analyzed the ED physician note for documentation of alcoholic ketoacidosis. We searched the medication administration record for use of dextrose and thiamine.

Results: After screening, 203 patients were identified. 42 (21%) had both an elevated anion gap and ketonuria. In six (6/42, 14%) of those meeting our definition of AKA, the diagnosis was made in the ED. A majority (64%) were admitted, twenty for >48 hours, eight to the intensive care unit, and three to a step-down unit. Compared to all patients (83/203, 41%), more with AKA were admitted (p=.006). A majority had an elevated ethanol concentration (median: 124 mg/dL, IQR: 58-276). Nine patients (21%) were discharged with a median length of stay of 412 minutes. Seventeen (40%) AKA patients received thiamine and seven (17%) dextrose. Only 3 patients in whom AKA was diagnosed at time of visit received either thiamine or dextrose, none both.

Conclusion: A minority of patients received recommended treatments for AKA and fewer had the diagnosis documented in the EMR. Though recognition may improve treatment, even if diagnosed, thiamine and/or dextrose was only given in half of cases. Not all cases may require treatments and their benefit in improving clinically relevant outcomes needs to be further explored.

014. Extract or Not? Examining the Presence of Different Alkaloids Found in Kratom Products

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Background: Kratom is a leaf product derived from the tropical tree *Mitragyna speciosa*, known for its stimulant or sedative properties depending on the amount consumed. An estimated 1.9 million Americans used Kratom in 2022, often to help manage opioid withdrawal or pain symptoms. However, the growing availability of products containing isolated 7-hydroxymitragynine (7-HMG) at levels higher than naturally occurring in kratom leaves and mitragynine pseudoindoxyl (MGPI), which is not naturally found in leaves, has raised safety concerns.

Hypothesis or Research Question: What alkaloid compounds are found in kratom products sold in the Boston area?

Methods: An analytical survey was conducted to examine the alkaloid composition of kratom products. Using a novel ultra-performance liquid chromatography–tandem mass spectrometry (UPLC-MS/MS) method, we quantified 12 kratom alkaloids: 7-HMG, MGPI, mitragynine (MTG), paynantheine (PAY), speciogynine (SPG), speciociliatine (SPC), mitraciliatine (MTC), isopaynantheine (IsoPAY), corynantheidine (CORY), corynoxine A (COX-A), corynoxine B (COX-B), and mitraphylline (MTP). Various commercial kratom products were purchased from retailers around the Boston area, including 3 powder, 13 tablet/gummy, and 15 liquid products were tested.

Results: Of the 31 kratom products tested, the most commonly detected alkaloids were MTG (93.5%), 7-HMG (67.7%), MGPI (64.5%), CORY (61.3%), and COX-A (58.1%). All three Remarkable Herbs Maeng Da powders (Green, White, and Red) contained an alkaloid profile comprising of 10 of 12 tested alkaloids, missing MGPI and MTP. Gummy/tablet products contained a mean of 4.92 [2.36] alkaloids, and liquid products contained a mean of 6.07 [2.99] alkaloids. Some gummy/tablet products contained up to 10 of 12 alkaloids (missing MGPI and MTP), whereas other gummy/tablet products only contained 2 of 12 alkaloids (containing 7-HMG and MGPI). Liquid products showed similar variability with some containing up to 10 of 12 alkaloids (7-HMG and MGPI), while others only had a singular alkaloid (MGPI).

Conclusion: This study demonstrates the high variability in alkaloid composition in kratom products available for sale around the Boston area. While MTG and 7-HMG were found in a majority of products, many products also contained MGPI which is not naturally found in kratom leaves. The high variation in the number of alkaloids detected in kratom gummy, tablet, and liquid products highlights the lack of product standardization and may indicate some products being more natural than others. Given that kratom alkaloids act on opioid receptors, these findings emphasize the need for greater regulation and accurate product labeling.

015. Mind the Gap: Severe Mixed Ethanol-Isopropanol Toxicity and Alcohol Withdrawal in a Pediatric Patient

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Background: Isopropanol is a toxic alcohol which can result in life-threatening toxicity with hypotension and ketosis. Cases of severe isopropanol toxicity are rarely described, and this report describes an unusual case of mixed acute ethanol and isopropanol intoxication with subsequent alcohol withdrawal in a pediatric patient in the setting of chronic alcohol use.
Methods: This is a single patient chart review. A three-year-old male with a history of autism and asthma was found unresponsive with foaming respiratory secretions by family members. He was intubated upon arrival for airway protection. He presented tachycardic, hypotensive and hyperglycemic requiring treatment with norepinephrine, epinephrine and insulin infusions. He initially had a detectable serum ethanol level of 198 mg/dL, an elevated serum lactic at 13.1 mmoL/L and an elevated osmolar gap of 64.5 as well as 2+ ketonuria. Initial blood gas revealed a respiratory and metabolic acidosis with a pH of 7.15, pCO2 63 mmHg and HCO3 of 22 mEq/L. He was later found to have an elevated acetone level at 103 mg/dL and an isopropanol level of 28 mg/dL.
Results: The patient was started on fomepizole for treatment of elevated osmolar gap prior to obtaining acetone and isopropanol levels and treated with supportive management. The patient had a normal lactate with down-trending osmolar gap by hospital day three. The patient's fomepizole was discontinued after receiving non-detectable ethylene glycol and methanol levels and a detectable isopropanol level. Gradually, he was weaned off vasopressors and insulin with discontinuation by hospital day four and successfully extubated. After extubation, the patient was noted to have residual tremors in the bilateral upper extremities and tongue fasciculations. The patient was treated with lorazepam with concern for alcohol withdrawal and was found to have elevated phosphatidylethanol levels at 56 ng/mL consistent with chronic ethanol use. After clinical improvement, the patient was discharged home with court ordered involvement with child protective services with concern for medical child abuse.
Conclusion: A mixed ethanol and isopropanol ingestion can cause an elevated osmolar gap and anion gap and profound hypotension, CNS depression, and hyperglycemia. While alcohol withdrawal in young children is extremely rare, medical child abuse with repeated ethanol and isopropanol ingestion may lead to alcohol dependence with risk for withdrawal.

016. Analysis of Urine Drug Screens for Opioids in a Prenatal Clinic

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Background: As the prevalence of opioid use disorder (OUD) in pregnancy rises, so does the use of opioids that may elude standard urine drug screens. Though substance use screening does take place in the prenatal environment, there may be an underdiagnosis of OUD, especially in the setting of falsely negative urine drug screens.
Hypothesis or Research Question: We hypothesize that with the increased fentanyl in the current drug supply, screening for fentanyl and other nonstandard opioids in the pregnant population will increase identification of opioid use.
Methods: This is a prospective observational study of urine samples collected from routine prenatal visits at a single obstetrics and gynecology clinic serving public insurance patients in New Orleans. We screened for opioids including buprenorphine, fentanyl, methadone, morphine, oxycodone, and tramadol. Xylazine was added if the sample was positive for fentanyl. Tests were marked as positive if the urine screened positive for one or more of the opioids listed. Demographic data including comorbidities, home Address Deprivation Index (ADI) and prior documented OUD were collected from the electronic medical record. The primary outcome was positive UDS. Secondary analysis was performed examining characteristics associated with positive UDS among patients without documented OUD. Categorical baseline characteristics were compared via Fisher's exact test while continuous variables were compared via Mann Whitney U test. Primary outcome was evaluated via chi square with odds ratio (OR) and 95% confidence intervals (95% CI). Secondary outcomes were evaluated via a regression model. Alpha was set at 0.05.
Results: Among 342 patients undergoing 828 urine drug screens, four (1.2%) had documented OUD. Patients with OUD were younger and lived in neighborhoods with lower deprivation while having higher prevalence of smoking, depression, and anxiety (table 1). OUD was highly associated with a positive UDS (75% vs 1.8%, OR 10.4, 8076.1).

Among 338 patients without documented OUD, six patients (1.7%) screened positive on UDS. Positive substances included one with buprenorphine, one with methadone, one with tramadol, and three with non-specific opioids. No fentanyl was identified. No statistical differences were observed in baseline characteristics between those with and without positive UDS. A model including smoking, body mass index, and bipolar disorder did not identify any significant associations with a positive UDS.
Conclusion: Pregnant patients with OUD were likely to have other comorbidities in pregnancy and were highly associated with a positive UDS. We expect that with expanded opioid screening to at-risk patients, more interventions and resources may be provided.

017. First Dose Naloxone Characteristics When Given by Bystanders Versus EMS

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Background: Community naloxone administration is a key strategy for managing pre-hospital opioid overdose and reducing time to reversal. However, limited data exist on how initial doses differ when administered by bystanders (family/friend/strangers) versus EMS providers. This analysis compared initial dosing, routes of administration, and clinical response by administrator type.
Hypothesis or Research Question: What are the differences in first-dose naloxone administration characteristics when administered by bystanders versus EMS providers?
Methods: The ToxIC RENDOR project is an ongoing study whereby EMS providers take enhanced and standardized histories on patients suffering from opioid overdoses in the prehospital environment. These histories emphasize naloxone administration characteristics by bystanders, police/fire personnel, and community health workers, which are often absent in EMS documentation. Details on administration (dose, route, indication, response) and administrators (bystander, police/fire personnel, EMS) are collected from five U.S. sites: Denver, CO; Detroit, MI; Pittsburgh, PA; Portland, OR; and San Francisco, CA. Group differences (bystander vs. EMS) were analyzed using Independent Samples T-Tests and Chi-Square Tests using R (v.4.5.5).
Results: Of 2,392 completed cases, 648 patients (27.1%) received their first naloxone dose from other administrators (e.g., police/fire personnel) and were excluded from the analysis. The analytic sample (n=1744) compared those who received their initial naloxone dose from a bystander (n=641) with those who received their initial dose from an EMS provider (n=1103). Patients receiving bystander naloxone were less likely to present to EMS providers with respiratory depression (39.3% vs. 71.8%, $p < 0.001$) or depressed consciousness (81.7% vs. 90.4%, $p < 0.001$), while rates of cardiac arrest presentation were similar between groups (6.4% vs. 5.3%, NS). Routes of administration varied markedly: bystanders used naloxone intranasally in 95% (n=608/641) of administrations, compared with 45% (n=497/1103) by EMS. Parenteral routes (IV/IM/IO) were used in 1% (n=8) of bystander cases versus 54% (n=596) by EMS. Median dose was higher among bystanders for all routes (IN: 4 mg vs. 2 mg, $p<0.001$; Parenteral: 4 mg vs. 2 mg, $p=0.046$). Response to the first dose differed significantly: a lack of response was more often reported after bystander administration (49.8% vs. 30.9%; *p<0.001*), while respiratory improvement was more frequently reported following EMS administration (18.6% vs. 43.2%; *p<0.001)*. Precipitated iatrogenic opioid withdrawal was uncommon and similar across groups (6.2% vs. 7.3%, NS).
Conclusion: Naloxone administered first by bystanders significantly differed from EMS-first administered naloxone by dose, route, clinical presentation, and response. Further research is needed to determine how these variations affect clinical outcomes.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

018. Recreational Nitrous Oxide Use and Associated Thromboembolic Disease: A Potentially Underrecognized Clinical Concern

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Background: The potential thromboembolic complications of chronic nitrous oxide (N2O) use are not widely known among clinicians and are likely underrecognized. Oxidation of vitamin B12 (cobalamin) with N2O use functionally disables it as a cofactor in the methylmalonic acid and homocysteine pathways, leading to elevated serum concentrations. Hyperhomocysteinemia in those genetically predisposed is associated with an increased thrombotic risk. The theorized pathophysiologic mechanism of hypercoagulability is multifactorial but ultimately leading to endothelial dysfunction.

Methods: This is a single patient chart review. A 32-year-old man with a history of daily N2O use for several months presented with acute encephalopathy and paresthesias of his lower extremities (LEs). Given concerns for N2O abuse, he was started on vitamin B12 supplementation while in the emergency department and admitted for observation. On hospital day (HD) two, he reported worsening chest discomfort and was notably hypoxic. Subsequent CT pulmonary angiogram revealed extensive bilateral pulmonary emboli (PE) including a saddle PE with associated right ventricular (RV) strain. He underwent a thrombectomy on HD three and was found to have significant right atrial and ventricular clot burden. On HD four, he developed acutely worsening hypoxia, with repeat imaging revealing bilateral embolic burden and persistent RV strain. He suffered a peri-intubation cardiac arrest prior to repeat thrombectomy and immediately underwent veno-arterial extracorporeal membrane oxygenation cannulation (ECMO). Thereafter, he had a successful second thrombectomy. Bilateral LE ultrasound revealed extensive deep vein thrombosis in his right LE. An IVC filter was placed on HD six and he was decannulated from ECMO. His hospital course was complicated by hospital-acquired pneumonia requiring re-intubation, but he was ultimately discharged on HD 23 neurologically intact and on room air.
Results: Vitamin B12 level returned low at 193 pg/mL [reference 213-816] on HD one and methylmalonic acid level returned elevated at 2.17 umol/L [reference 0-0.4] on HD two, consistent with N2O use. A homocysteine level drawn on HD one returned markedly elevated at 240.2 umol/L [reference, 5.0 - 15.4] and subsequently improved to 49.4 umol/L on HD six. His standard hypercoagulable workup was otherwise unrevealing.
Conclusion: There are significant thromboembolic risks with excessive recreational N2O use. The etiology is multifactorial, including the hypercoagulable state of hyperhomocysteinemia. Recognizing the thromboembolic risk of chronic N2O use can lead to prompt diagnosis and thereby reduce morbidity and mortality.

019. The Effect of Incorporating Addiction Medicine Consults on Medical Toxicology Service Volume

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Background: A portion of Medical Toxicology services have incorporated Addiction Medicine (AM) consultations into their practice. Most--if not all--AM consults fall within the purview of Medical Toxicology. Our group began performing AM consults in 2019.
Hypothesis or Research Question: AM consultations increase overall Medical Toxicology service volume and do not affect "traditional" toxicology consult volume.
Methods: This was a retrospective analysis of our consult list from 2015 to 2025 based on billing descriptions entered by attending toxicologists (e.g., "clonidine toxicity requiring IMV" or "OUD, BUP micro"). Average volumes were calculated over the 5 years prior to service initiation and 5 years since (2015 – 2019 and 2020- 2024). We counted only new consult encounters; follow-up evaluations were removed. Consults were divided by year and by type: "TOX" and substance use disorders (SUD) were subdivided into opioid use disorder (OUD), alcohol use disorder (AUD), OUD + AUD, OUD + substance use disorder (SUD), SUD, cocaine use disorder (CUD), and "wean/sedation". "Wean/Sedation" are consultations we perform to help sedate patients with high tolerances and/or facilitate ventilator liberation. All consults prior to 2019 were considered TOX. Total ED volume for the hospitals where we see >95% consults added for context to help interpret changes in overall patient volume due to COVID pandemic. Simple counts and percentages were used to document results.
Results: Despite an average reduction in ED volume by 24% from 2015-2019 and 2020-2024, addition of AM consultations resulted in a marked increase in volume by 71%. Consult volume in 2024 alone was triple the average volume in 2015-2019. Over time, types of SUD and polysubstance use consultations also increased but may reflect more complex descriptions of reason for consultation. Apparent TOX volume decreased slightly and was likely due to change in diagnosis description by attendings over time (e.g., evaluations for AUD in 2020-2024 cohort often include treatment of alcohol withdrawal that were coded as "TOX" in 2015-2019 cohort), but may be related to a non-specific "post COVID effect". Consultations for ICU sedation and ventilator weaning have increased consistently since initiation of that service component.
Conclusion: The addition of AM consultations to a medical toxicology service greatly increased overall Medical Toxicology Service volume and the apparent decrease in "traditional" medical toxicology service volume likely reflected changes in coding for reason for evaluation. This suggests that Medical Toxicologists fill a need for acute treatment of patients with SUDs in the hospital system where no established AM service exists.

020. ED Visits for Cannabis-Induced CNS Depression Predominantly Occur in Young Children

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Background: Increased cannabis availability has led to an increase in cannabis attributable ED visits. A potential cannabis antidote is being developed for use in acute cannabis-induced toxicity. Serious cannabis-induced CNS depression has been reported in young children and confirmation of symptoms present in different age groups may identify populations at greatest need of antidote treatment.
Hypothesis or Research Question: Primary objective was to compare symptoms between young children, older children/adolescents, and adult patients with cannabis attributable ED visits.
Methods: All cannabis related ED visits to an academic safety net hospital from (2019-2024) were identified by cannabis related ICD-10 codes. Demographics, insurance, and hospital disposition were uploaded from the local data warehouse. Trained chart reviewers identified cannabis attributable cases and recorded symptoms present at presentation. Reviewers captured the following symptoms, which were not mutually exclusive: cannabis hyperemesis syndrome (CHS), central nervous system (CNS) depression, intoxication, nausea/vomiting, psychiatric symptoms, and seizures. Richmond Agitations-Sedation Scale (RASS) was calculated for each case. The proportion of visits with each symptom category were stratified by age group: young children (0-5 years), older children/adolescents (6-17 years), and adults (18+ years).
Results: 4,197 cases were reviewed and 845 were confirmed as cannabis attributable. There were 30 cannabis attributable visits in young children, 169 in older children/adolescents, and 646 in adults (Table 1). CNS depression (n=14, 46.7%) and intoxication (n=15, 50%) were the most common syndromes in young children. In older children/adolescents, psychiatric symptoms (n=69, 40.8%) and intoxication (n=31, 18.3%) were most common. Psychiatric symptoms (n=310, 48.0%) and CHS (n=115, 17.8%) were the most common attributable symptoms in adults. RASS was more likely to be less than 0 in young children and more commonly above 0 in adolescents and adults. RASS was < 0 in 43.3% of case in young children, 9.5% older children/adolescents, and 10.1% adults and above 0 in 3.3% of cases in young children, 14.8% of older children/adolescents, and 33.4% of adults. The odds of CNS depression were 6.8 (95%CI:3.2-14.7) in young children compared to adults. ED discharge was the most common disposition in adolescents (n=118, 69.8%) and adults (n=344, 53.3%). The most common disposition in young children was admission (n=13, 43.3%).
Conclusion: The most common condition in young children with cannabis attributable visits was CNS depression. Older children/adolescent and adult syndromes more commonly involved psychiatric symptoms and CNS excitation. Antidote treatment that reverses cannabis-induced CNS depression could potentially treat the most serious cases in pediatric patients.

021. Emergence of 7-Hydroxymitragynine in Michigan: A Five-Year Review

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Background: The potent kratom alkaloid 7-hydroxymitragynine (7-OH) is more frequently encountered in the US market, with uncertain impact on clinical severity and resource use.
Hypothesis or Research Question: Emergence of 7-OH will lead to increasing case volume and higher-severity presentations versus non-7-OH kratom.
Methods: This is a retrospective consecutive-case review of poison center kratom exposures from January 1, 2020 – November 13, 2025. Cases were classified as 7-OH if specifically documented, all others were classified as kratom. Single-substance versus co-ingestion, toxicity, withdrawal, respiratory depression, naloxone use, buprenorphine (recommended/administered), death, and disposition were abstracted. Dispositions were classified as home, emergency department (ED) treat-and-release, floor, intensive care unit (ICU), psychiatric facility, and lost/refused care. Statistics are descriptive.
Results: We identified 347 total kratom exposures: 58 (2020), 62 (2021), 51 (2022), 41 (2023), 48 (2024), 87 (2025), with an 81% rise in 2025 versus 2024. The first 7-OH case appeared April 2025, and 24/87 (27.6%) of 2025 cases involved 7-OH. Overall, median age was 33 years (range 2d-69y), with 71% male. We identified two dominant presentations: toxicity in 234/347 (67.4%) and withdrawal in 70/347 (20.2%). Withdrawal was more common with 7-OH: 9/24 (37.5%). In single-substance exposures, withdrawal was present in 40% of 7-OH cases versus 21.6% for non-7-OH cases. Respiratory depression was higher for 7-OH cases 12.5% versus 9.5% and more frequent with co-ingestions versus single-agent cases (13.9% versus 6.7%). Naloxone was administered 6.9% overall and in 4.2% in 7-OH cases. Seizures identified in 7.8%. Buprenorphine was recommended in 35/347 (10.1%) and administered in 12/347 (3.5%) and more frequently used in 7-OH (recommended 20.8%; given 12.5%) versus non-7OH (recommended 9.3%;

given 2.8%). ICU admissions were 20.2% overall and more frequent with 7-OH (29.2%) versus non-7-OH (19.5%). ICU care occurred more often with co-ingestions (26.3%) versus single agent (16.2%). For 7-OH cases, ICU care occurred in 25% single agent and 50% co-ingestion cases. Disposition: floor 23.0%, ED treat-and-release 32.6%, psychiatric facility 4.6%, home-managed 6.9%, and lost to follow up/refused care 9.2%. Death occurred in 1.4%, all involving co-ingestants, with no deaths in 7-OH group.

Conclusion: In 2025, 7-OH appeared and incidence comprised over a quarter of kratom exposures with a higher withdrawal burden, more frequent buprenorphine use, and greater ICU utilization versus non-7-OH kratom. Findings correlated with known higher potency of 7-OH. Limitations include single-center, retrospective design, and reliance on poison center documentation.

022. Naltrexone Precipitated Withdrawal Six Months After Long-Acting Buprenorphine Injection Confirmed by Serum Quantification

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Background: Long-acting injectable buprenorphine (Sublocade®) is used for both opioid use disorder and chronic pain. Product information describes prolonged opioid receptor activity for at least two months. Pharmacokinetic data indicates that clinically significant serum concentrations persist well beyond this period. We present a case of naltrexone precipitated opioid withdrawal after six months of buprenorphine abstinence.

Hypothesis or Research Question: Naltrexone initiation can precipitate opioid withdrawal six months after discontinuation of long-acting injectable buprenorphine.

Methods: A 72-year-old male with pertinent past medical history of chronic back pain following prior laminectomy and alcohol use disorder presented to the emergency department with complaints of myalgias, anxiety and altered mental status.

He previously received five monthly subcutaneous injections of buprenorphine 300 mg for chronic back pain. The final injection was given approximately six months prior to presentation. At a routine clinic visit, he was noted not to be taking opioid therapy and was started on oral naltrexone 50 mg daily for alcohol use disorder. About 45 minutes after the first dose, he developed significant symptoms and presented for evaluation. Initially alcohol withdrawal was suspected. Intravenous benzodiazepines (2 mg midazolam and 30 mg diazepam) and 260 mg phenobarbital were administered without improvement. Review of his medication history revealed prior Sublocade® exposure. A trial of sublingual 16 mg buprenorphine-naloxone (Suboxone) resulted in rapid improvement of all symptoms.

Results: Serum blood samples were analyzed via liquid chromatography/tandem mass spectrometry (LC-MS/MS) for buprenorphine, norbuprenorphine, naltrexone and 6-beta-naltrexol. Buprenorphine concentration was 2.7 ng/mL and norbuprenorphine was 0.65 ng/mL. The parent compound naltrexone was not detected. The active metabolite 6-beta-naltrexol was present at 66 ng/mL, confirming recent naltrexone exposure. Pharmacokinetics of long-acting buprenorphine at 300 mg predict serum concentrations of approximately 5 to 10 ng/mL during active monthly dosing. Modeled concentrations remain above 2 ng/mL for up to nine months after discontinuation. Serum concentrations of 2 to 3 ng/mL are considered sufficient to produce therapeutic opioid receptor occupancy. The measured concentration of 2.7 ng/mL was consistent with ongoing opioid activity during naltrexone administration.

Conclusion: This case describes naltrexone-precipitated opioid withdrawal six months after the last Sublocade® injection. Rapid improvement with suboxone supported an opioid mediated mechanism. Clinicians should recognize that long-acting buprenorphine can persist at clinically active levels for many months and antagonists such as naltrexone may precipitate withdrawal.

023. Life-Threatening Mixed Intoxication From Synthetic Cathinones and Bromazolam Confirmed by LC-MS/MS

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Background: The designer cathinone 4'-methyl-α-pyrrolidinohexanophenone (MPHP) and its analog 3,4-methylenedioxy-α-pyrrolidinohexiophenone (MDPHP) are potent psychostimulants structurally related to pyrovalerone and methylenedioxypyrovalerone (MDPV). These synthetic cathinones, along with designer benzodiazepines such as bromazolam, continue to appear in the illicit drug market and may cause severe toxicity. This report describes a rare case of life-threatening mixed intoxication with confirmed MPHP, MDPHP, and bromazolam exposure.

Methods: This is a single patient chart review. A 34-year-old man with a history of polysubstance use disorder was found unresponsive at home after an estimated downtime of 18 hours. He was known to purchase various "research chemicals" online. On arrival to the emergency department, he was obtunded, tachypneic, and hypoxemic and was intubated. Initial examination showed hyperthermia

(39.8 °C), tachycardia (HR 118 bpm), sustained bilateral lower extremity clonus, dilated pupils, and diaphoresis. The ECG revealed sinus tachycardia with normal QRS and QTc intervals. Laboratory workup disclosed elevated AST and ALT, creatine kinase, troponins, and creatinine (5.4 mg/dL). Computed tomography of the brain demonstrated global hypoxic brain injury. His prescribed medications included only bupropion, and his mother provided the team with unlabeled drugs that the patient had purchased.

Results: Patient serum and provided drug samples were analyzed by liquid chromatography–tandem mass spectrometry (LC-MS/MS) at our toxicology laboratory. Detected compounds included isomers of MPHP and MDPHP, as well as bromazolam. Quantitative analysis in the serum and urine was performed. In serum, concentrations of 0.89 ng/mL and 2.14 ng/mL were found for the isomers of MPHP and MDPHP, respectively. In urine, concentrations of 25.6 ng/mL and 40.92 ng/mL were found for the isomers of MPHP and MDPHP, respectively. Bromazolam concentrations were 33.59 ng/mL in serum and 12.92 ng/mL in urine. No traditional opioids, cocaine, or other phenethylamines were detected. The patient was managed with supportive care, benzodiazepines, and external cooling. After one month of hospitalization, he was discharged without residual neurologic deficits.

Conclusion: As novel psychoactive substances such as MPHP and MDPHP become more widespread, awareness of their toxicity is essential. This is the first documented case of combined MPHP, MDPHP, and bromazolam intoxication confirmed by LC-MS/MS. Synthetic cathinones can cause profound sympathomimetic toxicity, while concurrent designer benzodiazepines may obscure the clinical picture. Bromazolam has been associated with fever, myocardial injury, seizures, and prolonged CNS depression. Early recognition, supportive care, and confirmatory testing are essential in managing patients exposed to novel psychoactive substances.

024. The Kratom Conundrum: Three Cases of Mitragynine Withdrawal

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Background: Kratom (*Mitragyna speciosa*) and its active alkaloids, mitragynine and 7-hydroxymitragynine, exhibit partial μ-opioid receptor agonism, producing both stimulant and opioid-like effects. Chronic use can lead to physiologic dependence and withdrawal, though standardized treatment strategies remain undefined. We present three cases of mitragynine dependence and withdrawal managed with buprenorphine, highlighting variable presentations and the need for pharmacologic intervention.

Hypothesis or Research Question: Does mitragynine create a true withdrawal state? Can we treat it with standard opioid management tactics.

Methods: Case 1**:** A 53-year-old man with alcohol use disorder (six pints of beer daily) and chronic 7-hydroxymitragynine use presented with vomiting, diarrhea, tremulousness, and anxiety. He exhibited features of concomitant alcohol and opioid-like withdrawal requiring inpatient management. The patient was treated with symptom-triggered benzodiazepines for alcohol withdrawal and a 7-day buprenorphine bridge for mitragynine withdrawal, with progressive improvement and successful discharge. Case 2: A 30-year-old man using 240–260 mg of 7-hydroxymitragynine daily for chronic leg pain presented after abrupt discontinuation due to loss of supply. He reported restlessness, myalgias, and anxiety, with a Clinical Opioid Withdrawal Scale (COWS) score of 8 (later worsening to 10). Diazepam, hydroxyzine, and clonidine failed to provide relief; buprenorphine induction resulted in rapid symptom resolution and sustained improvement. Case 3: A 21-year-old hospital employee with chronic daily 7-hydroxymitragynine use was brought in as a stroke alert for acute confusion and amnesia after using during his lunch break. He reported alternating diarrhea and constipation in preceding days. Neuroimaging, including head CT and CTA, was negative. His presentation was attributed to acute mitragynine toxicity and withdrawal overlap, and he was discharged with naloxone and Buprenorphine

Results: These cases illustrate the clinical variability of mitragynine-related presentations, ranging from isolated withdrawal to mixed withdrawal states and acute neurobehavioral changes. Traditional symptomatic therapies may be insufficient for significant withdrawal, whereas buprenorphine provided consistent symptom relief in each of these cases. These observations support mechanistic overlap between kratom alkaloids and traditional opioids. This suggests a potential therapeutic role for buprenorphine in managing kratom dependence.

Conclusion: Mitragynine and 7-hydroxymitragynine withdrawal can produce clinically significant distress similar to that of an opioid withdrawal and may respond to buprenorphine therapy when supportive measures fail. Increased clinician awareness and evidence-based protocols are needed as kratom use and dependence continue to emerge in everyday life

025. Bradycardia and Bradypnea Refractory to Naloxone Secondary to Medetomidine Stuffing

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Background: Medetomidine is an alpha-2-adrenergic agonist which is prevalent in much of the current opioid drug

supply in Pennsylvania. Up to 80% of samples tested in Q2 of 2025, and 68% in Q3 20251 contain medetomidine. When taken in overdose, patients present with bradycardia, hypotension, and sedation, not responsive to naloxone therapy.
Hypothesis or Research Question: Overdose patients with bradycardia and bradypnea refractory to naloxone therapy may be due to medetomidine adulteration in the opioid supply.
Methods: This is a single patient chart review of a patient who had overdosed on 13 stamp baggies of fentanyl with bradycardia refractory to activated charcoal and continuous naloxone infusion.
Results: He was initially treated at an outside hospital after admittedly ingesting 13 stamp baggies of fentanyl prior to arrival. Poison control was contacted who recommended activated charcoal if patient's mental status would allow and monitoring for potential naloxone treatment for respiratory depression if he became symptomatic. He eventually became somnolent and bradypneic and received a dose of naloxone. Computed tomography of the abdomen obtained at outside facility did not show radio-opaque foreign bodies in the GI-tract. He became increasingly bradycardic and bradypneic, despite administration of activated charcoal, requiring additional naloxone, and was ultimately transferred to our tertiary facility for additional treatment. He was maintained on a naloxone drip for the next 24 hours with improvement of his mental status, but continued bradycardia and bradypnea. He was off of the naloxone by the next morning. Unfortunately, he progressed into opioid and medetomidine withdrawal, requiring a dexmedetomidine infusion and additional adjunctive therapy. Symptoms resolved by the next day. He was discharged on hospital day 2. Comprehensive urine drug screen (Liquid-chromatography qTOF) obtained on admission confirmed the presence of fentanyl, fentanyl metabolites, medetomidine, and medetomidine metabolites.
Conclusion: While the degree of the patient's intoxication may have been secondary to the amount and steady release of the stuffed fentanyl bags, the refractory nature to naloxone and activated charcoal with persistent bradycardia is consistent with medetomidine toxicity. With the prevalence of medetomidine in the illicit opioid supply, it should be considered in cases refractory to naloxone

026. Fentanyl-Overdose Patients Reporting Self-Harm or Suicide Intent Versus Those Seeking Euphoria: A Comparison

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Background: The intent of opioid use is complex and multifactorial, and there is little data describing overdose intent, or the dose or route that is used related to that intent. We sought to evaluate the characteristics of patients who overdosed on fentanyl with the intent for self-harm/suicide and compare them to those who were pursuing euphoric effects.
Hypothesis or Research Question: Among those with fentanyl in their blood, subjects who use fentanyl for the purpose of self-harm/suicide use by a familiar route and have higher average fentanyl concentrations compared to those seeking euphoric effects.
Methods: The Toxicology Investigators Consortium (ToxIC) Drug Overdose Toxico-Surveillance (DOTS) Reporting Program included ED patients ages 13 and older who presented to 17 U.S. medical centers with suspected opioid or stimulant overdoses. Data collection included comprehensive chart reviews, detailed patient interviews, and qualitative and quantitative toxicology analyses conducted by the Center for Forensic Science Research and Education for the detection of over 1,200 drugs and metabolites. This analysis compared subjects who reported that the intent of substance use was self-harm/suicide to those who reported utilizing the substance for euphoria. Variables included use-history, fentanyl concentrations, and naloxone doses.
Results: There were 609 subjects who completed interview questions and presented as a clinical opioid overdose. Thirty subjects (4.8%) reported intent of self-harm, 326 (52.5%) were seeking euphoria, and 265 (42.7%) used for other reasons. The self-harm group had a median (ng/mL, IQR) fentanyl concentration of 2.6 (1.6, 5.5), whereas the euphoria-seeking group had a median concentration of 4.7 (2.3, 9.4). The self-harm/suicide group self-reported (# of subjects, %) having used the following drugs: opioids only (16, 53.3%), stimulants only (4, 13.3%), opioids and stimulants (3, 10.0%), pharmaceuticals only (1, 3.3%), and other combinations (6, 20%). Five (17.2%) subjects reported their first time using this drug. Subjects with the intent of self-harm/suicide were more likely to report ingestion/swallowing the overdose drug compared to those with euphoric intent (42.3% vs. 12.6%; respectively, p=0.004) and first time using the drug (17.2% vs. 11.3%; respectively, p=0.01).

Conclusion: Approximately five percent of all opioid overdoses reported their fentanyl use as a self-harm/suicide attempt. Our results are limited by reliance on subject self-report and recall bias, and self-harm/suicide may be underreported. These subjects had lower blood fentanyl

concentrations, used a familiar drug but not by the usual route, and had a high rate of ingestion of the drug rather than injection or smoking.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

027. THC and Metabolite Concentrations and Triage Temperature in Patients Presenting to the ED With Acute Cannabis Intoxication

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Background: Recreational and medical cannabis use has been legalized in many states. Some population statistics report daily cannabis use surpasses daily alcohol use. Hypothermia has been described in the setting of Acute Cannabis Intoxication (ACI) in animal models with a dose dependent relationship. There has been limited investigation into the effect of ACI on body temperature.

Hypothesis or Research Question: Given animal data supporting an indirect relationship between ACI and body temperature, we hypothesize the same relationship exists in humans.

Methods: This study was a secondary analysis of data collected as part of a prospective cohort study of adult and pediatric patients presenting to an Emergency Department at a single academic medical center. Subjects were identified by rolling review of disposition diagnoses associated with ACI. Charts were reviewed to ensure accuracy of diagnosis. Subjects with remnant biological samples collected as part of their routine clinical care met inclusion criteria. Remnant biological samples were identified in the clinical pathology laboratory and stored for analysis. Retrospective chart review and data extraction was performed for all subjects meeting inclusion criteria. Triage body temperature was measured with temporal contact thermometer. All serum samples were assayed for serum THC and metabolite concentrations.

Results: Fifteen patients were identified with ACI during the 10-month study period with a mean body temperature at triage of 36.4.(SD=.77). Waste blood was available for analysis in ten patients. The mean age of this cohort was 22.9 (10 mo – 72 years), 70% male, 50% white, and 70% were discharged from the ED. Mean body temperature at triage was 36.6 degrees Celsius. Mean serum THC concentration was 7.7 mg/dL (SD =11), THC-OH 1.2 mg/dL (SD=2.8), THC-COOH 134.4 (SD=107.4). Mean serum THC:THC-OH ratio was 55.7, THC:THC-COOH ratio was 0.07. Spearman coefficient (r) for triage body temperature and THC concentration was 0.32 (p=0.37), temperature and THC-OH was 0.11 (p=0.76), temperature and THC-COOH 0.13 (p=0.72). r-value for THC:THC-OH and body temperature was -.01 (p=0.99) and THC:THC-COOH was 0.37 (p=0.3).

Conclusion: We identified a weak positive correlation between serum THC concentration and triage body temperature which was not statistically significant. We found a weak positive correlation between THC:THC-COOH ratio and body temperature which was not statistically significant. In this limited data set we did not find evidence of a correlation between THC and metabolite concentrations and body temperature.

028. Medispa Sepsis Mimic: SIRS Response From NAD+ Infusion

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Background: Using food-grade products to compound infusions carries a known risk of microbial and endotoxin contamination. We present a case of systemic inflammatory response syndrome (SIRS) following nicotinamide adenine dinucleotide (NAD+) infusion suspected to be from a preformed endotoxin.

Hypothesis or Research Question: Intravenous administration of food grade supplements can provoke a SIRS response due to endotoxin accumulation.

Methods: This is a single patient retrospective chart review.

Results: A previously healthy 80-year-old female developed rigors and experienced a syncopal episode four hours after receiving an infusion of NAD+ at a medical spa. She presented to the emergency department with a blood pressure of 78/40 mmHg, pulse of 91 beats per minute, temperature of 38.5 °C, oxygen saturation of 95%, and respiratory rate of 26 breaths per minute. Labs revealed a lactate of 2.3 mmol/L, leukocyte count of 11.9 K/uL, and creatinine of 1.1 mg/dL. She was treated with intravenous fluids and empiric antibiotics. She had clinical improvement and normal vital signs after nine hours. Blood and urine cultures yielded no growth. Creatinine and leukocyte count normalized. Antibiotics were discontinued on hospital day one and she was discharged on hospital day two. On follow up in the toxicology clinic five days after discharge, the patient had normal vital signs and functional status. A serum endotoxin level was attempted during hospitalization but was unavailable.

Conclusion: Wellness centers may offer a variety of intravenous supplements, claiming various health benefits. NAD+ infusions are advertised to improve longevity and combat aging. These infusions are often compounded using food-grade products, which may be nonsterile. Specifically, gram-negative bacteria can release lipopolysaccharides, or endotoxins, that may persist in the compounded product.

When administered, these endotoxins may provoke a SIRS response. This patient's hemodynamic changes shortly after NAD+ infusion were consistent with exposure to pre-formed endotoxins. Bacteremia was less likely given negative microbial cultures and rapid clinical improvement. Similar reports of adverse events following NAD+ infusion have been issued to the Food and Drug Administration. Unregulated processing of infusion therapies poses significant safety concerns to patients seeking therapies at wellness centers.

029. Exploratory Ingestion of 7-Hydroxymitragynine Leading to Profound Respiratory Depression in Two Pediatric Cases

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Background: 7-hydroxymitragynine (7-OH), a mu receptor agonist, is a naturally occurring alkaloid from the *Mitragyna speciosa* plant, commonly found in kratom products. 7-OH has a greater binding affinity for the mu opioid receptor and is now being produced and sold as an unregulated, high potency standalone product. In July of 2025 the U.S. Food and Drug Administration (FDA) issued a warning of the toxicological concerns around 7-OH as an emerging opioid threat.
Hypothesis or Research Question: Ingestion of 7-OH-containing products can lead to severe sedative hypnotic syndrome requiring naloxone infusion and intensive care monitoring in pediatric patients.
Methods: This is a case series of two siblings, aged 20 months and two years, who received care from the Washington and Oregon Poison Centers following an exploratory ingestion of an unknown quantity of 7-OH–containing tablets. Case details were abstracted from Poison Center records. In both instances, the parents reported missing "7-hydroxymitragynine tablets," although the exact number ingested by each child was unknown. Expanded drug screening was obtained for both patients.
Results: Both children developed symptoms within approximately 10 minutes of ingestion, with notable somnolence, prompting EMS evaluation and transport to a healthcare facility. The 20-month-old was altered and hypotensive upon EMS arrival and responded initially to naloxone boluses but experienced recurrent desaturation events. He required three naloxone boluses before initiation of a continuous naloxone infusion, which was maintained for more than 12 hours until respiratory function stabilized. The two-year-old sibling required two naloxone boluses but did not require an infusion. Both children were discharged within 24 hours of the presumed ingestion time. Urine immunoassay testing was found to be positive for kratom and 7-hydroxymitragynine, conformation via liquid chromatography/mass spectrometry (LC/MS-MS) with a limit of detection of 1.0 ng/mL confirmed the presence of both mitragynine and 7-hydroxymitragynine.
Conclusion: 7-OH-containing products can cause severe respiratory depression in young children, requiring intensive supportive care and, in some cases, prolonged naloxone infusion akin to opioid toxicity management. Clinicians should be aware of the potential severity of pediatric exposures, particularly given the increasing availability of concentrated 7-OH products.

030. National and Regional Trends in Fentanyl-Associated Polysubstance Use: A Cross-Sectional Analysis of Urine Drug Testing in the United States, 2016–2025

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Background: The rise of fentanyl as a driver of overdose and death in the United States (US) has coincided with an evolving landscape of polysubstance use, where stimulants and other illicit drugs are increasingly used concomitantly. Understanding these patterns is critical to inform public health interventions and treatment strategies in response to the evolving opioid crisis.
Hypothesis or Research Question: Among patients in the US who use fentanyl, what are the national and regional patterns in concomitant use of other illicit drugs, and how have these trends changed over time?
Methods: This retrospective, cross-sectional analysis evaluated de-identified and aggregated definitive urine drug testing (UDT) results derived from testing with liquid chromatography-tandem mass spectrometry (LCMS/MS) on patient urine specimens from across the US. Specimens for UDT were submitted by healthcare providers as part of a comprehensive patient care plan between January 1, 2016 and August 31, 2025. This study assessed UDT specimens positive for fentanyl for co-detection of additional illicit drugs. The specimens included were from patients aged 18 years and older with a substance use disorder diagnosis code provided by the ordering provider. UDT positivity rates were modeled using logistic regression.
Results: A total of 1,626,286 UDT specimens representing 513,718 unique patients were included in the analysis. Fentanyl positivity rates increased dramatically after COVID-19 but remained stable or decreased after 2022. However, fentanyl and stimulant co-detection continued to increase

after COVID-19. Among fentanyl-positive patients, national methamphetamine prevalence increased from around 10% in 2016 to nearly 80% in 2025, while cocaine use declined over the same period, though detection has increased since 2022. Methamphetamine was detected at a higher rate in the West (84%) than nationally (71%), whereas cocaine detection was higher than the national average (28%) in the Northeast (67%), South (48%), and Midwest (42%). When evaluating fentanyl co-detection trends for commonly used fentanyl adulterants, xylazine, benzodiazepines, parafluorofentanyl, and other novel illicit opioids were all found at lower US rates in 2025 compared to 2024 and 2023, whereas acrylfentanyl and carfentanil co-detection rates increased over this same period. These results will be updated prior to poster presentation with the most recent data available.

Conclusion: This analysis demonstrates the role that fentanyl continues to play in polysubstance use, with methamphetamine emerging as the most prevalent co-detected substance nationally and within the majority of states. The growing overlap in fentanyl and stimulant use and regional differences in polysubstance use patterns highlight the evolving complexity of the opioid crisis.

031. Alkaloid Content Analysis of Kratom Products in Boston

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Background: Kratom (Mitragyna speciosa), a tropical tree native to Southeast Asia, is traditionally used for its stimulant and analgesic properties and has recently gained popularity. However, their chemical composition, particularly alkaloid content, varies widely among different commercial products. Sharma et al., 2025's recent study is the most comprehensive analysis of kratom alkaloid content, so this poster aims to compare these alkaloid profiles with those of the products purchased in Boston. Evaluating the consistency in the reported alkaloid concentrations contributes to understanding more about the differences in alkaloid content and the accuracy of labeling in products.

Hypothesis or Research Question: How does the alkaloid distribution in Boston kratom products compare to those found in Sharma et al., 2025?

Methods: This study conducted an analytical survey that examined the alkaloid composition of 31 kratom products purchased in Boston, including three powders, 13 tablets/gummy, and 15 liquid products. Alkaloid content was quantified using the ultra-performance liquid chromatography–tandem mass spectrometry. The formula calculates alkaloid concertation based on the grams of the substance, per 100 grams of the kratom product, presented as percent weight-by-weight. We quantified 10 alkaloids (mitragynine, 7-hyroxymitragynine, speciogynine speciociliatine, mitraciliatine, mitraphylline, paynantheine, corynantheidine, corynoxine A, and corynoxine B) and compared them to the expected alkaloid content.

Results: In prior studies, the expected ratio of mitragynine to 7-hydroxymitragynine in kratom products is 0.93:0.01. The ratio in our analysis for powder products was on average 1.064:0.004, higher than the expected ratio. Across the tablet/gummy kratom products, we found the ratio to be about 1.64:1.57, much smaller than the expected ratio. According to our analysis of liquid kratom products, the ratio was about 4.06:0.19, which is 4.32 times smaller than the expected ratio. Our analysis found that 9/15 liquid products reported being 7-hydroxymitragynine products had very little to no 7-hydroxymitragynine in the product. Suggesting the contents may not have been derived directly from naturally occurring kratom, or that varying production methods alter the alkaloid contents.

Conclusion: Our testing points to differences in alkaloid concentrations from the expected average; leading to varying consumer effects. The differing alkaloid profiles may contribute to differences in experienced effects, as higher concentrations of 7- hydroxymitragynine lead to higher potency. Given the differing alkaloid profiles and differing labeling, these results suggest a need for more research to better understand the variance of alkaloid contents between different products. This also points to a need for more regulation into what goes into the products and accurate content labelling.

032. Methamphetamine Exposures in Young Children and Infants Presenting to Acute Healthcare Facilities

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Background: Children under six years of age account for approximately 3% of all methamphetamine exposures reported to U.S. poison centers. Data characterizing the

clinical presentations of this population in acute healthcare settings are limited.

Hypothesis or Research Question: What are the characteristics of children under age 6 years who present to acute healthcare facilities with methamphetamine exposure?

Methods: This analysis is based on data obtained from the Toxicology Investigators Consortium (ToxIC) Core Registry. The ToxIC Core Registry collects detailed de-identified information on patients who are seen at the bedside by medical toxicologists. Cases in this analysis included children under 6 years of age with methamphetamine exposure who were entered into the ToxIC Core Registry between Jan 1, 2010 and Dec 31, 2024. Exclusion criteria were outpatient clinic evaluations or documentation of chronic exposure history.

Results: A total of 190 children under 6 years of age were reported to be exposed to methamphetamine during the study period (from 2010 – 2024). 5 were excluded for non-acute or outpatient presentations. Of these, 54.6% (n=101) were male. The majority of patients were identified as non-Hispanic White (44.3%, n=82), Hispanic (17.8%, n=33) and Black/African American (16.8%, n=31). Most patients (85.9%, n=159) were 2 years of age or younger, with 36.8% (n=68) <1 year old, and the median age being 1 year (12 months). A sympathomimetic toxidrome was reported in 57.3% (n=106) of patients, with common clinical findings including agitation (87.7%, n=93), tachycardia (79.2%, n=84), seizures (17.0%, n=18), and hypertension (5.7%, n=6). Most in this group were treated with benzodiazepines (75.5%, n=80), while 20.8% (n=22) received antipsychotic medications. An opioid toxidrome was reported in 7.6% (n=14), with 57.1% (n=8) exhibiting respiratory depression and 85.7% (n=12) with CNS depression. Of these, 64.3% (n=9) were treated with naloxone. Another 7.6% (n=14) presented with other toxidromes involving CNS depression, including "washout," sympatholytic, or sedative-hypnotic. There were no deaths. Co-exposures to other drugs of abuse were reported in 22.7% (n=42) of cases. These drugs included fentanyl, cocaine, MDMA, heroin, methadone, THC, buprenorphine, oxycodone, and nicotine.

Conclusion: In this study of children under age 6 years who presented to hospitals and were found to be exposed to methamphetamine, 85.9% were infants and toddlers 2 years of age or younger (85.9%, n=159). While the greatest proportion of cases presented with sympathomimetic toxidromes, opioid and sedative toxidromes were also observed, and 22.7% of children were exposed to multiple drugs of abuse.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

033. Severe Hyperchloremic Metabolic Acidosis Following Ornithine Supplement Ingestion

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Background: Ornithine is a nonessential amino acid marketed for athletic performance and liver detoxification and is generally regarded as safe, with no described human toxicity. We report the first known case of profound non–anion gap hyperchloremic metabolic acidosis associated with excessive oral ornithine ingestion in a previously healthy adult male, requiring continuous renal replacement therapy (CRRT).

Hypothesis or Research Question: Excessive oral ornithine ingestion may precipitate clinically significant non–anion gap hyperchloremic metabolic acidosis in adults

Methods: This is a single-patient case report based on chart review. A previously healthy 34-year-old male ingested escalating, unquantified doses of oral ornithine over several days. Clinical presentation, laboratory studies, imaging, toxicology screening, and therapeutic interventions were reviewed, including bicarbonate therapy parameters and continuous renal replacement therapy (CRRT) settings.

Results: The patient presented with tachypnea (respiratory rate 45/min), global weakness, nausea, and abdominal bloating. Initial venous blood gas demonstrated a pH <7.0 and carbon dioxide <5 mmol/L. Serum studies revealed severe non–anion gap hyperchloremic metabolic acidosis (chloride 123 mmol/L), mild renal insufficiency (creatinine 1.29 mg/dL), and hypokalemia (potassium 2 mmol/L). He remained hemodynamically stable but showed signs of respiratory distress. The patient had also been consuming homemade potassium bicarbonate but denied all other supplement or drug use. Imaging and a comprehensive toxicologic evaluation revealed no alternative etiology. Despite aggressive intravenous fluids and bicarbonate infusion at 75 mL/hr, his acidosis remained refractory at four hours, prompting CRRT initiation. Clinical improvement and laboratory normalization occurred within twenty-four hours of CRRT.

Conclusion: This case suggests a potential toxicologic effect of excessive oral ornithine ingestion, producing a metabolic acidosis similar to that observed with cationic amino acids such as arginine or with total parenteral nutrition–associated acidosis. Possible mechanisms include chloride-mediated acid load and proximal tubular bicarbonate wasting. Clinicians should be aware that unregulated supplement use, even with substances marketed as benign, can precipitate life-threatening acid-base disturbances.

034. Diethylene Glycol Toxic Ingestion With Confirmatory Testing and Serum Levels Pre/Post Dialysis

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Background: Diethylene glycol (DEG) is a common solvent in antifreeze products and brake fluid. DEG ingestion primarily causes gastrointestinal symptoms, metabolic acidosis, and acute kidney injury, with risk of delayed neurological issues. Typically, toxic alcohol panels screen for methanol, ethylene glycol, and isopropanol, not DEG levels.
Methods: This is a case report of a 62-year-old male in the Western United States.
Results: A 62-year-old male presented to a tertiary care emergency department with concern for ingestion. Initial labs reveal lactate of 9.3 mmol/L, pH 6.83, pCO_2 36 mmHg, HCO_3^- 5.8 mEq/L, anion gap >32, CK 15,000 (IU/L), AST 267 U/L. Serum drug screen negative for salicylates, acetaminophen, and ethanol. Toxic alcohol testing negative for ethylene glycol, propylene glycol, methanol, and isopropanol. The patient later confirmed that he ingested brake fluid. On day 1, DEG level was drawn at 0531 (46 mg/dL, reference value <5) using test NMS 1589SP. Fomepizole began at 15mg/kg on day 1 at 0530, continued for 48 hours at 10 mg/kg every 8 hours. He received 4 hours of intermittent hemodialysis (0958 to 1423) with removal of 800 mL. Hypotension necessitated transition to CRRT, pulling 100 mL/hour from 0700 to 2200 on day 2. At 1640 on day 2, repeat DEG level of 8.2 mg/dL. Day 1 osmolality of 338 mOsm/kg at 0400 (Osmol gap 37, anion gap >32) downtrended to 307 mOsm/kg at 0240 (osmol gap 7, anion gap 16) on day 2. On day 2, urine output decreased from 0.89 to 0.20 mL/kg/hour, then gradually improved. Creatinine peaked at 2.41 mg/dL on day 7 and stabilized at 1.6 mg/dL. He later developed small bowel ischemia requiring ileocecectomy with ileostomy and was discharged on day 37.
Conclusion: DEG metabolizes into 2-hydroxyethoxyacetic acid and Diglycolic Acid (DGA). DGA is associated with neurotoxicity and nephrotoxicity. Large DEG ingestions (1–1.5 g/kg) are lethal. Animal models show a dose-dependent relationship between ingested amounts and toxicity. Few studies include DEG concentrations; one study noted a median of 40.7 mcg/mL in 20 individuals with acute kidney injury. It is unclear what caused bowel ischemia; potentially, hypotension and vasopressor support. Because time and quantity of ingestion are unknown, delayed ingestion and metabolism might explain changes in osmolar and anion gaps. DEG levels positively correlate with the osmolar gap, which could be a useful surrogate in determining dialysis duration.

035. Impact of the COVID-19 Pandemic on Amphetamine Exposures in the United States

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Background: Patterns of amphetamine exposures are influenced by many factors, such as illicit trade and sociocultural elements. One recent influence on exposure trends was the COVID-19 pandemic. This study analyzed the National Poison Data System (NPDS) recordings of amphetamine exposures and compared pre- and post-pandemic periods.
Hypothesis or Research Question: The COVID-19 pandemic will influence exposure trends of amphetamines documented by the NPDS.
Methods: This is a retrospective ecological analysis of annual NPDS reports (2012-2023). Single exposures of "Amphetamines and Related Compounds," "Hallucinogenic Amphetamines," "Methamphetamine," "Methylphenidate," and "Other Stimulants (Excluding Amphetamines)" were identified and expressed as a proportion of all "Stimulants and Street Drugs" exposures per year. Logistic regression models evaluated annual trends of exposure odds for the pre-COVID period (2012-2019) and post-COVID period (2020-2023). Odds ratios (OR), 95% confidence intervals (CI), and p-values were reported.
Results: From 2012-2019, methamphetamine exposures increased from 2,207 to 4,022 cases (6.1% to 10.7% of stimulant/street drug exposures), demonstrating a significant upward trend, with a 7.6% annual increase in exposure odds (OR = 1.076, 95% CI = 1.070-1.082, $p < 0.001$). In contrast, exposures declined from 3,715 to 2,788 cases (9.2% to 5.7%) from 2020-2023, displaying a significant downward trend (see figure), representing a 16.2% annual reduction in exposure odds (OR = 0.838, 95% CI = 0.825-0.851, $p < 0.001$). All other amphetamine exposures displayed a downward trend, with a decrease in annual exposure odds during both time periods. Amphetamines and related compounds showed modest declines pre-pandemic (OR = 0.978, 95% CI = 0.974-0.981, $p < 0.001$) and greater reductions post-pandemic (OR = 0.921, 95% CI = 0.912-0.930, $p < 0.001$). Methylphenidate exposures decreased pre-pandemic (OR = 0.963, 95% CI = 0.959-0.967, $p < 0.001$) and remained relatively stable post-pandemic (OR = 0.966, 95% CI = 0.955-0.978, $p < 0.001$). Hallucinogenic amphetamine exposures declined throughout both periods, with a significantly larger decline post-pandemic (pre: OR = 0.872, 95% CI = 0.863-0.881, $p < 0.001$; post: OR = 0.642, 95% CI = 0.572-0.720,

$p < 0.001$). Similarly, other stimulants (excluding amphetamines) showed modest declines pre-pandemic (OR = 0.957, 95% CI = 0.941-0.973, $p < 0.001$) and greater reductions post-pandemic (OR = 0.765, 95% CI = 0.729-0.802, $p < 0.001$).

Conclusion: Amphetamine exposure odds declined following the COVID-19 pandemic, suggesting that societal disruptions (e.g. reduced public gatherings and altered supply chains) may have reshaped amphetamine use patterns. In particular, methamphetamine exposures, which steadily rose pre-pandemic, showed a pronounced post-pandemic reversal. Our findings highlight how large-scale societal events can influence national substance exposure trends.

036. A Case Series of Patients With Suspected Medetomidine Withdrawal

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Background: Medetomidine is an alpha-2 agonist composed of a racemic mixture of levomedetomidine and dexmedetomidine. Medetomidine has become increasingly common as an adulterant in the illicit opioid supply. Medetomidine overdose has been associated with hypotension and bradycardia not responsive to naloxone, with Essex County data from 2025 through September demonstrating 10% of seized heroin glassine bags contained medetomidine. We present a case series of three patients demonstrating symptoms consistent with medetomidine withdrawal associated with illicit opioid use.

Hypothesis or Research Question: Medetomidine withdrawal is a unique clinical syndrome whose treatment is dependent on alpha-2 agonist therapy.

Methods: This is a retrospective case series examining three patients presenting with opioid withdrawal with incomplete response to opioid agonist treatment who subsequently improved after treatment with high-dose alpha-2 agonists. Patients were identified as likely having alpha-2 agonist withdrawal due to their severe autonomic dysfunction including tachycardia, hypertension, diaphoresis, and tremulousness. Data was extracted to examine dosing of opioid agonists, benzodiazepines, and alpha-2 agonists, and the timing of clinical response. We describe pertinent symptoms and physical exam findings to define medetomidine withdrawal.

Results: All patients had opioid use disorder, and were being treated for opioid withdrawal or developed opioid withdrawal in the Emergency Department. During treatment for opioid withdrawal they developed symptoms consistent with alpha-2 agonist withdrawal. Mean pre-treatment peak heart rate and mean arterial blood pressure were 139, (range of 100-157) beats per minute and 152 (range 136-170) mmHg, respectively. All patients had impressive physical exam findings including tachycardia, tremulousness, diaphoresis, agitation, and intermittent leg shaking and spasms. After initiating alpha-2 agonist therapy, patients had an average reduction in heart rate of 48 bpm and MAP of 57 mmHg. One of the patients received 2 boluses of 1 mcg/kg dexmedetomidine in addition to oral clonidine 0.3 mg every 6 hours and the other two patients were started on dexmedetomidine infusions at 1-1.5 mcg/kg/hour. Multimodal symptomatic therapy was used to treat these patients including benzodiazepines, partial opioid agonists and alpha-2 agonists; clonidine, tizanidine, and dexmedetomidine. One patient was discharged after 15 days to a subacute rehab and two were discharged home after three days.

Conclusion: Medetomidine withdrawal is a unique syndrome seen in patients using adulterated illicit opioids predominantly characterized by refractory tachycardia, hypertension, diaphoresis, muscle spasms, and tremors. It is poorly responsive to treatment with standard therapies for opioid withdrawal and improves with use of an apha-2 agonist.

037. The Heart Withdrawals Too: Naltrexone-Induced Takotsubo Cardiomyopathy

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Background: Takotsubo cardiomyopathy is a type of non-ischemic cardiomyopathy characterized by an acute and transient decrease in left ventricular systolic function associated with high morbidity and mortality. This condition usually occurs after a significant emotional or physical stressor. Pathophysiology remains poorly understood and may be due to catecholamine surge.

Hypothesis or Research Question: Severe and acute precipitated opioid withdrawal is sufficient to cause takotsubo cardiomyopathy.

Methods: This is a single patient chart review.

Results: A 66-year-old male on daily methadone, 112mg, with no known cardiac history presented to the Emergency Department after an intentional single dose ingestion of his daughter's oral naltrexone to treat his opioid addiction. Shortly after, he developed symptoms consistent with severe precipitated opiate withdrawal. The patient was tachycardic, tachypneic, hypertensive on initial presentation, and became hypoxic secondary to aspiration and was placed on

mechanical ventilation. His opiate withdrawal was treated with droperidol, buprenorphine, hydromorphone, and propofol. He then had supraventricular tachycardia with aberrancy, rising troponins, and an ECG concerning for ST elevation myocardial infarction. He required inotropic/vasopressor support with norepinephrine and dobutamine and had a temporary mechanical assist device placed on hospital day two with removal on hospital day four. Cardiac catheterization demonstrated no evidence of occlusive coronary artery disease. Trans thoracic echocardiography showed findings consistent with takotsubo cardiomyopathy with findings of depressed left ventricular systolic function (estimated ejection fraction of five percent) dilation of basal segments, and relatively preserved contractility in the apical segments. He was extubated on hospital day five. Ejection fraction improved to 20-25 percent prior to discharge. He was discharged with a temporary external defibrillation device and was maintained on buprenorphine and beta blocker initiated prior to discharge.

Conclusion: Severe precipitated opioid withdrawal can cause takotsubo cardiomyopathy and should be considered when abnormal cardiovascular signs and symptoms occur in this clinical setting.

038. Marijuana Exposures and Poisonings of Children Aged 12 and Under

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Background: There are two principal data series tracking pediatric exposure to cannabinoids which have complementary strengths. One is the Pediatric Health Information System (PHIS) which is a database covering approximately 50 children's hospitals in the US. The second is the National Poison Data System (NPDS) which is administered by America's Poison Centers. Two recent studies with PHIS show sharp increases.

Hypothesis or Research Question: The purpose of this study is to provide an updated longitudinal report based on the NPDS data.

Methods: In 2000, the NPDS Annual Report had one category for marijuana (the term NPDS used). By the 2023 NPDS Annual Report, there were 17 subcategories of cannabinoids and analogs, categories which including marijuana: concentrated extracts, marijuana: dried plant, and marijuana: edible preparations. Using the NPDS Annual Reports for each year, the number of exposures of children under 6 years of age reported to NPDS was tabulated. NPDS began tracking the 6-12 age group in 2009. The subcategory of marijuana: edible preparations was added to the NPDS Annual Report in 2016.

Results: The numbers of reported exposures of children under age 6 was relatively stable between 2000 and 2009, ranging from 108 to 141, with 132 exposures in 2009. Beginning in 2010, a consistently rising number of reported exposures is seen, with the shape of the curve demonstrating acceleration. In 2023, there were 8400 reported exposures, an increase of 6363% from 2009. The percentage increase of reported marijuana exposures in the 6-12 age group since 2009 has been even greater than that of the under 6 group and that number has also been accelerating, though numbers of reported exposures in the 6-12 age group have been lower than those in the under-6 group, with 3023 exposures in 2023. Reported exposures of children under 6 to edible preparations rose from 7 in 2016 to 4795 in 2023, while those in the 6-12 group increased from 0 in 2016 to 2025 in 2023. Edible preparations were the predominant marijuana product involved in reported exposures of children, accounting for 57% of exposures in the under-6 age group in 2023, and for 67% in the 6-12 group.

Conclusion: Exposures of children under 6 and 6--12 have increased by >6000% since 2009. Edible preparations cause the majority of marijuana exposures in both age groups. Stronger regulation of the marijuana market is needed to reduce childhood exposures particularly to edible marijuana products.

039. Cardiotoxicity Surveillance After Osimertinib Overdose

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Background: Osimertinib is a third-generation Epidermal Growth Factor Receptor Tyrosine Kinase Inhibitor (EGFR-TKI) used as a first-line treatment for non-small cell lung cancer (NSCLC). Chronic use of osimertinib has been associated with an increased risk of cardiotoxicity including QTc prolongation, heart failure, and atrial fibrillation; however, no data exists regarding outcomes following intentional overdose. We present the first known report of an intentional osimertinib overdose.

Methods: This is a single patient case report. A 14-year-old female with a history of multiple suicide attempts ingested at least 2.4g (over thirty 80mg tablets) of osimertinib in a suicide attempt. Activated charcoal was given within 1 hour

of ingestion with subsequent nausea, vomiting, and diarrhea that improved with supportive measures. Due to concerns for QTc prolongation, serial Electrocardiograms (ECGs) were obtained, and prophylactic magnesium sulfate was administered. Medical Toxicology was consulted for evaluation and treatment upon admission to the Pediatric Intensive Care Unit (PICU).

Results: The initial ECG showed a QTc of 439 ms. Continuous cardiac telemetry, serial complete blood counts (CBC), complete metabolic panels (CMP), and magnesium/phosphorus levels were obtained in addition to a urine drug screen. Magnesium trended down to a nadir of 1.7 mg/dL and improved with oral and intravenous magnesium repletion. She was given 2 g of intravenous magnesium sulfate on day two and a second dose of 2 g on day three. A slight downtrend in the patient's hemoglobin (1 g/dL) and platelets (167,000/uL) was observed. The patient did not demonstrate dysrhythmias while on telemetry for 48 hours. QTc remained stable (428ms – 444ms) on serial ECGs. Intermittent headaches and sore throat improved with supportive measures. No imaging was obtained in the absence of red flag symptoms. On day two, a pruritic rash developed on her upper extremities that resolved with hydroxyzine. The patient remained hemodynamically stable over three nights and was transferred to the inpatient behavioral health unit four days post-ingestion.

Conclusion: An acute osimertinib ingestion may amplify known cardiotoxic and systemic complications; additionally, a nonspecific drug eruption or derangements in electrolytes or blood counts may occur. While no known antidote exists, cardiac monitoring and collaboration among multidisciplinary services can support favorable patient outcomes.

040. Intoxication From Transdermal Absorption of Isopropyl Alcohol

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Background: Isopropyl alcohol is one of the most common toxic alcohol ingestions, typically through the oral ingestion of various household and industrial products. Systemic intoxication from transdermal absorption is rarely reported and is primarily documented in the pediatric population. The possibility of clinical toxicity from transdermal absorption of isopropyl in adults is not well established.

Hypothesis or Research Question: Abundant dermal application of isopropyl alcohol can contribute to clinical intoxication in the adult population.

Methods: Presented as a single patient case report. A 49-year-old female with a history of asthma presented to the emergency department with acute intoxication. She endorsed consuming ethanol-containing beverages, but ethanol levels did not correlate with her degree of intoxication. Upon further questioning, she endorsed applying fabric soaked in isopropyl alcohol across her skin for long periods of time as a self-remedy for hot flashes. Laboratory studies revealed a high serum osmolality with a high calculated osmolar gap. Toxic alcohol levels were sent to a reference laboratory. Repeat chemistry studies were obtained at four hours and eight hours after presentation to monitor progression. The patient received supportive care and was discharged in improved condition after an extended period of observation.

Results: Initial serum ethanol was 62 mg/dL with serum osmolality of 358 mOsm/kg, yielding a calculated osmolar gap of 49.4 mOsm/kg. Initial anion gap was 17 mmol/L with bicarbonate 21 mmol/L. Toxic alcohol levels were obtained after consultation with medical toxicology, collected approximately six hours after presentation. Results returned multiple days later revealing an isopropyl alcohol level of 16 mg/dL and acetone level of 160 mg/dL, with undetectable methanol and ethylene glycol. Repeat chemistry studies at four hours and eight hours demonstrated resolution of anion gap to 10 mmol/L and 5 mmol/L respectively, with bicarbonate remaining 26 mmol/L. She improved with supportive care and was discharged in stable condition approximately 12 hours after presentation. She was advised to discontinue dermal application of isopropyl alcohol.

Conclusion: This case demonstrates that transdermal absorption of isopropyl alcohol may produce clinically significant intoxication in adults, or at least may be contributory when ingested with other agents. Clinicians should maintain suspicion for toxic alcohol exposure in patients with elevated osmolar gaps and intoxication disproportionate to measured ethanol levels, and should inquire about alternative remedies which may contain toxic products or environmental hazards.

041. Acute Nephrotoxicity Due to Intentional Chlorine Dioxide Supplementation

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Background: Solutions of 0.3% chlorine dioxide (Miracle Mineral Solution) have been promoted by alternative medicine practitioners for detoxification and treatment of COVID-19, autism, HIV, and malaria. Chlorine dioxide is a strong oxidizer associated with severe diarrhea, nephrotoxicity, and hepatotoxicity with significant exposures.

Hypothesis or Research Question: Ingesting "therapeutic" 0.3% chlorine dioxide solution may worsen existing gastrointestinal inflammation and cause end-organ damage.
Methods: This is a single-patient case report. A 50-year-old male with a history of hypertension and recent hospitalization for complicated diverticulitis presented to the emergency department with three days of left lower quadrant pain and one week of diarrhea. The patient reported the pain began a few hours after eating pumpkin seeds, and the diarrhea began shortly after starting a "water fast." The patient also reported ingesting 96 drops of 0.3% chlorine dioxide solution over the course of three days in the week prior to presentation. Basic laboratory studies, urinalysis, and CT of the abdomen and pelvis were obtained in the emergency department.
Results: Initial lab work demonstrated a serum creatinine of 3.16 mg/dL, from a baseline creatinine of 1.10 mg/dL, a serum bicarbonate of 18 mmol/L, and an anion gap of 11 mmol/L. Urinalysis was normal. Formal testing for chlorine dioxide was not performed. CT of the abdomen and pelvis without contrast demonstrated persistent wall thickening and inflammatory change of the distal descending colon with significant improvement noted from the patient's previous CT. The patient received fluid resuscitation with lactated ringers solution, IV antibiotics for presumed diverticulitis flare, and oral sodium bicarbonate therapy. On day three of hospitalization, the patient was observed to have scleral icterus. Liver enzymes were obtained which demonstrated an AST of 80 units/dl, an ALT of 86 units/L, an alkaline phosphatase of 160, a total bilirubin of 0.7 mg/dL, and a direct bilirubin of 0.4 mg/dL. On day four of hospitalization, the patient's creatinine was 1.49 mg/dL, his bicarbonate was 20 and his scleral icterus had resolved. Due to the improvement in his renal function he was discharged home on oral antibiotics.
Conclusion: Ingestion of "therapeutic" doses of 0.3% chlorine dioxide solution may worsen existing gastrointestinal inflammation and result in nephrotoxicity and hepatotoxicity. Limitations include lack of chlorate levels, unclear ingested dose, alternative etiologies of end-organ injury and nonexperimental single case design.

042. The Structural Similarity of Mexiletine to Amphetamines Can Result in False-Positives on Immunoassay-Based Drug Screens

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Background: Mexiletine is an oral Class 1B antiarrhythmic used to treat ventricular arrhythmias through the inhibition of sodium channels during phase zero of the cardiac myocyte action potential. Mexiletine and amphetamine share a benzene ring with a hydrocarbon tail terminating in an amine group. Immunoassays detect the amino group of amphetamines with high sensitivity, which leaves immunoassay-based urine drug screens (UDS) prone to false-positives.
Hypothesis or Research Question: Mexiletine can cross-react with the amphetamine immunoassay on a urine drug screen.
Methods: This is a single-patient case report. A 65-year-old male with history of ventricular fibrillation arrest status post left anterior coronary artery stent and automatic internal cardiac defibrillator placement, prediabetes, and chronic kidney disease presented to the emergency department for two months of auditory hallucinations after starting mexiletine 150 mg three times daily. He was otherwise asymptomatic. Clinical presentation was not suggestive of acute sympathomimetic intoxication. His blood pressure was 102/70 mmHg, heart rate 98 beats per minute, oxygen saturation 98%, and temperature 36.8 C. Physical examination was unremarkable. He did not have a prior psychiatric history. He denied use of alcohol, recreational drugs, supplements, herbal products, wild or foraged foods, methamphetamine, nasal sprays, decongestants, or antihistamines. His other medications comprised aspirin, atorvastatin, pantoprazole, ticagrelor, diclofenac, and over-the-counter acetaminophen. He had previously taken dexamethasone, but therapy was complete prior to presentation.
Results: Initial diagnostic studies including serum electrolytes, ammonia, thyroid screening, urinalysis, electrocardiogram, and CT head were unremarkable, although amphetamine was detected on immunoassay-based UDS. High resolution mass spectrometry of urine and serum detected only mexiletine and acetaminophen. The other medications the patient was taking would not be expected to cross-react with the amphetamine immunoassay. No alternative organic or psychiatric etiology for his hallucinations was identified.
Conclusion: The shared amino group in mexiletine and amphetamine can lead to cross-reaction of mexiletine with the amphetamine immunoassay on a UDS resulting in a false-positive.

043. Unusual Presentation of Diethylene Glycol Toxicity: Severe Metabolic Acidosis Without Renal or Neurologic Sequelae

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Background: Diethylene glycol (DEG) toxicity can cause high anion gap metabolic acidosis, renal failure, and neurologic injury. This case report describes a pediatric patient with significant DEG ingestion who developed marked anion gap metabolic acidosis without renal or neurologic sequelae.
Methods: A 15-year-old male with developmental delay and a history of seizures ingested approximately half a bottle of brake fluid over 30 minutes. The patient appeared sedated. On arrival to the emergency department, he was normotensive, tachycardic (131 bpm), and had an oxygen saturation of 95% on a non-rebreather. Initial labs showed bicarbonate 15 mEq/L, anion gap 18 mEq/L, and creatinine 0.7 mg/dL. The patient was intubated for airway protection and subsequently transferred to a tertiary hospital for further management.
Results: On arrival at the tertiary hospital, the patient remained acidemic (pH 7.09, bicarbonate 13 mEq/L, anion gap 31) but without evidence of renal injury (creatinine 0.79 mg/dL). Fomepizole was initiated, and nephrology was consulted for potential hemodialysis. Due to overnight staffing limitations, initial management prioritized a bicarbonate infusion, with hemodialysis performed the following morning. Over the next 24 hours, the anion gap (31 to 13 mEq/L), bicarbonate (11 to 28 mEq/L), and osmolar gap (32 to 3 mOsm/L) improved. No further sessions of hemodialysis were required. Despite developmental delays limiting neurological assessment, there was no evidence of new neurologic deficits. He had no seizures while in hospital and he was extubated by hospital day four. He was discharged home on day six with normal renal function (creatinine 0.53 mg/dL) and anion gap 12 mEq/L.
Conclusion: DEG poisoning is uncommon, and the natural course is not well reported on in developed countries. This case highlights that significant DEG ingestion may present with marked anion gap metabolic acidosis before effective treatment initiation without renal failure or clinically apparent neurologic injury. Recognition of this presentation can inform early supportive care and guide therapy. Further studies are needed to identify predictors of organ injury in DEG toxicity.

044. Chronic Nitrous Oxide Abuse Leading to Acute Limb Ischemia Requiring Amputation

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Background: Nitrous Oxide has become increasingly prevalent as a recreational drug. While its neurologic complications from functional Vitamin B12 deficiency are well-documented, its thromboembolic risk from an increase in hypercoagulability remains underrecognized in the medical community and rarely described in the literature.
Hypothesis or Research Question: Chronic nitrous oxide abuse leads to hyperhomocysteinemia, which causes a hypercoagulable state, with acute limb ischemia being a rare complication.
Methods: We conducted a single-patient chart review. A 29-year-old female with no significant medical history presents to the emergency department for severe pain and discoloration to the distal half of her left foot. Over the past few months, she has noted progressively worsening plantarflexion of both feet, gait disturbances, and decreased ability to walk long distances, requiring more assistance from family members for ambulation. Initially, this was thought to be the somatic manifestation of the patient's depression and PTSD. Over the past 7 days, the patient's left foot has felt cool to the touch. The distal half of that foot had developed purplish discoloration, accompanied by worsening pain. The patient had been using nitrous oxide ("whippets") for over a year, continuously inhaling it every single day.
Results: The patient was found to have an elevated homocysteine level of 35.1 μmol/L (ref 5-15) with an otherwise negative hypercoagulability work-up. Imaging revealed partial thrombosis of the abdominal aorta and left common iliac artery and complete thrombosis of the superficial femoral artery, popliteal artery, and tibial arteries. She was hospitalized for 20 days with a complicated course that involved multiple procedures, including embolectomy, faciotomy, tPA infusion with thrombectomy and angioplasty, and finally, a left below-the-knee amputation.
Conclusion: As recreational nitrous oxide use becomes more prolific, heightened clinician awareness of its potential complications is essential. While neurologic symptoms are well-characterized, thromboembolic events such as acute limb ischemia are rarely mentioned. Here, we present a case of chronic nitrous oxide use leading to acute limb ischemia requiring multiple procedural interventions and, ultimately, a left below-the-knee amputation. This case illustrates the potential for severe vascular morbidity associated with hypercoagulability from chronic nitrous oxide abuse and highlights the importance of recognizing this complication in clinical practice.

045. A Case of Survival After Massive Ibuprofen Ingestion With Severe Multiorgan Failure and Reversible Renal Concentrating Defect

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Background: Massive symptomatic ibuprofen overdoses are uncommon but can cause profound multi-organ dysfunction. The nephrotoxic effects are most commonly attributed to ibuprofen's inhibition of renal prostaglandin synthesis, which impairs afferent arteriolar vasodilation and precipitates prerenal azotemia or acute tubular injury. Less is known about ibuprofen's ability to cause renal concentrating defects. Management of ibuprofen toxicity is primarily supportive, as intermittent hemodialysis offers little to no benefit given the drug's high protein binding.
Hypothesis or Research Question: Does massive ibuprofen toxicity produce renal concentrating defects distinct from classic nephrogenic diabetes insipidus, and can extracorporeal therapy aid in management despite high protein binding?
Methods: This is a single-patient chart review with data obtained from electronic medical records, laboratory analysis. A 41-year-old woman ingested an estimated 200 grams (2107 mg/kg) of ibuprofen. She developed progressive somnolence and eventual non-convulsive status epilepticus, profound vasopressor-refractory shock, and severe lactic acidosis (peak lactate >13 mmol/L, nadir pH 7.09). Despite normal baseline renal function, she developed polyuria of six liters over the initial 24 hours of hospitalization. Her laboratory values suggested impaired renal concentrating ability with serum hypernatremia (sodium 152 mmol/L), elevated serum osmolality of 331 mOsm/kg (reference 275-295 mOsm/kg), elevated urine sodium of 137 mmol/L (reference 30-90 mmol/L), and a low-normal urine osmolality 197 mOsm/kg (reference 50-1,200) despite fluid restriction. Her creatinine peaked at 2.4 mg/dL on hospital day one.
Results: Continuous renal replacement therapy was initiated to treat profound metabolic acidosis and provide renal support, and therapeutic plasma exchange was performed despite ibuprofen's >99% protein binding. By hospital day three, the patient's acidosis improved and vasopressor needs declined. Her polyuria resolved with complete renal recovery by hospital day 14. She was discharged to inpatient psychiatry care without residual neurologic deficits. Analysis of initial serum samples demonstrated an ibuprofen concentration of 798 μg/mL (therapeutic range: 10-50 μg/mL).
Conclusion: This case represents survival after a massive ibuprofen ingestion. The coexistence of refractory shock, severe lactic acidosis, and transient polyuria supports a mechanism of reversible tubular dysfunction rather than true nephrogenic diabetes insipidus. Improvement after plasma exchange illustrates a potential role for extracorporeal therapies in select cases of highly protein-bound drug overdose. This case expands understanding of ibuprofen's toxic renal phenotype, underscores the limits of current management strategies, and emphasizes the importance of aggressive multimodal supportive care in massive ibuprofen overdoses.

046. Acute Toxicity of Recreational Substances: A 13-Year Retrospective Analysis

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Background: The use of drugs for recreational purposes is increasingly common, wich has led to a gradual increase in the number of acute poisonings requiring attention in emergency services.
Hypothesis or Research Question: What are the main demographic and clinical features related to recreational drug use in the emergency department of a Poison Control Center in Mexico State?
Methods: This is a retrospective, descriptive review of a Poison Control Center database at a hospital in Mexico State from February 2012 to October 2025. We included all patients who consulted the emergency department for acute intoxication by recreational drugs. Data analysis was performed using SPSS v25.
Results: From a database of 1852 patients, 491 (26.5%) met the inclusion criteria, of wich 52% (n=253) were male. The mean age was 22 years (range: 13–75 years), and 26% (n=127) were under 18 years old. The average number of cases per year was 35 (range: 10-60, p = 0.0217). Ethanol was the most common substance at 84.9% (n=417), followed by cannabinoids (13%, n=64) and cocaine (8.5%, n=24). The consumption of 2 or more substances was recorded in 140 patients (28.5%), with ethanol being the most common in combination with cannabinoids (n=28, 20%), cocaine (n=22, 15.7%), and benzodiazepines (n=18, 12.8%). The most frequent symptoms were vomiting (13.13%, n=24), drowsiness (12.68%, n=122), nausea (11.64%, n=112), and anxiety (6.55%, n=63). In patients who consumed ethanol, serum concentrations were measured in 66.8% of cases (n=328) with a mean of 210.6 mg/dL (range: 10–485). Toxicological screening was performed in 41% (n=202); of these, 33% (n=67) tested positive for cocaine and 17% (n=34) for cannabinoids (p <0.001). The majority of patients were discharged from the emergency department (69%; n=339), 14% (n=69) left voluntarily, 10% (n=51) were hospitalized, 2.85% (n=14) were admitted to the intensive care unit and one death was

recorded related to methamphetamine intoxication (0.2%). The mean emergency department stay was 13.3 hours (range: 1 hour to 16 days, p <0.001).
Conclusion: More than a quarter of the patients treated by the Poison Control Center were related to recreational drugs, primarily affecting adolescents and males. Ethanol was the most frequently implicated recreational substance, both alone and in combination with other drugs, what reflects its high accessibility and common use in the population. Most patients had a favorable outcome, although a smaller percentage required hospitalization or intensive care.

047. Pediatric Burn Injury From Cyanoacrylate Nail Glue Exposure Through Denim: A Case Report and Review of the Literature

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Background: "Super glues" are common household products; those containing ethel – and methyl – cyanoacrylates are often used in 'nail glue' cosmetics to affix artificial nails. Other than mild irritation and sticking skin to unintended objects, they have minimal harm with direct skin exposure. However, the combination of liquid cyanoacrylate glues with hydroxyl-rich, natural fibers (e.g., cotton, denim, wool) can trigger intense exothermic polymerization reactions causing full-thickness thermal burns. This cause of burn injury is rare, under recognized, and preventable.
Methods: "Super glues" are common household products; those containing ethel and methyl cyanoacrylates are often used in 'nail glue' cosmetics to affix artificial nails. Other than mild irritation and sticking skin to unintended objects, they have minimal harm with direct skin exposure. However, the combination of liquid cyanoacrylate glues with hydroxyl-rich, natural fibers (e.g., cotton, denim, wool) can trigger intense exothermic polymerization reactions causing full-thickness thermal burns. This cause of burn injury is rare, underrecognized, and preventable.
Results: A nine-year-old healthy female presented to the ED with severe pain and blistering to bilateral medial thighs 30 minutes after inadvertently spilling nail glue onto her denim jeans while on the school bus. This resulted in an immediate exothermic reaction producing first and second-degree burns involving 2% of total body surface area (images 1-3). Examination revealed erythema, blistering, and scattered open areas post-blister rupture (caused by blister adherence to the fabric when removed). Standard burn care was provided, including irrigation, debridement, topical antibiotics, and nonadherent dressing. Pain was controlled with oral analgesics. Follow up examination demonstrated minimal to moderate residual scarring, and no functional limitations.
Conclusion: This case highlights a recognized and preventable injury caused by enhanced polymerization of cyanoacrylate adhesives upon contact with hydroxyl-rich fabrics. Prior reports have experimentally shown temperatures exceeding 70°C within seconds of glue – cotton interaction, confirming risk for local full thickness burns. Severe adult and pediatric cases of glue related burns have been reported worldwide with some requiring split-skin grafting; however, medical and public recognition of this injury as well as manufacturer warnings and cautions remain low. Product labeling on these nail glues rarely warns against contact with natural fibers; and child resistant packaging is uncommon. Explicit multilanguage warnings and safe handling instructions are needed to increase recognition of this potential reaction for clinicians, regulators, and public safety.

048. Ten-Year Clinical Outcomes of Buprenorphine Exposures Reported to the California Poison Control System

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Background: Buprenorphine is considered to have a wide safety margin due to a "ceiling effect" on respiratory depression. However, clinically significant opioid toxicity has been reported.
Hypothesis or Research Question: Among patients using buprenorphine therapeutically, do acute exposures to buprenorphine warrant extended (≥12-hour) observation?
Methods: We retrospectively reviewed acute-on-chronic buprenorphine exposures in adult patients reported to the California Poison Control System between January 1, 2014 and December 31, 2024. Inclusion criteria were oral and/or parenteral exposure routes and management at a healthcare facility. Excluded were cases with other exposure routes, miscoded or incomplete cases, cases coded as effects unrelated to exposure, presentations attributed to a non-toxicologic etiology, co-ingestion with ethanol or sedative-hypnotic drugs, and those with known active abuse of full opioid agonists. Data abstracted included demographics, dose, route, symptoms, interventions and disposition.
Results: Forty-two cases were included. The median age was 41.5 years (IQR: 34-57 years), and the median exposure dose was 32 mg (IQR: 22-100 mg). Thirty-four (81%) patients were symptomatic on arrival. Medical outcomes were coded as major in eight (19%) cases, moderate in 19 (45.2%), minor in 10 (23.8%), minimal effects possible in 3 (7.1%), and no effect in 2 (4.8%). The most reported

symptoms were CNS depression (n=26), confusion/disorientation (n=5), vomiting (n=4), bradycardia (n=3), nausea (n=2), headache (n=2), and hypotension (n=2). Respiratory depression was noted in eight cases; however, 14 patients (33.3%) had some degree of respiratory depression documented on narrative review. Twenty-seven (64.3%) patients had documented observation for at least 12 hours and eight (19%) for less than 12 hours. The minimum period of observation was unidentifiable in seven (16.7%) cases. Naloxone was administered to 23 (54.8%) patients, of which 52.2% had a documented positive response and 34.8% had no response. Five (11.9%) were intubated. Nine (21.4%) were admitted to a critical care unit and eight (19%) to non-critical care units; twenty-two (52.4%) were discharged from the emergency department. Of the eight patients that were asymptomatic on arrival, none developed documented respiratory depression, need for mechanical ventilation, admission to a critical care unit or major effects.

Conclusion: Most acute-on-chronic buprenorphine exposures were coded as mild to moderate in severity; however, major effects were noted in nearly 20% of cases, with a higher than anticipated rate of mechanical ventilation (11.9%) and ICU admission (21.4%). No severe outcomes were noted for patients asymptomatic on arrival. Clinicians should maintain a cautious approach with acute-on-chronic buprenorphine exposures.

049. Takotsubo Cardiomyopathy From Combined Opioid and Alpha-2-Adrenergic Agonist Withdrawal

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Background: Combined opioid and alpha-2 adrenergic agonist withdrawal syndrome has recently been described in Pennsylvania due to adulteration of the illicit opioid supply with medetomidine. In addition to the symptoms of opioid withdrawal, this syndrome is characterized by agitation, profuse vomiting and hypertensive emergencies. Induced opioid withdrawal from opioid toxicity reversal has been shown to cause stress-induced cardiomyopathy. We report a case of potential stress-induced cardiomyopathy from severe withdrawal without an inciting event.

Hypothesis or Research Question: Withdrawal from medetomidine, an alpha-2-adrenergic agonist adulterant found in regional opioid supply, can lead to stress-induced cardiomyopathy without preceding exposure to an opioid antagonist.

Methods: A single patient case report of a 64-year-old female with opioid use disorder who presented to the Emergency Department (ED) after being found unresponsive.

Results: The patient presented to the ED with encephalopathy and vital signs notable for tachycardia, with normal blood pressure and oxygen saturation. Initial EKG displayed sinus tachycardia to 123 with 1-2 mm of ST elevation in the anterolateral leads without reciprocal depressions. Echocardiogram was obtained in the ED and was consistent with Takotsubo cardiomyopathy with reduced ejection fraction (15-20%) and troponin returned severely elevated to 19,228ng/L. Interventional cardiology was consulted from the ED and recommended against catheterization given atypical story and lack of chest pain. Medical Toxicology was consulted for suspected severe withdrawal syndrome from opioids and the novel adulterant, medetomidine, an alpha-2 agonist. She was placed on dexmedetomidine infusion to manage alpha-2 agonist withdrawal symptoms and underwent buprenorphine induction for management of opioid withdrawal. She had clinical improvement in her mental status over the following day and successfully transitioned to oral alpha-2 agents and sublingual buprenorphine. Stroke and infectious workups were ultimately negative. Troponins peaked at hospital day zero at 19,302ng/L and began to downtrend that night and were 3489ng/L at discharge. Her mental status returned to baseline, and she was downgraded to the floor. She was ultimately discharged on hospital day four with plans for close outpatient follow up with Cardiology including plans for repeat echocardiogram shortly after discharge.

Conclusion: This case report describes stress-induced cardiomyopathy from severe withdrawal without an inciting event. Providers should be aware of the potential severe complications that can occur from this withdrawal state and the need for early aggressive treatment.

050. A Retrospective Review of Heavy Metal Ordering Practices and Results

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Background: Although uncommon in the United States, heavy metal poisoning remains a consideration in patients with unexplained neurologic or systemic symptoms. Current guidelines discourage heavy metal screening without a suspected or confirmed exposure source, yet provider adherence to these recommendations is unclear.

Hypothesis or Research Question: Providers who adhere to established heavy metal testing guidelines will document a suspected or known exposure prior to ordering a urine heavy metal screen.

Methods: We performed a retrospective analysis of all adult patients receiving emergency department or inpatient care within a large academic health system who had urine heavy metal screens between January 1, 2015, and December 31, 2024. Demographic and clinical data were extracted from the electronic medical record, and descriptive statistics were calculated using R (R Foundation for Statistical Computing).
Results: A total of 211 urine heavy metal screens from 192 patients were reviewed; 83.3% were random collections and 11.8% were 24-hour collections. Twelve (8.3%) were invalid due to interference from recent iodinated contrast. Screens were most frequently ordered or recommended by neurology (n=108, 56.2%), internal medicine (n=27, 14%), and psychiatry (n=13, 6.7%). Medical toxicology was consulted in 31 cases and ordered nine screens. Only 32 (22.2%) patients had a potential exposure source documented, and complete exposure or occupational histories were documented only by medical toxicology. Common indications included neuropathy (n=53, 41%), altered mentation (n=35, 27%), and psychosis (n=18, 13.9%); 30 orders lacked an associated diagnosis. Ninety (62.5%) initial screens detected at least one elevated metal concentration, most often zinc (n=81, 56%), copper (n=44, 29.6%), cadmium (n=30, 15%), and arsenic (n=28, 14.5%). Forty-two (46%) patients underwent repeat testing; three had multiple repeats, including one with eight total tests. No patients received chelation or other specific treatment after screening.
Conclusion: Urine heavy metal screening was often performed without documentation of exposure history and rarely involved toxicology consultation. Most samples were random rather than 24-hour collections, and zinc, the most frequently detected metal, was likely due to contamination from medications or topical agents. Despite frequent abnormal results, testing did not change management. These findings highlight gaps in understanding of the indications and limitations of heavy metal testing and underscore the need for improved provider education and early involvement of medical toxicology when considering the diagnosis of heavy metals poisoning.

051. Characterizing Patient Exposures to Levomilnacipran, a Newer Antidepressant Medication

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Background: Levomilnacipran is a serotonin-norepinephrine reuptake inhibitor (SNRI) that was approved in July 2013 for the treatment of major depressive disorder in adults. It is unique from the other SNRIs due to increased potency in inhibition of norepinephrine reuptake compared to serotonin reuptake. Due to its relative recency in availability, limited data is available regarding its clinical manifestations in toxic exposures.
Hypothesis or Research Question: What are the clinical characteristics and outcomes of levomilnacipran ingestions reported to US Poison Centers?
Methods: This is a retrospective review using the National Poison Data Systems of any single-substance ingestion of levomilnacipran reported to any US Poison Control Center from 1/1/2013 to 1/1/2025. Data was collected on patient demographics, reason for ingestion, location of call, medical outcome, clinical effects, and therapies provided. Descriptive statistics were used to analyze data.
Results: During the study period, 229 patient exposure cases were identified with 183 (79.91%) being female. In the total cohort, there were no reported major outcomes or deaths. The most commonly observed outcome was tachycardia which occurred in 30 (13.1%) cases. There was one case of QRS prolongation, one case of QTc prolongation. There were three reported cases of seizure. There were no cases of ventricular dysrhythmias or clonus. Of the total cohort, 45 (19.65%) cases were documented as intentional self-harm ingestions. The most common effect remained tachycardia, followed by drowsiness/lethargy. The most common therapy provided was intravenous fluids.
Conclusion: Levomilnacipran toxicity is consistent with primarily norepinephrine reuptake inhibition with tachycardia as the primary effect seen without any major outcomes reported in this cohort. Additionally, minimal serotonergic effects reported with no documentation of clonus. This study is limited due to its retrospective nature and limited granularity of information provided by the database. Continued monitoring and documentation of significant ingestions are needed to further characterize levomilnacipran's toxicity profile.

052. Unintentional Edible Cannabis Exposure in Children Six to Twelve Years of Age

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Background: Unintentional exposures to edible cannabis products in children less than six years of age have increased dramatically over the last decade coincident with increasing availability of these products. Although unintentional exposure to any substance is less common in older children edible cannabis products may be an exception.
Hypothesis or Research Question: Are unintentional ingestion of edible cannabis products increasing in children aged six to twelve years?

Methods: This is a retrospective study of children aged six to twelve with reported unintentional ingestion of an edible cannabis product reported to a single poison center from 2016 through October 2025. Cases were retrieved through a search of the National Poison Data System (NPDS). Each chart identified was retrieved and the narrative reviewed in order to confirm exposure route and correct any miscoding of the substance within the Cannabinoid category. Data collected for each case included year of exposure, age, sex, exposure site, management site and, for children seen at a health care facility (HCF), the level of care, clinical effects, therapies and outcomes (as defined by NPDS). Results are reported using descriptive statistics.
Results: Reported exposures increased dramatically from only eight cases reported from 2016 to 2020 to an average of 60 cases yearly from 2021-2025. Edibles accounted for less than one per thousand calls to the poison center for ingestion of any substance prior to 2021 in this age group to over 28 per 1000 calls since. Exposures were evenly distributed between males and females (52.8%, 47.2%). Two hundred eighty-seven of the exposures occurred at home (88.3%) and 1.4% (n = four) at school. There was a progressive decline in unintentional exposures with increasing age (91 at age six to 21at age twelve). Of the 208 children evaluated at a HCF 22.8% were admitted (4% CCU, 18.8% non-CCU) while 77.1% were treated and released. Coded clinical outcomes for children seen at a HCF were no effect in 21 (10.1%), minor in 133 (64%), moderate in 51 (24.5%) and major in three (1.4%). The most common clinical effects were CNS depression, predominantly mild, vomiting, tachycardia and mydriasis. There were two cases of respiratory depression with one receiving intubation. The most common therapy was fluids.
Conclusion: Unintentional exposures to edible cannabis products in children 6-12 years have increased dramatically over the past 5 years, with most cases resulting in minimal toxicity. The majority of exposures occurred at home. Parental education regarding safe storage is imperative.

053. Subcutaneous Buprenorphine Induction in the Emergency Department – Descriptive Analysis of Implementation of the Game-Changer

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Background: Opioid use disorder (OUD) is a major cause of death in the U.S. population. Buprenorphine has become a primary means of medication assisted treatment (MAT). The risk of precipitated withdrawal is a deterrent to starting buprenorphine in the emergency department (ED). Precipitated withdrawal is not only uncomfortable but can also cause serious patient harm by deterring individuals from buprenorphine inductions in the future. In 2023, The FDA approved a subcutaneous (SQ) buprenorphine. A one-time SQ buprenorphine injection simplifies administration and mitigates the risk of precipitated withdrawal due to the slow onset kinetics, with peak serum concentration achieved 24 hours after administration.
Hypothesis or Research Question: We hypothesize that SQ buprenorphine administration in the ED is a safe and effective method for induction and minimizes the risk of precipitated withdrawal.
Methods: This is a retrospective single center chart review. We reviewed charts of all patients who received a 24 mg SQ buprenorphine injection in the ED since its addition to the hospital formulary. Patient demographics, protocol adherence, withdrawal symptoms, and engagement with outpatient chemical dependency (CD) treatment clinics were abstracted. Additionally, the state prescription monitoring program was queried to identify buprenorphine prescriptions from outside providers after the index ED visit.
Results: Between June 2024 and October 2025, 17 cases were identified. 59% were male with a median age of 37 years (min 31, max 68). 65% of individuals were white, 24% were black or African American, and 12% were Hispanic. All patients had withdrawal symptoms documented as mild-to-moderate prior to buprenorphine administration. 53% of patients reported that their last opioid use was within 12-24 hours of ED presentation. 11% reported last use within 12 hours. There were no recorded injection site reactions, and there was one case of reported withdrawal symptoms in the ED. 44% of patients had a buprenorphine prescription written by another provider after the index ED visit, and 30% of individuals had a documented or reported follow-up with CD treatment. One patient reported significant withdrawal symptoms after SQ buprenorphine, and one patient was subsequently seen in the ED for opioid overdose.
Conclusion: While limited by the size and retrospective nature of this study, SQ buprenorphine offers a safe and easy-to-use option to start MAT for OUD in the ED. The SQ buprenorphine formulation simplifies induction strategy for both providers and patients while also mitigating the risk of precipitated withdrawal.

054. Inhalational Methanol Poisoning: A Case Report of Methanol Poisoning From Recreational Huffing of Methyl Acetate

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Background: Methyl acetate is an organic solvent used in certain nail polish removers that can be hydrolyzed to liberate methanol and acetic acid. We present a rare case

of severe methanol poisoning from recreational huffing of methyl acetate containing nail polish remover.

Methods: This is a single patient case report. A 47-year-old female presented to the emergency department via EMS after an episode of loss of consciousness. The patient reported that she poured 3 6 oz bottles of nail polish remover into a towel and began huffing it when she lost consciousness. She reported she woke with the towel over her face approximately 24 hours later. Hospital staff investigated the bottle's ingredient label and discovered the product contained methyl acetate. The patient's reported vital signs were temperature 97.1F, pulse 78 bpm, respiratory rate 18, blood pressure 138/77, and SpO2 99% on RA. She appeared drowsy and intoxicated. Initial laboratory studies included arterial pH 7.123, arterial pCO_2 15, serum $HCO3^-$ 5, and anion gap 21. 3 hours into the ED course, the patient developed blurry vision. Toxicology was consulted and a 15 mg/kg loading dose of fomepizole was administered and nephrology consultation for hemodialysis was recommended. The patient was admitted to the ICU for further management of suspected methanol poisoning.

Results: The patient's initial serum methanol concentration was 35 mg/dL. A bicarbonate infusion was started by nephrology, and she underwent emergent hemodialysis. Fomepizole was continued as recommended by toxicology. The patient's ICU course was uncomplicated—her mental status improved, and her ocular symptoms resolved. A post-hemodialysis methanol concentration was obtained and was 5 mg/dL. Fomepizole and bicarbonate were discontinued, and her acid-base status stabilized. She was deemed medically stable and discharged in stable condition on hospital day 2.

Conclusion: This is a rare case of methanol poisoning secondary to inhalational exposure of methyl acetate. During follow up, the patient reported she had been recreationally huffing nail polish remover for years and this is the first time she had ever lost consciousness. It was unclear whether she had been suing acetone or non-acetone nail polish removers. She was advised of the dangers of using methyl acetate containing nail polish removers and methanol poisoning. It was advised that she seek treatment for addiction. Ultimately, this case underscores the importance of proper exposure identification and how it can guide management of the poisoned patient.

055. Prescription Drug Overdose in Tertiary Care in India: Insights From Isolated and Mixed Drug Intentional Self Harm

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Background: Prescription-drug overdoses form a substantial portion of toxicology presentations, particularly among young adults with access to multiple household medications. The majority of cases involve benzodiazepines, often taken impulsively or intentionally for self-harm, with frequent co-ingestion of other drugs simply because they are readily available. Benzodiazepine (BZD) overdoses are among the most common drug-related emergencies in India with significant fatalities.

Hypothesis or Research Question: We hypothesized that the majority of prescription drug overdoses, whether isolated or mixed present with minimal clinical toxicity and a benign course, provided timely supportive care is administered.

Methods: We present cases referred to the toxicology department of a tertiary care institute during a 12 month period of patients who had consumed prescription drugs, either alone or in combination with other over the counter medications. Data collected included presenting symptoms, vital signs, mental status, type of medication consumed, laboratory and ECG findings, emergency interventions, and patient outcomes.

Results: In 4 out of 18 cases of prescription drug overdose, benzodiazepines were taken. Other frequently reported drugs included Salicylates, Antibiotics, Sedative hypnotics, Anti depressants, Anti psychotics, Oral Hypoglycemic Agents and Paracetamol. Management largely remained supportive, with only two patients presenting in the golden period for gastric lavage. Most had some intervention at a hospital or a primary care facility before referral. No need for antidotes such as flumazenil, and minimal for advanced airway interventions were noted. One case of near hanging patient died during the course of hospitalization due to hypoxia brain injury. Most patients were observed, reassessed clinically, and referred for psychiatric evaluation once medically stable, recovered uneventfully and did not require prolonged hospitalizations.

Conclusion: This pattern reinforces the importance of structured clinical assessment, careful monitoring, and comprehensive medico-legal documentation. Lack of specialized healthcare personnel, advanced lab testing and socioeconomic barriers to healthcare access are frequent challenges in LMICs. A toxicology service provides clear treatment oversight and effective care. There is a need for routine psychiatric intervention as well as follow up, given the emotional and behavioral drivers of these ingestions are far more significant than the toxicological effects of the medications themselves.

056. Niacin-Induced Hepatotoxicity

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Background: Niacin is an essential vitamin metabolized primarily through a high-affinity, low-capacity amidation pathway. Nicotinamide and pyrimidine metabolites produced via this pathway can induce hepatotoxicity by triggering hepatic cellular apoptosis. We present a case of acute liver failure after massive niacin ingestion.
Hypothesis or Research Question: Niacin overdose can cause acute liver failure, and treatment with N-acetylcysteine (NAC) and supportive care may lead to clinical improvement.
Methods: A 38-year-old man with diabetes, alcohol use disorder, and methamphetamine use disorder presented with nausea and abdominal pain after ingesting 75 grams of niacin. He took 150 capsules of 500 mg niacin over five days in an attempt to "self-detoxify" from marijuana, methamphetamine, and alcohol. He reported no immediate symptoms and denied co-ingestants. Initial laboratory studies showed AST 2122 U/L, ALT 5136 U/L, INR 4.9, total bilirubin >4.2 mg/dL, and platelets 85 $\times 10^9$/L. Creatine kinase was normal (74 U/L), hepatitis B and C testing was nonreactive, and ethanol and acetaminophen concentrations were undetectable. Abdominal ultrasound suggested possible underlying cirrhosis. The Oregon Poison Center was consulted and recommended N-acetylcysteine (NAC) along with supportive care. The transplant hepatology service agreed that his acute hepatic failure was most consistent with niacin toxicity and recommended continued medical management. The patient received NAC for four days, with marked improvement in liver enzymes (AST 89 U/L, ALT 663, INR 1.4) at discharge.
Results: This patient developed fulminant hepatic failure following massive niacin ingestion and demonstrated significant clinical and biochemical improvement after stopping niacin and receiving supportive care with NAC. Limitations include the inability to quantify niacin concentrations and the absence of baseline liver function tests in a patient with alcohol use disorder; however, the undetectable ethanol level and rapid improvement in liver tests strongly support acute niacin toxicity as the primary cause of his presentation.
Conclusion: Niacin overdose is an uncommon but notable cause of acute liver injury. This case highlights the risks associated with high dose over the counter supplement use and the potential harms of non-evidence based "detoxification" practices.

057. When Relief Becomes Risk: Magnesium Citrate Overdose Associated With Hypermagnesemia, Seizure and Paralytic Ileus

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Background: Magnesium (Mg) is a divalent cation that can be found in oral cathartic agents, which in overdose can elevate the serum magnesium level. Hypermagnesemia is associated with muscle weakness, respiratory depression, gastrointestinal upset, depressed mental status, hypotension and cardiac conduction disturbances.
Hypothesis or Research Question: Patients with chronic kidney disease (CKD) are not only at increased risk of hypermagnesemia in overdose with saline based Mg containing cathartic agents, but also may develop a degree of tolerance to chronic hypermagnesemia without experiencing significant respiratory or hemodynamic compromise.
Methods: This is a single patient chart review. An 80-year-old female with prior medical history of CKD, congestive heart failure, diabetes, hypertension and atrial fibrillation arrived at the emergency department with altered mental status and weakness after reportedly drinking three bottles of magnesium citrate for constipation. Serial Mg levels were obtained with a peak initial level of 16.0 mg/dL. Initial EKG demonstrated sinus rhythm with QRS interval of 56 milliseconds and QTc interval of 504 milliseconds. She received one liter of isotonic fluids and one gram of calcium gluconate on arrival.
Results: The patient underwent a four-hour session of iHD on hospital day one with repeat serum Mg concentration of 3.1 mg/dL. However, the following day it rebounded to 10.6 mg/dL, and remained elevated despite four additional iHD sessions. She was started on a norepinephrine infusion at 3 mcg/min on presentation which was discontinued on hospital day two. CT of the abdomen and pelvis demonstrated paralytic ileus. For her persistent hypermagnesemia, she was started on furosemide IV 80 mg BID. On day four, she had an episode of seizure-like activity which resolved with 2 mg lorazepam and was started on levetiracetam 500 mg BID by neurology. At that time, she had an ionized calcium of 1.11 mmol/L and this seizure-like activity was not likely a result of hypocalcemic clonus or tetany, and she denied history of chronic alcohol or benzodiazepine use. She returned to her neurological baseline on day seven with a serum Mg concentration of 2.4 mg/dL.
Conclusion: This is a novel case of hypermagnesemia associated with seizure-like activity and paralytic ileus. Her cardiac and respiratory tolerance to such a high concentration and potential seizure imply that she may have had underlying chronic hypermagnesemia prior to her acute overdose prompting her hospitalization.

058. Severe Salicylate Toxicity With CSF Salicylate and Glucose Concentrations Obtained

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Background: Hypoglycorrhachia is abnormally low glucose in the cerebrospinal fluid (CSF), even with a normal serum glucose. Hypoglycorrhachia is a commonly cited potential complication of salicylate overdose and is thought to be related to increased glycolysis and uncoupling of oxidative phosphorylation.
Hypothesis or Research Question: Hypoglycorrhachia may not occur, even in the setting of severe salicylate toxicity and elevated CSF salicylate levels.
Methods: This is a single patient case report. A 64-year-old male was brought to the Emergency Department (ED) by paramedics after a traffic stop by police for concern of driving under the influence. On arrival at the ED, the patient was encephalopathic, diaphoretic, and complaining of dyspnea. Initial vitals revealed: blood pressure 91/54 mmHg, heart rate 81 beats/min, temperature 99.3 F (rectal), respiratory rate 32 breaths/min, and SpO2 100% on room air. Lumbar puncture was obtained 1.5 hours after arrival. Approximately 1.75 hours after arrival, serum salicylate level returned at 92 mg/dL and bicarbonate infusion was initiated. Prior to ICU admission, the patient's encephalopathy and hemodynamics worsened. He was intubated, given activated charcoal, and started on vasopressors. A six-hour run of emergent hemodialysis was performed. Salicylate level normalized after hemodialysis and bicarbonate infusion was discontinued. The patient was extubated on hospital day three and ultimately did well.
Results: Initial laboratory studies revealed VBG with pH 7.31, pCO2 27 mmHg, and bicarbonate 13 mEq/L, lactate 9.3 mmol/L, potassium 4.4 mmol/L, chloride 110 mmol/L, anion gap 17 mmol/L, glucose 273 mg/dL, and serum creatinine 1.74 mg/dL. Initial salicylate concentration in the serum was 92 mg/dL. CSF glucose concentration of 179 mg/dL and CSF salicylate concentration of 52.38 mg/dL were noted. CSF studies were otherwise without evidence of infection. Ethanol, methanol and ethylene glycol were not detected.
Conclusion: The true incidence of hypoglycorrhachia in salicylate toxicity is unknown. This patient presented undifferentiated and CSF samples were obtained while the patient was encephalopathic, prior to dextrose administration and hemodialysis. The CSF glucose level was not consistent with hypoglycorrhachia. However, hyperglycemia on arrival may have been protective. It is also possible that hypoglycorrhachia is a pre-terminal event and less commonly identified clinically. CSF salicylate levels are difficult to interpret, and no reference range is established. This is the highest reported CSF salicylate level to date. Regardless, the treatment for severe salicylate toxicity is hemodialysis which should not be delayed for lumbar puncture, nor if dextrose administration improves encephalopathy.

059. You Kanna't Be Serious: The Rising Trend of Kanna Related Presentations to the Emergency Department

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Background: Kanna (*Sceletium tortuosum*) related products have gained popularity in recent years, both for recreational use to elicit euphoria and for medicinal purposes to purportedly treat anxiety and depression. The kanna plant is native to South Africa and contains alkaloids including mesembrine and mesembrenone which have shown inhibition of 5-HT transporters in vitro. Kanna products are available online as gummies, pills, powders, and tinctures. We sought to evaluate the clinical effects and outcomes of kanna exposures.
Methods: We conducted a retrospective review of ingestions of kanna containing products reported to U.S. poison centers. We extracted data from the National Poison Data System from January 1, 2005, to September 30, 2025, and included both single and multiple substance ingestions of products containing kanna. Age, intent, clinical effects, and therapies associated with single substance ingestions were analyzed.
Results: A total of 101 cases of kanna ingestions were identified, 56 being single substance exposures. The most common co-ingestions in multiple substance exposures were ethanol (n=7) and marijuana (n=7). In single substance ingestions, 48% (n=27) were children 0-12 years, 9% (n=5) were adolescents aged 13-18, and 43% (n=24) were adults aged 19 or over. All cases of children were unintentional ingestions; symptoms reported were abdominal pain, hyperthermia, vomiting, agitation, and drowsiness. Adolescents' ingestions were all intentional; symptoms reported were chest pain, dyspnea, headache and tachycardia. In adults, 75% (n=18) of cases were intentional. The most common symptoms on presentation in adults were agitation (n=6), confusion (n=4), hypertension (n=4), and vomiting (n=3). There were two cases of seizures and one case with clonus reported. A majority of adults (62%) and adolescents (60%) were treated in a health care facility while only 7% of children were. Therapies given included antiemetics, antihistamines, benzodiazepines, IV fluids, magnesium, and potassium.
The median yearly cases between 2011 and 2023 was 5 cases. There has been an increase in cases in 2024 and 2025, with 17 and 16 reported, respectively.
Conclusion: Kanna exposures reported to U.S. poison centers remain uncommon, but recent increases suggest rising availability and use. Most pediatric cases were unintentional

and rarely resulted in symptoms; adults more often presented with symptoms to health care facilities. Clinicians should be aware of the potential for kanna to cause symptoms that may require treatment, such as benzodiazepines. The clinical significance of possible serotonin-mediated effects remains uncertain.

060. Essentially Toxic: Clove Oil-Induced Hepatotoxicity

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Background: Clove oil is extracted from the plant (*Syzygium aromaticum)* native to the islands of Indonesia, with its use dating back to the Chinese Han dynasty and modernly used as a topical anesthetic. Eugenol exerts its hepatotoxic effects by inhibiting voltage-gated calcium channels and prostaglandin synthetase, resulting in anti-inflammatory and analgesic effects, while still posing a risk for glutathione-dependent hepatotoxicity.
Hypothesis or Research Question: How can this case help shape our poison center guidelines for anticipated hepatotoxicity per ingested volume?
Methods: This is a single patient chart review. A 2-year-old fully immunized and previously healthy female ingested approximately 3.75 mL of over-the-counter clove oil toothache relief liquid kit containing 85% eugenol. Within 20 minutes of ingestion, she developed perioral irritation and drowsiness. She was referred to the emergency department in consultation with the poison center. She was noted to be hemodynamically stable, tolerating PO, playing appropriately, and protecting her airway. Her laboratory studies showed an AST 540 U/L, ALT 732 U/L, total bilirubin 0.8 mg/dL, platelets 410 THOU/uL, PT of 14.4 sec, and INR 1.1. N-acetylcysteine was administered in a traditional 3-bag system (150 mg/kg in D5W over 1 hour, 50 mg/kg in D5W over 4 hours, and 100 mg/kg in D5W over 16 hours). Repeat liver function studies revealed an AST 100 U/L, ALT 388 U/L, total bilirubin 0.4 mg/dL, PT 14.8 sec, and INR 1.1.
Results: Post-ingestion liver enzymes and hepatic function studies were obtained. All demonstrated a degree of hepatic injury, without the development of fulminant hepatic failure. External causes including coingestants (salicylates, ethanol, acetaminophen) and viral testing (RPP, HAV, HBV, HCV) were obtained and negative. Ingestion amount and eugenol concentration were calculated to be a total dose of 330 mg/kg. Following 21 hours of N-acetylcysteine, her AST:ALT ratio was < 0.4, with normal hepatic function, and NAC was discontinued. Her liver function fully recovered and she was referred for follow-up with her pediatrician.
Conclusion: The WHO recommends an ingestion threshold of 2.5 mg/kg/day with no observed studies demonstrating toxicity. Currently, there are no guidelines for referral to a healthcare facility or liver function testing. We recommend using a threshold of > 2.5 mg/kg for liver function testing, given observed eugenol-mediated hepatotoxicity in our single case ingestion of 330 mg/kg, successfully treated with N-acetylcysteine.

061. A Linguistic Survey of Cannabis Product Packaging Targeting University Students

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Background: Product packaging reveals intended impressions of the product and consumer characteristics. Prior researchers have evaluated cannabis products' packaging appeal to children, but little has been studied about the university student population, many of whom cannot legally consume cannabis.
Hypothesis or Research Question: This study aims to more clearly delineate the type of cannabis product packaging geographically adjacent to a university and what it suggests about retailers' target demographic.
Methods: This convenience sample product survey considers cannabis products (n=55) purchased within a half mile of a university from the time of decriminalization to the present. Product packages were evaluated manually; name and content labeling legal violations, warnings present, dosing information, and marketing language around selected topics - "natural", "traditional", "social", "cool", "risk to consumer", and "legality of product" - were recorded.
Results: Product types found were edibles (n=46), vapes (n=4), pre-rolls (n=3), oil (n=1), and incense (n=1). All but four of the edibles were sweet foods or candies. Gummies (n=16) and fruity candies (n=11) made up 59% of all edibles, demonstrating significant "sweet", "candy", and "fruity" preferences. These flavor preferences are all associated with youth. Forty-seven (86%) products have "youth appeal" features, most commonly "lookalike product" (n=28, 51%) and "taste descriptors" (n=28). Cartoons also featured prominently (n=19, 35%). Thirty-eight products violated at least one of the cannabis name restrictions in state-level administrative code, the most common being "recreation" (n=38). The most prominently missing information is "use instructions" (71% missing). Twenty-two (40%) products omit cannabinoid content laboratory results, ten (18%) omit serving size, and thirteen (24%) omit warning labels. Thirty-six (65%) of products contained a "legal consumption age" warning. One product contained a "research purposes only"

warning. Sixteen products (29%) did not reference any of the six selected marketing topics. Six products (11%, 38% of products with no identified marketing language) came in unmarked clear packaging. "Legal" language surrounding the product's delta-9 content or the 2018 Farm Bill was the most common product packaging language (n=35, 64%). "Natural" language surrounding sustainability, "vegan", "organic", or "all-natural" was the second-most common tactic (n=13, 24%).

Conclusion: Sellers of cannabis products geographically near a university target inexperienced consumers. Most products advertise the legality of their delta-9 content, undermining any warning messaging present. Minimal consumer benefits were specified on packaging. Youth appeal factors like fruity taste descriptors, edible gummy and candy formulations, and cartoon designs abound. Sporadically-printed use instructions, serving size listing, and warning labeling increase these inexperienced consumers' risk of adverse health events.

062. Naloxone Blood Concentrations After Treatment of Presumed Opioid Overdose Among Patients Presenting to Emergency Departments at 17 U.S. Medical Centers

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Background: Limited research exists describing naloxone blood concentrations after naloxone treatment of suspected opioid overdose. Naloxone blood concentrations may inform real-world analyses of naloxone's effectiveness, particularly comparing intranasal (IN) versus intramuscular/intravenous (IM/IV) administration.

Hypothesis or Research Question: Among patients presenting to the emergency department (ED) after suspected opioid overdose, do blood naloxone concentrations differ between IN and IM/IV administration, and is there a correlation between total dose and measured concentrations?

Methods: The Toxicology Investigators Consortium (ToxIC) Drug Overdose Toxico-Surveillance (DOTS) Reporting Program consisted of ED patients ages > 13 who presented to 17 U.S. medical centers with suspected opioid or stimulant overdose. Data collection included chart reviews, patient interviews, and qualitative/quantitative toxicology analyses conducted by the Center for Forensic Science Research and Education. Medians and interquartile ranges [IQR] were calculated for naloxone concentrations above the level of quantitation (LOQ), and Spearman's correlation coefficients were computed for total naloxone dose and naloxone blood concentrations drawn within 2 hours of ED presentation.

Results: There were 414 subjects with opioid overdose whose blood was drawn within 2 hours, and 306 (73.9%) received naloxone. Of these 306, half (159/306; 52.0%) had naloxone concentrations above LOQ. Overall, there was a statistically significant difference in naloxone concentrations and route of administration ($p<0.001$). Among those who received IN naloxone only with quantitative naloxone concentrations above the LOQ (n = 66), the median concentration was 2.1 ng/mL (IQR: 1.8, 3.2) for those who received < 2.0 mg, 3.7 ng/mL (IQR: 1.9, 5.8) for doses 2.1 – 4.0 mg, 6.6 ng/mL (IQR: 4.2, 14.0) for doses 4.1 – 8.0 mg, and 7.9 ng/mL (IQR: 3.7, 10.6) for doses > 8.0 mg. There was a moderate correlation (rho: 0.42, $p=0.002$) between the total IN dose administered and blood concentrations. There were no statistically significant correlations between naloxone concentrations and total dose for IV only naloxone, IM only naloxone, or combination IV/IM/IN naloxone.

Conclusion: Among subjects who had blood drawn within 2 hours of ED presentation after receiving naloxone, only half had naloxone concentrations above the LOQ. Overall, blood naloxone concentrations were statistically different among routes of administration. When stratifying by route of administration, there was a moderate correlation between the total naloxone IN dose administered and naloxone quantitative concentrations, but this association was not detected for other routes of administration.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

063. Ethylene Glycol Toxicity Complicated by Massive Osmotic Diuresis Leading to Hypovolemic Shock Treated With Hemodialysis

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Background: Ethylene glycol toxicity is primarily mediated by its metabolites, glycolic acid and oxalic acid, which cause severe metabolic and acid-base derangements and multi-organ dysfunction. While oliguria is a common late

finding, the characteristic early hyperosmolarity is mediated by unmetabolized ethylene glycol and may rarely cause marked polyuria and osmotic diuresis as an early feature of toxicity.

Hypothesis or Research Question: Large-volume osmotic diuresis can be treated with hemodialysis in ethylene glycol toxicity.

Methods: This is a single patient case report. A 53-year-old man with history of polysubstance use and schizoaffective disorder presented after intentional ingestion of approximately one-half gallon of antifreeze (Prestone® Prime 50/50 Prediluted Antifreeze/Coolant), which contained 45-55% ethylene glycol, 45-55% water, 1-5% 2-ethyl hexanoic acid, 0-5% diethylene glycol, per Safety Data Sheet.

Results: The patient was initially alert, with temperature 36.7° C, heart rate 109 beats per minute, blood pressure 144/98 mmHg, respiratory rate of 20 per minute, and oxygen saturation of 94% on ambient air. Workup for coingestions was negative with normal electrocardiogram intervals and undetectable serum concentrations of acetaminophen, salicylate, and ethanol. Initial labs were notable for serum osmolality greater than reporting range of 500 mOsm/kg (280 - 300 mOsm/kg), venous pH 7.21 (7.33-7.43), venous blood gas lactate 9.1 mmol/L (1.0-1.4 mmol/L). Calculated serum ethylene glycol concentration exceeded 1122 mg/dL. Patient was loaded with fomepizole at 15 mg/kg, and additionally administered intravenous thiamine and pyridoxine. Within an hour, the patient became obtunded and required intubation for airway protection. Urine output exceeded ten liters in under six hours, reaching a rate of approximately three liters per hour, and the patient developed refractory hypovolemic shock despite more than 12 liters of crystalloid resuscitation, necessitating norepinephrine infusion. Emergent hemodialysis was initiated and continued for eight hours, with resolution of polyuria, discontinuation of vasopressor support, and resolution of encephalopathy. Patient was discharged to inpatient psychiatric facility on hospital day four.

Conclusion: While ethylene glycol toxicity typically results from toxic metabolites, glycolic acid and oxalic acid, this patient's osmotic diuresis resulted from unmetabolized ethylene glycol. This transient phase, preceding acidosis, is an underrecognized etiology for significant morbidity and mortality as severe polyuria from osmotic diuresis can precipitate hypovolemic shock, worsen acidosis, and decrease perfusion. While fomepizole and fluid resuscitation are often sufficient therapeutic interventions for ethylene glycol, massive diuresis secondary to extreme hyperosmolality is an indication for hemodialysis even in the absence of other hard indications like refractory acidemia or renal failure.

064. Kratom Deaths Are Not Secondary to 7-Hydroxymitragynine Adulteration

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Background: Kratom has emerged as a potentially fatal herbal product in the United States, with mitragynine and its active metabolite 7-hydroxymitragynine as possible causes. Recent legislation has moved to ban the intentional enhancement of kratom products with 7-hydroxymitragynine. Yet, it is unclear whether documented deaths from kratom are the results of exposure to kratom in its natural form or from adulteration with 7-OH. We sought to compare quantitative postmortem concentrations of mitragynine (MTG) and 7-hydroxymitragynine (7-OH) from Florida medical examiner cases and compare them to recently published pharmacokinetic data in healthy human subjects..

Hypothesis or Research Question: We hypothesize our subset of mitragynine related deaths will have similar concentrations of 7-OH as is found in kratom leaf powder in phase-one trials. While a dangerous alkaloid of mitragynine, 7-OH may not be solely the cause of all mitragynine deaths.

Methods: From January 2017 through December 2023, we contacted all 25 medical examiner districts in Florida to identify subjects with mitragynine induced fatalities (MIF) and cases in which mitragynine found on autopsy (MFA) but not determined to be the cause. For each case, autopsy and toxicology reports were reviewed for quantitative alkaloid results, demographics, and co-ingestants. Concentrations were reported in nanograms per milliliter (ng/mL). Ratios of 7-hydroxymitragynine to mitragynine were calculated for MIF group and compared to two control groups which included published human data from controlled trials and the MFA group.

Results: We identified 38 mitragynine-induced fatalities across Florida. Quantitative levels for both mitragynine and 7-hydroxymitragynine were available in 6 cases (18 %) and 37 MFA subjects. Mitragynine concentrations in MIF ranged from 270–5,696 ng/mL (mean = 2,199.5 ng/mL), and 7-OH concentrations ranged from 70–966 ng/mL (mean = 410 ng/mL). The mean 7-OH to MTG ratio was 0.22 (range: 0.16-0.24; 95%CI 0.18-0.26) in the MIF group. In the MFA cohort, the mean ratio was 0.28 (95%CI 0.13-0.43) Recently published data on humans indicates that this ratio after single dose of MTG is 0.20 to 0.29; while after multiple doses ranges from 0.16 to 0.21.

Conclusion: Quantitative analysis of mitragynine-induced fatalities in Florida demonstrates a consistent 7-hydroxymitragynine-to-mitragynine ratio comparable to human

pharmacokinetic findings. This concordance suggests that 7-hydroxymitragynine appears in predictable proportion to mitragynine in postmortem settings, supporting its role as a secondary metabolite rather than as an adulterant in fatal cases. State and federal regulators should consider stronger regulation on kratom rather than a myopic focus on 7-OH.

065. Iatrogenic Clonidine Toxicity Secondary to Epidural Dosing Error in a Pediatric Patient

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Background: Clonidine is a central alpha2-adrenergic agonist sometimes added to epidural solutions to potentiate the effects of local anesthetics and opioids. It is rarely reported for pediatric patients to develop significant clonidine toxicity after an epidural block.
Methods: A healthy 6-month-old male, weighing 7.59kg, developed clonidine toxicity following an epidural injection for an outpatient genitourinary procedure. During the procedure, he received general anesthesia and was administered an epidural injection believed to contain 10 mcg of clonidine and 7 mL of 0.25% bupivacaine w/ epinephrine 1:200,000. There were no notable intraoperative vital sign abnormalities. During PACU monitoring over 2-3 hours, his parents voiced concern about persistent lethargy. He was found to be hypotensive (SBP 70-80 mmHg), bradycardic (HR 60-70 bpm), and hypothermic (rectal temperature 34.4°C) with normal blood glucose (130 mg/dL) while in the recovery area. He was then returned to the operating suite for management of presumed local anesthetic toxicity. He received 350 ml normal saline and 2 doses of 0.8 mL epinephrine followed by 12 mL of 20% IV lipid emulsion resulting in no significant changes to vital signs or mental status. He was transferred to the ED and on arrival he was somnolent with pinpoint pupils. He received 2 mg of naloxone IV without improvement. The ED then consulted toxicology for further recommendations. His heart rate improved from 70s to 100s over his 3-hour ED course.
Results: Laboratory studies included CMP, CBC and urinalysis that were unremarkable. Electrocardiogram showed normal sinus rhythm with normal intervals. Toxicology was concerned for clonidine toxicity and had admitted the patient to the pediatric intensive care unit for neurologic checks and continuous vital sign monitoring. He was discharged home without further sequalae 24 hours after his mental status returned to baseline. An iatrogenic dosing error was confirmed related to accidental inversion of the intended clonidine/bupivacaine ratio resulting in administration of between 700-900 mcg clonidine (intended 10 mcg) and 0.1 mL bupivacaine/epinephrine (intended 7 mL after 2 mL test dose). 12 hours after epidural administration, the serum clonidine level was 8.6 ng/mL (0.5-2.0 ng/mL expected 2 hours after oral dosing). Plasma bupivacaine level was undetectable. Urine immunoassay drug screen was negative, and urine liquid chromatography/mass spectrometry overdose panel was positive for naloxone (given in the ED) and clonidine.
Conclusion: This is the first known report of clinically significant cardiovascular and neurologic toxicity following a dosing error of epidural clonidine.

066. Brugada-Like Electrocardiographic Pattern Following Reported Kratom Use in a Young Adult: A Case Report

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Background: Kratom (Mitragyna speciosa) is a botanical product that is increasingly used for stimulant and opioid-like effects. Its cardiac electrophysiologic impact remains poorly characterized, though several alkaloids exhibit adrenergic or sodium-channel activity.
Hypothesis or Research Question: Kratom exposure may be temporally associated with acquired Brugada-like electrocardiographic (ECG) changes.
Methods: This is a single-patient case report. Clinical information was obtained through chart review at Brigham and Woman's. A twenty-six-year-old man with asthma, generalized anxiety disorder, major depressive disorder, and prior opioid use disorder in remission was found unresponsive at home with agonal respirations and pulselessness. Emergency medical services achieved return of spontaneous circulation after approximately fifteen minutes of cardiopulmonary resuscitation. Initial rhythm showed wide-complex tachyarrhythmia requiring multiple defibrillation shocks. On hospital arrival, he was intubated and sedated with propofol and midazolam (propofol later discontinued). Management included electrolyte repletion, amiodarone bolus and infusion, and lidocaine. ECG demonstrated sinus bradycardia with RSR′ in V1–V2, down-sloping ST-segment elevation, and terminal QRS notching consistent with a type I Brugada pattern. Transthoracic echocardiogram revealed mildly reduced left-ventricular systolic function (ejection fraction ≈ 48 %), right-ventricular dilation with impaired function, and biatrial enlargement. Toxicology screening was positive for cannabis; his partner reported daily kratom use and

recent ingestion of hallucinogenic mushrooms (> 24 hours prior). There was no evidence of opioid overdose, and no other cardiotoxic substances were identified. No mitragynine (MTG) or alkaloid testing was performed. During hospitalization he developed fever (T max 101.3 °F), a known precipitant of ventricular arrhythmias in Brugada syndrome.
Results: Recurrent polymorphic ventricular tachycardia required repeated defibrillation. Following aggressive electrolyte correction (K 4.0–5.0 mEq/L, Mg > 2.0 mg/dL) and discontinuation of sodium-channel–active agents, arrhythmias stabilized. Electrophysiology confirmed a Brugada type I pattern, and an implantable cardioverter-defibrillator was placed for secondary prevention. Genetic testing for Brugada syndrome was negative.
Conclusion: This case illustrates a possible association between kratom exposure and Brugada-like ECG changes. While causality cannot be established, awareness of this potential relationship may guide clinicians in evaluating unexplained ventricular arrhythmias among polysubstance users. Further investigation into kratom's electrophysiologic effects is warranted.

067. A Case of 5-Oxoprolinemia From Acetaminophen Ingestion

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Background: 5-Oxoproline (pyroglutamic acid) is an uncommon cause of elevated anion gap metabolic acidosis.
Methods: This is a single patient chart review. A 54-year-old female with a history of Crohn's disease presented after her husband found her at home confused. He reported that the patient had taken more than 100 tablets of 650 mg acetaminophen "arthritis formula" along with ibuprofen and diphenhydramine over the preceding week. Her initial pH was 7.04 with a bicarbonate 10 mmol/L, anion gap 19 mmol/L, acetaminophen concentration 22 mcg/mL, AST 60 units/L, ALT 565 units/L, lactate 0.8 mmol/L, and serum beta hydroxybutyrate 0.8 mmol/L. Salicylate and toxic alcohols were undetectable. Urine drug screen was negative for all classes tested. While in the Emergency Department the toxicology service additionally recommended urine organic acid testing. She received N-acetylcysteine (NAC) (150 mg/kg bolus over 1 hour and 12.5 mg/kg/h continuously) and a sodium bicarbonate infusion (150 mEq/L at 150 mL/h). She was admitted to the intensive care unit (ICU). Her mental status improved with symptomatic care, NAC was discontinued on hospital day 2, with normalization of her pH on day 2. Urine organic acid testing by gas chromatography with mass spectrometry (GC-MS) revealed "extremely high" (3+) excretion of 5-oxoproline.
Results: This patient had an unremarkable initial workup for acidosis, but a presentation consistent with 5-oxoprolinemia. Her comorbidities put her at increased risk, and further testing confirmed the diagnosis. Acidosis in acute acetaminophen ingestion is from mitochondrial disruption as opposed to chronic ingestion where glutathione depletion may result in elevated 5-oxoproline in at risk populations**.**
5-oxoproline is an intermediate in the production of glutathione. Accumulation of 5-oxoproline can occur in congenital defects of the g-glutamyl cycle and in other clinical scenarios such a chronic acetaminophen ingestion. Both scenarios result in decreased glutathione and cysteine concentrations. The decrease in glutathione results in increased g-glutamyl cysteine that is then metabolized to 5-oxoproline. Falling glutathione concentrations decrease accumulation of g-glutamyl cysteine and 5-oxoproline. Abstinence from acetaminophen and administration of NAC are the mainstay of treatment along with supportive care. NAC repletes cysteine and glutathione and may improve the acidosis.
Conclusion: This case highlights the importance of considering 5-oxoprolinemia as a cause of high anion gap metabolic acidosis in the setting of chronic acetaminophen poisoning.

068. Case Report: Recreational Rectal Administration of Compounded Topical Ketamine Gel

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Background: Compounded topical ketamine preparations in Lipoderm bases are relatively new, infrequently prescribed, and poorly described in the literature. Reports of toxicity from these compounded creams are sparse. Rectal administration of these preparations has not been previously reported and likely results in unpredictable and enhanced systemic absorption. We describe the first reported case of acute neuropsychiatric toxicity following intentional recreational rectal administration of a compounded ketamine gel.
Methods: This is a single patient chart review.
Results: A 49-year-old female was found on the bathroom floor at work with altered mental status after reportedly rectally inserting an unknown quantity of her prescribed compounded ketamine hydrochloride 10%, gabapentin 10%, and ketoprofen 10% in Lipoderm base. She had been dispensed 240 grams one month earlier for topical treatment of chronic arm pain. On arrival to a rural emergency department, she was intermittently responsive, oriented only to self and place, actively vomiting, demonstrating bilateral horizontal nystagmus, and "making weird noises, gasping for air". Initial vital signs were notable for tachycardia (136

bpm) and blood pressure of 155/131 mmHg, with an oxygen saturation of 94% on room air. Electrocardiogram showed sinus tachycardia with normal QRS and QTc intervals. Labs were unremarkable apart from a mildly elevated anion gap at 16.3. CT angiography of the head and neck and chest radiograph showed no acute abnormalities. She received one liter of intravenous normal saline and ondansetron. No additional interventions were required. Her mental status improved gradually over the course of the next four hours, though horizontal nystagmus and tachycardia persisted. Approximately nine hours after presentation, patient was back at her baseline mental status with normal oral intake and all symptoms and vital sign abnormalities had resolved.
Conclusion: Rectal administration of a compounded ketamine cream intended for dermal use resulted in significant but self-limited neuropsychiatric and autonomic toxicity. The variable and unpredictable bioavailability of rectal ketamine, especially when delivered via a formulation intended for dermal use, likely contributed to systemic toxicity. As the use of compounded topical ketamine increase, clinicians should be aware of the potential for toxicity when these preparations are misused or administered by non-intended routes.

069. Clinical Characteristics and Treatment Outcomes of Patients With Self-Poisoning at the Poison Control Center, Bach Mai Hospital, 2024–2025

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Background: Self-poisoning is a major global public health challenge, ranking suicide as the third leading cause of death among individuals aged 15–29 years. In Vietnam, epidemiological data on self-poisoning remain limited, and clinical patterns vary across regions. A detailed assessment of clinical features, poisoning severity, and outcomes is essential to improve preventive strategies and optimize the management of high-risk groups.
Hypothesis or Research Question: To describe the epidemiological, clinical and laboratory findings, poisoning severity, interventions, and treatment outcomes of patients with self-poisoning treated at the Poison Control Center, Bach Mai Hospital, from 2024 to 2025.
Methods: A cross-sectional descriptive study with retrospective data analysis was conducted using electronic medical records. Collected variables included age, sex, psychiatric and medical history, toxic agents, reasons for self-harm, clinical manifestations, laboratory parameters, Poisoning Severity Score (PSS) at admission and discharge, treatment interventions, and final outcomes.
Results: Among 810 patients included, the mean age was 33.9 ± 16.29 years (range: 12–94), with a male-to-female ratio of 0.9. Seasonal distribution showed the highest number of admissions in summer (29.8%), followed by autumn (25.9%), spring (23.7%), and winter (20.6%). The most common triggers for self-poisoning were family conflict (29.1%), psychological stress (16.8%), and underlying psychiatric disorders (9.0%). The leading toxic agents included analgesics/antipyretics (22.0%), herbicides (16.8%), sedatives (16.0%), rodenticides (12.7%), pesticides (9.1%), and others (23.4%). Psychiatric comorbidity was present in 22.2% of cases, and 4.7% had a history of previous self-poisoning. At admission, PSS distribution: asymptomatic (25.19%), mild (40.99%), moderate (20.12%), and severe (13.7%). All patients received intravenous fluid therapy. Specific antidotes 35.6% and gastrointestinal decontamination with laxatives in 33.3%. At discharge, 20.4% had mild symptoms, 3.1% moderate, and 4.1% severe; the mortality rate was 0.5% (4 cases). Overall, 86.2% were discharged in stable condition, 9.4% were transferred to psychiatric care, 4.0% left in severe condition against medical advice, 0.5% died. Among severe and fatal cases, herbicides accounted for the majority (63.9%), pesticides (11.1%) and corrosive cleaning chemicals (8.3%).
Conclusion: Self-poisoning predominantly affects young adults and was closely linked to family conflict, psychological burden, and psychiatric illness. While most cases present with mild-to-moderate severity and respond well to treatment, herbicide poisoning is strongly associated with severe outcomes and mortality. These findings highlight the need for strengthened mental health support and targeted prevention programs in high-risk populations.

070. Therapeutic Plasma Exchange in Pediatric Amatoxin-Induced Acute Liver Failure: A Life-Saving Case Report

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Background: Amatoxin-containing mushroom ingestion is a rare but life-threatening. Despite advances in supportive care, no specific antidote is available, and liver transplantation is

often inaccessible in resource-limited settings. Therapeutic plasma exchange (TPE) has emerged as a potential adjunctive therapy, but evidence in pediatric amatoxin poisoning remains limited.

Hypothesis or Research Question: In pediatric patients with amatoxin-induced acute liver failure managed in resource-limited settings without immediate access to liver transplantation, can early scheduled therapeutic plasma exchange, combined with intensive supportive care, improve liver function and enable transplant-free survival?

Methods: Medical history was obtained from the patient and family, including details of mushroom ingestion. Clinical symptoms and physical examinations were recorded throughout hospitalization. Serial laboratory tests—liver enzymes, bilirubin, ammonia, coagulation profile, complete blood count, renal function, electrolytes, and arterial blood gases were used to monitor disease progression. Viral hepatitis markers were tested to exclude infectious causes; autoimmune hepatitis. Therapeutic plasma exchange sessions were performed using standard protocols.

Results: A 11-year-old boy developed severe acute liver failure following ingestion of wild mushrooms from the mountainous area of Che La commune, Ha Giang province, Vietnam. The patient presented on day 4 after ingestion with vomiting, diarrhea, jaundice, markedly elevated liver enzymes (AST/ALT 3846/4011 U/L), hyperammonemia (103.8 μmol/L), and profound coagulopathy (PT 14%, INR >5). The diagnosis of amatoxin poisoning was supported by the ingestion history, delayed gastrointestinal symptoms, and family member. The clinical course marked by severe hepatocellular injury, coagulopathy, and hyperammonemia was consistent with amatoxin toxicity. Other virus negative with HAV IgM, HBcAb IgM, HCV Ab, HBsAg, HEV/HAV/CMV/EBV IgM testing, absence of autoimmune hepatitis. The combination of exposure history, clinical progression, laboratory findings, and exclusion of alternative etiologies confirmed amatoxin-induced acute liver failure. Comprehensive management included N-acetylcysteine, vitamin K, antibiotics, fluid resuscitation, and therapeutic plasma exchange. Four daily TPE sessions were performed, exchanging 1.7 L of plasma per session. Rapid improvement in liver function was observed after TPE initiation, with AST decreasing from 3846 to 64 U/L, INR normalizing from >5.0 to 1.05, bilirubin falling from 48.9 to 3.7 μmol/L, and ammonia decreasing from 104.3 to 37.8 μmol/L. The patient fully recovered without requiring liver transplantation.

Conclusion: Therapeutic plasma exchange may play a critical role in improving outcomes in pediatric amatoxin poisoning complicated by acute liver failure. Early recognition and prompt initiation of intensive supportive care combined with TPE can be life-saving.

071. Reversible Cerebellar and Peripheral Neurotoxicity Following Chronic High-Dose Metronidazole Use

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Background: Metronidazole is a widely used antibiotic generally considered safe. However, prolonged high-dose use is rarely associated with reversible central nervous system (CNS) encephalopathy and peripheral nervous system (PNS) toxicity.

Hypothesis or Research Question: This case report describes the clinical, radiological, and electrophysiological findings of concurrent metronidazole-induced CNS and PNS neurotoxicity.

Methods: This is a single-patient case report. We describe the clinical course, laboratory findings, neuroimaging, and electrodiagnostic results of a patient presenting to the National Poison Control Center, Bach Mai Hospital, Vietnam, with progressive neurological deficits.

Results: A 52-year-old male with a history of hepatitis B self-administered daily oral metronidazole for approximately four months for dental pain. He presented with dysarthria, ataxia, left-sided weakness, and bilateral leg numbness. Initial brain MRI revealed characteristic symmetric T2/FLAIR hyperintensities in the bilateral cerebellar dentate nuclei and surrounding the fourth ventricle. Electromyography (EMG) confirmed a sensory axonal polyneuropathy, primarily in the lower limbs. CSF studies were normal. Metronidazole was discontinued, and supportive care was provided. The patient's central symptoms (dysarthria, ataxia) improved markedly within one week. A follow-up MRI 12 days after admission demonstrated complete resolution of all cerebellar lesions. The peripheral numbness improved but persisted mildly at discharge.

Conclusion: Metronidazole-induced neurotoxicity should be considered in patients with unexplained cerebellar syndromes or polyneuropathy, particularly with prolonged use. This case highlights the classic, reversible MRI findings of central toxicity and the concurrent, slower-to-resolve peripheral nerve damage. Prompt cessation of the drug is critical and can lead to rapid radiological and significant clinical recovery.

072. Buprenorphine Microinduction: A Case Series

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Background: Buprenorphine is a first-line medication for the treatment of opioid use disorder (OUD), but initiation may be hindered by the need for ongoing full opioid agonist therapy or recent use of long-acting opioids such as methadone. Microinduction offers an alternative for transitioning to buprenorphine, though protocols vary across clinical settings.
Hypothesis or Research Question: We hypothesize that a 3-day buprenorphine microinduction protocol provides a safe, effective, and replicable approach for transitioning patients from full agonist opioids to buprenorphine.
Methods: This retrospective case series was conducted at an urban tertiary care hospital. Patients who received intravenous (IV) buprenorphine during hospitalization were identified, and records were reviewed to determine eligibility, defined as completion of a microinduction protocol. Microinduction was characterized by initiation of IV buprenorphine at a dose below 1 mg, followed by gradual dose escalation. Data were entered into REDCap and descriptive statistics were completed for analysis.
Results: Forty-eight patients were included in this analysis; 52.1% (25/48) were female, 47.9% (23/48) were male. Fifty percent of patients (24/48) identified as Black, 37.5% (18/48) as White/Caucasian, 4.2% (2/48) as American Indian or Alaska Native, 6.3% (3/48) as Other, and 2.1% (1/48) Unknown. The median hospital length of stay was 11.5 days. Microinduction was initiated by the toxicology service in 58.3% (28/48) of cases, and by the palliative service in 41.7% (20/48). The most common reasons for microinduction were: history of OUD with acute pain requiring full agonists (29.2%, 14/48), long-term opioid therapy without history of OUD (22.9%, 11/48), and transitioning from methadone to buprenorphine (22.9%, 11/48). Patients receiving full opioid agonists inpatient received a median 84 OME (inter-quartile range, 23 – 143.5) in the 24 hours leading up to microinduction initiation, and most patients required continued full opioid agonist therapy at the start of microinduction (70.8%, 34/48). A 3-day microinduction regimen was satisfactory in 43.8% (21/48) of cases, and microinduction was completed in 72.9% (35/48) of cases. The most common reasons for noncompletion were: patient left the hospital (10.4%, 5/48) and inadequate pain control (8.3%, 4/48). PW symptoms occurred in 16.7% (8/48) of patients; none reported worse than "mild" symptoms and no patient stopped the microinduction due to PW symptoms. Other adverse events were rare but included headache, tremor, nausea, opioid toxicity, and cardiac arrhythmia (supraventricular tachycardia).
Conclusion: This case series suggests buprenorphine microinduction is a safe, effective method to transition patients from full opioid agonists to buprenorphine, though individual alterations to a standard 3-day regimen may be necessary.

073. The Evolution of Central Alpha-Agonists in Minnesota: Xylazine and Medetomidine

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Background: The opioid epidemic has been marked by the widespread detection of synthetic opioids and, increasingly, adulterants including the central alpha-2 receptor agonists (CARs) xylazine and medetomidine. We describe the evolving detection of CARs in a community public health surveillance program for novel substances of abuse.
Hypothesis or Research Question: We describe the evolving prevalence of the central alpha agonists, xylazine and medetomidine, in Minnesota, identified through a collaborative effort of the state department of health and bedside emergency clinicians.
Methods: Observational public health surveillance of emergency departments (EDs) at two large healthcare systems in the upper Midwest. Residual biological samples of ED patients seen for presumed unintentional drug overdose were evaluated with non-directed liquid chromatography/high resolution mass spectrometry against a large library (>1,300) of substances. As new substances were identified or suspected, additional standards were added. We describe the evolving prevalence of CARs over the first 5 years of screening via this surveillance project.
Results: Between January 2020 and September 2025, 5077 patients meeting inclusion criteria presented to affiliated EDs; biological samples were available in 1,149 (22.6%) of these. CAR testing was limited to xylazine until November 20 2024 when medetomidine testing was subsequently initiated. CARs were first identified in 2020 (n=1 of 277 cases, 81 samples [1.2% of samples]), with steadily increasing identifications through 2025; over this period, xylazine identifications initially increased, followed by a decrease in 2025 (see Graph). Medetomidine was first identified in December 2024, and increasingly identified over the next 9 months. CARs were co-identified in 21% of samples positive for opioids or opioid metabolites (123/586 cases). CARs were most commonly identified among white and Black/African American patients, with lower detection proportions observed across other racial and ethnic groups. Medetomidine detection in this sample increased at the end

of the observation period, while instances of xylazine detection decreased.
Conclusion: CARs are now detected in [blinded for review] in biological samples collected from patients following unintentional overdose when analyzed as part of this collaborative public health surveillance project. Within this data, xylazine and medetomidine were commonly co-identified with opioids. As xylazine observations have decreased, medetomidine detection has increased, but further data collection is needed to detect a sustained change in the prevalence of both. This data may help clinicians shed light on atypical cases, such as cases of opioid overdoses refractory to naloxone, and may be considered in other regions.

074. Characteristics of Overdoses in Patients Who Obtain Opioids From an Unfamiliar /Atypical Source Versus a Usual/Typical Source

Tricia Dowis[1], Daniel Tirado[1,2], Rachel Culbreth[3], Paul Wax[3], Jeffrey Brent[4], Kim Aldy[3,5], Alyssa Falise[3], Alex J Krotulski[6], Robert G Hendrickson[1], On Behalf of the ToxIC DOTS Study Group
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Background: People who use fentanyl often use multiple times a day and rely on a consistent source for this supply. Obtaining drugs from a new dealer or an unfamiliar source may increase the risk of overdose. There is little data describing the clinical characteristics and blood concentrations of fentanyl in patients that use fentanyl from an unfamiliar source.
Hypothesis or Research Question: Is using fentanyl from an unfamiliar source associated with higher blood fentanyl concentrations among subjects who overdose?
Methods: The Toxicology Investigators Consortium (ToxIC) Drug Overdose Toxico-Surveillance (DOTS) Reporting Program (2022 – 2024) consisted of ED patients ages 13 and older who presented to 17 U.S. medical centers with suspected opioid or stimulant overdose. DOTS data collection included chart reviews (e.g., prehospital naloxone), subject interviews, and qualitative/quantitative toxicology analyses conducted by the Center for Forensic Science Research and Education. Summary data includes medians and interquartile ranges [IQR] for skewed data, and Wilcoxon Rank Sum Tests with Continuity Corrections and Chi-Square or Fisher's Exact Tests were used for testing statistical differences.
Results: Among 587 subjects with an opioid overdose presentation, 361 used drugs from their usual source, and 226 used from an unfamiliar source. Subjects who used fentanyl from an unfamiliar source generally used a single drug (61%) and by a usual route (66%) and had similar markers of risk-taking behavior than those with a usual source (e.g. previous overdose, previously incarcerated, using more than one drug, p=NS). Of those with unfamiliar sources, there was no difference in fentanyl concentration amongst subjects who used someone else's medication (3.1 ng/mL; IQR 1.6, 9.8), usual dealer but not usual brand (4.8 ng/mL; IQR 3.0, 11.0), not usual dealer but usual brand (4.0 ng/mL; IQR 2.1, 8.7), not usual dealer and not usual brand (4.9 ng/mL; IQR 2.8, 7.0). However, a higher percentage of subjects that used fentanyl from an unfamiliar source received 2 or more naloxone doses (50.4% vs. 43.2%, p=0.03) and had a higher average IN dose (unfamiliar source: mean: 6.5 mg; median: 4.0 mg; IQR: 4.0, 8.0 vs. usual source: mean 5.1 mg; median: 4.0 mg; IQR: 2.0, 8.0; p=0.02).
Conclusion: Subjects who used fentanyl from an unfamiliar source had similar characteristics to those who used from a usual source but were treated with more doses of naloxone and had higher average IN naloxone doses. Our results are limited by subject self-report and the potential for recall bias.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

075. Acute Loss of Vision Attributed to Serum Acidemia With Rapid Resolution During Hemodialysis

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Background: We present a case of acute reversible blindness caused by ketoacidosis. The blindness was initially thought to be caused by acute methanol toxicity, but was later attributed to severe ketoacidosis in the context of recent alcohol ingestion as well as being on a ketogenic diet. Severe acidemia has been previously described as a possible cause of acute vision loss.
Methods: This is a single patient chart review. A 50-year-old female with a history of seizure disorder, hypertension, and bulimia presented to the emergency department with acute onset painless vision loss that began about one hour prior to arrival. She was in her normal state of health except for generalized weakness and slurred speech for three days preceding her presentation. The night prior she had drank a pint of vodka and endorsed regular alcohol use (about 2-3 drinks per week). She also endorsed being on a ketogenic diet. On examination there was complete bilateral loss of

all visual fields. Pupils were non-reactive to light bilaterally. Her exam was otherwise unremarkable with no other focal neurological deficits noted. A venous blood gas revealed a pH of 6.78. She had a bicarbonate level of 3 mmol/L with an anion gap of 54 mmol/L. The glucose level was 164 mg/dL, creatinine 3.0mg/dL, BUN 30mg/dL, and lactate 8.8 mmol/L. A beta-hydroxybutyrate level was 8.5 mmol/L, and blood ethanol was 98 mg/dL. CT imaging of the head did not reveal any acute abnormalities or perfusion deficits. The patient was started on a continuous bicarbonate infusion and received fomepizole, thiamine, and folate. In consultation with nephrology, emergent dialysis was started for presumed methanol toxicity.

Results: During dialysis she began having rapid resolution of the vision loss. The next day, a serum methanol and ethylene glycol level, collected prior to hemodialysis, resulted as negative. The patient was discharged on day six of admission in her normal state of health. Case reports have previously described acute vision loss associated with metformin-associated lactic acidosis, as well as a few case reports of acute vision loss associated with severe diabetic ketoacidosis and alcoholic ketoacidosis.

Conclusion: Serum acidemia can be a source of acute loss of vision, which may rapidly resolve with correction of serum pH with hemodialysis.

076. 7-Hydroxymitragynine: A Small Modification, a Major Reaction and Complicating Buprenorphine Management

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Background: Kratom (Mitragyna speciosa) primary alkaloid, mitragynine, metabolizes to 7-hydroxymitragynine (7-OH), a potent μ-opioid receptor partial agonist. Purified commercially available "7-O" products are now widely accessible. 7-OH can have as much as 10 to 22 times greater binding affinity for the mu-opioid receptor than morphine. Numerous 7-OH withdrawal cases have been described, but consensus management of these withdrawal syndromes has not been established. We describe a case of 7-OH withdrawal with a full agonist bridge to buprenorphine.

Hypothesis or Research Question: Buprenorphine, a strong, partial opioid agonist, induction for 7-OH withdrawal may precipitate withdrawal.

Methods: This is a single patient chart review. A 49-year-old-male man presented to a tertiary medical center with congestive heart failure, atrial fibrillation, and hypertension with restlessness, myalgias, rhinorrhea, and gastrointestinal distress. Substance-use history and urine comprehensive toxicology panel using liquid chromatography with tandem Quad-Time of Flight (QTOF) mass spectrometer obtained.

Results: He endorsed daily use of this product varied between 200-400mg of 7-OH per day for 3 months and had tried to wean himself off 7-OH. He denied using any other opioids. His symptoms were consistent with moderate opioid withdrawal (Clinical Opiate Withdrawal Scale (COWS) 14) and were complicated by atrial fibrillation with rapid ventricular response. His last dose of 7-OH occurred one hour before arrival. Due to active arrhythmia and uncertain pharmacokinetics of 7-OH, buprenorphine induction was deferred. He was monitored overnight with an oxycodone bridge with 5mg every 6 hours with last dose at midnight. By morning, COWS increased to 24, last 7-OH dose was 18 hours prior, and he was successfully induced with 12 mg sublingual buprenorphine without precipitating withdrawal. Symptoms improved significantly (COWS 6), though atrial fibrillation persisted. His opioid and fentanyl immunoassay were negative: LC-MS confirmed mitragynine and 7-OH.

Conclusion: Purified 7-OH withdrawal poses substantial clinical management challenges related to its opioid receptor potency, rapid redistribution, prolonged terminal elimination (half-life up to 25 hours with chronic use), and inconsistent product composition. This results in severe withdrawal and uncertainty regarding buprenorphine induction timing. Furthermore, this patient did not tolerate the oxycodone bridge well. Given that 7-OH is a partial opioid agonist, he may have tolerated buprenorphine induction sooner without risk of precipitating withdrawal. Finally, increasing availability and legislative changes related to these semi-synthetic kratom derivatives may increase withdrawal cases and highlight the need for improved surveillance and further study of buprenorphine induction to optimize induction strategies.

DAY 2: PLATFORMS, ABSTRACTS 077-080

077. Longitudinal Assessment of PFAS Body Burden: Implications for Patient Screening and Monitoring

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Background: Per-/poly-fluoroalkyl substances (PFAS) persist in the environment, may have long biological half-lives, and may cause adverse health effects. Many chemicals comprise PFAS, and effects can be additive.

Hypothesis or Research Question: This study examined PFAS combinations in serum/ plasma (SP) samples from a large population, compared the National Academies of Sciences, Engineering, and Medicine (NASEM) summation results recommended action concentrations, and assessed changes over four years of testing.

Methods: The laboratory information management system was queried for SP results from cases submitted between January 1, 2022, and June 30, 2025. Testing included 13 analytes (PFHxA, ADONA, branched and linear PFOA, PFHxS, PFNA, PFDA, branched and linear PFOS, PFHpS, PFUnDA, MeFOSAA, and PFDoDA). NASEM summation results came from this dataset. The timeframe was expanded to capture patients with five annual tests. Baseline and year 1 testing included PFBS, PFHpA, PFNA, linear PFOA, linear PFOS, and PFHxS; years 2-4 included all 13 analytes. For comparability, only results above the reporting limits of the larger assay were analyzed. Statistical analyses were performed in R (2025).
Results: Over 20,000 SP samples were analyzed; 99.2% were positive for at least one PFAS. Linear PFOA, PFHxS, branched/ linear PFOS, and PFNA were detected in >95.5% of samples. Most cases (96.4%) contained 5–8 PFAS; the most common combination (28%) included branched/ linear PFOS, linear PFOA, PFHpS, PFHxS, and PFNA. NASEM summations showed 3.2% of samples were <2.0 ng/mL, 87% were 2.0–19 ng/mL, and 9.8% were ≥20 ng/mL. Forty-nine patients had annual testing; 44 had at least one PFAS positive at all five time points. PFHxS (median [IQR]: 2.70 [1.60–4.55] ng/mL at baseline vs. 2.30 [1.15–2.90] at year 4), PFNA (0.37 [0.28–0.46] vs. 0.28 [0.21–0.36]), and linear PFOA (1.30 [0.87–1.70] vs. 0.91 [0.57–1.20]) significantly decreased over 4 years (paired Wilcoxon, $p < 0.001$). There was no difference between baseline (4.35[2.32-6.35]) and year 4 (4.15[2.70-5.50] for linear PFOS but concentrations significantly increased between baseline and year 3 (5.05[3.13-7.4]; $p = 0.005$; $r = 0.52$).
Conclusion: Most individuals' body burden comes from mixtures of 5-8 PFAS. Most patients may need additional medical screening and monitoring. Reported half-lives of PFAS with 4-year dataset can range from years to decades. The decrease in concentration observed is consistent with published literature. Interpretation of the causes for concentration changes is limited by a lack of information on changes in exposure between data points.

078. Healthcare Utilization Increased After PCB Exposure at a Contaminated School: A Cohort–NHANES Comparison

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Background: An estimated 26,000 U.S. schools have been built with PCB-containing materials. These PCB sources can off-gas volatile PCBs, become airborne, adhere to dust, carpets and surfaces and can result in spikes from failing PCB-containing fluorescent ballasts. At a Seattle-area school site (SS), environmental testing and remediation records document sustained airborne PCB contamination with intermittent peaks prior to 2016. Given the well-documented toxicity of PCBs, the health status of students and staff were of concern.
Hypothesis or Research Question: Did healthcare utilization among the School Site-exposed children and adults increase after exposure, compared with (1) their pre-exposure period and (2) a nationally representative reference sample (NHANES)?
Methods: We conducted a retrospective observational analysis of medical records abstracted by trained registered nurses using a standardized, double-checked workflow. Visits were limited to outpatient sick/well/follow-up encounters (ER/inpatient excluded). Two time points were aligned to NHANES cycles: 2009 (before exposure for the majority) and 2017 (during and after exposure). The outcome (visits in prior year) was categorized as 0, 1, 2–3, or ≥4. We used ordinal logistic regression (adjusted for age and sex) to compare School Site with NHANES and Cochran–Mantel–Haenszel tests for distributional differences; analyses were stratified by children (1–17) and adults (≥18).
Results: Before exposure (2009), SS children and adults had visit frequencies comparable to NHANES (children OR=1.18, 95% CI: 0.77–1.82; adults OR=1.32, 95% CI: 0.79–2.21). After exposure (2017), utilization was markedly higher in the SS cohort (children OR=7.10, 95% CI: 4.39–11.49; adults OR=5.50, 95% CI: 3.22–9.39). These comparisons used NHANES weighting and demonstrate parity at prior to exposure with significant divergence after exposure.
Conclusion: Healthcare utilization rose substantially among School Site exposed children and adults after documented PCB exposure, relative to national expectations and their own pre-exposure utilization. Findings are consistent with increased symptom burden prompting care-seeking. Strengths include objective medical records, standardized abstraction, and an external national comparator; limitations include observational design and a non-random sample not intended for population generalization.

079. AMIGO: An Explainable AI Model for Predicting Hospital Need in Poison Control Home Calls

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Background: One in five home exposures that Poison Control Centers (PCCs) refer to hospital do not need medical treatment. Existing decision support tools require laboratory data that

those calling from home do not have. To assist PCC triage for home calls, we developed AMIGO ("AM-I-GO"), an explainable artificial intelligence clinical decision support tool.
Hypothesis or Research Question: How accurately does AMIGO identify exposures that require medical evaluation using only information available from a call from the public?
Methods: We trained a machine learning model, XGBoost, using cases from the Lyon PCC in France from 2000–2024 to predict whether the caller should stay at their current location or go to hospital. We excluded incomplete cases and calls from hospitals. Our model predicted three outcomes: stay at home, stay in a facility, or go to hospital. We developed the model on an 80% random sample of the data and evaluated it on the remaining 20%. We evaluated model performance using the F1 score (harmonic mean of precision and recall) and area under the receiver operating characteristic curve (AUC ROC). We used SHAP values (SHapley Additive exPlanations) to identify predictive features. We benchmarked AMIGO against published algorithms optimized for specific exposures.
Results: Of 549,098 screened cases, 215,914 met inclusion, representing 1,453 unique exposures. The PCC dispositions were: stay at home, 66.6%; stay at healthcare facility, 7.4%; and go to hospital, 25.4%. The median age (interquartile range) was 9 (2–36.5) years, 49.8% male, and 50.2% female. XGBoost obtained an AUC ROC of 0.87 (95% CI 0.87–0.89); accuracy 0.79 (0.78–0.80); sensitivity 0.71 (0.70–0.72); specificity 0.65 (0.64–0.66); F1 0.67 (0.67–0.68). Intent, time from exposure to call, and symptomatology were the most predictive features. AMIGO achieved F1 0.69 (0.66–0.71) for paracetamol versus 0.76 from prior models and 0.64 (0.61–0.67) for diphenhydramine versus 0.75 from prior models.
Conclusion: AMIGO, a generalist explainable AI triage tool trained on PCC data from public calls, predicts the need for medical evaluation using clinically plausible variables. It covers 1,453 substances and performs comparably to models that are optimized for specific exposures and require laboratory testing. Single-center data and retrospective design are limitations of our study. AMIGO's significant but not total agreement with clinician recommendations demonstrates alignment with clinical rationale and provides an avenue to improve PCC home triage recommendations.

080. Patients Report Similar Experiences With In-Person Versus Virtual Peer Recovery Coaches

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Background: Peer recovery coaches are increasingly deployed in hospital settings to assist people with substance use disorders (SUD) in navigating their recovery. From a window of lived experience, peer coaches provide motivational interviewing and linkage to resources. Although telehealth (virtual) peer coaches have been demonstrated to be feasible in both emergency department and inpatient settings with similar rates of short-term emergency department utilization, there are concerns that using a telehealth format may negatively affect therapeutic alliance between patients and peer coaches.
Hypothesis or Research Question: Patients seen by an in-person peer coach report higher therapeutic alliance than those seen by a telehealth peer coach using a tablet device.
Methods: This is a convenience sample survey of patients with SUD seen at a single hospital from 6/2023-8/2025. Peer coaches met with patients at the bedside (in-person) or via a tablet device (virtual). After the encounter, research assistants approached patients and asked them to complete an anonymous survey about the encounter. Patients reported the peer coach they had seen, and completed the Session Rating Scale (SRS) Version 3, which includes four questions on a 0-100 scale covering domains of relationship, goals and topics, approach or method, and overall. We performed a permutation ANOVA to evaluate for differences in SRS scores between the in-person and virtual arms, and reviewed quotes for common themes.
Results: We enrolled 177 participants, 78 of whom saw an in-person and 99 a virtual peer coach. SRS scores were high overall, with respective mean scores (95% CI) of 97.4 (96.2-98.5), 94.8 (92.9-96.6), 94.7 (92.3-96.8), and 94 (92-97), in the domains of relationship, goals and topics, approach or method, and overall. Mean (95% CI) "overall" scores were 96.4 (94.0-98.3) in the in-person group and 93 (89.2-96.2) in the virtual group (p=NS for comparison). Free text quotes were similar across the two arms (Table), generally conveying gratitude for peer coach services, however several patients expressed that they wanted more time for personal conversation.
Conclusion: In this survey of hospitalized patients with SUD seen by a peer coach, there were no reported differences in perceived therapeutic alliance when comparing patients seen in-person or using a tablet device. Patients in both arms reported that peer coaches sometimes seemed short on time, possibly reflecting pressures of the busy hospital environment. As the survey was conducted anonymously, we could not analyze possible relationships between SRS scores and patient-level outcomes such as subsequent substance use or rehospitalization.

DAY 2: MODERATED POSTERS, ABSTRACTS 081-087

081. Variation in Buprenorphine Induction Practices by Medical Toxicologists Across 36 Medical Centers

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Background: Despite growing adoption of emergency department (ED)-initiated buprenorphine, limited research has characterized variation in clinical practice across ED/inpatient settings in the U.S.
Hypothesis or Research Question: What are the patient-level characteristics and site variability associated with low-dose, high-dose, and standard dose buprenorphine induction practices by medical toxicologists across the US?
Methods: This analysis was conducted using the Toxicology Investigators Consortium (ToxIC) Opioid Use Disorder (OUD) Sub-registry, a database consisting of patients over 13 years of age that received a medical toxicology consultation in the ED/inpatient setting for OUD related visits, including opioid overdose, withdrawal, and OUD evaluations (e.g., patients on maintenance buprenorphine who are admitted for surgery). Data collection includes presentation type (opioid overdose, withdrawal, and/or OUD evaluation), patients' medication for OUD (MOUD) prior experience, induction dose, location of induction, and other clinical data. This analysis focused on patients undergoing buprenorphine inductions. The initial buprenorphine dose was classified using the American Society of Addiction Medicine's Guidelines (2023) for low-dose buprenorphine (LDB; 0.25 – 2.00 mg), standard-dose buprenorphine (>2.00 – 8.00 mg), and high-dose buprenorphine (HDB; 8.01 – 16.00+ mg). Mixed effects ordinal regression models were computed to determine patient-level characteristics associated with buprenorphine induction doses while simultaneously assessing site variation via intra-class correlation (ICC).
Results: Cases in the ToxIC OUD Sub-Registry (N = 1465) represented 36 institutions across 17 states, and 492 (34%) were initiated on buprenorphine. Almost half received initial doses of LDB (48%), 36% received standard doses, and 16% received HDB. Patients who presented with opioid withdrawal or after an overdose were more likely to receive HDB compared to those seen for OUD evaluations (18.9% and 18.2% vs. 9.8%, p=0.01). HDB was more likely to be administered in the ED compared to inpatient settings ($p<0.001$). Patients with prior MOUD experience were more likely to be initiated on LDB (47.3% vs. 39.8%, p=0.04). In the model controlling for age and sex, OUD evaluations were less likely to receive HDB (OR: 0.34; 95% CI: 0.18, 0.63), and there was significant site variation in buprenorphine induction practices (ICC = 0.39; $p<0.001$).
Conclusion: Thirty-nine percent of the total variation in buprenorphine practices was attributed to differences in medical centers rather than patient-level factors. Future research should examine hospital-level protocols and policies that may impact site characteristics for buprenorphine inductions.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

082. Optimizing Best Practice Advisory (BPA) Alerted Kit in Hand Naloxone Distribution From the Emergency Department

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Background: Recent data suggest opioid related deaths are declining. This may be in part due to increased access to intranasal naloxone. Emergency department (ED) visits for unintentional opioid overdoses pose an opportunity to dispense kits to those at highest risk. Following implementation of an electronic health record best practice advisory (BPA) at our institution, naloxone kit in hand increased from 15.6% to 56.1%; but additional improvement could be made.
Hypothesis or Research Question: The BPA alerts by emergency medicine (EM) physician assigned ICD-10 diagnosis code related to opioid use disorder and opioid overdose at discharge (F.11 & T.40). We suspected clinicians are assigning broader drug overdose diagnosis codes for opioid overdose patients that are not causing the BPA to trigger. We hypothesized broadening diagnosis codes would improve naloxone kit in hand at ED discharge and not result in significant alert fatigue for clinicians.
Methods: In June 2024, we adjusted the BPA to alert for ICD codes for drug overdose (T50) in addition to those previously used. We then retrospectively analyzed unintentional opioid overdoses presenting to the same urban academic ED over an additional 3-month period and compared these to our previously published data. Patients who were incarcerated, expired, or required hospital observation or admission were excluded. Data are reported descriptively.
Results: In total, 192 patients were screened and 86 met inclusion criteria; median age 42 years (IQR 35-57.5), 66% male, 49% white, 84% non-Hispanic. Overall, 76.7%

of patients with unintentional opioid overdose were discharged from the ED with a naloxone kit in hand; a significant improvement from the previous BPA evaluation cycle (56.1%). Of the 20 patients that did not receive naloxone, 6 refused a kit and 14 had an unknown reason. There were only 3 patients (3.5%) that had a naloxone prescription written instead of the kit in hand order. This was improved from the initial BPA implementation where 22 patients (11.6%) had a prescription written and only 1 prescription was picked up; thereby showing the ED naloxone kit in hand program to be a superior process.

Conclusion: Broadening the BPA trigger to include ICD 10 codes for drug overdose in addition to opioid use disorder and overdose resulted in an improved rate of ED naloxone kit in hand distribution in the patient population at highest risk for opioid overdose.

083. Retention in Opioid Agonist Therapy Following Nonfatal Opioid Overdose

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Background: Emergency department (ED) discharge after opioid overdose is a high-risk period for repeat overdose and death, representing a critical opportunity to initiate opioid agonist therapy (OAT). Retention in OAT reduces mortality, yet retention following overdose in the era of synthetic opioids and non-opioid adulterants has not been well characterized.

Hypothesis or Research Question: Has retention in OAT initiated after ED discharge for opioid overdose declined in the era of synthetic opioids?

Methods: We conducted a population-based cohort study of patients initiated on OAT following an ED presentation for non-fatal opioid overdose between January 1, 2014, and June 30, 2024, in Ontario Canada. Patients admitted >24 hours were excluded. The primary outcome was retention in OAT (buprenorphine, including immediate and extended-release formulations) methadone, sustained-release oral morphine (SROM), or combination therapy (methadone and SROM)) initiated within 7 days of ED discharge. We assessed temporal trends in initiation and retention across the study period. Secondary outcomes included median OAT dose at treatment discontinuation for immediate-release buprenorphine, methadone and SROM.

Results: We identified 1,762 individuals initiated on OAT within one week of ED discharge following opioid overdose. The median age was 32 years (IQR 27-40) and 69.7% were male. Buprenorphine was the most frequently initiated OAT (n=836; 47.4%) followed by methadone (n= 754; 42.8%). Over the study period, the median duration of OAT retention was 17 days (IQR 4 to 86). Median retention was longest with SROM (29 days; IQR 7-154) followed by methadone (20 days; IQR 4 to 126), buprenorphine (13 days; IQR 3 to 70) and combination therapy (13 days; IQR 2 to 70). Although overall OAT initiation increased by 1,014% over the study period, median retention declined sharply for buprenorphine (-91%) and methadone (-70%), while SROM remained consistently low and combination therapy showed a modest upward trend. Median OAT dose at time of treatment discontinuation remained low throughout the study, with final-year median doses of buprenorphine 8 mg, methadone 40 mg and SROM 200 mg.

Conclusion: Among patients discharged from ED following opioid overdose, initiation of OAT increased over time but retention was extremely poor irrespective of OAT initiated and declined markedly over time. These findings highlight a major gap during a period of elevated overdose risk and underscore the need for strategies to improve OAT retention after overdose, including optimization of dosing in the context of increasingly potent synthetic opioids.

084. Geographic Variation in Pre-EMS Naloxone Administration in the United States

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Background: Naloxone administration given prior to EMS arrival (pre-EMS) has become increasingly common in the United States (US) since the FDA's approval of over-the-counter naloxone in 2023. Pre-EMS naloxone is typically administered by bystanders (family/friends/strangers) or non-medical first responders (fire/police personnel). Limited

research has investigated regional variation in patterns of naloxone administration prior to EMS arrival, which is critically needed to focus saturation efforts in the US.

Hypothesis or Research Question: Do patterns of pre-EMS naloxone administration for suspected opioid overdose differ across the US?

Methods: The RENDOR project is an ongoing, prospectively collected, observational study of patients who had EMS encounters between June 2024 and September 2025 and received pre-hospital naloxone for a suspected opioid overdose. RENDOR collects naloxone doses, routes of administration, administrators, and clinical responses from EMS personnel on patients who received naloxone prior to EMS arrival, which is not routinely or systematically collected in EMS's clinical documentation. Study sites include Pittsburgh, PA, Detroit, MI, Denver, CO, Portland, OR, and San Francisco, CA. Chi-square tests and Fisher's exact tests were used to determine statistically significant differences between five geographical sites. All analyses were conducted in R v.4.5.5.

Results: Among all RENDOR overdose cases with completed data (n = 2,986), 1,623 patients (54.4%) received pre-EMS naloxone. The proportion of cases receiving their first naloxone dose from pre-EMS responders ranged from 34.4% in Detroit to 66.0% in San Francisco ($p<0.001$). Overall, half of the patients who received pre-EMS naloxone received only one pre-EMS dose, ranging from 55.6% of cases in Pittsburgh to 27.7% in San Francisco ($p<0.05$). Of the patients who received one or more pre-EMS naloxone doses, naloxone was administered by bystanders in 26.3% of cases in Detroit, 44.4% in Pittsburgh, 53.1% in Denver, 55.9% in Portland, and 86.3% in San Francisco ($p<0.05$). Naloxone was administered by police in 13.5% of cases overall, ranging from 3.8% in San Francisco ($p<0.05$) to 21.4% in Denver.

Conclusion: Pre-EMS naloxone administration patterns and practices differed markedly across sites, reflecting local differences in community overdose response and first responder roles. These variations underscore the need for expanded training and bystander naloxone access to ensure equitable overdose responses nationwide.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

085. Medical Toxicology Service Increases Volume Through Addiction Medicine Consults

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Background: Medical toxicologists have expertise in treatment of illness from exposure to drugs, chemicals, and biological agents. Expertise in the treatment of addiction and withdrawal conditions are also well-documented.

Hypothesis or Research Question: Medical toxicologists expand inpatient volumes by incorporating addiction medicine services into their practice.

Methods: This is a retrospective review of patients treated by a single inpatient medical toxicology service that has incorporated addiction care. Face-to-face encounters at 5 hospitals within one healthcare system in a medium-sized metropolitan city were assessed. The service database was queried for addiction-specific conditions defined as diagnoses of "withdrawal", "buprenorphine induction", "methadone induction", or "methadone maintenance". Descriptive statistics were used to compare patients with the above assessments as compared to the entire medical toxicology inpatient cohort. Univariable linear regression was performed to measure how patient volume changed over time.

Results: Total unique face-to-face encounters performed in a fiscal year by the medical toxicology service were as follows: 2016 (n=1346); 2017 (n=1476); 2018 (n=1486); 2019 (n=1496); 2020 (n=1467); 2021 (n=1833); 2022 (n=1599); 2023 (n=1564); 2024 (n=1507); 2025 (n=1766). Number of unique patients with addiction-specific conditions were as follows: 2016 (n=178); 2017 (n=194); 2018 (n=275); 2019 (n=419); 2020 (n=522); 2021 (n=786); 2022 (n=684); 2023 (n=613); 2024 (n=674); 2025 (n=885). The percentage of addiction-specific conditions based on total unique face-to-face encounters were as follows: 2016 (13.2%); 2017 (13.1%); 2018 (18.5%); 2019 (28.0%); 2020 (35.6%); 2021 (42.9%); 2022 (42.8%); 2023 (39.2%); 2024 (44.7%); 2025 (50.1%). In regression analyses, we observed that each successive year was associated with a mean decrease of 45 non-addiction-related encounters (95% CI: 27-63, $p<0.001$) but also a mean increase of 76 addiction-related encounters (95% CI: 49-102, $p<0.001$), resulting in an overall increase of 31 total encounters per year (95% CI: 0-61, $p=0.048$).

Conclusion: The percentage of addiction-related conditions increased over time during the study period. The percentage more than tripled in fiscal year 2025 as compared to 2016. Many hospital services have witnessed decreases in patient volumes since the COVID-19 pandemic. Treatment of patients suffering from addiction can increase or maintain patient volume for medical toxicology services. Although much overlap exists between addiction diagnoses and toxicology diagnoses in this cohort, the percentage of patients with addiction-specific assessments clearly increased during the study period based on the search criteria.

086. The First 8 Years of the Minnesota Drug Overdose and Substance Use Surveillance Activity

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Background: Unintentional drug overdoses, driven by the opioid epidemic and clandestinely produced fentanyl as well as other drug classes, contribute substantially to morbidity and mortality frequently addressed in emergency departments (EDs). Few programs monitor the emergence of novel substances of abuse at a community level via the collaboration of public health and clinical entities.
Hypothesis or Research Question: We describe the community prevalence of drugs of abuse through a collaborative effort of a state department of public health and bedside emergency clinicians.
Methods: This is an ongoing public health surveillance project by the Minnesota Department of Health (DoH) and two large hospital systems in the state. Emergency medicine clinicians identified unintentional overdoses due to substance abuse and characterized them as "atypical" or "typical" presentations of the presumed substance. In early 2024, as previously "atypical" presentations became commonplace, the program expanded to include all unintentional overdoses due to substance abuse. De-identified clinical history was collected and any unused biological specimens were delivered to the state's public health laboratory for non-directed liquid chromatography/high resolution mass spectrometry against a library of >1,300 substances. Results were returned to hospitals for timely situational awareness of emerging/novel substances within the catchment area, while enhancing interagency collaboration to inform public health alerts and responses and national biosurveillance initiatives (CDC-OD2A).
Results: Over 8 years of surveillance, 5519 subjects were identified (1542 at site 1 [27.9%]; 3977 at site 2 [72.1%]), 1316 (23.8%) with discarded biological samples available for analysis (616 at site 1 [46.8%]; 700 at site 2 [53.2%]). Detected compounds were grouped into drug classes. Amphetamines metabolites (810, 62%), opioids and metabolites (805, 61%), cannabinoids and metabolites (556, 42%), benzodiazepines and metabolites (510, 39%), and cocaine and metabolites (354, 27%) were the most frequently identified categories. Notable emerging substances included xylazine (110, 10% of 2020–2025 cases), medetomidine (21, 8% of 2024–2025 cases), mitragynine (26, 2%), carfentanil (9, <1%), and nitazenes (7, <1%).
Conclusion: Population-based definitive testing for drugs of abuse is rare, particularly in non-fatal overdoses. Through DoH and bedside clinician collaboration, extensive data characterizing the community-level presence of substances of abuse is now available in near-real time to clinicians, public health entities, and others to augment bedside management and epidemiologic surveillance. This insight into the evolving landscape of drugs of abuse and emerging threats extends beyond available hospital discharge data, ICD-10 codes, and other similar efforts, and provides a framework for other regions to do the same.

087. How Well Do Online Triage Systems Agree With a Poison Control Call Center? an Assessment of Low-Risk Exposures

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Background: Two-thirds of exposures can be managed on-site ("home"). If online systems, like PoisonHelp and webPOISONCONTROL, triaged these low-risk exposures, they could free resources for more complex cases. Both systems consider xenobiotic, amount, and time of exposure. PoisonHelp considers symptom presence and refers to a Poison Control Call Center (PCC) or home; webPOISONCONTROL, specific symptoms and to PCC, the Emergency Department (ED), or home.
Hypothesis or Research Question: Do online triage systems agree with PCC recommendations for low-risk exposures?
Methods: This is a retrospective study comparing recommendations from webPOISONCONTROL and PoisonHelp with those from one PCC. We included public calls of single-substance ingestions from the 15th day of each month from September 2023 to August 2024. We excluded information requests, exposures involving multiple substances, pregnant individuals, self-harm, pets, age <6 months or >79 years, or self-triage to a healthcare facility (HCF). Two abstractors, blinded to PCC recommendations, re-entered cases into each application. Our primary outcome was inter-rater reliability, quantified with Cohen's κ (0=chance agreement, 1=perfect agreement), between PCC and each application in triaging to home versus further evaluation ("call PCC" for PoisonHelp; "call PCC" or "go to ED" for webPOISONCONTROL). We computed sensitivity and specificity to PCC recommendations. True positives were cases that PCC and an application triaged to home.

Results: Of 1,274 cases screened, 800 met inclusion criteria, of which 351 (44%) involved children <6 years. Nonpharmaceutical exposures were more common than pharmaceutical ones, 433 (54%) vs 367 (46%). PCC recommended home care in 740 cases (93%), webPOISONCONTROL, 499 (62%), and PoisonHelp, 138 (17%). webPOISONCONTROL triaged to home 496 (67%) of the 740 cases that PCC did; PoisonHelp 137 (19%). Agreement with PCC was fair for webPOISONCONTROL (κ=0.218 [95% CI: 0.166–0.270]) and at chance level for PoisonHelp (κ=0.03 [0.019–0.041]). Agreement between webPOISONCONTROL and PoisonHelp was minimal (κ=0.137 [0.098–0.177]).Sensitivity and specificity were 67% [64%–70%] and 95% [86%–99%] for webPOISONCONTROL, and 19% [16%–22%] and 98% [91%–100%] for PoisonHelp. Of the 60 cases PCC triaged to HCF, webPOISONCONTROL triaged 52 (87%) to PCC, five (8%) to the ED, and three (5%) to home- two were PCC guideline deviations and one required social support. PoisonHelp referred 59 (98%) to PCC and one (2%) to home, which PCC referred to the ED for social support.
Conclusion: webPOISONCONTROL's recommendations align significantly more with PCC than do PoisonHelp's. Neither matched PCC performance. Data from 12 days at one PCC do not capture the full spectrum of exposures.

DAY 2: POSTERS, ABSTRACTS 088-152

088. Socio-Demographic, Clinical and Seasonal Profiles of Mushroom Poisoning Events Caused by Exsudoporus Ruber Cf. and Boletus Species in the Himalayan Region of Nepal

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Background: Wild mushroom poisoning events are common in Himalayan region of Nepal and studies on mushroom poisoning are very scarce. It is not known what factors are associated with poisoning events. We conducted a retrospective descriptive analysis to find out the socio- demographic, clinical and seasonal profile of mushroom poisoning events in Manang, Nepal from 2022 to 2024.
Hypothesis or Research Question: What are the socio-demographic, clinical and seasonal profiles of wild mushroom poisoning events in Himalayan region of western Nepal?
Methods: This is a retrospective descriptive analysis to investigate events in the Himalayan district of Manang from August 2022 to August 2024. Medical records of patients visiting Manang Hospital, Nepal in this time frame after consuming wild mushrooms were evaluated. Suspected Cases were defined as people who had history of vomiting, diarrhea, or abdominal pain and who consumed mushrooms in last 6 hours. Suspected cases with supporting clinical and laboratory evidence were classified as confirmed cases. District Forestry officials identified the wild mushrooms with expert help. Data were analyzed for descriptive characteristics including age, gender, incubation period, seasonality, clinical features, and outcomes.
Results: A total of 25 migrant workers ate wild mountainous mushrooms. Attack rate was 100%. Outbreaks mostly occurred from May to August. Mean age was 32.5 years (Range: 14-58 years). 80% of cases were males. Median incubation period was 51.6 minutes (range: 45 minutes- 2.5 hours). Common manifestations involved gastrointestinal (96%), nervous (64%), musculoskeletal (60%), cardiovascular (40%), and visual (20%) symptoms. Mean systolic blood pressure was 104.4 ± 17.57 mmHg and mean diastolic blood pressure was 68.8 ± 13.32 mmHg. Renal Function Tests showed mean urea concentrations of 35.22 ±14.61 mg/dl and mean creatinine concentration of 0.98 ± 0.23 mg/dl. Mean pulse rate was 97.52 ± 22.17 bpm. All patients were hospitalized for 16 to 48 hours. The mushroom was identified as *Exsudoporus ruber cf*. in 18 cases and Boletus species in rest cases. There was no associated mortality. The mushroom was unknowingly picked by the patients in all cases.
Conclusion: The poisoning event was due to consumption of wild mushrooms of genus *Exsudoporus* and Boletus. Migrant workers of male gender are found to be predisposed to mushroom poisoning events, particularly in monsoon season. Our study provides valuable insight into the impact of poisoning events and highlights the need for further studies on classifying poisonous wild mushrooms to mitigate such incidents in the future.

089. Cyclones and Carbon Monoxide Poisoning Among Children in Pennsylvania and Delaware: A Time-Series Analysis

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Background: Carbon monoxide poisoning is a preventable cause of morbidity and mortality among children, who are uniquely vulnerable to its effects. Identifying behavior-modifying environmental exposures that influence the incidence of carbon monoxide poisoning may guide preventative

counseling and aid quick diagnosis. Though cyclones have been strongly associated with power outages, a risk factor for carbon monoxide poisoning, their impact on pediatric carbon monoxide poisoning in the mid-Atlantic region of the United States is not well characterized.

Hypothesis or Research Question: We hypothesized cyclone events would increase the incidence rate ratio of National Poison Data System (NPDS) cases for carbon monoxide poisoning.

Methods: To assess the relationship between storm events and carbon monoxide poisoning, we used a county-level time-series study design, applying generalized additive mixed-effects models with a negative binomial distribution. We included NPDS cases from a blinded dataset with a substance coded as "carbon monoxide" (generic code: 106000) from January 2020 to December 2024 for individuals under the age of 20 in Delaware and Pennsylvania with geographic data available. Residing in county within 100 km of a cyclone track between the day of the case to three days prior constituted exposure.

Results: In total, 755 cases met inclusion criteria, and cyclone exposure was associated with a significantly increased incidence rate ratio (IRR) for carbon monoxide poisoning (IRR: 4.43, 95% CI: 1.70-11.52). However, co-occurrence of cases with cyclones was uncommon (N=16, 2%). Days by county (county-days) with cyclones constitute a rare exposure, occurring in 0.59% of total county-days (N=750). Most cases (N=505, 64%) occurred in the heating season (October-March), which primarily falls outside of the cyclone season in the North Atlantic (June-November).

Conclusion: Cyclones constitute a potent, albeit rare, risk factor for carbon monoxide poisoning among children in the mid-Atlantic. While the point estimate should be interpreted with caution given wide confidence intervals and infrequent co-occurrence of cyclones and carbon monoxide cases, cyclones likely represent a moment for intervention and education for carbon monoxide poisoning prevention. The results also suggest winter-related events affect population-level carbon monoxide rates more than cyclones in the region given low co-occurrence events and seasonality of cases, warranting further attention and research.

090. Hypersensitivity to Both Fab and F(ab')2 Rattlesnake Antivenoms

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Background: Acute hypersensitivity reactions (AHRs) to rattlesnake antivenom are uncommon and can be secondary to foreign proteins and possibly alpha-galactose carbohydrate moieties. Typically, patients with reaction to one of the available antivenoms tolerate the other. From 2018-2023, 1051 antivenom administrations were recorded in the ToxIC NASBR registry, with 50 AHRs to individual antivenoms but only one to both.

Hypothesis or Research Question: Acute hypersensitivity reactions to both Fab and Fab_2 antivenoms can occur in a single individual.

Methods: This is a case report of a 3-year-old, 18.9 kg boy with no history of atopy or asthma who was envenomated by a rattlesnake in Arizona.

Results: Initial exam showed significant swelling to the hand. He did not demonstrate systemic toxicity, hemotoxicity, or allergic reaction to the envenomation. Six vials of Fab antivenom were administered 105 minutes after the envenomation with an initial rate of 1.6 vials/hr. Upon rate increase to 8 vials/hr, generalized urticarial rash with hives developed. Antivenom was paused, and IV methylprednisolone and IV diphenhydramine were administered. AHR resolved and Fab was resumed at 2 vials/hr. It was tolerated over 3 hours without reaction and initial control was achieved. The patient was transferred to a toxicology referring center 18 hours post-envenomation and progressive edema to the upper extremity was noted. 4 vials of F(ab')2 were administered at 0.8 vials/hr after pretreatment with diphenhydramine. Upon rate increase to 2 vials/hr the patient developed hives, flushing, facial edema, and vomiting. He was treated with epinephrine infusion, dexamethasone, additional diphenhydramine, and antivenom was stopped. 24 hours post-bite, F(ab')2 at 0.4 vials/hr, was given for progressive edema. Upon increase to 0.8 vials/hr 1.5 hours later, hives recurred and antivenom was stopped. No additional antivenom was given as swelling had not progressed. He remained in the hospital for one week due to complexity of care and difficulty of follow up. He was discharged home with mild swelling and ecchymosis on exam, no evidence of hemotoxicity, and without medications.

Conclusion: This case report describes recurrent rate-related acute hypersensitivity reactions after administration of both Fab and Fab_2 antivenom in a child not known to be sensitized to antivenom components or atopy.

091. A Case Series of Serum Sickness Mimicking Cellulitis Following Treatment of Rattlesnake Envenomation With F(ab')2 Antivenom

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Background: Incidence of serum sickness (SS), a type III hypersensitivity reaction caused by immune complex formation after exposure to nonhuman serum proteins, is significantly lower with fragment antivenoms than with whole IgG products. Nonetheless, SS remains a clinically relevant sequela of antivenom treatment.
Hypothesis or Research Question: Serum sickness after antivenom for rattlesnake envenomation can mimic cellulitis.
Methods: Case series describing the development of SS misdiagnosed as cellulitis in two patients after receiving F(ab')2 antivenom for rattlesnake envenomation.
Results: Case 1: A 51-year-old previously healthy woman received 20 vials of F(ab')2 antivenom for progressive swelling after a rattlesnake bite to the foot. At clinic follow-up (day 6 post-envenomation/post-antivenom) she reported return to work with mild increase in pain and swelling but denied rash, fever, or arthralgias. She re-presented for worsening pain and swelling in her foot with subjective fever the next day (day 7 post-antivenom dose). Exam was notable for a confluent erythematous, warm rash to the envenomated foot with extension proximally into scattered wheals. Labs were unremarkable for leukocytosis, eosinophilia, or AKI. She was admitted to the hospital and antibiotics were initiated for suspected cellulitis. On hospital day 2 (day 8 post-antivenom) symptoms persisted and pruritus developed. Steroids were administered due to concern for SS with significant improvement within hours. She was discharged with a prednisone taper. Case 2: A 60-year-old man with history of HTN, HLD, DM2 received 24 vials of F(ab')2 for swelling and hemotoxicity after a rattlesnake bite to his finger. He was recovering well following discharge until day 6 post-envenomation (day 5 post-final antivenom dose) when he re-presented for subjective fever and new redness with swelling to the hand and forearm. Extremity exam was remarkable for erythema with edema and warmth from his hand to the mid forearm. There was no joint pain or tenderness. Labs were unremarkable for leukocytosis, eosinophilia, or AKI. He was admitted and IV antibiotics were initiated for cellulitis. On hospital day 2 (day 8 post-antivenom) symptoms persisted and full body exam revealed a diffuse maculopapular rash to the back, flanks, and other extremities. Prednisone and diphenhydramine were administered for presumed SS. There was significant improvement in rash within 3 hours and he was discharged with a prednisone taper.
Conclusion: Serum sickness can be misdiagnosed as cellulitis after antivenom administration. Despite lower incidence with fragment antivenoms, serum sickness should be considered in patients presenting with rash and localized edema after antivenom therapy.

092. Fatality Following Intramuscular Injection of Diquat Herbicide

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Background: Diquat is a highly toxic bipyridyl herbicide, and toxicity is most reported following ingestion. Intramuscular (IM) injection is extremely rare, with only 2 prior case reports identified, one of which was fatal. We describe the clinical effects of a rare case of fatality from IM injection of diquat.
Hypothesis or Research Question: Intramuscular injection of diquat bypasses the dermal and gastrointestinal barrier and will result in significant acute toxicity.
Methods: This is a single patient chart review. A 44-year-old male with history of chronic kidney disease, hypertension, prior deep vein thrombosis, and ulcerative colitis injected 30 mL of 37.3% Diquat dibromide herbicide (approximately 11.2 grams, 116 mg/kg) in a suicide attempt, ultimately developing multiorgan failure, refractory shock, and death at 30 hours post injection.
Results: 44-year-old male presented to the Emergency Department within 1 hour of IM injection of 30 mL of 37.3% Diquat into the right thigh. His initial vital signs: T 98.4ºF, HR 82 bpm, BP 169/112, RR 27/min, Oxygen saturation 97% on 6L NC. He was alert and noted to be flushed. Pertinent initial diagnostics: APAP negative, ASA negative, ETOH over 300 mg/dL, CO_2 25 mEq/L, BUN 12 mg/dL, Cr 0.9 mg/dL, AST 111 U/L, ALT 58 U/L, PT 10.3 sec, INR 0.96, CPK 968 U/L, Chest X-ray mild to moderate interstitial coarse markings. Over the next several hours, patient became agitated and progressively more tachycardic and hypotensive, requiring vasopressors at 10 hours post injection (PIn). Due to oliguric renal failure, renal replacement therapy was initiated for a short period of time. Creatinine peaked at 5.17 mg/dL at 24 hours PIn. Lactate peaked at 6.8 mMol/L at 13 hours PIn. AST peaked at >1500 units/L and ALT 661 units/L. Creatine kinase peaked at 22000 units/L. Patient was intubated for respiratory failure at 24 hours PIn, at which time he was on maximal doses of norepinephrine, vasopressin, and phenylephrine. Repeat Chest X-ray demonstrated worsening diffuse patchy consolidative changes. Patient's code status was changed, and he suffered bradycardia and ultimately asystole at 30 hours post injection.
Conclusion: In an adult male, IM injection of reported 30 mL of 37.3% Diquat led to multiple organ system injury including refractory shock, renal failure, respiratory failure, and eventual death 30 hours post injection. To our knowledge, this is only the second case report of a fatality by diquat injection.

093. The Bite Beneath the Surface: Abdominal Wall Abscess Mimic From Loxosceles Reclusa Envenomation

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Background: Loxosceles reclusa envenomation can present with variable dermatologic and soft tissue findings occasionally demonstrating deeper tissue involvement resulting in additional imaging. In children, these findings may raise other clinical considerations, and there are no published studies describing CT imaging characteristics of Loxosceles reclusa envenomation in the pediatric population.
Hypothesis or Research Question: In a child, Loxosceles reclusa venom-induced dermatonecrosis can extend into deeper dermal layers, producing a sterile fluid collection that can mimic soft tissue abscess on CT imaging.
Methods: This is a case report. An unimmunized, 11-year-old female presented with two days of fever, left lower quadrant abdominal pain, and rash. The patient's rash was characteristic of acute generalized exanthematous pustulosis (AGEP), a finding associated with Loxosceles reclusa envenomation and systemic loxocelism. She had ecchymosis without erythema along the left lower quadrant with extension to the flank and groin. Lab work and CT of the abdomen and pelvis were obtained for concerns of an intraabdominal infection.
Results: Initial lab work demonstrated thrombocytopenia with platelets of 116 K/cumm (150.0 – 400.0 K/cumm) and anemia with a hemoglobin of 12.0 g/dL (11.5 - 15.5 g/dL) without leukocytosis. CT imaging showed a left lower quadrant fluid collection. General surgery and interventional radiology (IR) were consulted due to concerns of an abdominal wall abscess requiring drainage. The consulting teams recommended initiation of antibiotics therefore patient was started on ceftriaxone and metronidazole. Repeat lab work showed pancytopenia with white blood cell count of 3.6 (4.5-13.5 K/cumm), hemoglobin of 9.9 g/dL (11.5 - 15.5 g/dL), and platelets of 80 K/cumm (150.0 – 400.0 K/cumm). On additional discussion with the patients' father, he noted multiple instances where he discovered brown recluse spiders crawling near the patient. With this new information, the general surgery and IR consulting teams decided against abscess drainage, and antibiotics were discontinued. Her blood work normalized within a few days of admission and did not require blood product transfusion.
Conclusion: Loxosceles reclusa envenomation can cause deeper soft tissue damage which can be mistaken for other conditions. This case report hypothesizes that a Loxosceles reclusa envenomation can create a sterile fluid collection deep in the skin that mimics an abscess on CT imaging.

094. A Light Roasting: How the r/Coffee Online Forum Challenged Misinformation Around Mycotoxins and America's Favorite Beverage

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Background: Misinformation about contaminants in food is widespread online, yet little is known about how communities respond to such claims. Wellness influencers and companies who market "mold-free" coffee products claim that "regular" coffee contains enough mycotoxins to cause diverse gastrointestinal and neuropsychiatric symptoms. Scientific analyses have found that coffee generally contains mycotoxin levels below recommended safety limits.
Hypothesis or Research Question: How do online communities discuss and respond to claims about harmful amounts of mycotoxins in coffee?
Methods: This is a retrospective analysis of all posts to the r/Coffee Reddit forum. r/Coffee began in March 2008 and has 1.9 million members. We included posts from March 2008 to July 2025 that contained the word "mycotoxin" or a spelling variant. Both authors independently reviewed every comment and resolved disagreements by consensus. We coded comments as (1) refuting, (2) questioning, or (3) agreeing with misinformation. For each comment we recorded upvotes, a measure of community endorsement of the comment's content, and whether comments provided explanations or cited sources.
Results: Of 5,591 total posts, we included 35, yielding 865 unique comments, a median of 16 comments per post (IQR 10-20). Of 130 comments that directly addressed the claim, 99 (76%) refuted it, 8 (6%) questioned it, and 23 (18%) concurred. Among the 99 refuting comments, 61 (62%) provided explanations referencing coffee production, roasting, or the origin of the misinformation, and 19 (19%) cited outside sources. Among the accepting comments, 9 (39%) provided explanations referencing coffee production, roasting, or personal experience, and 2 (9%) cited outside sources- one being a blog and one being an FDA webpage. Twenty-five of the 35 (71%) total posts contained at least one refuting comment. Refuting or questioning comments received more upvotes (median 4, IQR 2–9) than comments supporting the misinformation (median 2, IQR 1–2).
Conclusion: In one subreddit, misinformation about mycotoxins was challenged through domain-specific explanations and the citing of academic and governmental sources. The community engaged more with critical comments than non-critical ones. Limitations include examining one community, one social media platform, reliance on manual review, and inability to access deleted posts. Our findings suggest

that some online communities may effectively self-correct misinformation, highlighting opportunities for collaboration between domain experts and engaged lay communities.

095. Severe Coagulopathy From Dermal Superwarfarin Exposure in a Pest Control Worker

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Background: Superwarfarins are highly-potent vitamin K antagonists that cause prolonged coagulopathy often requiring extended, high-dose vitamin K_1 therapy. Most human poisonings result from ingestion; transdermal exposure is exceedingly rare, with only a few cases reported in the literature.
Hypothesis or Research Question: Occupational skin contact with superwarfarins can produce clinically significant anticoagulant toxicity in the absence of ingestion.
Methods: A 61-year-old pest control worker presented with three days of progressive dysphonia, dyspnea, and oral bleeding. Further enquiry revealed several months of easy bruising. He had been handling rodenticides without personal protective equipment. Examination revealed diffuse hematomas and supraglottic edema. Computerized tomography of the neck revealed bilateral supraglottic soft tissue swelling with moderate to severe airway narrowing. Flexible nasopharyngoscopy demonstrated extensive edema and hematoma. Coagulation testing showed an INR > 10 and aPTT > 150 seconds, with normal fibrinogen and liver function. Factor activity assays confirmed severely reduced activity of vitamin K-dependent clotting factors (II, VII, IX, X), consistent with vitamin K antagonist toxicity.
Results: There was no report of deliberate ingestion. His occupational history implicated dermal absorption as the likely route of exposure. He was intubated urgently for airway protection and given 3,000 units of four-factor prothrombin complex concentrate (PCC) and 10 mg intravenous vitamin K_1, normalizing coagulation parameters within hours. Oral vitamin K_1 was then initiated at 20 mg four times daily. Within 36 hours, the INR rose to 2.31 and aPTT to 40 seconds, prompting an increase in the vitamin K_1 dose to 40 mg four times daily. He recovered fully and was discharged after nine days, with oral vitamin K_1 gradually tapered over three weeks and INR remaining stable. The relatively short treatment course, compared with typical durations exceeding 100 days, likely reflects a lower body burden of superwarfarin owing to unintentional exposure.
Conclusion: This case represents a rare presentation of presumed transdermal superwarfarin toxicity resulting in life-threatening coagulopathy. Diagnosis rested on the combination of an isolated vitamin K-dependent factor deficiency and a clear occupational exposure history. Management required airway protection and PCC followed by prolonged high-dose vitamin K_1 and close INR monitoring to prevent rebound coagulopathy. Clinicians should consider atypical exposure routes in unexplained coagulopathy and reinforce proper use of protective equipment among individuals handling rodenticides.

096. Disruption of Barriers: Evaluating the Effects of Di(2-Ethylhexyl) Phthalate on Trophoblast Function in the Blood-Placenta Barrier and Its Implications for Obstetrical Pathologies

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Background: The blood-placenta barrier (BPB) is a dynamic and highly specialized structure that safeguards fetal development by regulating the exchange of nutrients, gases, and waste while protecting against harmful agents. At the forefront of this barrier are trophoblast cells, which orchestrate the invasion of maternal tissues, spiral artery remodeling, and immune regulation critical to pregnancy, obstetrical morbidities and outcomes. Emerging evidence suggests that exposure to environmental toxicants such as di(2-ethylhexyl) phthalate (DEHP)—a widely used plasticizer—poses a significant risk to trophoblast function and BPB integrity. DEHP, a known endocrine-disrupting chemical (EDC), has been implicated in adverse reproductive outcomes, including preeclampsia, intrauterine growth restriction, and preterm birth. Moreover, DEHP have been found in the waters of Biscayne Bay and South Florida's drinking water.
Hypothesis or Research Question: This study investigates the molecular and cellular mechanisms by which DEHP disrupts trophoblast-mediated BPB function, focusing on key proteins such as Synctin-1, Placenta Growth Factor, Vascular Endothelial Growth Factor, YAP 1, JAM1, MMP2, Connexin-43, and IL10.
Methods: The research employs in vitro trophoblast and placenta endothelial culture models. Analysis include QPCR's, western blots, transwell assays with fluorescent tracers such as FITC-dextran, and immunostaining to test the BPB permeability and gene expression of key proteins that modulate pregnancy and birth outcomes.
Results: The outcomes of this research aim to elucidate the pathophysiological links between DEHP and viral exposure to trophoblast dysfunction, BPB integrity, and obstetrical morbidities. The findings have implications for environmental health policy, prenatal care strategies, and

the development of therapeutic interventions targeting BPB integrity.
Conclusion: This study seeks to offer practical and real time response measures to environmental exposures that pose risk to maternal-fetal health in South Florida.

097. Not Feeling Very Gourd: A Case Report of 'Toxic Squash Syndrome'

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Background: The *Cucurbitaceae* plant family, composed of edible squash, melons, and gourds, has been used outside of Western medicine to treat a variety of diseases. Cucurbitacin's bitter-tasting and cytotoxic compounds are thought to be harmful when ingested in high concentrations, though few cases have been reported.
Hypothesis or Research Question: Ingestion of calabash (*Lagenaria siceraria*), a gourd in the Cucurbitaceae plant family, can expose patients to toxic levels of cucurbitacin leading to distributive shock.
Methods: This is a case report of a 64-year-old male with a past medical history of hypertension, type 2 diabetes mellitus, hyperlipidemia, and gout, who presented with abdominal pain, emesis, and generalized weakness after ingesting juiced calabash for the first time one hour prior to arrival to the emergency department (ED). Emergency Medical Services (EMS) found the patient to be hypotensive with dyspnea and hives. Upon presentation to the ED, he was in acute distress with ongoing emesis. Blood pressure (BP) was 78/53 mmHg [mean arterial pressure (MAP) of 61], heart rate 70 beats per minute, respiratory rate 18 breaths per minute, oxygen saturation 95% on room air, and afebrile. Medical toxicology was consulted for management guidance.
Results: One liter of lactated Ringer fluid was administered, and the patient was started on a titratable norepinephrine infusion in addition to 4 mg of intravenous (IV) ondansetron, 40 mg IV pantoprazole, and 100 mcg IV fentanyl. Vasopressors were titrated following completion of the fluid bolus (BP 112/56 mmHg, MAP of 75). Labs showed leukocytosis (17.8 thou/uL) and elevated lactate (3.2 mmol/L). Computed tomography scan of the abdomen and pelvis was suggestive of enterocolitis. He was admitted to the intensive care unit for continued vasopressor support. The following day he stabilized and was transferred to the floor for further management. Labs after admission showed a downward trend in lactate and white blood cells, but a rise in liver enzymes (peak alanine aminotransferase 364 U/L, aspartate aminotransferase 192 U/L). Gastroenterology work up, including a duplex ultrasound and coagulation, ferritin, and hepatitis serologies, were all normal. He was discharged after one week and was followed outpatient to trend his liver enzymes which normalized one month after initial presentation.
Conclusion: Though an unusual culprit, calabash and other cucurbitacin-containing produce should be considered in cases of distributive shock and managed aggressively.

098. A Case of Symptomatic Drug-Induced Liver Injury Associated With Bupropion in a Young Adult

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Background: Bupropion is an aminoketone antidepressant that is widely prescribed to treat depression and smoking cessation. It is generally deemed safe with infrequent mild elevations of liver enzymes. However, rare cases of clinically significant acute liver injury have been reported in otherwise healthy individuals. The mechanism is incompletely understood, but oxidative stress and mitochondrial dysfunction have been implicated in pre-clinical models.
Hypothesis or Research Question: Bupropion can induce symptomatic acute liver injury in healthy individuals, manifesting as hepatitis with marked transaminase elevation and clinical symptoms, which resolve upon discontinuation of the drug.
Methods: This is a single case of a 28-year-old healthy adult female who developed symptomatic acute liver injury following bupropion initiation. Clinical presentation, laboratory findings, exclusion of other etiologies, and temporal association with bupropion exposure are detailed. Liver function tests and imaging were performed. Drug withdrawal and follow-up were documented.
Results: The patient was started on bupropion 150 mg daily, and 28 days later, she began to develop itching on her hands and feet, without a rash, despite topical hydrocortisone use. On day 37 after initiation of bupropion, the patient continued to have itching and started to notice darker urine, lighter stools, and yellowing of her eyes so she self-discontinued the bupropion. Outpatient labs were collected on day 39, showing a marked increase in her AST (416 U/L), ALT (575 U/L), alkaline phosphatase (780 U/L), and total bilirubin (4.3 mg/dL), with prior normal liver function tests. She went to the ED the following day, where her initial presentation was

significant for scleral icterus and right upper quadrant tenderness. Infectious hepatitis serologies were negative, the acetaminophen level was undetectable, and her right upper quadrant ultrasound was normal. Discontinuation of her bupropion led to rapid clinical and biochemical improvement, with labs at 24 days after discontinuation showing a decrease in AST (94 U/L), ALT (14 U/L), alkaline phosphatase (447 U/L) and total bilirubin (0.8 mg/dL). A Roussel Uclaf Causality Assessment Method (RUCAM) score of 6 suggests a "probable" association between bupropion and the observed clinical picture of drug-induced liver injury.
Conclusion: Bupropion can rarely cause symptomatic acute liver injury in healthy individuals, with resolution following drug withdrawal. Clinicians should be aware of this potential adverse effect, especially as bupropion use expands. Early recognition and discontinuation are critical for recovery. Further research is needed to clarify the incidence and mechanisms of drug-induced liver injury from bupropion.

099. Look What the Cat Dragged In: A Case of Critically-Ill Coral Snake Envenomation

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Background: Eastern coral snake (*Micrurus fulvius*) envenomation is rare but potentially life-threatening due to the delayed onset of neurotoxicity. Unlike pit viper bites, local tissue injury is minimal, which may obscure severity. The rarity of cases, limited antivenom supply, and potential for respiratory failure highlight the need for early recognition and appropriate management.
Methods: A 40-year-old woman presented 90 minutes after a confirmed coral snake bite to the wrist. Initial symptoms included nausea and paresthesias, with reassuring vitals and normal laboratory evaluation. She was transferred to a tertiary center, where she received 5 vials of North American Coral Snake Antivenin (NACSA). Despite initial stability, she developed bilateral ptosis, dysarthria, and dysphagia at 11 hours post-bite. The consulting clinical toxicologist emphasized that redosing antivenom is uncommon, but a second 5-vial course was administered due to progression. At 15 hours, she developed copious secretions and was intubated for airway protection. She was extubated within 24 hours and discharged on hospital day 3 with mild residual ptosis and dysarthria.
Results: This case illustrates the delayed and progressive neurotoxicity of coral snake envenomation and the diagnostic uncertainty surrounding optimal antivenom dosing. Evidence guiding redosing strategies remains sparse, and this case underscores the rarity of requiring multiple doses. Additionally, the patient's airway course raises questions about altered pharmacodynamics of neuromuscular blockers during rapid sequence intubation in the setting of elapid neurotoxins.
Conclusion: Coral snake envenomation requires early transfer to facilities with antivenom, close neurologic monitoring, and readiness for airway intervention. Further research is needed to clarify antivenom dosing protocols and intubation pharmacology in this unique patient population.

100. Fatal Methemoglobinemia and Hemolysis: Hereditary Alkaptonuria Decompensation Presenting Without Xenobiotic Trigger

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Background: Toxicologists are recognized authorities in managing methemoglobinemia and are frequently called upon for guidance in these cases However, not all cases arise from toxicologic causes. In these situations, conventional therapies are often ineffective. Recognition of non-toxicologic etiologies enhances the toxicologist's ability to better inform patient care.
Methods: We report a single case from an urban, tertiary care hospital.
Results: Toxicologists are experienced in diagnosing and treating methemoglobinemia, yet not all clinically significant cases result from exogenous oxidant exposure. Hereditary alkaptonuria (AKU) is an autosomal recessive disorder caused by deficiency of homogentisate-1,2-dioxygenase, leading to accumulation of homogentisic acid (HGA). Oxidation of HGA produces benzoquinones, which can polymerize into melanin-like deposits in connective tissues, creating a pro-oxidant environment. This process generates reactive oxygen species, disrupts red blood cell membrane integrity, and promotes tissue damage. A rare syndrome of methemoglobinemia with rapidly progressive hemolytic anemia has been reported in AKU patients under metabolic stress. Literature review identified 11 documented cases of methemoglobinemia and/or hemolysis in AKU, with only five demonstrating both features. All reported cases were fatal. We describe a 72-year-old woman with AKU who presented to the emergency department with respiratory distress after several days of gastrointestinal symptoms. Oxygen saturation by pulse oximetry was 74% despite high-flow oxygen, and she exhibited central cyanosis and pallor. Laboratory evaluation revealed normocytic anemia with hemoglobin of 9.3 g/dL (5.4 g/dL drop from two months

prior), acute renal failure, metabolic acidosis, and methemoglobinemia >30% (Figure 1 & 2). Persistent hemolysis precluded accurate measurement of potassium, AST, and ALT. CT angiography was unremarkable. Despite aggressive supportive therapy - including methylene blue (6 mg/kg over 15 hours), enteral ascorbic acid, renal replacement therapy, and three units of packed red blood cells - the patient's hemolysis progressed. Hemoglobin fell to 5.9 g/dL, and she developed worsening hyperlactatemia with persistent acidosis. She expired approximately 18 hours into hospital course. Limitations include the absence of Coombs testing, LDH, and haptoglobin. No home medications were deemed likely contributors, though patient did have access to topical lidocaine (Figure 3).

Conclusion: Toxicologists play a central role in managing methemoglobinemia. Awareness of non-toxicologic causes of oxidative stress is critical. This case illustrates that severe methemoglobinemia and hemolysis can occur in the absence of xenobiotic exposure and highlights the limitations of conventional therapies. Multidisciplinary collaboration and consideration of novel interventions, such as nitisinone or exchange transfusion, may improve outcomes in this otherwise universally fatal scenario.

101. Persistent Hypoglycemia Following Overdose of Semaglutide

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Background: Glucagon-like peptide-1 (GLP-1) agonists are a class of medication originally developed to treat Type II Diabetes Mellitus. Since being approved for use in treatment of obesity the rate of prescription increased dramatically. Unlike some other diabetic medications like sulfonylureas, they are rarely associated with hypoglycemia. Here we present a case report of a patient who overdosed on semaglutide and developed multiple episodes of hypoglycemia.

Methods: This is a case report of a single patient treated at our institution. The patient is a 45-year-old female with a history of prediabetes and bipolar disorder, and is on semaglutide for weight loss. She takes no other antidiabetic medications. She was recently switched from another GLP-1 agonist to semaglutide. About 16 hours prior to presentation, she mistakenly injected 8mg of semaglutide instead of the prescribed 2mg due to unfamiliarity with the injector. She then developed nausea, vomiting, headache, dizziness and tremors and inability to tolerate oral intake, and then presented to the ED. The patient's vitals and exam were normal. Her initial blood glucose was 67mg/dL, and the remainder of her labs were normal. She was given antiemetics and oral glucose which she was unable to tolerate. She was then treated with a 25g of intravenous dextrose. Her glucose improved to 85mg/dL but then again dropped to 68mg/dL. She was started on an infusion of 10% dextrose in normal saline and admitted to the hospital for glucose monitoring. The patient had 4 further episodes of hypoglycemia overnight with a nadir of 54 for which she received 7 additional doses of 25g intravenous dextrose. By the following morning, 36 hours after overdose, she was able to tolerate oral intake and had no further episodes of hypoglycemia. She was removed from the dextrose drip on day 2 of hospitalization and discharged on day 3.

Results: Due to their mechanism of action of increasing glucose induced insulin release, GLP-1 agonists are not expected to commonly cause hypoglycemia. Studies of GLP-1 agonist exposures reported to poison centers have shown rates of hypoglycemia ranging from 2.5-3.4%. One study which evaluated patients who presented to the ED found a higher rate of 10%.

Conclusion: Although rare, overdoses of GLP-1 agonists have the potential to lead to persistent hypoglycemia. Clinicians should be aware of and vigilant for potential toxicity of this class of medications especially in overdose.

102. Possible Attenuating Effect of Ethanol in Zinc Phosphide Poisoning

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Background: Zinc phosphide (Zn_3P_2) is an inorganic phosphorus salt commonly used as a rodenticide, which, upon contact with water or an acidic environment, produces phosphine, a gas that inhibits the mitochondrial respiratory chain

Hypothesis or Research Question: The alcoholic concentration could have influenced the rate of phosphine release and clinical evolution

Methods: We describe two cases of potentially lethal zinc phosphide ingestion associated with co-ingestion of ethanol. Case 1: A 42-year-old male presented two hours after ingesting 100 g of Zn_3P_2 diluted in one liter of 38% ethanol (aguardiente). Prior to arrival he had experienced diarrhea and a syncopal episode. On presentation he was disoriented, diaphoretic, with tremor and a strong rotten-fish odor.

Laboratory studies revealed mixed acidosis. Gastric decontamination was performed with vegetable oil and a single dose of activated charcoal. He received intravenous fluids, N-acetylcysteine, and oral vegetable oil every six hours, with favorable evolution and discharge at 96 hours. Case 2 A 32-year-old female presented four hours after consuming 50 g of Zn_3P_2 diluted in 750 ml of 12% ethanol (wine). She arrived in convulsive status and subsequently developed cardiopulmonary arrest, with return of spontaneous circulation after six minutes of advanced resuscitation. Laboratory tests showed respiratory acidosis, hyperlactatemia, elevated troponin, and mild coagulopathy. She received respiratory and hemodynamic support, along with oral olive oil every six hours, evolving favorably and being discharged at 72 hours.
Results: The toxicity of Zn_3P_2 is related to the rate of phosphine release. Since Zn_3P_2 is practically insoluble in ethanol, it requires moisture or an acidic environment to hydrolyze and generate phosphine. Alcohol concentrations ≥20–40% have been reported to inhibit gastric secretion and reduce intragastric aqueous content, thereby decreasing the availability of free protons (H^+) required for acid-dependent hydrolysis. Consequently, ethanol co-ingestion may modify the intragastric chemical environment by reducing water activity and failing to provide free protons as hydrochloric acid does, delaying phosphide hydrolysis and therefore phosphine generation. A more gradual release may result in a less abrupt clinical course, which was more evident in the first case.
Conclusion: When a metal phosphide is ingested together with ethanol, higher ethanol concentrations may exert a partial protective effect by delaying phosphine formation. However, despite the favorable clinical outcomes observed, ethanol has no therapeutic role, and larger series would be required before such an effect could be confirmed.

103. 2 Drops or 2 Milliliters? A Pediatric Case Report of a 292,000 IU Daily Vitamin D Dosing Error

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Background: Vitamin D toxicity is typically observed in chronic presentations and predominantly in adults. The classic presentation includes hypercalcemia and its sequelae (nephrolithiasis, abdominal upset, shortened QT interval, weakness, mental status changes). Most cases are supratherapeutic exposures over time. There is a paucity of literature on pediatric vitamin D toxicity and its management. We present a case of vitamin D toxicity in a three-year-old secondary to a dosing error.
Methods: This is a single patient chart review of a three-year-old female with a history of Alagille Syndrome status-post recent liver transplant, who presented for one week of generalized weakness. On initial labs, she was hypercalcemic to 13.2mg/dL with no significant EKG changes (Qtc was 438msec). She was admitted for intravenous hydration at a rate of 2x maintenance and endocrinology evaluation. On Hospital Day 2 (HD#2), the patient's mother discovered that she had inadvertently been administering 2mL daily of cholecalciferol liquid (292,000 IU daily) to the patient instead of 2 drops (4000 IU/drop, formula 365 drops/10mL) for the past 2 weeks. The patient was also prescribed tacrolimus, enalapril, mycophenolate, and prednisolone. Toxicology was consulted for further management. Calcium and Vitamin D levels were drawn serially.
Results: Prednisone was increased to 2mg/kg/day on HD#2. Despite this and IV hydration, calcium continued to rebound. Calcitonin 4u/kg twice daily was started HD#4-HD#6. She was also started on a low calcium/vitamin D diet. Intravenous fluid administration was stopped on HD#4. After calcitonin was discontinued, the calcium remained mildly elevated but never again exceeded >12mg/dL. Endocrinology and toxicology recommended bisphosphonate therapy for anticipated continued hypercalcemia, but the family declined. The patient was discharged HD#8 to follow up with her transplant team. Vitamin D levels peaked on HD# 3 at 650 ng/mL and remained elevated at 330 ng/mL on discharge on HD#8. Calcium levels peaked on HD#2 at 13.9mg/dL and was 10.7mg/dL on discharge.
Conclusion: This is a single-patient case report demonstrating vitamin D toxicity in a pediatric liver transplant patient secondary to a dosing error. There is limited literature reflecting the pediatric population, especially with Alagille Syndrome. This case also stresses the importance of clear labels on formulations and the importance of discussion with families when preparations have changed. Vitamin D toxicity can be difficult to manage due to its distribution/lipophilicity and rebound hypercalcemia.

104. Zonisamide-Induced Toxic Epidermal Necrolysis

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Background: Zonisamide is a newer generation broad spectrum antiepileptic approved as an adjunctive therapy for seizures in 2000. Although newer generation antiepileptics have a greater safety profile, serious cutaneous adverse events have been linked to their use. The mechanism by which hypersensitivity reactions occur is ill-defined but is thought to be associated with accumulation of arene oxide derivatives. Zonisamide itself is also a sulfonamide derivative, potentially adding to the risk for hypersensitivity reactions.

Hypothesis or Research Question: Zonisamide can produce rapidly progressive toxic epidermal necrolysis.
Methods: This is a single case report of an 80-year-old gentleman with a history of traumatic brain injury on alprazolam who presented with 3 to 4 days of fevers at home, sore throat, odynophagia, injected conjunctiva, and an erythematous, non-desquamating rash not involving the palms, soles or mucosa. The patient reported that his neurologist started him on zonisamide 24 days prior due to a breakthrough seizure.
Results: On initial presentation, the patient was hemodynamically stable and afebrile but became progressively more tachypneic during his emergency department stay. His workup was notable for a creatinine of 1.4 mg/dL, elevated transaminases (AST: 924 U/L; ALT: 1538 U/L) and a total bilirubin of 4.7 mg/dL, but otherwise a normal white blood cell count without eosinophilia. His workup revealed an undetectable acetaminophen concentration, normal creatine kinase and hepatitis panel, proteinuria and hematuria on urinalysis, and a markedly elevated ferritin at 2617 ng/mL. Aside from zonisamide initiation, the patient did not have any other medication change, making an adverse drug reaction the most likely. The patient was started on steroids, prophylactic antibiotics, and N-acetylcysteine. Zonisamide was discontinued and he was initiated on lacosamide. Toxicology cautioned the use of lacosamide since it has also been associated with toxic epidermal necrolysis. The patient had a history of having tolerated lacosamide, so the primary team opted to continue lacosamide for seizure precautions. Despite maximal supportive care at a burn center, the patient continued to deteriorate, rapidly progressed to nearly 100% total body surface area involvement of desquamation by hospital day 4. He died 8 days after initially presenting.
Conclusion: Although newer antiepileptics are considered to have a better safety profile, zonisamide is capable of producing profound and rapidly progressing toxic epidermal necrolysis. The initial presentation may be subtle or present as a mild allergic reaction. It is important for clinicians to be aware of this rare and potentially lethal hypersensitivity profile.

105. Exercise-Induced Neurocobaltism Associated With a Low Cobalt Concentration

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Background: Cobaltism is a well-described syndrome with symptoms including cardiomyopathy, thyroid dysfunction, and neurotoxicity classically associated with serum cobalt concentrations greater than 10 mcg/L. Patients with cobalt-alloy orthopedic implants are at increased risk of developing systemic cobaltism, particularly in the setting of prosthetic wear and failure. However, it is believed that symptoms do not typically occur with serum cobalt concentrations less than 10 mcg/L.
Hypothesis or Research Question: Cobalt neurotoxicity can be observed even at mildly elevated serum cobalt concentrations.
Methods: This is a single case report of a 49-year-old previously very physically active physician, with a remote history of a cobalt-chromium metal-on-metal hip implantation, who presented due to increased hip pain, fatigue, and cognitive functional decline. Her decrease in functionality corresponded to a strenuous mountaineering expedition. As part of her workup, a serum cobalt concentration and an MRI of the hip were obtained. Alternative diagnoses and sequalae of cobaltism were investigated by obtaining a brain MRI, thyroid function tests, and cardiac studies. The patient was managed in consultation with the Oregon Poison Center in the outpatient setting, throughout multiple admissions, and after explantation was performed.
Results: Serum cobalt was mildly elevated at 7.1 mcg/L. A hip MRI performed after a period of intense exertion demonstrated early signs of synovitis near the prosthetic hip. Thyroid function studies and echocardiography were unrevealing. A brain MRI did not reveal an intracranial pathology. The patient was initiated on oral N-acetylcysteine (NAC) with minimal improvement. However, she became progressively more impaired in performing her professional and personal daily tasks prompting multiple hospital encounters where she received IV NAC with marginal improvement in functionality. Despite progressively increasing doses of oral NAC culminating at 1200 mg daily to mitigate her symptoms but her neurocognitive symptoms persisted to the point of being unable to perform her duties as a physician. She underwent a revision arthroplasty, where her prosthetic hip was replaced with a cobalt-free metal-on-plastic material. The operative site demonstrated early prosthetic failure and metal staining of the synovium. The patient's neurocognitive symptoms progressively improved in the ensuing weeks.
Conclusion: Despite not having a serum concentration greater than 10 mcg/L, patients with cobalt-based implants may still be at risk of developing cobalt neurotoxicity.

106. Thallium Exposure in a Family of Seven

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Background: While Prussian blue is the less-invasive treatment for thallium toxicity compared to hemodialysis, the exact duration of treatment is not entirely clear based on persistent symptoms despite diminishing thallium concentration.
Hypothesis or Research Question: Does hemodialysis improve the clearance of thallium compared to Prussian

blue alone and should concentration clearance be the endpoint of treatment?

Methods: This is a seven-patient case series at a single academic institution. A family of five (a 56-year-old male, 52-year-old female, 23-year-old female, 18-year-old female and an 11-year-old male) traveled to India and stayed for one week. Approximately one month later, five of the seven family members developed abdominal pain, non-bloody diarrhea, fatigue and severe burning paresthesias, predominantly in the bilateral feet followed by alopecia. Urine testing confirmed thallium exposure (117 micrograms [mcg]/gram [g] creatinine [Cr] to 12536 mcg/g Cr). The oldest family member (81-year-old female who did not travel to India) received four, four-hour sessions of hemodialysis (HD) due to a high concentration (12536 mcg/g Cr) and minimal symptoms. All family members were started on Prussian blue at three grams by mouth, three times per day. Urine thallium concentrations were obtained weekly for the duration of treatment and all family members were advised to live outside of their primary residences until the source was identified.

Results: The 81-year-old patient who underwent four HD sessions had a marked decrease (12536 mcg/g Cr pre-HD) in urine thallium concentrations (620 mcg/g creatinine post-HD) compared to the remaining four family members who were treated with Prussian blue only. All five patients were treated with Prussian blue until urine concentration was less than 50 mcg/g Cr. The 81-year-old had a similar treatment duration with Prussian blue despite a markedly higher concentration. Notably, alopecia and reported paresthesias were persistent for several weeks after the Prussian blue was stopped.

Conclusion: Hemodialysis clears thallium faster than Prussian blue alone, however resolution of symptoms lag behind urine thallium concentration clearance. We used lab confirmation with thallium/creatinine random urine ratio as the endpoint of therapy. Notable limitations of this case series include lack of knowledge of the exposure source and not all patients performed twenty-four-hour urine thallium testing.

107. Is More Nuance Necessary Regarding Antivenom Choice?

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Background: Crotalidae immune $F(ab')_2$ (equine) is often suggested to be the preferred antivenom for all North American rattlesnake envenomations. However, it is not clear if Crotalidae polyvalent immune Fab (ovine) is more efficacious in some envenomations.

Hypothesis or Research Question: Crotalidae immune $F(ab')_2$ (equine) is the preferred treatment for the neurotoxicity and hematotoxicity from Southern Pacific rattlesnakes.

Methods: This is a single patient case encounter. A 62-year-old male was envenomated by a Southern Pacific rattlesnake to his hand when he reached into a cabinet. He developed pain and swelling to his hand that progressed to his wrist and forearm. He developed diffuse and painful myokymia as well as numbness and dysarthria. Initially in the emergency department, he received morphine, midazolam, and intravenous fluids. His initial laboratory testing demonstrated thrombocytopenia to 126 ($X10^3$/mL) and hypofibrogenemia (175 mg/dl). Six vials of Crotalidae polyvalent immune Fab (ovine) were ordered. Following receipt of antivenom, the pain to his hand slightly improved but he continued to have painful myokymia. While the dysarthria improved, his speech was still not normal. Repeat testing demonstrated a persistent thrombocytopenia (62 $X10^3$/mL) but improvement of his fibrinogen. Another four vials of Crotalidae polyvalent immune Fab (ovine) were ordered. His thrombocytopenia, myokymia, and dysarthria improved. A blister to his hand was deroofed.

Results: On hospital day #3, it was noted that he could not walk due to worsening neurotoxicity and was having worsening paresthesias and myokymia. It was discovered that pharmacy had substituted Crotalidae immune $F(ab')_2$ (equine) instead without adjusting the MAR or informing toxicology. Six vials of Crotalidae polyvalent immune Fab (ovine) were ordered and administered, which was confirmed with pharmacy. The next day the patient's pain was much improved, he was not demonstrating any neurotoxicity and could walk without difficulty, and he was discharged. At follow up, the wound to his hand and swelling were improving and there was no recrudescence of the neurotoxicity. He did have slight recurrence of his thrombocytopenia without bleeding that improved by day #14. Limitations: The patient did receive both antivenoms which could confound comparisons regarding their efficacy. While there was no confirmation of the snake with a photograph, the only native venomous snakes in our area are the Southern Pacific rattlesnake.

Conclusion: Crotalidae polyvalent immune Fab (ovine) effectively treats the neurotoxicity from Southern Pacific rattlesnakes. While Crotalidae immune $F(ab')_2$ (equine) treated the initial hematotoxicity, it did not prevent delayed thrombocytopenia.

108. Protobothrops Mangshanensis Envenomation: Convalescence of Coagulopathy After Haemato-Polyvalent Snake Antivenom Administration

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Background: *Protobothrops mangshanensis* (Mang Mountain viper) is an endangered pit viper endemic to the Guangdong and Hunan provinces of Southern China. There are three published cases of *P. mangshanensis* envenomation, all of which were complicated by severe coagulation dysfunction. We report a case of an envenomation by P. *mangshanensis* and the patient's subsequent coagulopathy treated with Haemato-polyvalent snake antivenom (HPAV).
Hypothesis or Research Question: Dose adjustment of HPAV by coagulation lab monitoring can lead to resolution of coagulopathy in *P. mangshanensis* envenomations.
Methods: A 28-year-old male with a history of an MI presented to the ED with a snake bite to the left second digit by a captive *P. mangshanensis*. The patient had a prior history of envenomation by Crotalus horridus in 2023. He reported pain to the bite site and the left axilla. Initial vitals were stable and examination revealed two punctate wounds to the distal second digit with mild edema to hand and wrist. Following an initial coagulopathy workup that revealed normal prothrombin time/international normalized ratio (PT/INR), D-Dimer, fibrinogen, platelets, and a mildly increased activated partial thromboplastin time (aPTT), he was transferred to a regional snake bite center for potential anti-venom administration.
Results: The patient was stable on arrival to ED and was admitted to a monitored unit. His workup 12 hours post-envenomation revealed evidence of consumptive coagulopathy with undetectable fibrinogen levels, increasing PT/INR and aPTT, and worsening function evidenced on thromboelastography. He complained of worsening pain at this time. Treatment with HPAV was initiated 18 hours after the envenomation with four vials of HPAV, followed by an additional five vials 12 hours later. Coagulopathy labs peaked with PT/INR 20.4/1.8, aPTT 42.6, and fibrinogen<35 at 24 hours post-bite before near-resolution 24 hours after treatment with normal PT/INR, aPTT 41.3, and fibrinogen=155. The patient also reported improvement in left upper extremity pain after administration of antivenom. He was discharged from the hospital and returned three days later for follow up labs which showed normalization of PT/INR and fibrinogen, and aPTT 41.8.
Conclusion: This case describes an acute *P. mangshanensis* envenomation complicated by consumptive coagulopathy with apparent resolution after escalating doses of HPAV. On follow up, the patient reported only mild pain 5 days after the bite. While there are limited reports of prolonged coagulopathy in the setting of *Protobothrops* envenomation, this patient's coagulation labs appeared stable 120 hours after the event.

109. Adverse Effects and Clinical Outcomes Associated With Individual Glucagon-Like Peptide-1 Receptor Agonists

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Background: Although the number of GLP-1 receptor agonist (GLP-1 RA) prescriptions are rapidly increasing, the data on overdoses of these agents are limited. This is important given the high incidence of online procurement of these drugs, which raises questions of quality control and risk of dosing errors.
Hypothesis or Research Question: Describe adverse effects and clinical outcomes associated with GLP-1 RA exposures.
Methods: This is an analysis of prospectively collected data from the Toxicology Investigators Consortium (ToxIC) Core Registry of adult and pediatric patients who had a GLP-1 RA exposure between January 2010 and August 2025. Descriptive statistics were calculated for baseline characteristics, presenting symptoms and outcomes (treatment and disposition). Additionally, outcomes were compared across GLP-1 RA subtypes and between single and multi-drug ingestions.
Results: Seventeen patients met inclusion criteria. Ages ranged from two to 80 years (mean=39 years) and four (24%) patients were male. Three (18%) patients had a history of diabetes mellitus. The most common reason for exposure was attempt at self-harm (n=5, 29%). Semaglutide was the most common agent (n=9, 53%). Patients who took semaglutide had no hypoglycemia and presented with nausea/vomiting (n=3, 33%) and diarrhea (n=1, 11%). Six (75%) patients who took a non-semaglutide GLP-1 RA presented with hypoglycemia. Six (75%) patients reported taking other hypoglycemic medications, either therapeutic or supratherapeutic. Six patients (67%) in the semaglutide group received a therapeutic intervention, with the most common being intravenous (IV) fluids (n=5, 57%). All patients who took a non-semaglutide GLP-1 RA received treatment, with the most common being dextrose-containing IV fluids (n=7, 88%). Four patients in the semaglutide group were discharged from the ED (44%), and five were admitted to the floor (56%). Patients who took other GLP-1 RAs were all admitted to hospital either to the floors (n=4, 50%), to the intensive care unit (n=3, 38%), or unknown (n=1, 13%). Compared to patients who only took a GLP-1 RA, those who had multiple medications had higher incidence of hypoglycemia (n=1, 10% vs. n=5, 71%), were more likely to require treatment (n=7, 70% vs. n=7, 100%), and had higher rates of hospital admission (n=6, 60% vs. n=6, 86%).

Conclusion: Intentional self-harm comprised nearly one third of GLP-1 RA exposures. Patients who took a GLP-1 RA with other hypoglycemic medications were more likely to be hypoglycemic, receive therapeutic interventions, and require escalation of care. The low incidence of GLP-1 RA exposures in light of their increasing prevalence suggests that exposures are likely underreported.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

110. CHANTER Syndrome: Clinicoradiographic Diagnosis With Diverse Toxicologic Exposures

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Background: Cerebellar, Hippocampal, and basal Nuclei Transient Edema with Restriction diffusion (CHANTER) is a clinicoradiographic syndrome defined by a distinct, non-vascular distribution pattern seen on MRI. It is most often recognized in patients presenting with altered mental status, ranging from disorientation to unresponsiveness, after opioid or polysubstance overdose. Recent cases suggest that CHANTER may have several variants and may be associated with more diverse exposures than previously believed.
Hypothesis or Research Question: CHANTER syndrome may be broadened to include "incomplete" or "subacute" variants, along with exposures beyond opioids.
Methods: This is a retrospective case series of 14 patients evaluated at our center between 2018-2025. Cases with a diagnosis of "CHANTER syndrome" (CS), "incomplete CHANTER syndrome" (iCS), or "subacute CHANTER syndrome" (sCS) were assessed for a toxicologic exposure using history, laboratory analysis, and, if available, review of medical toxicology consultation or confirmatory testing. CS was defined as having all three areas (i.e. cerebellum, hippocampus, and basal nuclei) with diffusion restriction on MR imaging and a presentation of "unresponsive" or "altered". iCS was defined as two of three areas with diffusion restriction and consistent presentation. sCS was defined as T2/FLAIR abnormality without diffusion restriction and consistent presentation. Mortality and neurological outcome (Modified Rankin Score) were assessed through chart review.
Results: Five patients met the case definition for CS, eight patients met the case definition for iCS, and one patient met the case definition for sCS. Of the CS patients, 40% (2/5) were associated with opioids, 40% (2/5) were associated with cocaine, and 20% (1/5) were associated with a combined opioid and cocaine. Of the eight iCS patients, 25% (2/8) were combined opioid and cocaine, 50% (4/8) were cocaine only, and 25% (2/8) were other exposures, specifically hydrogen sulfide and ethylene glycol. The sCS patient's toxic exposure was cocaine. Overall mortality was 28.6% (4/14) and among those who survived the mean Modified Rankin Score was 4.3 upon discharge. There was not a statistically significant difference in Modified Rankin Scores between CS and iCS groups (5.2 vs 4.625, p=0.4192).
Conclusion: CHANTER syndrome is a clinical presentation and radiographic diagnosis associated with a variety of toxicologic exposures, with possibly more variations than previously described.

111. Pediatric Prairie Rattlesnake Envenomation Resulting in Myokymia

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Background: Myokymia, spontaneous, involuntary muscle contractions seen as rippling movements of the overlying skin, is associated with envenomation from several North American rattlesnake species. While neurotoxicity is reported in human cases from other snake species, we could not find prior reports from Praire rattlesnake (*Crotalus viridis)* envenomation.
Hypothesis or Research Question: C. viridis envenomation can produce neurotoxic findings similar to other North American rattlesnakes.
Methods: This is a case report of an 18-month-old male in Montana.
Results: An 18-month-old male residing in western Montana was bitten near his right eyebrow by a rattlesnake. Definitive identification was not possible; however, C. viridis is the only known species of rattlesnake in this region. The patient was treated at a local hospital. He received 3 vials of crotalidae polyvalent immune Fab (ovine) followed by two 10-vial doses of crotalidae immune $F(ab')_2$ (equine). He was intubated for airway protection prior to transfer to a tertiary care center. On arrival to the pediatric hospital, the patient had significant facial swelling (around the right eyebrow, eyelid, and involving the bilateral face to the level of the maxilla). Laboratory results were: platelets 200, PTT 32, PT 16.3, INR 1.3, and fibrinogen 154. On hospital day 3, the patient was extubated. Later that evening he developed diffuse, rhythmic

twitching without other concerning findings on neurologic exam. He had no respiratory difficulties. Symptoms were refractory to treatment with benzodiazepines. Additional doses of F(ab')2 and Fab antivenom failed to treat the symptoms, but they resolved spontaneously over the next 36 hours. The remainder of the clinical course was uncomplicated, and he was discharged from the hospital on day 8 without evidence of coagulopathy or recurrence of neurotoxicity.

Conclusion: While some rattlesnake venoms have a phospholipase A2 neurotoxin (e.g., C. scutulatus, C. horridus, C. oreganus helleri, C. oreganus), but none of these species are found in Montana. C. viridis venom in Montana populations has been shown to be comprised of >50% myotoxin A. This toxin is known to cause tetanic contractions in murine models. While we were unable to find prior human reports of neurotoxicity in human C. viridis envenomation, this may be the result of a dose-dependent phenomenon whereby a small child was exposed to a large amount of venom per kilogram bodyweight. The delayed nature of the myokymia may have been masked by the initial control doses of antivenom, however both antivenoms failed to ameliorate the effects.

112. Hypothermia Cases Reported to the National Poison Data System (2005–2024)

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Background: Hypothermia is a medical emergency that may reflect the severity of overdose. Although it can serve as a clue to certain toxidromes and may complicate the medical management of poisoned individuals, the epidemiology and etiologies of hypothermia in adult overdose patients remain poorly characterized.

Hypothesis or Research Question: This study aims to describe the age distribution, substance associations, clinical manifestations, and therapeutic interventions among adult cases reported to U.S. poison centers in which hypothermia was documented as a clinical finding.

Methods: This was a retrospective review of adult cases reported to U.S. poison centers in which hypothermia was reported as a clinical effect. Data were extracted from the National Poison Data System (2005–2024) and included single substance exposures among patients 20 years of age or older. Age, associated substances, clinical effects, therapies, and reason for exposure were descriptively analyzed.

Results: Over the 20-year study period, a total of 17,876 hypothermia cases were reported. Annual case counts ranged from a low of 439 cases in 2007 to a high of 1,319 cases in 2019, averaging approximately 894 cases per year. The overall trend showed a rising incidence through the late 2010s, with a subsequent plateau from 2020 through 2024 (annual counts consistently above 1,200 cases). Cases were evenly distributed across 10-year age groups and included ages 20-29 years (17%), 30-39 years (18%), 40-49 years (18%), and 50-59 years (19%). The most commonly implicated substances were ethanol (5.1%), benzodiazepines (5.0%), and atypical antipsychotics (4.7%). Severe CNS depression/coma and moderate CNS depression were reported in 40% and 30% of cases, respectively. Other frequently reported clinical effects were hypotension (36%), acidosis (32%), tachycardia (30%), electrolyte abnormalities (27%) and elevated creatine (21%). Management most frequently involved intravenous fluids (68%) and oxygen supplementation (57%). More advanced interventions included intubation (45%) and mechanical ventilation (44%).

Conclusion: The study findings revealed a sustained burden of hypothermia reported to poison centers over the past two decades, with notable increases through the late 2010s. The high proportion of cases linked to ethanol, sedatives, and antipsychotics points to the influence of psychoactive substances in thermoregulatory failure.

Therapeutic interventions indicate that many cases required intensive management, including intubation and ventilation, suggesting significant clinical severity. The frequent occurrence of systemic complications, including CNS depression, hypotension, acidosis, electrolytes and creatinine abnormalities, further emphasizes the potential for hypothermia to be an indicator of a life-threatening condition when associated with toxic exposures

113. The Hidden Cost of Having an Exotic Venomous Snake

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Background: Although exotic pet ownership in the United States is common, the number of privately owned non-native venomous snakes is unknown. National tallies on treatment of exotic snakebites suggest a community of enthusiasts. Exotic envenomation poses not only clinical and logistical challenges for the healthcare team, (e.g., most exotic-species antivenins aren't FDA-approved; they must be obtained through zoo-based or poison center sources; are often difficult to obtain through red-tape and distance); as with domestic snakebites, they also pose a significant unanticipated financial challenge to those bitten.

Methods: This case report describes the clinical healthcare pathway costs after a neurotoxic envenomation from a privately owned non-native snake. This case describes an index patient's journey though care and is illustrated

with charge estimates derived from representative billing data that is intentionally presented as averages/estimates to accommodate regional variability in costs.
Results: A previously healthy 30-year-old male was bitten on the left hand by his pet leucistic monocled cobra (Naja kaouthia) while cleaning the enclosure. He immediately presented with severe localized pain to a critical-access hospital (ED charge: $2,000). Due to concern for neurotoxicity and absence of the required antivenom, he was transferred via EMS to a regional university hospital (EMS charge: $5,000). His ED evaluation (ED charge: $2,000) confirmed impending neurotoxic symptoms and the need to have antivenom available. Immediate transfer (Medical helicopter: $14,000) was arranged to bring the patient to the nearest center with access to Thai neuro-polyvalent antivenom (unavailable locally). On arrival he received 10 vials of antivenom ($1,500 total), spent one night in the ICU (ICU charge: $3,500), and completed a three-day hospital stay (in-patient/day: $2,400 x3). Total estimated charges were approximately $35,200 (excluding additional medications, consultants, etc). These figures are purposely estimated to reflect regional variation but illustrate the substantial unanticipated financial burden of exotic-snake ownership/envenomation.
Conclusion: This case describes the profound financial cost of a single bite, and the additional underappreciated risk (besides death) associated with keeping exotic venomous snakes. These costs are directly related to issues of accessing and obtaining non-FDA-approved antivenins, long range transport of the patient or antivenin, and ICU care. All accumulate rapidly into the tens of thousands of dollars. As with domestic snakebites, the long lasting financial impact of exotic snakebite care often exceeds the acute clinical episode—information that should be imparted to our patients that benefits of their exotic pet ownership should be balanced by their economic risks.

114. Paint in the Glass: Succimer Monotherapy in an Artist Presenting With Lead Colic

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Background: Historically, moderate to severe lead toxicity was treated with multiple chelation agents. Artists have long experienced toxic exposures, including lead, dating back centuries. We present a unique case of a painter with chronic ingestion of leaded oil paints during recurrent self harm attempts successfully treated with oral succimer monotherapy.
Hypothesis or Research Question: Is oral succimer monotherapy an effective treatment for moderate to severe lead toxicity without encephalopathy?
Methods: This is a single patient chart review. A 27-year-old woman presented with lead colic after intentionally consuming leaded paint and button batteries for two months. We initiated treatment with succimer monotherapy empirically based on symptoms of lead colic and sign of anemia. The patient was treated for six days prior to obtaining lead results, and the patient was continued on succimer monotherapy for the remainder of the 14 day treatments. Furthermore, we explore the decision to treat her empirically with succimer monotherapy while awaiting levels, and examine the patient's response through lead levels, symptom resolution, and rebound blood levels.
Results: The initial lead level was 126.1 μg/dL. This level did not result until six days after the patient's initial presentation. Oral succimer monotherapy was started on HD one, and after three days of therapy, a repeat level was, 78.9 μg/dL. She was continued on succimer with a decrease in the blood lead level to 21 μg/dL over the next three weeks. She had a measured rebound to 42.5 μg/dL two months after the completion of her succimer.
Conclusion: Chelation is an essential component of treating lead toxicity in both adults and children. While several guidelines suggest treatment of lead toxicity with oral monotherapy up to levels of 70 ug/dL in pediatrics, or 100 ug/dL in adults, there may be a role for monotherapy in select patients with levels above these thresholds. This case highlights the effectiveness of oral succimer monotherapy in moderate lead toxicity.

115. Persistent Hypersensitivity Reaction to N-Butyl Cyanoacrylate With Acute Kidney Injury

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Background: Tissue glues commonly contain cyanoacrylate derivatives which have been associated with hypersensitivity reactions. Internal use may result in persistent exposure causing prolonged hypersensitivity reactions and end organ damage.
Hypothesis or Research Question: Prolonged hypersensitivity reactions to internal use of cyanoacrylate derivatives may result in end organ damage.
Methods: This is a single patient case report. A 66-year-old female with a history of varicose veins, type one diabetes and hypothyroidism presents to the emergency department with persistent pruritic bodywide rash 18 days after varicose vein gluing with n-butyl cyanoacrylate (VenaSeal). She had no prior allergy history. She denied any new substance

exposures, new medications, new household products or changes in her diet. She had been taking over-the-counter antihistamines without relief of symptoms.
Results: Diffuse urticaria and associated pruritus were noted on physical examination. Laboratory testing revealed no leukocytosis, but did show an eosinophil count of 400 cells/uL. The creatinine and BUN were 5.14 mg/dL and 68 mg/dL, respectively from a baseline of 0.52 mg/dL and 17 mg/dL, consistent with acute kidney injury (AKI). Her initial blood pressure was 87/54 which improved to 133/57 after receiving one liter of intravenous lactated ringers solution, 20 mg of intravenous famotidine, 125 mg of intravenous methylprednisolone and 0.5 mg of intramuscular epinephrine. Despite these interventions, the patient's rash and pruritus persisted and the patient was admitted to the hospital for observation. During her hospitalization, she was evaluated by nephrology who attributed her AKI to drug-induced hypersensitivity syndrome and persistent hypotension. No tissue biopsy was performed. Her symptoms resolved 21 days after the procedure and she was discharged. On the day of discharge, the creatinine and BUN had improved to 1.42 mg/dL and 53 mg/dL respectively.
Conclusion: Persistent hypersensitivity to internally used cyanoacrylate derivatives may result in end organ damage including kidney injury. Limitations include nonexperimental chart review, atypical presentation, alternative etiology of her hypersensitivity reaction and alternative etiologies of her end organ damage.

116. Hypersensitivity Reaction to F(ab')2 Following Fab in an Alpha-Gal Sensitized Patient

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Background: Both Fab and F(ab')2 are available for the treatment of North American pit viper envenomation. While immune reactions to newer antivenom products are infrequent, some hypersensitivity reactions may be related to underlying alpha-gal (galactose-α-1,3-galactose) sensitization.
Hypothesis or Research Question: In a patient inadvertently administered F(ab')2 for an *Agkistrodon contortrix* envenomation following initial treatment with Fab, is a hypersensitivity reaction potentially related to alpha-gal sensitization?
Methods: This is a single patient chart review.
Results: A 12-year-old male with no past medical history presented to an outside hospital after an *Agkistrodon contortrix* envenomation. His initial exam demonstrated two puncture wounds over the dorsolateral left foot with erythema, edema, ecchymosis, and tenderness extending to the ankle. CBC, CMP, INR, and fibrinogen were within normal limits. He received 2 vials of Fab prior to transfer and tolerated this antivenom without reported reaction. Upon arrival to a pediatric hospital, his examination by the toxicology consultant was consistent with the outside hospital report. He was admitted for observation. By hospital day 2, his pain progressed up to the thigh. The toxicology consultant recommended an additional 2 vials of Fab and entered the order in the electronic health record on behalf of the primary team per their request. During the second antivenom infusion, the patient developed throat discomfort and itching concerning for a possible hypersensitivity reaction. He did not have any vital sign abnormalities, rash, or other symptoms. The antivenom infusion was stopped. Following this, his symptoms gradually resolved with a single dose of oral diphenhydramine 25 mg and oral cetirizine 10 mg. His leg pain improved, and he was discharged home the following day. Further investigation revealed that he had received 2 vials of F(ab')2 rather than Fab due to a pharmacy administration error. Alpha-gal IgE testing obtained prior to discharge was positive at 1.63 kU/L (normal range 0-0.34 kU/L), confirming alpha-gal sensitization.
Conclusion: This patient with previously undiagnosed alpha-gal sensitization tolerated Fab but subsequently had a hypersensitivity reaction to F(ab')2. F(ab')2 has a higher alpha-gal content compared to Fab. Geographical regions with higher alpha-gal syndrome prevalence have increased rates of hypersensitivity reactions to F(ab')2. Together, these implicate alpha-gal sensitization as a cause of this patient's hypersensitivity reaction.

117. Use of Succimer to Treat Copper Sulfate Induced - Hemolysis

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Background: Copper sulfate ingestion is an uncommon but potentially life-threatening exposure associated with oxidative hemolysis, methemoglobinemia, and hepatic injury. Chelation therapy options for copper toxicity are limited and not well standardized. Dimercaptosuccinic acid (succimer, DMSA) has rarely been described as a therapeutic agent in acute copper toxicity.
Hypothesis or Research Question: Can oral succimer be an effective and well-tolerated chelating agent in the management of acute copper sulfate poisoning complicated by hemolysis?
Methods: This is a single-patient case report. A 60-year-old female accidentally ingested a copper sulfate–containing

fungicide. She presented three days later with vomiting, cramping abdominal pain, and darkened urine. Initial laboratory studies revealed total bilirubin 7.8 mg/dL (indirect bilirubin 7.1 mg/dL), and hemoglobin 12.2 g/dL. Over the subsequent 24 hours, bilirubin peaked at 13.1 mg/dL and hemoglobin fell to 7.5 g/dL, consistent with hemolysis. Chelation with oral succimer was initiated at 700 mg every 8 hours x 5 days. Other supportive measures included N-acetylcysteine, oral zinc acetate, and three packed red blood cell transfusions. Hepatic and renal function were monitored serially.

Results: Within 48 hours of starting succimer, total bilirubin decreased from 8.0 mg/dL to 2.4 mg/dL. Hemoglobin stabilized and increased from a nadir of 6.2 g/dL to 8.3 g/dL after transfusion and continued therapy. Direct bilirubin and transaminases normalized within four days. Haptoglobin, initially undetectable, became measurable by day 5, consistent with resolution of hemolysis. Neither significant methemoglobinemia, nor acute kidney injury occurred. No adverse effects attributable to succimer were observed.

Conclusion: Succimer, an orally bioavailable dithiol compound, binds divalent metals including copper and has a favorable safety profile. This case demonstrates biochemical and clinical improvement temporally associated with succimer therapy. Succimer may represent a viable alternative chelating agent in acute copper toxicity, particularly when D-penicillamine or dimercaprol are unavailable or contraindicated. Further study is warranted to discern its potential role in copper sulfate poisoning when other supportive measures have not been successful.

118. Detection and Species-Specific Patterns of Harmful Algal Toxins in Cold-Stunned Sea Turtles From Massachusetts

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Background: Harmful algal blooms (HABs) produce toxins that can bioaccumulate in marine animals and pose risks to wildlife and human health.

Hypothesis or Research Question: The prevalence and patterns of harmful algal toxins in stranded sea turtles will vary by species, sex, and year.

Methods: Post-mortem liver samples from three sea turtle species stranded on Cape Cod, Massachusetts, between November 2023–January 2024 and November 2024–January 2025 were analyzed for 14 regionally specific toxins, including neosaxitoxin, saxitoxin, cylindrospermopsin, anatoxin A, domoic acid, microcystins, nodularin, okadaic acid, brevetoxins, and teleocidin. Descriptive statistics summarized toxin prevalence and concentration. Differences in detection by species, sex, and season were evaluated using Pearson's Chi-square or Fisher's Exact tests, and the relationship between time from death to analysis was examined using the Wilcoxon rank-sum test ($\alpha = 0.05$).

Results: Eighty-six samples were analyzed: 42 *Lepidochyelys kempii*, 22 Caretta caretta, and 22 *Chelonia mydas*. Toxins were detected in 41% of the samples: one toxin in 29 (34%), and two in six (7%). The most prevalent toxins in the 2023-24 and 2024-25 seasons were domoic acid and microcystin YR respectively. The four most prevalent toxins were Microcystin YR (N=13, 15.1%), Domoic acid (N=8, 9.3%), Brevetoxin-3 (N=7, 8.1%) and Microcystin LR (N=7, 8.1%) which were further evaluated in statistical analyses. Detection prevalence varied significantly among species ($\chi^2 = 7.44$, df = 2, p = 0.024), highest in *C. mydas* (59%), followed by C. caretta (45%), and *L. kempii* (29%). The four most prevalent toxins were observed in 36% of the samples during the 2023-24 season and in 33% of the samples during the 2024-25 season. No significant differences were observed in toxin detection across seasons ($\chi^2 = 0.002$, df = 1, p = NS), sexes (Fisher's Exact Test, p = NS) or interval between death and sample analysis (W = 972, p = NS).

Conclusion: Harmful algal toxins were detected in over one-third of sea turtle liver samples collected across two consecutive stranding seasons, suggesting ongoing exposure in Massachusetts coastal waters. These findings provide baseline data to assess biotoxicosis and its potential impact on marine wildlife health. Enhanced monitoring of harmful algal toxins may inform marine wildlife conservation and human health risk assessment. Continued surveillance and cross-disciplinary monitoring are warranted to enhance preparedness for emerging algal-toxin–related health threats.

119. Metformin Overdose and Outcomes in the U.S. National Poison Data System, 2020-2024

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Background: Metformin is the first-line oral antihyperglycemic in the United States, with more than 553 million prescriptions dispensed between 2000 and 2015. While

therapeutic use is generally safe, overdose may result in metabolic acidosis with elevated plasma lactate, gastrointestinal disturbances, electrolyte abnormalities, and death. Most prior analyses of metformin toxicity are regional or single-center, limiting generalizability. A national, updated characterization of metformin overdose is needed to inform toxicovigilance and guide management practices.

Hypothesis or Research Question: What are the current epidemiology, severity, and outcomes of metformin overdoses reported to U.S. poison control centers?

Methods: This was a retrospective descriptive analysis of human single-substance metformin exposures reported to the National Poison Data System (NPDS®) between January 1, 2020, and December 31, 2024. Single-substance cases were included if metformin was listed as the primary substance. Children under 12 years were excluded to minimize misclassification of exposure intent. Annual trends in exposure incidence and mortality were compared across study years and stratified by age group. Descriptive statistics were calculated, and multinomial logistic regression identified demographic, clinical, and treatment factors associated with major effects or death.

Results: Among 15,142 reported human exposures (mean age 50 ± 20.7 years; 64% female), most were attributed to therapeutic error (72%). Case volume increased significantly from 2020 to 2024 ($p < 0.001$), though trends among adolescents and intentional exposures were not significantly different during the time period. Nausea (26%), vomiting (24%), and acidosis (23%) were the most frequently reported clinical effects. Mean reported dose was 3.8 g ± 7.1 g; intentional ingestions involved higher doses and younger patients (21.8 g, 33 years) than unintentional exposures (3.4 g, 54 years; $p < 0.001$). Overall mortality was 1.9%. Multinomial regression identified increasing age, hypotension, ECMO, intubation, vasopressor use, and CRRT as significant predictors of major effect or death (all $p \leq 0.034$).

Conclusion: Single-substance metformin exposures have increased nationally, likely reflecting therapeutic errors among older adults receiving antihyperglycemic therapy. The identified predictors, particularly age, hypotension, and need for critical care interventions, align with clinical indicators of severe poisoning. Presentation with acidosis and elevated creatinine were not independent predictors of mortality, though NPDS codes acidosis dichotomously limiting severity assessment. These findings reinforce the importance of early recognition and consideration of extracorporeal support in critically ill patients. National surveillance data demonstrate increasing metformin exposures with consistent mortality rates and clinical patterns. Continued evaluation of extracorporeal treatment use and outcomes is warranted to optimize care in severe metformin toxicity.

120. Encephalopathy Following Unintentional Repeated Supratherapeutic Levetiracetam Use

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Background: Levetiracetam is a first-line antiepileptic regularly prescribed for treatment of epilepsy. Its antiepileptic activity is a result of presynaptic calcium channel inhibition as well as inhibition of neurotransmitter exocytosis via modulation of synaptic vesicle glycoprotein 2A (SV2A). Due to its wide therapeutic window, there are few reported cases of levetiracetam toxicity.

Hypothesis or Research Question: Repeated supratherapeutic levetiracetam use can lead to levetiracetam toxicity.

Methods: This is a single patient chart review of a 35-year-old female with a history of seizure disorder on levetiracetam who presented to the emergency department with somnolence, encephalopathy, nausea, and headache. Initially while in the ED, the patient was too somnolent to provide history. The patient's work-up including basic metabolic panel, complete blood count, levels for acetaminophen, salicylate, ethanol, urine drug screen, and CT head were unrevealing. Initial levetiracetam level obtained in the ED was 518.6 μg/mL. Her headache and nausea resolved after treatment with intravenous ketorolac, prochlorperazine, and ondansetron, although she remained somnolent while in the emergency department. Repeat levetiracetam level obtained prior to admission 2 hours later was increased at 671.5 μg/mL. Her home levetiracetam regimen was held, and she was admitted to the medical toxicology service for monitoring.

Results: Serial levetiracetam levels obtained while inpatient returned to therapeutic range within 30 hours of hospital stay (184.2 μg/mL at hour 16, 119.0 μg/mL at hour 20, 32.5 μg/mL at hour 30, 18.4 μg/mL at hour 34). During this time, the patient had accompanying gradual resolution of encephalopathy and other symptomology, returning to baseline mental status, and additional history was obtained. According to the patient, she had been compliant with her prescribed antiepileptic regimen of 1g levetiracetam daily and had been experiencing chronic headaches. For months the patient had also been taking multiple tablets daily from an unlabeled pill bottle that she presumed was prescribed by her PCP for abortive headache therapy. The patient was able to reproduce this unlabeled bottle of medication, which was determined by a web-based pill-identifying application to also be levetiracetam, presumably an older prescription. She was instructed on proper medication practices, including pharmacologic labeling and dosing. She was restarted on her home antiepileptic regimen, provided follow-up

information for an outpatient neurologic clinic for ongoing management of seizure disorder and headaches, and was discharged from the hospital.
Conclusion: Chronic levetiracetam toxicity, though rare, can cause encephalopathy, somnolence, and nausea. Cases can be managed with inpatient observation for monitoring and symptomatic control.

121. Severe Ethylene Glycol Poisoning Presenting With Early Hypernatremia and Polyuria

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Background: Ethylene glycol toxicity classically presents with high–anion gap metabolic acidosis, elevated osmolar gap, and acute kidney injury. The case below describes a patient with early hypernatremia and polyuria that have not been previously described as associated clinical features.
Methods: We present a 62-year-old woman presenting for suspected ingestion of multiple medications who developed severe acidemia and hypernatremia. On presentation, she was agitated requiring sedation, intubation, and admission to the intensive care unit. Initial labs included serum sodium 143 mEq/L and bicarbonate 18 mEq/L; within 18 hours, serum sodium rose to 159 mEq/L and bicarbonate became undetectable. Serum osmolality was unavailable at this hospital. Anion gap was 27 mEq/L with venous pH 6.83. She had significant polyuria (>3 L) in 12 hours. Based on polyuria and hypernatremia, ethylene glycol poisoning was suspected.
Results: Subsequent urinalysis demonstrated calcium oxalate crystals. She received fomepizole, thiamine, pyridoxine, and emergent hemodialysis (HD) for suspected ethylene glycol toxicity, later confirmed at 264 mg/dL on hospital day 2. Due to limited resources, she received one session of HD and was subsequently transferred for additional dialysis. The patient was continued on HD with rapid normalization of anion gap. Her course was complicated by recurrent seizures requiring benzodiazepines and antiepileptics, with multifocal cerebral infarcts revealed on MRI. Despite metabolic improvement, she became anuric with creatinine rising from 0.7 mg/dL to 4.6 mg/dL, suggestive of oxalate-mediated renal injury. By hospital day three, she was following commands and was extubated, and remained dialysis-dependent for one month.
Conclusion: Rapid development of hypernatremia with marked polyuria may represent a previously unreported presenting feature of ethylene glycol intoxication, presumably due to osmotic diuresis. Recognition of this pattern could support earlier consideration of toxic alcohol ingestion and prompt early initiation of therapy, especially if the osmol gap is unavailable. Future studies may evaluate whether early hypernatremia and polyuria can serve as reliable clinical predictors in suspected ethylene glycol exposures.

122. Naloxone Administration in Relation to Fentanyl, Xylazine and Central Nervous System Depressants in Suspected Opioid Overdose: A Pilot Study

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Background: Fentanyl is a potent opioid that has supplanted heroin in the illicit drug supply in the United States (US). Because of this change, some have advised that routine naloxone dosing should be higher to reverse fentanyl's effects during the resuscitation of opioid overdoses. There is limited research evaluating the need for this change. Opioid overdose resuscitation has been affected by the emergence of xylazine, a common adulterant in fentanyl. Xylazine is not reversed by naloxone and has prolonged sedating effects that mimic opioid overdose. Other central nervous system (CNS) depressants (e.g., benzodiazepines, opioids, muscle relaxants, hypnotics) often complicate opioid overdose. The impact of xylazine and other CNS depressants on bystander- and pre-hospital naloxone dosing is unknown.
Hypothesis or Research Question: In this pilot, observational study, we aimed to estimate how much naloxone was needed for suspected opioid overdose patients in the presence of fentanyl, xylazine, and other CNS depressants.
Methods: Adults (≥18 years-old) with suspected opioid overdose occurring in the community and presenting to two, academic, urban, US emergency departments (EDs) were recruited between October 2022 and January 2025. Naloxone parenteral equivalents administered prior to patient arrival in the ED was compared to toxicological analyses of their blood obtained at ED presentation. Quantitative and qualitative analyses for the presence of fentanyl, xylazine, and CNS depressants (benzodiazepines, opioids, muscle relaxants, hypnotics) were performed using a Waters TQS liquid chromatograph tandem mass spectrometer (LC-MS/MS). Naloxone administered was compared to detected fentanyl and xylazine blood concentrations, as well as the presence or absence of CNS depressants.
Results: Of 106 participants, 64% were male; median age was 43 (IQR 33-55); 72% had detectable fentanyl, while

49% had xylazine and 44% CNS depressants. There were 50 (47%) participants with fentanyl and xylazine detected; among these participants, xylazine levels increased moderately as fentanyl levels increased (Spearman ρ=0.45, p<0.001). Mean naloxone parenteral equivalents administered was 1.98 mg (SD 1.1); the median was 2 mg (IQR 2-2). Mean naloxone parenteral equivalents administered were similar whether xylazine (3.64 vs. 3.26 mg; p<0.36) or CNS depressants (3.41 vs. 3.48; p<0.87) were present vs. absent. Participants with detected fentanyl concentration in the 4th quartile received more naloxone (4.67 mg) than those in the 3rd (3.22 mg), 2nd (3.25 mg) and 1st quartiles (2.74 mg) (p<0.01 all comparisons).
Conclusion: Naloxone utilization was generally greater with higher exposures to fentanyl. Xylazine did not confer a greater need for naloxone. A larger, multi-site investigation is needed to confirm these findings.

123. Dexmedetomidine for the Treatment of Toxicologic Conditions in the Emergency Department: A Dual-Center Retrospective Cohort Study

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Background: Dexmedetomidine (DEX) is a sympatholytic which has been used in the treatment of toxicologic conditions. Previous research on DEX in toxicology is limited by missing data, narrow patient selection, and small sample sizes.
Hypothesis or Research Question: How is DEX used in the treatment of patients with toxicologic conditions in the emergency department (ED), and what adverse events are associated with its use?
Methods: This is a retrospective cohort study of patients with toxicologic conditions treated with DEX in the EDs of Barnes-Jewish Hospital (BJH) and Hennepin County Medical Center (HCMC). The study period was from the launch of the current electronic medical record (EMR) system (HCMC January 2007, BJH June 2018) through November 2024. We included patients who had a toxicologic condition and received intravenous DEX in the ED. Patients were identified via EMR query for DEX administration in the ED. Toxicologists screened charts and adjudicated the presence and nature of toxicologic condition(s). Trained abstractors then manually abstracted data from the EMR using a standardized electronic instrument. Data were summarized using descriptive statistics. Multilevel logistic regression was used to explore factors associated with intubation post-DEX.
Results: We screened 1,081 patients and included 321 (HCMC 108, BJH 213). The most common toxicologic conditions were acute poisoning/intoxication (n=231, 72%) and withdrawal (n=99, 31%). The top xenobiotics were ethanol (n=143, 45%), opioids (n=114, 36%), and sympathomimetics (n=120, 38%). The median time-weighted average DEX infusion rate was 0.3 mcg/kg/h (IQR 0.2-0.6), with a median infusion duration of 2.5h (IQR 1.4-4.2). Other sedating drugs were administered in all cases, most commonly benzodiazepines (n=223, 70%) and antipsychotics (n=144, 45%). Post-DEX, the median heart rate (HR) was 76bpm (IQR 65-93) and the median mean arterial pressure (MAP) was 76mmHg (IQR 64-90). One hundred twenty patients (62%) were intubated pre-DEX; of the remaining 200, 43 were intubated in the ED post-DEX (22%, 95% CI 16-28). On multi-level regression, only 3.3% of variation in intubation frequency was explained by inter-site differences; none of the factors tested (blood ethanol or co-administration of benzodiazepines, antipsychotics, or ketamine) was significantly associated with intubation.
Conclusion: DEX was used in combination with other sedatives to treat patients with toxicologic conditions in the ED, primarily intoxication and withdrawal from psychoactive substances. Post-DEX median HR and MAP were normal. There was a relatively high frequency of intubation following DEX administration, which was not associated with ethanol intoxication or the use of specific additional sedatives.

124. A Case of Tirzepatide-Associated Drug-Induced Liver Injury and Gallbladder Disease

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Background: Glucagon-like peptide-1 (GLP-1) agonists, a second-line agent for the treatment of type II diabetes mellitus, are being prescribed more frequently due to their emerging role in weight loss, and US poison centers have seen a nearly 1,500% increase in related calls since 2021. While many GLP-1 agonist exposures are associated with mild symptoms, serious adverse effects have been reported in clinical trials and in case reports.
Hypothesis or Research Question: Tirezepatide is a subcutaneous GLP-1 that can cause self-remitting drug-induced

liver injury (DILI) and gallbladder disease by an unknown mechanism.

Methods: This is a single patient chart review. A 34-year-old female with a history of polycystic ovarian syndrome presented for evaluation of progressively worsening epigastric abdominal pain, nausea, and vomiting. Laboratory analysis was notable for leukocytosis of 13,200 cells/mL. Initial AST/ALT levels were elevated to 256/192 U/L, respectively, which peaked at 1556/1505 U/L over the course of one day. Alkaline phosphatase and lipase was unremarkable. The patient denied any alcohol use but noted that she had been using internet-purchased subcutaneous injections of NAD+ once or twice weekly for the past two months and tirzepatide 20mg once weekly for the past seven months. Her husband also used the NAD+ injections and was asymptomatic. The patient was treated with N-aceylcysteine for its hepatic protective effect. A thorough evaluation revealed no clear cause for her transaminase elevations. Imaging showed cholelithiasis without complication. Despite this, she underwent a laparoscopic cholecystectomy due to persistent abdominal pain.

Results: Surgical pathology showed chronic cholelithiasis, but no clear cause for her transaminase elevations. The patient's pain ultimately resolved, her transaminase improved to 43/413 U/L, and she was discharged on hospital day 4 with instructions to abstain from tirzepatide and NAD+. A R value of 5.8 was calculated from the patient's laboratory values, suggesting a hepatocellular picture. A Roussel Uclaf Causality Assessment Method (RUCAM) score of 6 suggests a "probable" association between tirzepatide and the observed clinical picture of DILI. This picture of hepatocellular toxicity is consistent with another case found in the peer-reviewed literature.

Conclusion: Clinical trials have shown an increase in acute gallbladder disease and transaminase elevations without significant hepatotoxicity in Tirzepatide-treated patients compared to those in placebo groups. Given the rise in GLP-1 use, medical toxicologists should be aware of possible adverse reactions that can be seen with these medications.

125. Sodium Monofluoroacetate Poisoning in Veracruz, Mexico

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Background: Sodium monofluoroacetate (SMFA), also known as compound 1080, is a rodenticide whose toxicity is due to the irreversible inhibition of the citric acid cycle. Although its sale is prohibited in Mexico, it continues to be used due to its effectiveness and low cost; the objective of this study was to assess the frequency and clinical characteristics associated with SMFA poisoning in two hospitals in Veracruz, Mexico.

Methods: We conducted a two-center retrospective, observational and descriptive study from January 2023 to October 2025. Records of patients admitted to the emergency department were reviewed to identify cases of sodium monofluoroacetate poisoning. The main demographic and clinical features were analyzed using SPSS version 25.

Results: Of patients admitted to the emergency department, 9 met inclusion criteria. All of them were intentional and 55.5% were female (n=5). The mean age was 32 years old (range: 23-56 years). The same product (10 ml dropper bottle) was identified in 55.5% of the cases, as well as ingested dose, which ranged from 2 to 10 ml (mean 6.4 ml). The time between exposure and hospital admission ranged from 1 to 10 hours (mean 4 hours). The most frequently observed clinical manifestations were dyspnea in 66% (n=6), loss of consciousness in 44% (n=4), diaphoresis in 44% (n=4) and hypotension in 33% (n=3). Gastric decontamination measures were applied in 88.8% of patients (n=8), and 77.7% received ethanol (n=7). The hospital stay ranged from 1 to 17 days (mean 6 days), with a favorable outcome in 55.5% of the patients, and a mortality rate of 44.4%.

Conclusion: In Mexico, SMFA continues to be sold despite its prohibition, which raises the possibility that it could be used as a method of suicide. Even though it showed a high mortality rate, the time of arrival at the hospital and the timely administration of ethanol appeared to play an important role in survival. It is essential that the emergency physician be familiar with SMFA poisoning so that a timely diagnosis can be made and their therapeutic interventions can improve patient survival.

126. Cut It Out! Rethinking Elevated Compartment Pressures Due to Rattlesnake Envenomation

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Background: Rattlesnake envenomation can cause signs and symptoms concerning for compartment syndrome. Historically, fasciotomy was performed for elevated compartment pressures in pit viper envenomations, though more recent evidence demonstrates limited benefit and potential harm compared with antivenom therapy alone. Expert

consensus now emphasizes medical management with antivenom over surgical management.

We present a pediatric case of rattlesnake envenomation with elevated compartment pressures successfully treated with antivenom alone.

Methods: This is a case report of a previously healthy 5-year-old girl who presented obtunded three hours after complaining of right leg pain after walking outside. On arrival, she was tachycardic (160 bpm), normotensive (95/67 mmHg), and hypoxic (SpO_2 88% RA) with shallow respirations. She was emergently intubated on arrival. Examination revealed myokymia and severe right leg edema and ecchymoses. Laboratory work was notable for thrombocytopenia (50,000 cells/uL) and hypofibrinogenemia (160 mg/dL). Two puncture wounds on the posterior calf were clinically consistent with rattlesnake envenomation. Lower extremity X-rays did not demonstrate a fracture.

Initial superficial posterior compartment pressure measured 44 mmHg with a diastolic pressure of 60 mmHg (delta pressure 16 mmHg), meeting criteria for compartment syndrome. Orthopedic surgery was consulted and recommended emergent fasciotomy. Medical toxicology was also consulted and recommended against fasciotomy in favor of treatment with antivenom.

Results: Fasciotomy was deferred in favor of medical management with crotalidae immune $F(ab')_2$ antivenom and supportive care. She received 10 vials of antivenom initially. She developed hypotension, which persisted after fluids and required epinephrine infusion. At this time, she received an additional 10 vials of antivenom.

At 12 hours post-envenomation, doppler ultrasound confirmed triphasic distal arterial flow; however, repeat compartment pressures remained elevated prompting a third 10-vial dose. By 20 hours, compartment pressures had decreased and coagulopathy resolved. She was extubated and vasopressors were discontinued by hospital day 2. She was discharged on day 7 ambulatory with a walker. Her only immediate complication was the development of hemorrhagic bullae on her right leg.

At follow-up on post-envenomation day 25, she had near-complete recovery with only a mild limp and demonstrated no tissue loss.

Conclusion: This case illustrates that even in severe pediatric rattlesnake envenomation with respiratory failure, shock, and elevated compartment pressures, aggressive antivenom therapy alone can reverse systemic and local effects without surgery. We propose the term *Venom-Induced Pressure Elevation Reversible Syndrome (VIPERS)* to emphasize the reversibility of this toxin-mediated process and differentiate it from traumatic compartment syndrome.

127. Rise of the Platelets: An Accidental Ingestion of Avatrombopag in a Pediatric Patient

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Background: Avatrombopag is an oral direct thrombopoietin receptor agonist approved for the treatment of adult thrombocytopenia in the US in 2018. Although avatrombopag is also used to treat a variety of thrombocytopenic conditions in children, its safety in this population is not well studied. Moreover, to our knowledge, there have not been any reports of avatrombopag overdose in either adult or pediatric populations.

Hypothesis or Research Question: Accidental avatrombopag overdose will be well tolerated in a healthy pediatric patient.

Methods: This is a single patient case report. A two-year-old male with no past medical history accidentally ingested five tablets of 20 mg avatrombopag that belonged to a family member. The patient was immediately taken to a community emergency department, where our poison center was consulted. The patient had normal vital signs and an unremarkable physical exam. Activated charcoal was given and baseline labs were drawn. The patient was discharged with instructions to repeat platelet count every other day for at least three draws. Subsequently, the patient was brought to a specialized pediatric emergency department nine days later with concern about rising platelet levels. The patient was asymptomatic and again found to have a normal exam. Both our toxicology service and pediatric hematology were consulted for management recommendations. Ultimately, we jointly recommended continued monitoring of the platelet count in the outpatient setting, with a platelet count of 1000 TH/μL (normal: 140-440 TH/μL) being an indication for further evaluation in hematology clinic. Our poison center was not contacted about the case again and he did not present to hematology clinic (as had been instructed if the platelet count were to increase over 1000 TH/μL). He was ultimately lost to follow-up.

Results: At the initial time of presentation, the patient had a normal platelet count of 200 TH/μL. On day three, the platelet count increased to 400 TH/μL, then to 460 TH/μL on day five. On day seven, the platelet count peaked at 674 TH/μL. Although acetylsalicylic acid was considered by both our toxicology service and pediatric hematology, no specific therapy was recommended or given. The patient was monitored closely in the outpatient setting and to our knowledge did not develop complications from his secondary thrombocytosis.

Conclusion: This is the first report of an accidental avatrombopag overdose. Our patient developed secondary thrombocytosis that was left untreated. No adverse complications occurred.

128. Comparison of Tachycardia and Seizures in Venlafaxine Overdose

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Background: Venlafaxine is a serotonin and norepinephrine reuptake inhibitor antidepressant available in immediate-release and sustained-release formulations. Venlafaxine toxicity is associated with tachycardia in a dose-dependent manner. It is also associated with seizures, with delayed seizures (up to 21 hours post ingestion) being of clinical concern. Tachycardia has been observed to serve as a useful clinical predictor of seizure occurrence in cases of bupropion overdose, an antidepressant also associated with delayed-onset seizures. We hypothesize that venlafaxine may have a similar relationship between tachycardia and seizure.
Hypothesis or Research Question: Is there a correlation between tachycardia and seizures in acute venlafaxine ingestions?
Methods: This is a retrospective chart review cohort study of all patients who presented with acute venlafaxine ingestion and were evaluated at our academic health system, including a children's hospital, from January 1, 2013, to October 1, 2024. Patients who did not ingest a supratherapeutic dose of venlafaxine or had missing data were excluded. A preplanned subgroup analysis was performed for patients who co-ingested or were prescribed xenobiotics known to lower heart rate. Data extracted on chart review included patient demographics, reason for ingestion, vital signs, EKG intervals, clinical outcomes, & hospital length of stay. Primary outcome was occurrence of seizures. Descriptive statistics were applied to clinical outcomes of interest. Predictive values and likelihood ratios were calculated for clinical data and seizure occurrence.
Results: A total of 160 cases met inclusion criteria and were included for final analysis. Of these cases, 90.62% (n = 145) were female. Median age was 17.3 years (IQR 14.37 – 28.15). There were 23 cases with a reported co-ingestion of a heart rate lowering medication. Seizures occurred at a rate of 7.5% (n = 12). The longest time to seizure occurrence was 15.5 hours. All patients who developed seizures, except for one patient who co-ingested propranolol, were tachycardic within eight hours of presentation. Highest heart rate within eight hours without heart-rate lowering co-ingestants is correlated with development of seizure (AUC 0.84, 95% CI 0.73-0.94), with a threshold of 119bpm maximizing Youden's J (0.6, 95% CI 0.53-0.81), yielding 100% sensitivity and 60% specificity.
Conclusion: Seizures occurred infrequently in this cohort of venlafaxine ingestions. When a medication that lowers heart rate is not present, the absence of tachycardia within eight hours of presentation may be useful for identifying patients at low risk of developing delayed seizures. A low sample size, retrospective nature, and low rate of seizures observed limit the Conclusion: s of this study.

129. Withdrawn

130. Reversal of Coagulopathy in Children With Acute Liver Failure From Acetaminophen

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Background: Acetaminophen is one of the most common xenobiotics to cause acute liver failure in children, characterized by a significant coagulopathy and elevated transaminases.
Hypothesis or Research Question: We hypothesize that patients who underwent reversal of their coagulopathy were sicker, but that reversal would not be associated with improved outcomes.
Methods: This study is a retrospective review of pediatric patients (< 18 years old) admitted to one of six tertiary care medical centers with a diagnosis of either liver failure or acetaminophen toxicity. All records were reviewed to select patients with liver injury (AST or ALT > 1000 IU/L) due to acetaminophen toxicity. Patients with liver failure from other etiologies, as well as patients with acetaminophen toxicity who did not exhibit liver injury, were excluded. The threshold to reverse coagulation parameters is variable, as reversal can be associated with increased thrombotic complications and impair the ability to follow improvements in synthetic function, and was performed at the treating physician's discretion. We defined reversal as receiving fresh frozen plasma, factor 7 replacement, or pooled complex concentrates. Vitamin k administration was not considered a reversal agent.

Results: A total of 202 patients were identified. Thirty patients had their coagulopathy reversed. The median maximal prothrombin time (PT) among those who were reversed was 42.8 seconds compared with 21.4 seconds in patients without reversal, median difference of 21.4 (95% CI 14.2-32.9). Seven patients met the composite endpoint of death or transplant, six of which had their coagulopathy reversed (OR 42.75, 95% CI 3.9-472.2). Thrombotic complications occurred in five patients, four of which had coagulopathy reversal (p=0.002). Quantile median regression indicates that reversal of coagulation is associated with a median length of stay (LOS) increase of 4 days (95% CI 1.6-6.4) (p=0.001).

Conclusion: Potentially unnecessary reversal resulting in over-correction of a patient's coagulopathy was associated with increased length of stay and possibly increased thrombotic complications. However because the individuals who were reversed were sicker than those who were not, and given the lack of randomization, we could not establish causality.

131. Response to High-Dose Insulin Euglycemia Therapy in Calcium Channel Blocker Poisoning Stratified by Echocardiogram Phenotypes

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Background: High-dose insulin euglycemia therapy (HIET) is theorized to benefit cardiogenic shock but worsen distributive shock in calcium channel blocker poisoning, and echocardiograms may help differentiate underlying shock physiology.

Hypothesis or Research Question: Response to HIET in calcium channel blocker poisoning varies by echocardiogram phenotype: normal, reduced, or hyperdynamic left ventricular ejection fraction (EF).

Methods: This is a retrospective chart review-based cohort study of cases 1/1/2020—12/31/2024 from one regional poison center. Patients included were >18-years-old with suspected calcium channel blocker poisoning receiving vasopressors or HIET, with an echocardiogram utilized to guide decision-making on HIET. Patients with co-ingested beta blockers or missing data were excluded. The primary outcome was in-hospital mortality. Secondary outcomes were: 1) days alive and free (DAF) of hemodynamic support (vasopressors, inotropes, or mechanical support) within seven days post-case start and 2) need for additional hemodynamic support post-HIET. Echocardiogram phenotypes were categorized by qualitative description or, when quantitative EF was available, by previously validated ranges: normal 50-70%, reduced <50%, and hyperdynamic >70%. DAF in normal EF patients receiving vs. not receiving HIET were compared via Wilcoxon rank sum test.

Results: Twenty-nine patients (76% dihydropyridine poisoning) were analyzed: 22 (76%) normal EF, two (7%) reduced, and five (17%) hyperdynamic. The median age was 62 (56–69) years, and 19 (66%) patients were female. Six (21%) died. Fourteen (48%) received HIET, and the median maximal HIET infusion rate was 2 (1—4) units/kg/hour. Median pre-HIET number of vasopressors was two (IQR 2—3).

In normal EF patients, mortality was 18% (2/11) with HIET versus 9% (1/11) without. Median DAF was 0 (0—2.2) days in normal EF patients receiving HIET vs. 5.7 (4.3—5.9) in those not receiving HIET (p<0.001). Seven (64%) patients required increased support post-HIET. Both reduced EF patients survived (one with HIET, one without). Median DAF was 5.5 (2.5—5.7) days. The patient receiving HIET did not require increased vasopressors post-HIET. In hyperdynamic EF patients, mortality was 100% (2/2) with HIET vs. 33% (1/3) without. Both patients receiving HIET required increased vasopressors post-HIET. Median DAF in this group was 0 (0.0—5.0) days.

Conclusion: In this small retrospective study, outcomes and response to HIET varied by echocardiogram phenotype. While unable to prove harm from HIET, these results suggest patients with hyperdynamic EF, and most patients with normal EF, did not clearly benefit from HIET. Limitations include varying timing of echocardiograms and amount of hemodynamic support during echocardiograms.

132. Use of Hydroxocobalamin and Methylene Blue in the Management of Shock: A Retrospective Review of a Single Poison Center

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Background: Vasoplegic shock, characterized by profound hypotension and low systemic vascular resistance refractory to catecholamines, remains a critical and often

fatal complication in toxicologic exposures. Therapies such as hydroxocobalamin and methylene blue are occasionally recommended as rescue therapies for refractory vasoplegia. Despite their use, there is limited data describing the utilization and outcomes of these agents in toxic exposures.

Hypothesis or Research Question: When are hydroxocobalamin or methylene blue recommended by poison centers for the management of shock in toxic exposures, and what are the associated outcomes?

Methods: A single poison center, retrospective chart review was conducted of all consultations between January 2021 and October 2025 in which hydroxocobalamin or methylene blue were recommended for the management of shock. While vasoplegic shock is not a discrete selection within the database, cases were included after selecting for hypotension, followed by a narrative review confirming refractory hypotension related to a toxic exposure. Variables included demographics, exposure, therapies administered, and patient outcomes. Descriptive statistics are reported.

Results: Forty-seven cases were identified where methylene blue or hydroxocobalamin were recommended for the management of refractory shock (hydroxocobalamin: 17; methylene blue: 42). Case distribution increased over time, with one case identified in 2021, four cases in 2022, 11 cases in 2023, 10 cases in 2024, and 21 cases in the first 10 months of 2025. Sixty-four percent of patients were male (30/47), and ages ranged from 18 to 88 years. Intentional exposures accounted for 37 cases (79%). Calcium channel blockers were the most frequently associated exposure (30/47, 64%), with amlodipine representing the majority (21/30, 70%). The remaining exposures included metformin, beta-blockers, and bupropion. Of the 42 cases where methylene blue was recommended, it was administered in 36 cases (86%). Hydroxocobalamin was used in only seven of the 17 cases (41%) for which it was recommended. In all cases, these agents were recommended only after initiation of multiple vasopressors. Overall mortality was high: 24 of 47 patients (51%) expired. In 19 of those 24 cases (79%), the recommended agent had been administered.

Conclusion: Hydroxocobalamin and methylene blue were infrequently but increasingly recommended as adjunctive therapies for presumed toxin-induced shock, most commonly in severe calcium channel blocker overdoses. Despite their use, mortality remained high, reflecting the severity of underlying toxicity and the limited evidence base guiding these interventions. These findings underscore the need for further research to clarify optimal patient selection, timing, and comparative effectiveness of hydroxocobalamin and methylene blue in the treatment of vasoplegic shock.

133. Cannabis Product Landscape Around a U.S. Public University: August 2025

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Background: Cannabis stores face additional federal penalties within 1000 feet of university campuses as they may entice underage consumers (21 U.S. Code §860). We seek to compare the packaging language of cannabis products sold within this distance to products purchased in previous years.

Methods: This convenience sample product survey considers cannabis products purchased by researchers from the front counters of four shops within 1000 feet of a university in August 2025. Cannabis product packaging was manually evaluated based on naming and content labeling legal violations, consumer warnings, dosing information, youth appeal, and participation in six marketing discourses: “natural”, “traditional”, “social”, “cool”, “risky”, and “legal”. Findings were then compared to products purchased in previous years (2020-2024). Batch one refers to products purchased in previous years (n=55). Batch two refers to products purchased in August 2025 (n=12).

Results: Edibles (n=4), vapes (n=4), pre-rolls (n=2), and cannabis flower (n=2) were the formulations present in batch two. On average, 1.5 state-level product naming violations were present in batch two while there were 2.5 violations per product on average in batch one. Batch two averaged 7.6 consumer warnings while batch one averaged 3.7 consumer warnings. Every product (100%) provided a QR code for cannabinoid laboratory results in batch two, while 60% of products in batch one provided one. Serving size was printed in 33% of the products in batch two and 18% in batch one. Half of batch two products (50%) and 29% of batch one products contained use instructions. Youth appeals remained relatively constant (1.4 in batch one and 1.0 in batch two). Batch two used appeals to “natural” in 42% of products and “legal” sensibilities in 100% of the products. Batch one appealed to these in 24% and 64% of products, respectively. Batch two contained fewer health claims (8.0% of products) than batch one (29%).

Conclusion: Youth appeal indicates a young target demographic, matching nearby university students. Vendors are providing more warnings and use information to consumers than previously. This trend, along more mentions of product content legality, and fewer product name violations and health claims, suggests that sellers want to legitimize their products in the eyes of consumers and the law. These products undergo no standardized quality assurance,

endangering consumers who may develop a false sense of security from the seemingly standard packaging.

134. Prophylactic Lidocaine Infusion After Delphinium Denudatum (Jadwar) Ingestion in a Resource-Limited Emergency Department

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Background: Delphinium Denudatum (Jadwar) is a traditional South Asian herbal root that contains aconitine-like diterpenoid alkaloids, which cause persistent sodium-channel activation, early afterdepolarizations, and malignant ventricular dysrhythmias. Human poisoning data are sparse from low- and middle-income countries where such remedies are widely used and advanced cardiotoxicant care is limited.
Hypothesis or Research Question: In significant Delphinium Denudatum ingestion, a mechanism-guided approach using lidocaine—an intravenous class Ib sodium-channel blocker—combined with structured monitoring can safely suppress ventricular dysrhythmias in a resource-limited emergency department.
Methods: This is a single-patient case report from a tertiary-care emergency department in Pakistan with constrained access to cardiothoracic surgery, extracorporeal support, and specialty toxicology consultation. A 28 year old male with diabetes mellitus ingested a finger-breadth piece of Delphinium Denudatum root and presented about one hour later with flushing, sweating, and palpitations but no syncope, chest pain, or neurologic deficits. Initial evaluation included vital signs, basic laboratories, and 12-lead electrocardiogram (ECG). The patient was placed on continuous telemetry, received lidocaine 100 mg intravenous bolus followed by 4 mg/min infusion for twenty-four hours, and was observed for forty-eight hours with electrolyte optimization and supportive care.
Results: On arrival, he was hemodynamically stable with normal examination and laboratory values except for mildly elevated C-reactive protein. Initial ECG demonstrated atrial fibrillation with frequent ventricular ectopy and non-sustained polymorphic ventricular tachycardia with ST-segment changes consistent with sodium-channel–mediated toxicity. After lidocaine initiation, ventricular ectopy and polymorphic ventricular tachycardia resolved within approximately six hours without hypotension, bradycardia, or progressive conduction block. Following discontinuation of the infusion at twenty-four hours, an additional twenty-four hours of telemetry revealed no recurrent dysrhythmias. Repeat ECG at forty-eight hours showed normal sinus rhythm with improved ST segments and corrected QT interval, and the patient was discharged asymptomatic with counselling regarding herbal medicine toxicity.
Conclusion: Accidental Delphinium Denudatum ingestions are under-recognized and can result in an aconite-like poisoning in regions where traditional medicines are common and critical-care resources are scarce. This case illustrates how understanding delphinium toxicity can guide the selection of lidocaine as a targeted, affordable intervention, and emphasizes the importance of early recognition, electrolyte optimization, and 24-48 hour cardiac monitoring to prevent sudden ventricular arrhythmias and enable safe discharge from resource-limited emergency departments.

135. Red, White, and Blue – Gout, Anemia, and Methylene Blue

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Background: Pegloticase is a recombinant uricase used in the treatment of refractory gout. Although contraindicated in patients with G6PD deficiency due to a risk of hemolytic anemia and methemoglobinemia, these adverse effects have rarely been reported.
Methods: This is a single-patient case report of a 60-year-old man with gout who presented to the emergency department for shortness of breath and dark urine. He had previously received two infusions of IV pegloticase and reported to have negative G6PD testing before initiation of therapy. His last infusion was four days prior to presentation. On exam, he was hypoxic and notably jaundiced.
Results: Abnormal labs included a white blood cell count of 24.2 x 10^3/μL, hemoglobin 6.8 g/dL, platelets 240 x 10^3/μL, creatinine 1.7 mg/dL, fibrinogen 69 mg/dL, LDH 2,200 U/L, total bilirubin 5.8 mg/dL, indirect bilirubin 5.0 mg/dL, methemoglobin 11.8%, and INR 4.02. Coombs testing was negative. CT imaging demonstrated segmental and subsegmental pulmonary emboli. Given worsening hemolytic anemia, we recommended against methylene blue, and the patient received IV ascorbic acid. Hematology recommended therapeutic plasma exchange (TPE) with suspicion of underlying G6PD deficiency. He underwent three sessions of TPE, received nine units of pRBCs, multiple units of platelets, cryoprecipitate, and FFP. Repeat methemoglobin was 0.6%. He was discharged on hospital day nine with a final hemoglobin of 8.1 x 10^3/μL.
Conclusion: We describe a patient with hemolytic anemia and methemoglobinemia after pegloticase infusion. Few cases describe these adverse developments, all in patients with known or later diagnosed G6PD deficiency. There is

no established treatment for these complications, but TPE may be beneficial.

136. Sulfhemoglobinemia From Chronic Phenazopyridine Exposure

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Background: Sulfhemoglobinemia is a rare dyshemoglobinemia caused by irreversible incorporation of sulfur into the porphyrin ring of heme, preventing oxygen binding. Oxidant drugs associated with methemoglobinemia and hemolysis may also induce sulfhemoglobin formation. We report a case of sulfhemoglobinemia following chronic phenazopyridine exposure.
Hypothesis or Research Question: Phenazopyridine induced oxidant stress can cause sulfhemoglobinemia and hemolysis without methemoglobinemia.
Methods: This is a single-patient chart review of a 95-year-old woman with a medical history significant for hypertension, chronic kidney disease, colonic adenocarcinoma, and renal cell carcinoma status post hemicolectomy and nephrectomy.
Results: This woman presented from a skilled nursing facility with apparent refractory hypoxemia. On presentation, pulse oximetry demonstrated an oxygen saturation of 74% despite administration of 15 L/min supplemental oxygen via non-rebreather mask. She appeared mildly cyanotic with dark nail beds and pale with dark arterial blood but remarkably asymptomatic. Her medication administration record review was notable for phenazopyridine 100 mg three times daily prescribed 20 days prior for dysuria which she was still receiving. Medical toxicology was consulted for suspected methemoglobinemia. Arterial blood gas analysis revealed a PaO_2 > 488 mmHg (normal 80-100 mmHg) hemoglobin 8.4 g/dL, negative carboxyhemoglobin, and methemoglobin 2.6%. The marked saturation gap was suggestive of a dyshemoglobinemia, though not accounted for by the methemoglobin level. Computed tomography angiography for pulmonary embolism was negative, and no alternative etiology for low pulse oximetry reading was identified. A presumptive diagnosis of sulfhemoglobinemia was made. Phenazopyridine was discontinued, and methylene blue therapy was deferred. The hospital course was complicated by anemia with hemolysis (hemoglobin nadir 6.4 g/dL) requiring transfusion of two units of packed red blood cells. Serial arterial blood gases off supplemental oxygen demonstrated stable oxygen saturations between 85–89%. Following transfusion her anemia stabilized and she was discharged back to her facility. Confirmatory testing subsequently demonstrated an elevated sulfhemoglobin level of 11% (reference < 2%). At her two-month follow-up her anemia resolved.
Conclusion: Sulfhemoglobinemia poses a diagnostic challenge because rapid testing is not readily available. Decreased pulse oximetry saturations occurs at lower concentrations than in other dyshemoglobinemias. Patients can appear cyanotic without symptoms of hypoxia. Although rare, sulfhemoglobinemia should be considered in patients presenting with hypoxemia and an elevated saturation gap following exposure to oxidant agents. Medical providers should be aware that prolonged use of phenazopyridine may cause these dyshemoglobinemias and hemolysis.

137. Unintended Consequences: Negative Health Effects of Herbal Medicine Ingredients in Regulated Cannabis Products

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Background: Cannabis use is decriminalized or legal in 47 US states, the District of Columbia, and 3 US territories, but the resulting patchwork of state and territorial laws leads to vast regulatory differences in areas such as labeling, retail availability, and allowed formulations, which has created public health safety concerns. Colorado collects reports of adverse health events related to retail cannabis products and, in 2022, received notification of hepatotoxicity after consumption of a cannabis product purchased through a licensed dispensary.
Hypothesis or Research Question: There may be unintended consequences of including herbal medicine ingredients in cannabis product formulations in the regulated cannabis market.
Methods: This is a case series of cannabis consumers reporting hepatotoxicity after consuming a cannabis product containing the herbal additive, Corydalis or Stephania. Notification of the first case was received in March 2022 and by May 2024 fifty-two reports of potential cases had been collected. Data were obtained via self-report and clinical interviews with the consumer, their health care provider, or a dispensary worker. The Colorado Department of Public Health and Environment (CDPHE) attempted contact with all reports and coordinated with other agencies to gather

comprehensive information on the product, ingredients, and clinical health impacts.

Results: All successfully contacted cases reported similar histories of regular consumption of a tablet sold through cannabis dispensaries and subsequent development of symptoms reminiscent of liver injury, prompting medical attention to be sought. Many received extensive medical testing in an attempt to determine the cause, including ultrasounds, MRI and CT scans, blood tests, and liver biopsy. Blood tests revealed a pattern of markedly elevated liver enzymes, specifically alanine transaminase (ALT) and aspartate transaminase (AST). Symptoms resolved following product cessation. The product, marketed for sleep aid, initially claimed to contain 100mg of Corydalis Rhizome extract per serving. Product analysis determined the inclusion of Tetrahydropalmatine (THP), a compound derived from Corydalis or Stephania extract, depending on the date manufactured. Scientific literature on THP, though minimal, implies a potential for hepatotoxicity.

Conclusion: The inclusion of THP, derived from Corydalis or Stephania, in a regulated cannabis product formulation led to multiple cases of hepatotoxicity among previously healthy individuals seeking sleep aid from products with natural ingredients. This liver injury was determined to be caused by the herbal additive, specifically THP, not a cannabinoid or interaction with cannabinoids, highlighting the urgent need for caution and rigorous study when incorporating any herbal medicine ingredients into newly regulated cannabis formulations.

138. Edible Cannabis Product Information Captured Within Patient Poison Center Records

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Background: Poison centers are encountering increasing numbers of calls pertaining to cannabis exposed patients following the decriminalization of marijuana in numerous states.

Hypothesis or Research Question: This study was performed to determine if the exact products involved with edible cannabis exposure were being captured in the records and to delineate those products.

Methods: This retrospective patient records review surveys Toxicall records from 1 January 2024 to 31 December 2024. Edible cannabis product human exposure records from calls to a single poison center (n=157) were manually evaluated for demographics, therapy outcome, and product information. Documented data were analyzed, focusing on 1) missing product information, and 2) differences in pediatrics aged 0-9, aged 10-20 years, and legal-aged consumer (≥21 years) exposures & product information provided.

Results: Demographics, therapy outcome, and amount of product ingested were always requested per documentation. Minimal product vendor (n=6), cannabis use history (n=11), or labeled serving size (n=12) information was requested. Twelve cannabinoids and 16 POISINDEX codes were recorded; unknown cannabinoids characterize 23% of cases. Of the products described (n=119), most (n=95, 80%) were gummies. The next most common formulations were non-gummy lookalike candy (products with significant naming / font / color similarities to a branded non-cannabinoid product, n=8), chocolate candy (n=5), and brownies (n=3). Product type was most described in pediatrics aged 0-9 (84.3%), followed by legal-aged consumers (71.2%) and ages 10-20 (69.2%). Six of the eight lookalike products were reported in pediatric cases. One-third of cases (n=52) recorded specific product names. In 73% of cases where a product name was requested, the product name was provided. Information exchanged in exposures involving adult patients were slightly more likely to include a product name in comparison to exposures involving pediatric patients (25.7% versus 22.4%). Pediatrics aged 0-9 constituted half (n=3) of major-severity exposures. Average time from call to end-of-care (EOC) was highest for major-severity young pediatrics (17.3 hours, compared to 15.9 for ages 10-20 & 7.5 for legal-aged consumers with major-severity exposures). All ages' moderate-severity exposures averaged a call-to-EOC time between nine and ten hours.

Conclusion: When specifically requested, most callers provided the ingested product name, but this information was not requested in two-thirds of cases. Gummy formulations were particularly common. Young pediatric patients are disproportionately reflected in edible cannabis product exposures. Improved poison center data collection pertaining to specific cannabis products would enable better tracking of adverse outcomes with those products as a critical part of public health surveillance.

139. Non-Antidotal Methylene Blue Exposures Reported to a Regional Poison Center, 2015-2025

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Background: Methylene blue (tetramethylthionine chloride) is an antidote for acquired methemoglobinemia that is also used to treat vasoplegic shock. It is an oxidizing agent and a monoamine oxidase inhibitor that, at high doses, has been associated with methemoglobinemia and serotonergic effects. Recently, methylene blue has been promoted

on social media platforms for non-specific health benefits. However, the public may be unaware of the potential for adverse effects and drug-drug interactions.

Hypothesis or Research Question: Research Question: Are non-antidotal exposures to methylene blue increasing and are they associated with toxic effects?

Methods: This is a retrospective review of methylene blue (MB) exposures reported to a regional poison center from January 1, 2015, through October 16, 2025. Case narratives over this timeframe were searched for the term "methylene blue" in patients > 18 years old. Cases where MB was recommended by the poison center or administered therapeutically by health care facility providers were excluded. We excluded cases of miscoding (where no actual MB exposure was documented) and cases that were managed at home due to lack of objective data. De-identified case narratives were then reviewed for circumstances of exposure, clinical effects, treatments, and medical outcomes.

Results: The initial search yielded 13 cases. Three were excluded (2 managed at home, 1 miscode), leaving 10 included for analysis. There were no exposures from 2015-2020, 1 in 2021, 1 in 2022, 2 in 2023, 1 in 2024, and 5 in 2025 (January through mid-October). The 10 patients included 5 males and 5 females with a median age of 35.5 years (range 28-75). Patients reported ingesting MB for a variety of reasons, including to treat mental health conditions, COVID-19, and Lyme disease, or to improve brain function. None involved self-harm attempts. In one 65-year-old male taking MB to treat mood disorder, providers were concerned about serotonin toxicity because he had been on citalopram and became delirious after starting MB (though without documented clonus or hyperreflexia). That patient was admitted to a critical care unit, improved with supportive care, and was transferred to behavioral health. None of the other patients had medical outcomes more serious than minor effect.

Conclusion: While our study is limited by small sample size, non-antidotal use of methylene blue among the public appears to be increasing. Although all patients had favorable outcomes, there is potential for systemic toxicity with unsupervised self-administration of this agent.

140. Acute Drug-Induced Pancreatitis Following Acetaminophen Overdose With Rechallenge of Other Medications

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Background: Drug-induced pancreatitis is a rare phenomenon, which is poorly understood. While acetaminophen's potential for hepatotoxicity is well known, cases of pancreatitis associated with acetaminophen use are extremely rarely reported and almost always without medication rechallenge.

Hypothesis or Research Question: Acetaminophen may be associated with acute pancreatitis in the absence of other etiologies.

Methods: This is a single patient case report. A 14-year-old girl with only past medical history of anxiety, depression and gastroesophageal reflux presented to a community emergency department with nausea and vomiting. She subsequently admitted to ingesting approximately 15 grams of acetaminophen in a suicide attempt the night prior to presentation.

Results: Serum acetaminophen concentration obtained approximately 14 hours after ingestion was 128.1 μg/mL (10-20 μg/mL), with serum aspartate aminotransferase 38 U/L (9-24 U/L), alanine aminotransferase 36 U/L (3-28 U/L), lactic acid 6.0 mmol/L (1.0-2.4 mmol/L), and international normalized ratio 1.4. Patient was started on N-acetylcysteine and admitted to an academic children's hospital. Patient required N-acetylcysteine infusion to be continued for a total of 69 hours before demonstrating laboratory evidence of hepatic recovery. She was discharged to inpatient psychiatric facility on hospital day 4. On hospital day 3, however, approximately 62 hours after ingestion, serum lipase was sent and resulted at 1884 U/L (9-82 U/L), approximately 23 times the upper limit of normal. Triglycerides were 46 mg/dL (0-89 mg/dL) and remainder of lipid testing was within reference range. Right upper quadrant ultrasound showed mild hepatomegaly, no gallbladder distention, no gallbladder wall thickening or pericholecystic fluid, no evidence of gallstones, and no evidence of biliary ductal abnormalities. Serum lipase obtained 6 months earlier was 27 U/L. Computed tomography imaging obtained 6 months prior showed mild constipation and no other abnormalities within the abdomen and pelvis. There was no family history of hepatopancreatobiliary pathology. Home medications included sertraline once daily, 0.1 mg clonidine nightly, 25 mg hydroxyzine twice daily, omeprazole 20 mg daily, and as needed ibuprofen. These were all restarted after lipase value was obtained and continued at discharge. All subsequent lipase values continued to decline, and all symptoms continuously improved prior to discharge. Pediatric gastroenterology consultation attributed acute pancreatitis to acetaminophen. Repeat labs obtained seven days after discharge showed further improvement of lipase and transaminases.

Conclusion: Drug-induced pancreatitis is a rare complication reported in association with acetaminophen use. Signs and symptoms of acetaminophen poisoning and acetaminophen-induced hepatic injury may mask acute pancreatitis.

141. A Novel Case of Microdosing-Induced Nutmeg Toxicity in the United Arab Emirates: Case Report and Literature Review

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Background: Nutmeg, also known as Myristica fragrans, has been reported to cause toxicity usually after single large ingestions. This is largely attributed to the myristicin and related phenylpropenes that it contains, which can cause anticholinergic-like and sympathomimetic neurotoxicity. Published reports predominantly describe intentional recreational use causing toxicity at levels involving ≥5-10g. To our knowledge, cases arising from repeated small-dose exposure ("microdosing") have not been documented, nor has nutmeg intoxication previously been reported from the United Arab Emirates (UAE), where nutmeg use in traditional foods and beverages is culturally common.
Hypothesis or Research Question: Repeated microdosing of nutmeg over several days can precipitate clinically significant neuropsychiatric toxicity comparable to that seen after acute large-dose ingestion.
Methods: This is a case report that was written up using a single-patient chart review, describing the presentation, diagnostic evaluation, and therapeutic response of a previously healthy woman who developed acute hallucinosis and insomnia after a week of small, repeated nutmeg ingestion. Findings were analyzed in the context of existing toxicology literature to assess the plausibility and significance of microdosing-related toxicity. Clinical findings were compared with existing published cases and toxicology literature.
Results: A 45-year-old woman presented with 72 hours of insomnia, severe dizziness, headache, and visual hallucinations after adding small amounts of nutmeg to Arabic coffee and chewing nutmeg intermittently for perceived calming effects for a week. This resulted in repeated low-dose exposure, with no large bolus ingestion. She was dehydrated but hemodynamically stable; neurologic examination was normal. Laboratory evaluation showed microcytic anemia, mild hypoglycemia (3.8 mmol/L), and sterile pyuria. CT head was normal. The patient improved rapidly with 2 L IV normal saline fluids and 1 mg IV lorazepam, with complete resolution of hallucinations during a 6-hour ED observation period. The poison center was consulted and supported the diagnosis of nutmeg-induced neurotoxicity. This appears to be the first documented case of nutmeg intoxication within the UAE and the first to implicate likely microdosing rather than a single high-dose ingestion as the precipitating exposure pattern.
Conclusion: This case expands the clinical understanding of nutmeg toxicity by demonstrating that repeated small-dose consumption may accumulate sufficiently to cause hallucinosis, profound insomnia, and dizziness. Recognition is particularly important in regions where nutmeg is used culturally in beverages or as a home remedy. Clinicians should consider spice-related exposures in unexplained neuropsychiatric presentations.

142. Inadvertent Intramuscular Administration of Methylergometrine to a Neonate Resulting in Respiratory Distress

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Background: Methylergometrine is an amine ergot alkaloid used to stimulate uterine contraction to prevent or treat postpartum hemorrhage. In overdose, the widespread vasoconstriction caused by ergot agents has been associated with lethargy, nausea, vomiting, seizures, feeding intolerance, elevated liver enzymes, oliguria, renal failure, and respiratory distress. The case report below discusses a neonate that developed respiratory distress after receiving 0.068 mg/kg of methylergometrine.
Methods: A neonate was born at 39 weeks gestation via vaginal delivery with no complications; APGAR scores were 9 at 1 minute and 9 at 5 minutes. While in the delivery unit, the patient was given 0.2 mg (0.068mg/kg) IM of methylergometrine (Methergine) intended for the mother for treatment of postpartum hemorrhage 1 hour after delivery instead of the intended 0.5 ml intramuscular phytonadione. The patient was initially asymptomatic, and vitals were heart rate 155, temperature 36.4 C, respiratory rate 58, and blood pressure 73/35 mmHg. The poison center was contacted, and toxicology recommended 24-hour NICU admission to monitor for respiratory distress, vomiting, convulsions, electrolyte abnormalities, and renal dysfunction and administration of sodium nitroprusside if the patient became symptomatic.
Results: Approximately 1 hour post-administration, the patient developed apnea and respiratory distress. A heel blood gas was drawn at this time and showed pH 7.255, PCO2 48, HCO3 19.2, and PO2 59 with an O2 saturation of 90%. The child was started on 2L O2 via nasal cannula which was escalated to CPAP 6L/21% then to NIPPV 6L/21%. A chemistry and complete blood count were unremarkable. Chest radiograph showed prominent perihilar markings, but no other abnormalities were seen. He remained on NIPPV for approximately 12 hours before being weaned to CPAP

and then room air, requiring a total of 24 hours of respiratory support. Laboratory studies remained normal, no other symptoms developed, and he was discharged on day 3. The post-delivery setting provides significant opportunities for medication errors give providers are taking care of two patients in close proximity. This patient exhibited normal respiratory function at birth but developed respiratory distress requiring CPAP and NIPPV shortly after inadvertent administration of methylergometrine intended for the mother. The likely cause of his respiratory symptoms is vasoconstriction in the pulmonary vasculature caused by the ergot alkaloid.
Conclusion: Accidental administration of maternal medications such as methylergometrine to neonates has potential severe consequences even in small doses. Close monitoring for respiratory decompensation and other sequelae is needed.

143. Autoimmune Encephalitis From Mercury Intoxication

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Background: Elemental mercury exposure is known to cause neurologic manifestations, which typically improve with source removal and chelation.
Hypothesis or Research Question: Autoimmune encephalitis secondary to mercury poisoning can cause persistent encephalopathy despite chelation and declining mercury concentrations.
Methods: This is a single patient chart review.
Results: A two-year-old girl, as well as her parents and sibling, developed cough and congestion, which were initially attributed to a respiratory infection. For the next month, the child had intermittent fevers, fussiness, and decreased oral intake, and eventually developed a pruritic rash with erythema to her distal extremities. She was then admitted to the hospital after presenting with persistent tachycardia, hypertension, diaphoresis, and dehydration. Assays for heavy metal exposure were positive for mercury in the patient's urine (35 mcg/g creatinine) and whole blood (11 mcg/L). The source of mercury exposure was determined to be a small vial of elemental mercury that had been spilled in the home shortly before her initial development of symptoms. Toxicology consultation was obtained, and she was treated with a 19-day course of dimercaptosuccinic acid (DMSA) prior to being discharged from the hospital. Remediation efforts were conducted in the patient's home, and a peak mercury vapor concentration in the room where the mercury spill occurred was measured at 500 mcg/m3. Despite home remediation and completion of the DMSA chelation course, the patient's clinical condition continued to deteriorate after she was discharged, with progressive worsening of behavior, intermittent episodes of agitation, and poor sleep quality. She was readmitted to the hospital due to continued altered mental status. A lumbar puncture was performed, and an autoimmune encephalitis panel was sent to an outside laboratory. Her urine mercury concentration upon this admission was undetectable. The autoimmune encephalitis panel returned positive for contactin-associated protein-2 IgG antibodies (CASPR2-IgG). She was then started on intravenous immunoglobulin (IVIG) and high-dose methylprednisolone, after which there was clinical improvement in her behavior and oral intake.
Conclusion: CASPR2-IgG autoimmune encephalitis is a complication after mercury intoxication that may persist despite chelation and clearance of mercury, necessitating therapy with IVIG and high-dose steroids.

144. A Case of Lethargy and Vomiting After Moxidectin Overdose

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Background: Moxidectin, a macrolytic lactone of the macrolide class, is FDA approved for use in humans in the treatment of onchocerciasis. Phase I clinical trials tested a maximum dose of 36 mg oral moxidectin with adverse effects described including headache and orthostatic hypotension, however overdose of moxidectin has not previously been reported in the literature.
Hypothesis or Research Question: Oral overdose of moxidectin causes lethargy and gastrointestinal distress.
Methods: This is a single patient case report. A previously healthy 36-year-old male presented to the emergency department eight and a half hours after intentionally ingesting 80 mg moxidectin due to a feared helminth infection. His chief complaint was lethargy, however the patient also noted several episodes of non-bloody emesis prior to arrival. Complete blood count, complete metabolic panel, magnesium, phosphate, and electrocardiogram were obtained upon patient arrival. He was examined and observed in the emergency department, toxicology was consulted, and it was recommended that he be admitted for further monitoring.
Results: Vital signs were within normal limits upon patient arrival and throughout his stay in the emergency department. Physical examination was notable for somnolence, though the patient was arousable to voice and was able to participate

in interview for short periods of time before falling back asleep. Speech was somewhat slurred. Neurological exam was otherwise without weakness, ataxia, clonus, cranial nerve deficit, or decreased sensation. No laboratory or electrocardiogram abnormalities were noted. No dysrhythmias were noted on telemetry during several hours of observation. He eloped from the emergency department prior to admission.
Conclusion: Oral ingestion of 80 mg moxidectin, a higher dose than previously observed in humans, appears to cause lethargy and gastrointestinal distress without immediate impact on acid/base status, cardiac conduction, , liver function, electrolytes, or hematologic cell lines. This Conclusion: is limited by its nature as a case report, the patient having eloped prior to continued monitoring, and lack of confirmatory laboratory testing for moxidectin.

145. Fatal Poisoning Due to Atractylis Gummifera in Children

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Background: Atractylis gummifera (Addâd) is a highly toxic plant endemic to the Mediterranean basin. In rural Algeria, it is often mistaken for the edible plant "Guernina", particularly by children who chew its latex, leading to frequent accidental intoxications. This report describes a severe pediatric poisoning involving three children following ingestion of the plant and highlights diagnostic challenges, treatment strategies, and outcomes.
Methods: This retrospective clinical case study describes the clinical presentation, biological findings, imaging, therapeutic interventions, and evolution of three related children admitted to the pediatric emergency unit of EPH Souk-Ahras, Algeria. Diagnosis was based on clinical features, history of ingestion, and toxicological confirmation by plant identification. Therapeutic management was supportive, including antivirals, antibiotics, mannitol, N-acetylcysteine (NAC), vitamin K, plasma transfusion, and intensive monitoring.
Results: The eldest child (X.K, five years old) presented 24 hours post-ingestion with coma (GCS 7/15), hypertonic seizures, cerebral edema on CT, severe hepatocellular failure, rhabdomyolysis, and multiorgan dysfunction. Despite intensive care and NAC therapy, the child died on day 7 due to refractory hyperkalemia, acute pulmonary edema, and cerebral edema. The other two children (X.M two years and Y.N, four years old) presented with acute hepatocellular cytolysis (AST 5000 IU/L, ALT 4300 IU/L) but preserved coagulation. Early supportive treatment and hydration allowed full recovery within 10 days. Toxicological analysis performed three days post-ingestion was negative due to delayed sampling.
Conclusion: The main toxin of *Atractylis gummifera* "Atractyloside", induces severe hepatic and renal failure through mitochondrial inhibition. Due to the absence of a specific antidote and the limits of toxicological detection, early clinical recognition is crucial. Management is purely supportive, and prevention relies on public education and strict control of plant distribution.

146. Toxicity Following Antiemetic Ingestions Reported to the Toxicology Investigators Consortium Core Registry

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Background: There are a variety of medications used to treat nausea. Although antiemetics are not frequently implicated in overdose, several side effects may develop idiosyncratically or following large ingestion.
Hypothesis or Research Question: What are the demographics, clinical features, treatments, and outcomes of prochlorperazine, ondansetron, and metoclopramide ingestions reported to the American College of Medical Toxicology's Toxicology Investigators Consortium (ToxIC) Core Registry?
Methods: This was a review of prospectively collected de-identified patient information reported to the ToxIC core registry by medical toxicologists providing bedside care for poisoned patients between January 1, 2010 and December 31, 2024. Data regarding patient demographics, clinical features, antidote administration, other interventions, and outcomes were reviewed.
Results: There were 85 cases entered into ToxIC during the study period. Promethazine was implicated in 58 (68.2%) cases, while ondansetron accounted for 14 (16.5%) exposures. Metoclopramide was ingested in 13 (15.3%) cases. The median patient age was 24 years, range 4 months – 74 years. Females accounted for 45 (52.9%) patients. The most common clinical feature following promethazine ingestion was antimuscarinic toxicity, which was reported in 22 (37.9%) cases. Agitation was observed in 16 (27.6%) cases, while central nervous system depression was seen in 14 (24.1%) patients. One patient had both QRS widening and QT prolongation on electrocardiogram. Thirty-five (60.3%) patients received treatment. Benzodiazepines were administered to 18 (31%) patients. Physostigmine was used in seven (12.1%) cases. Endotracheal intubation and mechanical ventilation was performed on four (6.9%) patients. Central nervous system depression was observed in six (42.9%)

ondansetron exposures. Two (14.3%) patients had seizures. Dystonia was reported in one case. One patient had QT prolongation, and another had respiratory depression and myocardial injury. Treatment was administered to five (35.7%) patients. Endotracheal intubation and mechanical ventilation was performed on two (14.3%) patients. One patient with a metoclopramide ingestion developed neuroleptic malignant syndrome. Dystonia was observed in six (46.2%) cases. One patient had both QRS widening and QT prolongation. Seizure was described in one case. Nine (69.2%) patients received treatment. Three patients received diphenhydramine. Three patients were treated with benzodiazepines. Bromocriptine, magnesium sulfate, and an unspecified antipsychotic were administered in one case each.
Conclusion: A variety of clinical features may develop antiemetic ingestions. Central nervous system depression is frequently reported. Promethazine may cause antimuscarinic toxicity. Electrocardiographic abnormalities may be observed. Treatment may include antidotal therapy, general supportive care, and in severe cases, endotracheal intubation and mechanical ventilation.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

147. Unrecognized Central Nervous System Depression in a Patient With Acetazolamide Toxicity

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Background: Acetazolamide is a reversible carbonic anhydrase inhibitor, resulting in reduced hydrogen secretion at the renal tubule and increased renal excretion of potassium, sodium, bicarbonate, and water. It has multiple effects, including decreased aqueous humor production and thus is a common treatment for acute angle closure glaucoma. We present a case of central nervous system toxicity from acetazolamide in a patient with end-stage renal disease.
Methods: This is a single patient chart review.
Results: A 68-year-old patient with a history of hypertension, diabetes, end-stage renal disease on hemodialysis presented to the Emergency Department with 2 weeks of left eye pain, visual acuity changes, and headache. The patient was diagnosed with acute angle closure glaucoma and treated with topical brimonidine, dorzolamide-timolol, and pilocarpine in addition to intravenous acetazolamide, 500mg every 6 hours. While still in the Emergency Department, the patient was noted to be globally weak and lethargic concerning for an acute stroke. A computed tomography and computed tomography angiography of the head were negative for pathology. After receiving a total of 1500mg of intravenous acetazolamide, the patient was transferred to our facility for evaluation by a retina specialist. On arrival to our facility, the patient continued to be encephalopathic and unable to follow commands. A continuous electroencephalogram was completed, and showed no evidence of seizure activity. Medical toxicology was consulted on hospital day four. Acetazolamide central nervous system toxicity was suspected, and the patient underwent a full hemodialysis session that day. The patient had subsequent improvement with hemodialysis completed on hospital days four and seven. He was discharged to long-term acute care on hospital day eight.
Conclusion: The exact mechanism of acetazolamide central nervous system toxicity has not been completely elucidated. Acetazolamide is readily removed with hemodialysis and central nervous system effects reverse or significantly improve with hemodialysis. This case demonstrates the effectiveness of hemodialysis for treatment of acetazolamide toxicity once recognized, and the importance of early recognition of acetazolamide as a significant cause of depressed mentation, lethargy, or coma.

148. Fast Facebook Mycology: A Case of Toxicity From Amanita Chrysoblema

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Background: *Amanita chrysoblema* or American fly agaric is a yellow-orange variant of *A. muscaria* and thought to contain muscimol and ibotenic acid. Cases of toxicity after ingestion of this variant have not previously been reported.
Hypothesis or Research Question: A social media group can emergently identify a mushroom in a symptomatic ingestion.
Methods: This is a single-patient chart review. A 9-month-old previously healthy female presented to the hospital within an hour of a witnessed mushroom ingestion with ataxia and lethargy. The patient was unable to stand or crawl. Family was able to provide pieces of the mushroom to the physician and pictures were uploaded by a poison center specialist to a Facebook group to assist with identification.
Results: Within 15 minutes of posting pictures of the ingested mushroom to the Facebook group, the mushroom was identified as *Amanita chrysoblema* by three independent mycologists. This identification was consistent with the patient's symptoms of mild CNS depression and ataxia. There was no gastrointestinal upset. No medical interventions were administered. The patient was admitted for observation and returned to baseline by 10 hours post-ingestion. To understand the reliability of the mycologists on this Facebook group, our poison center reached out to the group administrators. After discussions, we determined that this group does have an internal vetting process

for all participating mycologists and will not confirm an identification until there is agreement among independent mycologists.
Conclusion: Ingestion of *Amanita chrysoblema* resulted in mild CNS depression that resolved within 10 hours. Specialized social media groups can be a helpful tool for quick mushroom identification though management should be guided by clinical presentation.

149. Spuriously Elevated Bicarbonate Level in the Setting of Elevated Lactate Dehydrogenase

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Background: Laboratory errors can produce inaccurate results which may lead to excessive or unnecessary workup, prolonged inpatient stays, and inefficient use of time and resources. This abstract describes a spuriously elevated bicarbonate due to an elevated lactate dehydrogenase (LDH).
Methods: This is a case report of a patient seen at a high-volume tertiary care hospital in an urban setting. The patient's laboratory results were securely reviewed in the electronic medical record (EMR) throughout hospital admission.
Results: A 65-year-old female patient presented to the Emergency Department (ED) with significant hepatotoxicity following a repeated supratherapeutic ingestion of acetaminophen. She was managed with a combination of N-acetylcysteine, fluid resuscitation, and CRRT. The patient's laboratory results are as noted in Table 1. Evaluating the chemistry panel using the Stewart approach to acid base disturbances, a chemical imbalance was immediately obvious as the values fail to maintain electroneutrality. Furthermore, a venous blood gas was obtained showing a pH of 7.34, pCO2 39, and base excess -4.4 which was incongruent with the chemistry results. The most likely underlying mechanism was thought to be interference from elevated LDH in the sample. The enzymatic assay used to determine bicarbonate concentration measures the decrease in NADH absorbance at 340 nm. Under normal conditions, the only reaction that consumes NADH is a bicarbonate-dependent reaction. However, when LDH is markedly elevated, it reacts with components of the assay mixture and consumes NADH nonspecifically. This additional NADH consumption is interpreted by the analyzer as increased bicarbonate, leading to a spuriously elevated result. As the patient's condition progressed, hemolysis led to LDH levels exceeding 1200 U/L further exacerbating this artifact. Notification was later received from the assay manufacturer that certain lots of the test reagent were especially prone to this interference. We tested our patient's samples using an assay from a different manufacturer employing the same enzymatic methodology. These comparison results didn't show any abnormal elevation, supporting that the original reagent lot was the source of the interference.
Conclusion: The Stewart approach to acid base disturbances is an efficient and effective framework for interpreting the validity of laboratory values, even without additional studies for comparison. When contradictory results are noted, this should raise providers' concerns for errors in the laboratory process and prompt engagement in multidisciplinary conversations with laboratory leadership, and consideration of comparison with alternate methodologies.

150. Naja Melanoleuca Envenomation and Subsequent Management

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Background: The Forest Cobra (Naja melanoleuca) is a venomous elapid snake endemic to Central and Western Africa. Envenomations are medically significant, leading to local tissue injury, neuromuscular weakness, and death via respiratory failure.
Hypothesis or Research Question: What is the optimal management and expected clinical course for Naja melanoleuca envenomation?
Methods: A 21-year-old male presented to the emergency department the morning after being bitten on his right 2nd toe by his snake, identified as Naja melanoleuca. He reported pain and numbness throughout the right lower extremity, perioral paresthesias and shortness of breath. The envenomation site was ecchymotic, with swelling up to the mid-tibia. Presenting vital signs included a heart rate of 102 bpm, blood pressure of 142/88 mmHg, respiratory rate of 12 per minute, oxygen saturations of 100% and a temperature of 36.8 °C. White blood cell count was 18k cells/μL, hemoglobin 16.5 g/dL, creatine kinase 222 U/L, platelets 293 k/μL, fibrinogen 250 mg/dL, and INR 1.1. X-ray showed no evidence of foreign bodies. The limb was elevated and intravenous fentanyl was provided for pain. A zoo was contacted for antivenom, and fifteen vials of SAIMR Polyvalent Antivenom were obtained. Despite

a 2023 expiration date, the antivenom was deemed appropriate for use, and three vials were administered without adverse effects. Hematologic profile remained unchanged, and he did not develop neuromuscular weakness or require respiratory support. He experienced ongoing pain, and intravenous cefepime and metronidazole were started empirically, though no further evidence of infection developed. Podiatry was consulted regarding ecchymosis and necrosis of the 2nd and 3rd toes, but no further intervention was recommended. He was discharged home on hospital day 8 in stable condition.

Results: Proteomic analysis of Naja melanoleuca venom has identified post-synaptic α-neurotoxins, phospholipases, and metalloproteinases, among many others. Effects are generally rapid in onset, with a descending paralysis and subsequent respiratory failure. Local necrosis, pain and swelling may persist for several days. Our patient did not develop any true neuromuscular weakness. Evidence of local tissue injury suggests a mild envenomation.

Conclusion: Exotic envenomations, though rare in the United States, may be life threatening. Species identification may guide directed therapies; procuring antivenom may be limited by availability, knowledge of availability, and expiration status. Poison centers may assist with these concerns. Antivenom may remain viable past listed expiration dates, and providers must determine the benefit of administration compared to the risks of utilization in these cases.

151. Angioedema, Shock, and Refractory Coagulopathy After Crotalus Horridus Horridus Envenomation

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Background: Despite well-documented thrombocytopenia, angioedema and cardiovascular collapse after Crotalus horridus horridus envenomation are rarely reported in crotalid-naive individuals.

Hypothesis or Research Question: Crotalus horridus horridus envenomation can be associated with life-threatening angioedema, circulatory collapse, and antivenom-refractory thrombocytopenia.

Methods: This is a single patient case review. A 58-year-old male without significant previous crotalid exposure who initially presented to the outside hospital Emergency Department after a rattlesnake envenomation sustained while camping. He was bit in the finger and developed progressively worsening dyspnea and airway swelling en route to the Emergency Department. On initial hospital assessment, he was hypotensive and bradycardic. He was given 6 vials of F(ab') fragments (CroFab), methylprednisolone, epinephrine, diphenhydramine, and underwent RSI with ketamine to facilitate endotracheal intubation. The initial labs demonstrated thrombocytopenia with platelets 11x10^9/L. He received an additional 5 vials of F(ab') fragments and was accepted for transfer to a tertiary care facility.

Results: While preparing for transportation, patients suffered endotracheal tube failure with initial desaturations, with resolution after repositioning. Later, during helicopter transport, the tube became dislodged. Emergent cricothyrotomy, although initially avoided due to thrombocytopenia, was subsequently pursued with success by flight medics. Post procedure, the patient experienced a wide complex dysrhythmia necessitating defibrillation. On arrival to the tertiary care center, the patient developed lower gastrointestinal bleeding and received 2-unit platelet transfusion. He received 2 vials F(ab')2 fragments but developed worsening angioedema so the remainder of the dose was withheld. Patient underwent revision operative tracheostomy and was admitted to the ICU. Platelets showed biphasic improvement with subsequent nadir of 4 x10^9/L by hospital day #8. Over the course of his hospitalization, the patient received a total of 10U platelet transfusion without sustained improvement in thrombocytopenia until discharge (241x10^9/L on HD#22). The remainder of the patient's hospital course was complicated by pneumonia and acute renal failure necessitating intermittent hemo dialysis that did not recover.

Conclusion: This case highlights an inordinately severe case of toxicity after Crotalus horridus horridus envenomation. We present a rare presentation of angioedema, circulatory collapse and severe coagulopathy after relatively minor appearing envenomation in a crotalid naive individual. Despite bleeding risk from thrombocytopenia, emergent cricothyrotomy was successfully performed in a resource-austere setting. Furthermore, this case redemonstrates prolonged platelet recovery and ongoing destruction seen with Crotalus horridus horridus envenomation.

152. Recurrent Methemoglobinemia in a Chronic Alcohol User: Congenital Cytochrome B5 Reductase Deficiency

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Background: Methemoglobinemia is a rare but potentially life-threatening disorder in which the iron within the heme prosthetic group is oxidized, impairing hemoglobin's ability

to deliver oxygen. While most cases are acquired after exposure to oxidizing agents, recurrent or unexplained episodes may be indicative of an underlying enzymatic deficiency.
Hypothesis or Research Question: What are the alternative etiologies of methemoglobinemia apart from exposure to oxidizing agents?
Methods: This is a single-patient case report. Data were obtained from the patient's presentation, laboratory studies, hospital course, and retrospective review of prior encounters. Confirmatory enzyme activity testing was performed during hospitalization.
Results: A 66-year-old man with a history of chronic, heavy alcohol use, hypertension, transient ischemic attack, and left below-knee amputation was found unresponsive outdoors. On arrival to an outside facility, the patient was noted to be hypoxic and started on 100% oxygen. Supplemental oxygen failed to correct the hypoxia. Both arterial and venous blood gases demonstrated methemoglobin levels >30%. Methylene blue was unavailable, and the patient was transferred to our tertiary care facility. At the tertiary care facility, the patient received two doses of methylene blue (1 mg/kg IV over 15 minutes each), resulting in rapid improvement with methemoglobin levels trending down to 4.9%, 1.9%, and 1.6% over the next 24 hours. He also received empiric thiamine, folate, and pyridoxine. Chart review revealed multiple prior episodes of idiopathic methemoglobinemia or shortness of breath dating back to 2018. Given no putative source was identified; an enzymatic analysis was sent out, which later revealed reduced cytochrome b5 reductase activity (2.4 U/g; reference >7.5 U/g), consistent with congenital cytochrome b5 reductase deficiency. Additional concerning lab finding was vitamin C <0.1 mg/dL. The current episode resolved with therapy and vitamin supplement, and the patient was discharged with vitamin C supplementation and counseling for alcohol cessation.
Conclusion: This case highlights congenital cytochrome b5 reductase deficiency as a rare but important cause of recurrent methemoglobinemia, especially in individuals with heavy alcohol use. Alcohol-related nutritional deficiencies and hepatic dysfunction can lead to oxidative stress, likely contributing to patient presentation. Clinicians should maintain a high index of suspicion when confronted with recurrent or idiopathic cases. In particular, type 1 congenital cytochrome b5 reductase deficiency presents with lifelong methemoglobinemia and is best managed with daily vitamin C supplementation, rather than methylene blue alone. Early recognition can be helpful in diagnosis and long-term management.

DAY 3: LIGHTNING ORALS, ABSTRACTS 153-159

153. Are Toxicologists Using Telemedicine?

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Background: Telemedicine use has grown rapidly in recent years, enabling many specialties to extend their reach and billing. Understanding how medical toxicologists use telemedicine is important, as it may expand access to specialist consultation across multiple remote medical centers.
Hypothesis or Research Question: Medical toxicologists are increasing their use of telemedicine.
Methods: This is a review of the Toxicology Investigators Consortium (ToxIC) Core Registry from January 2018-March 2025 for all medical toxicology patients evaluated via telemedicine. Patients of all ages with a telemedicine encounter were included. Data was abstracted by a single author to a standardized data abstraction form. The method of telemedicine consultation (e.g., via video, phone, or by chart review only), rational for telemedicine use, and whether the consultation was billed were recorded. Other abstracted data included demographic information, reason for the consultation, and if an antidote or treatment was administered.
Results: There were 1,851 telemedicine encounters reported, including 143 in 2020, 122 in 2021, 206 in 2022, 324 in 2023, 670 in 2024, and 386 through the first 5 months of 2025. The relative frequency of telemedicine consultations among all patient encounters in the ToxIC Core Registry was 2.1% in 2020 (143/6668), 1.4% in 2021 (122/8552), 2.9% in 2022 (206/7206), 4.4% in 2023 (324/7392), 7.6% in 2024 (670/8868), and 10.1% in the first half of 2025 (386/3813). The mean patient age was 42 years old (range 2-88 years) with 864 identifying as female (47%), 966 as male (52%), and 21 as transgender (1%). Video encounters were most common (n=1033; 56%), while 34% of consultations involved a chart review only. Telehealth was primarily used for offsite patients (n=908; 49%), lack of bedside privileges (n=298; 16%), or off-hours consultations (n=190; 10%). Nearly 80% of all encounters (n=1,463) were billed, increasing every year from 52% (n=74) in 2020, 62% (n=200) in 2023, and 87% (n=582)

in 2024. Addiction medicine consultations were performed in 34% of patients (n=632). More addiction consults were for complications of ethanol use than for opioid withdrawal (n=747 vs n=185). Buprenorphine was administered to 206 patients, methadone to 57, and naloxone prescriptions or take-home doses to 162. Naltrexone or acamprosate was given in 152 encounters.

Limitations: While we know how many encounters were billed, data regarding collections was not available.

Conclusion: Toxicology telemedicine consultations increased from 2020-2025, along with the percentage that were billed. Future research should examine collections for telemedicine consults by toxicologists and site variation which may contribute to trends in telemedicine consults.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

154. Comparison of Self-Reported Drug Use and Adjudicated Causality in Polydrug Overdose Presentations

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Background: Polydrug exposure is increasingly common in overdose. Discrepancies between patient-reported drug use and toxicology laboratory findings complicate efforts to determine which substances primarily contribute to a patients toxicity. Evaluating the relationship between patient-reported substance use and causality can be best determined by quantitative and qualitative blood testing.

Hypothesis or Research Question: How does patient-reported substance use compare with laboratory determined causality in overdose patients?

Methods: This analysis utilized data from the Toxicology Investigators Consortium (ToxIC) Drug Overdose Toxico-Surveillance (DOTS) Reporting Program. Patients aged >13 years who presented to emergency departments at 17 participating medical centers with an apparent life-threatening opioid or stimulant overdose between April 2023 and September 2024 that provided informed consent were included. Structured interviews on current and past drug use patterns were conducted. Extensive state-of-the-art qualitative and quantitative blood toxicology analyses were performed by the Center for Forensic Science Research and Education. Two medical toxicologists independently reviewed each case for tolerance, precipitated withdrawal or stimulant unmasking after naloxone, and substances contributing to the overdose. Any discrepancies were resolved by a third reviewer or group consensus. Descriptive statistics compared self-reported substance use associated with the overdose presentation, detected substances, and case adjudicated causality.

Results: Among 995 patients enrolled, 875 had complete case adjudication at time of this analysis. Presentations were deemed not drug related in 30 cases (3.4%) and uninterpretable in 94 (10.8%). Opioids alone were the most common causality determination (N=563, 64.3%), followed by stimulants alone (N=82, 9.4%). When patients were asked the substances used prior to the overdose, 71 (8.1%) denied any use and 57 (6.5%) declined the interview or to answer substance use questions. Of those that denied any drug use leading to the overdose, only 11 (15.5%) were determined to be non-drug related after toxicologist review. Of those cases with a causality determination where the patient reported using at least one substance prior to the overdose (N=667, 76.2%), case adjudication implicated the same drug class in 343 patients (51.4%). Stimulants were the sole intended drug reported by 124 patients (18.6%), however more than half (N=73; 58.9%) were attributed to opioid only toxicity which was due to fentanyl, fentanyl analogues, or nitazenes. Only one of these patients reported knowingly utilizing an opioid within 24 hours of the overdose.

Conclusion: Patient self-reports often underestimate opioid involvement, particularly in cases of intended stimulant use, highlighting the value of quantitative and qualitative blood tests allowing for the definitive determination of causative substances.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

155. Homicide by Poisoning- a Comparative Review of the Uniform Crime Reports and the CDC WONDER Database

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Background: Homicidal poisoning is a relatively uncommon but consequential form of malicious violence, sitting at the intersection of criminal investigation and public health. Examining characteristics and victim-offender context of past homicidal poisoning cases is essential for better monitoring and targeted prevention.

Methods: The United States Federal Bureau of Investigation's Uniform Crime Reports (UCRs) and CDC Wide-Ranging Online Data for Epidemiological Research (WONDER) were queried for all cases of homicidal poisonings from 2018-2022 and 2018-2023, respectively. We descriptively assessed the demographic characteristics including gender, race, and age of victims. The demographic characteristics of the offender and the relationship of offender to victim were only available from the UCR database.

Results: There were 1,272 homicidal poisoning cases reported to the UCRs involving 1,623 offenders. A total of 134,192 homicidal poisonings were identified in the CDC WONDER database. The victims of homicidal poisoning were most often males (62.5% UCR, 67.3% WONDER). Victims were most commonly White (83.5% UCR, 70.9% WONDER), Black/African American (9.7% UCR, 24.6% WONDER), and American Indian or Alaska Native (2.9% UCR, 0.9% WONDER). The UCR data revealed victims were often among the 20–39-year-old age group (50.9%). The CDC WONDER data showed victims were most frequently in the 25-44-year-old age group (36%). The second most frequently reported age range was the 0-4 year-old age group (28.4%). The demographic characteristics for the offenders were found to be similar to victims. Offenders were commonly males (50.9%), White (63%), and within the 20–39-year-old age range (57.9%). In regard to relationship of offender to victim, the most frequently reported relationships were "acquaintance" (28.8%), "friend" (11%), and "other – known to victim" (10.7%).

Conclusion: Age patterns diverged between the two data sources, with CDC WONDER data suggesting both adult and very young child vulnerability, highlighting the importance of pediatric toxicology awareness alongside adult-focused interventions. Offender-victim relationship data indicate most perpetrators are known to the victim, suggesting prevention and detection strategies should prioritize close social networks rather than stranger-danger relationships.

156. Comprehensiveness of State-Level Legislation Related to Substance Use Disorder Treatment Insurance Coverage and Drug-Related Overdose Mortality

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Background: Substance use disorders (SUDs) are a major public health concern in the United States, and comprehensive insurance coverage for evidence-based SUD treatment is a critical component of the continuum of care.

Hypothesis or Research Question: We hypothesized that there would be an association between the comprehensiveness of state-level SUD treatment insurance coverage legislation and drug-related overdose mortality rates.

Methods: We performed a retrospective serial cross-sectional analysis of individuals 15 years and older with overdose mortality. Age-adjusted drug-related overdose mortality rates per 100,000 individuals were obtained from the Centers for Disease Control and Prevention Wide-Ranging Online Database for Epidemiologic Research. Insurance coverage data were obtained from the State Substance Use Disorder Insurance Laws Database (SSILD), which assigns scores (0–9) for each year and insurance sector in each state based on comprehensiveness of SUD treatment insurance coverage laws. Scores were weighted by insurance sector population coverage in each state. State-year scores were analyzed in quintiles. We performed multivariable population-averaged negative binomial regression to assess associations with overdose mortality, adjusting for state demographics, SUD-related legislation (naloxone access laws, good Samaritan laws, and recreational cannabis legalization), and the percentage of the state population with Medicare, Tricare, or VA insurance (as these insurance types were not scored in the SSILD). Time was grouped into two-year increments to balance temporal granularity and statistical power.

Results: From 2010 through 2020, there were 794,944 drug-related overdose deaths among Americans age 15 and older. Overdose mortality rates increased almost universally across all demographic and substance subgroups. In adjusted analyses, overdose mortality in state-years in the second (incidence rate ratio [IRR] 1.027, 95% confidence interval [CI] 0.850-1.241), third (IRR 1.123, 95% CI 0.891-1.416), fourth (IRR 1.188, 95% CI 0.950-1.485), and fifth (IRR 1.178, 95% CI 0.928-1.497) quintiles, representing more comprehensive legislation, did not differ significantly from mortality in state-years within the first quintile, representing the least comprehensive legislation.

Conclusion: Greater comprehensiveness of state-level SUD insurance coverage laws was not associated with lower drug-related overdose mortality rates from 2010 to 2020. These findings suggest that legislative efforts to expand insurance coverage for SUD treatment, in isolation, may be insufficient to reduce overdose deaths. Limitations of this study include the use of high-level legal variables—focusing on parity, mandatory coverage, definitions, and enforcement and compliance—as opposed to specific overdose-related legislation, inability to infer causality, and influence of unmeasured confounders.

157. Risk of Hydrogen Sulfide Off-Gassing From Victims After Lethal Exposure

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Background: Hydrogen sulfide (H2S) gas is a lethal cellular asphyxiant. The gas is an occupational hazard in certain industries and can be used as a means of suicide. Multiple reports exist of risks to first-responders and medical personnel due to off-gassing of H2S from victims.
Hypothesis or Research Question: After a significant H2S exposure, does H2S off-gassing from the victim pose a health risk to rescuers and medical personnel?
Methods: As part of a hazmat exercise, we set up a chemical suicide model by producing H2S in an enclosed vehicle with a clothed rescue mannequin in the driver's seat. H2S was made by combining 1.7 L of 29 wt % lime sulfur (calcium polysulfide) solution and 1.2 L of 31.4 wt % hydrochloric acid in a 5-gallon bucket on the front passenger's seat, then immediately closing the door. Concentrations of H2S were monitored in the vehicle real-time with MiniRae 3000 photoionization detectors installed by drilling through the ceiling of the vehicle. Rescue of the mannequin to a warm zone 20 feet upwind of the vehicle commenced after H2S peak. Continuous measurements of H2S off-gassing from the rescuer and mannequin were then recorded with MiniRae 3000 photoionization detectors. The exercise was performed twice. Across exercises, the rescuers wore different protective gear (structural turnout gear vs. Tychem® 6000 chemical protective clothing) and the mannequin was disrobed in exercise two. All involved in the warm and hot zones were in level A personnel protective equipment.
Results: Exercise 1: Vehicle H2S peaked at 1079 ppm. Immediately following rescue, off-gassing from the rescuer in Tychem® 6000 measured 11.6 ppm and returned to 0 ppm in 90 seconds, while clothed mannequin measured 6.9 ppm and returned to 0 ppm in 150 seconds. Exercise 2: Vehicle H2S peaked at 6359 ppm. Immediately following rescue, off-gassing H2S from the rescuer in structural turnout gear measured 36.6 ppm and 7.6 ppm at 180 seconds, while the unclothed mannequin measured 3.6 ppm and returned to 0 ppm in 45 seconds. See figure 1.
Conclusion: After exposure to extreme concentrations of H2S, off-gassing from the rescue mannequin was immediately below the permissible exposure limit of 10 ppm. Off-gassing from rescue gear fell to the permissible exposure limit within 3 minutes. Rapid transfer of victims to medical personnel in lower level personal protective equipment nearby would be safe without health risk to medical personnel.

158. National Trends in CroFab and Anavip Utilization in US Emergency Departments, 2016 – 2024: A Cross-Sectional Study Using Epic Cosmos

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Background: Anavip and CroFab are the two available antivenoms approved to treat North American crotalid envenomation in the United States (US). National utilization patterns of these two antivenoms are not well described.
Hypothesis or Research Question: Describe the national demographic, geographical, temporal, and seasonal patterns of CroFab and Anavip use in patients presenting to US emergency departments (EDs).
Methods: We conducted a cross-sectional study of patients who presented to US EDs from 2016 to 2024 and were treated with either CroFab or Anavip. Data used in this study came from Epic Cosmos, a dataset created in collaboration with a community of health systems using Epic representing more than 300 million patient records from over 1,800 hospitals as of October, 2025. For each eligible encounter we extracted age, sex, race, ethnic group, US Census region, state of residence, month of presentation, ED disposition, and hospital disposition. Annual utilization rates were normalized per one million ED visits.
Results: We identified 8,639 antivenom administrations, including 7,405 CroFab and 1,314 Anavip administrations, with combined annual use increasing from 22.5 to 26.8 administrations per million ED encounters from 2016 to 2024. CroFab use per million ED visits declined over the study period. EDs began using Anavip in 2019 and use per million ED visits increased from 0 in 2016-2018 to 7.7 in 2024. CroFab predominated in the Southeast and Mid-Atlantic, while Anavip was concentrated in the West and Southwest. Most patients were male (64%), White (78%), and non-Hispanic (83%). Seasonal patterns, ED disposition, hospital disposition were similar between both groups. Dual administration of CroFab and Anavip was rare.
Conclusion: Use of crotalid antivenoms in patients presenting to US EDs increased over the nine-year study period. Anavip use increased from 2019 to 2024 while Crofab use remained the same or decreased. Regional variation is likely due to variation in local snake species and influence of hospital formularies. Seasonal and demographic distributions seen in our data align with previously published studies. We saw a higher ratio of CroFab compared to other studies. These findings provide the largest and most contemporary national EHR-based dataset available to date and offer a clearer understanding of trends in CroFab and Anavip use compared to prior registry-based publications.

159. Concordance Between Self-Reported Drugs and Blood Toxicology Results: Findings From the ToxIC DOTS Program

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Background: People who use drugs are frequently unaware of the spectrum of potential adulterants in the drug supply; however, most studies examining discrepancies in self-report are limited to typical drug use patterns and rely on urine or hair sample testing.
Hypothesis or Research Question: What is the concordance between self-reported overdose drug and blood toxicology results among patients with an opioid or stimulant overdose?
Methods: This analysis was conducted using data from the Toxicology Investigators Consortium (ToxIC) Drug Overdose Toxico-Surveillance (DOTS) Reporting Program. Patients ages >13 presenting to emergency departments (EDs) with a severe opioid or stimulant overdose at one of 17 medical centers between April 2023 – September 2024 were eligible. All patients provided informed consent. Interviews assessed current drug use, sociodemographics, and treatment history. Qualitative and quantitative blood toxicology tests were conducted by the Center for Forensic Science Research and Education. Analyses included descriptive statistics and McNemar's Chi-Square Tests.
Results: Among the 995 patients enrolled, 933 completed the interview. Almost half of patients (43%) reported taking opioids only, 19% reported stimulants only, 13% reported opioids and stimulants, and 25% reported other drug combinations (e.g., alprazolam, cannabis). Among those who reported taking fentanyl only (n = 199), 189 (95%) had fentanyl present, but only 31% of those who reported no fentanyl (n = 734) had no fentanyl, norfentanyl, or fentanyl analogues ($p<0.001$). Three percent of patients who reported taking heroin only (n = 96) had morphine in their blood, and none had heroin or 6-monoacetylmorphine ($p<0.001$). Among patients who reported cocaine only (n = 86), 64% had cocaine, benzoylecgonine, or cocaethylene. However, 48% (n = 31) had no benzoylecgonine, cocaethylene, nor cocaine ($p<0.001$), and 5 of these 31 patients had methamphetamine. 52/54 patients who reported taking methamphetamine had methamphetamine present in their blood, and 65% who said they did not take methamphetamine had no methamphetamine present ($p<0.001$). 36/54 had amphetamine present (67%; $p<0.001$). Notably, fentanyl was detected in 72% of patients who reported taking cocaine only and 50% of patients who reported taking methamphetamine only.
Conclusion: Patients who self-reported fentanyl or methamphetamine use prior to their overdose had high sensitivity (>0.90) and concordant analytes; however, the specificity for self-reported fentanyl and methamphetamine was poor. There was considerable discordance for both cocaine and heroin. Future analyses should incorporate timing of blood collection. These findings could be compared with the illicit drug supply to inform public health messaging.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

DAY 3: MODERATED POSTERS, ABSTRACTS 160-166

160. The Provision of Outpatient Services by Medical Toxicologists

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Background: Many medical toxicologists provide non-emergent evaluations in outpatient clinics, yet their practices in this setting are not well described. Because medical toxicologists offer specialized expertise, understanding the availability of these services matters to individuals seeking toxicological evaluations and to communities responding to an environmental chemical or radiological exposure incident.
Hypothesis or Research Question: What is the reach of medical toxicologists who provide outpatient clinical services?
Methods: The American College of Medical Toxicology (ACMT) maintains a public "Find a Toxicologist" directory of clinicians offering medical toxicology services. In 2024, ACMT invited previously listed members to update their profiles and offered all other members the option to opt in. Between January and July 2025, 48 directory members who provide outpatient services were invited to complete a questionnaire on clinic location, setting, fellowship affiliation, staffing, clinic hours, patient volume, surge capacity,

and interest in a national network. Descriptive statistics were calculated.

Results: Thirty-seven individuals (78%) responded to the survey, 32 (86%) of whom reported providing outpatient medical toxicology services. Two duplicate records were excluded, yielding 30 unique clinics. Clinics were in 21 of 51 (41%) states/jurisdictions, including Washington, DC. Twenty-five (83%) clinics were affiliated with medical toxicology fellowships. Monthly clinic time dedicated to outpatient care ranged from <1 hour to >16 hours, with a median of 5–8 hours per month (IQR 1–4 to 9–16 hours per month). Median patient volume was 1-5 patients/month (IQR 1–5 to 6–10 patients per month). Clinics were staffed with a median of 2-3 attending medical toxicologists (IQR 1–4; range 1-8 attendings). All clinics (100%) saw environmental exposures; most also saw occupational exposures (97%) and provided toxicological laboratory result evaluations (97%); many saw unexplained symptoms with suspected toxicological exposure (87%), envenomation follow-up (67%), and post-acute overdose follow-up (60%); fewer reported seeing mold exposures (43%) and providing addiction medicine evaluations (20%). Twenty-four clinics (80%) reported the capability to expand clinic hours in response to an environmental toxicological emergency. Interest in a national outpatient clinic network was nearly universal: 29 (97%) were interested in participating.

Conclusion: Outpatient medical toxicology clinics were identified in 41% of U.S. states/jurisdictions, predominantly within academically affiliated clinical settings. These clinics report generally modest baseline clinic hours, but the majority reported their ability to surge in their capacity for consultation following an emergency. Strong interest (97%) supports exploring a national outpatient clinic network to coordinate communication and facilitate best-practice and resource exchange.

161. Does It Pay to Practice Medical Toxicology? Preliminary Results of a 2025 Compensation Survey

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Background: Specialty-specific physician compensation reports (e.g., emergency medicine) are widely available, but benchmarks for medical toxicologists are limited. This knowledge gap constrains healthcare organizations' ability to track market trends and physicians' ability to evaluate compensation and career options.

Hypothesis or Research Question: What are current compensation patterns in medical toxicology practice?

Methods: In August 2025, the American College of Medical Toxicology (ACMT) distributed an anonymous web-based survey to 186 medical toxicologists. Because practice patterns vary widely among board-certified medical toxicologists (e.g., many practice emergency medicine full time), we targeted fellowship faculty for this initial survey, assuming greater engagement and uniformity in medical toxicology practice. The questionnaire collected data across eight domains: (1) credentials/training; (2) total workload and professional time allocation across roles; (3) employment and academic context; (4) practice geography; (5) activities across the emergency department, inpatient/outpatient medical toxicology services, and poison center; (6) total professional compensation; (7) employer support; and (8) perceived compensation fairness. Analyses were descriptive; state-level and income categories with small counts were suppressed to protect confidentiality.

Results: Among respondents with compensation data (N=86; response rate 46%), total annual income most often fell between $300,000–$399,999 (57%); 26% reported $200,000–$299,999, and 17% reported ≥$400,000. Most common board certifications were Emergency Medicine (92%) and Addiction Medicine (30%). Most reported working 41–60 hours/week (59%); 22% worked 21–40 hours/week and 19% worked 61–80 hours/week. Primary employment setting was university-affiliated teaching hospital (80%); smaller proportions reported community hospital (5%), non-university teaching hospital (7%), poison center (5%), outpatient clinic (1%), government/public health (1%), or multiple settings (1%). Respondents averaged 11.6 years since fellowship completion, with total earnings associated with career stage ($P = 0.003$). The median proportion of total professional income supported by medical toxicology activities was 21–40%. Mean professional time allocation was dominated by emergency medicine (44%), followed by administration (17%), poison center services (16%), inpatient medical toxicology services (15%), research (14%), teaching (12%), inpatient SUD (10%), outpatient SUD (8%), legal/consulting (5%), and outpatient medical toxicology services (4%). Perceived compensation fairness was evenly split (50% yes; 50% no).

Conclusion: In 2025, total annual income for U.S.-based medical toxicology fellowship faculty most often fell between $300,000–$399,999, with higher earnings at later career stages. Findings reflect a self-selected sample; generalizability may be limited due to wide practice variability. Subsequent surveys should broaden sampling beyond fellowship faculty to better represent the full diversity of medical toxicology practice models.

162. Lifetime Exposure to Lead and Burden of Steatosis of the Liver Among Persons Living With or a History of IV Drug Use in Baltimore, Maryland

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Background: Baltimore City faces a persistent lead exposure crisis, with the highest percentage of children in Maryland exhibiting elevated blood lead levels. Because nearly 90% of the city's housing stock was built before 1980, chronic lead exposure remains a major environmental health concern. The AIDS Linked to the Intravenous Experience (ALIVE) Study, conducted at the Johns Hopkins Bloomberg School of Public Health since 1988, provided an opportunity to investigate the relationship between chronic lead exposure and hepatic steatosis among individuals living with or at risk for HIV infection.
Methods: Data were drawn from participants in the ALIVE Study, a prospective cohort of people who inject drugs in Baltimore, Maryland. Hepatic steatosis was identified using ultrasound-based Controlled Attenuation Parameter (CAP) scores, with values greater than 247 defining steatosis. Chronic lead exposure was measured noninvasively using X-ray fluorescence (XRF) bone lead analysis in approximately 500 participants. Demographic, socioeconomic, and clinical data were collected through structured interviews and examinations. Multivariable logistic regression models adjusted for age, sex, race, income, and body mass index (BMI) were used to assess associations between bone lead levels and steatosis.
Results: Among 129 participants with complete data, 82% were Black, 40% female, and 47% obese. One-third had bone lead levels ≥10 μg/dL. Elevated bone lead concentrations were strongly associated with hepatic steatosis among participants with a normal or underweight BMI (≤24.9), with an adjusted odds ratio (AOR) of 4.25 (95% CI: 1.46–12.32). Female participants in this BMI group also demonstrated higher odds of steatosis (AOR = 3.01; 95% CI: 1.05–8.69). No significant associations were observed among overweight or obese participants after adjustment.
Conclusion: Chronic lead exposure was significantly associated with hepatic steatosis among individuals with normal or low BMI, suggesting that environmental lead may contribute to liver fat accumulation independent of metabolic risk factors. These findings underscore the importance of addressing environmental determinants of liver disease in socioeconomically disadvantaged populations, particularly those enrolled in long-term cohorts such as the Johns Hopkins ALIVE Study.

163. Environmental Toxicants, Placental Dysfunction, and Fetal Brain Inflammatory Activation: A Transwell Co-Culture Model of Maternal-Fetal Interface

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Background: Placental dysfunction is a central contributor to intrauterine growth restriction (IUGR) and preeclampsia, leading causes of perinatal morbidity and mortality. Emerging evidence implicates environmental toxicants such as di(2-ethylhexyl) phthalate (DEHP), perfluorooctanoic substances (PFOAs), and other endocrine-disrupting chemicals in altering trophoblast function and impairing placental-fetal communication.
Hypothesis or Research Question: This study aimed to model these disruptions using an in vitro co-culture system mimicking the maternal-fetal interface as to understand downstream consequences on fetal neuroinflammation.
Methods: A transwell culture system was established using human trophoblast cells seeded in the apical chamber and placental endothelial cells in the basal chamber. Cultures were exposed to DEHP and PFOAs at physiologically relevant concentrations for 48 hours over four dosages. Conditioned media from the trophoblast-endothelial transwell cultures were collected, centrifuged to remove debris, and then applied to fetal human brain microvascular endothelial cells (HBMECs).
Results: Following exposure, trophoblast cells demonstrated significant downregulation of Connexin 43 (GJA1) and Syncytin-1 (ERVW-1), suggesting impaired cellular communication and fusion. Conversely, there was marked upregulation of Placental Growth Factor (PGF) and SLC2A1 (GLUT1), indicating a compensatory response to hypoxic or stress-induced conditions. Exposure to toxicant-conditioned media resulted in notable modulation of tight junction proteins and inflammatory markers in fetal human brain vascular endothelial cells. Specifically, Claudin-5 (CLDN5) was significantly downregulated, while Occludin (OCLN) was upregulated. There was also pronounced upregulation of interleukin-6 (IL-6), a key pro-inflammatory cytokine implicated in fetal neuroinflammation. Additionally, estrogen receptor expression and SLC2A1 were significantly downregulated, suggesting endocrine disruption and impaired glucose transport across the fetal blood-brain barrier.
Conclusion: These findings demonstrate that environmentally relevant toxicant exposures can disrupt trophoblast

signaling and induce pro-inflammatory and barrier-altering effects in fetal brain endothelial models. The transwell co-culture system provides a physiologically relevant platform for studying maternal-fetal toxicant transfer and neurovascular outcomes. These insights may inform mechanistic understanding of how environmental exposures contribute to adverse perinatal outcomes, and highlight the need for stronger environmental health surveillance and preventive strategies during pregnancy.

164. Ingestions of Guanfacine in Children Under 6 Years Old: Are We Too Conservative?

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Background: Alpha-2 agonists are increasingly prescribed for management of Attention Deficit and Hyperactivity Disorder and may be more accessible to siblings of children prescribed these medications. A common practice among poison centers is to automatically refer naïve alpha-2 agonist children to a healthcare facility. Our objective was to assess clinical effects and outcomes of ingestions that were ≤1mg and 0.05mg/kg in guanfacine naïve children.
Methods: This was a retrospective chart review of guanfacine ingestions reported to two poison centers from January 1st, 2010 to December 31st 2024. We included only single substance ingestions of guanfacine in patients under 6 years old. Patients who were not naïve to guanfacine were excluded. We assessed clinical effects including CNS depression, bradycardia, and hypotension.
Results: During the study period, there were 300 cases of single substance ingestions of guanfacine by children under 6 years old. Of these, there were 14 cases of patients who were naïve to guanfacine that ingested ≤1mg and 0.05mg/kg. Of these cases, 10 were referred to the emergency department and none of the 14 children developed symptoms. One child received activated charcoal.
Conclusion: Our pilot study demonstrated good outcomes, with no development of symptoms for guanfacine-naïve children who ingested low doses, defined as ≤1mg and 0.05mg/kg. Recommendations for home management of guanfacine ingestions in children under 6 years old is limited. A larger study looking at outcomes in guanfacine-naïve children is warranted to determine a safe, less conservative threshold for healthcare facility referral to avoid unnecessary pediatric emergency department visits.

165. State Cannabis Legalization and Pediatric Cannabis Poisonings in the United States: A Cross-Sectional Analysis Using Epic Cosmos

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Background: Unintentional pediatric cannabis poisoning is a growing public health concern as legalization expands across the United States (U.S.). Prior studies have shown increased exposures following legalization within individual states, but national analyses remain limited.
Hypothesis or Research Question: How is state recreational cannabis legalization associated with the incidence and evaluation of unintentional cannabis poisonings among young children?
Methods: We conducted a retrospective cross-sectional study of pediatric emergency department (ED) encounters (January 2015–June 2025) using Epic Cosmos, a database encompassing over 296 million patients across all 50 U.S. states. All ED encounters among children ≤5 years old (N = 1,497,705) were included. The primary outcome was the rate of cannabis poisoning encounters (identified by ICD-10 codes) per total pediatric encounters by state-month. State legalization status (prohibited vs. recreational) was linked to patient state of residence. Interrupted time series analyses were performed for states that legalized during the study period, using the recreational sales start date as the intervention. Multivariable logistic regression assessed demographic and socioeconomic predictors of cannabis poisoning and compared diagnostic utilization (blood testing, urine drug testing, computed tomography head imaging, and lumbar puncture) before and after legalization.
Results: From 2015–2025, 20 states began recreational cannabis sales, and 3,018 pediatric cannabis poisoning encounters were identified. Annual cases increased from 11 in 2015 to 768 in 2024. Interrupted time series analysis showed a transient decrease immediately after legalization ($p = .033$) followed by a sustained increase ($p < .001$). In multivariable models, older age (OR = 1.33, 95% CI 1.30–1.36), Black (OR = 2.04, 95% CI 1.88–2.25) and Native American (OR = 2.65, 95% CI 2.16–3.26) race, Medicaid (OR = 1.28, 95% CI 1.19–1.39) or self-pay insurance (OR = 3.03, 95% CI 2.43–3.78), and higher Social Vulnerability Index (OR = 2.20, 95% CI 1.91–2.53) were associated with increased risk. Female sex (OR = 0.75, 95% CI 0.70–0.81), Hispanic

ethnicity (OR = 0.63, 95% CI 0.56–0.70), and Asian (OR = 0.44, 95% CI 0.32–0.61) or Pacific Islander (OR = 0.36, 95% CI 0.16–0.79) race were associated with decreased risk. No significant differences were observed in diagnostic testing or imaging.
Conclusion: Unintentional pediatric cannabis poisonings have risen significantly nationwide, with legalization associated with increased healthcare encounters among young children. Diagnostic evaluation remained unchanged. Prevention efforts, such as child-resistant packaging, caregiver education, and targeted interventions in socially vulnerable communities, are warranted as legalization expands.

166. Two Decades of Accidental Pediatric Poisoning Deaths: A Retrospective Analysis of Epidemiologic Patterns and Emerging Challenges

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Background: This study is aiming to describe the epidemiologic and clinical patterns in accidental pediatric poisoning deaths from a pediatric poisoning centre
Methods: This retrospective study included all cases of fatal unintentional poisonings in patients under 18 years of age admitted to the Pediatric Poison Center between January 2004 and December 2024. Data were collected from hospital medical records and toxicological reports. Statistical analyses were performed using XLSTAT and MedCalc software.
Results: A total of 32 pediatric deaths due to accidental poisoning were recorded between 2004 and 2024.The majority of patients were male (59.3%), with a male-to-female ratio of 1.5:1. The highest number of cases was recorded in 2006 (6 cases), followed by 2005 and 2013 (4 cases each). Analysis of temporal trends revealed a significant decline after 2006 (linear regression, $R^2 = 0.42$, $p = 0.01$). Regarding residence, 71.8% of the cases(n= 23) originated from rural areas. The average age was 4.71 years and the average hospitalization period 8.65 days(with a maximum of 36 days). The 0–5 years age group was the most commonly affected (65.6%, n = 21). The most frequently implicated substances were organophosphate insecticides (34.3%, n= 11), followed by caustic agents(15.6%, n = 5), and pharmaceutical drugs (n= 4), including paracetamol and diltiazem. Other toxic agents included non-organophosphate insecticides and toxic mushrooms (n = 3 each), while hydrocarbons, carbon monoxide, and nitrites were each involved in two cases. The most frequent organ dysfunction involved the CNS, with coma and convulsive status commonly leading to death. Pulmonary complications, including acute pulmonary edema, acute respiratory failure, and occasional pneumothorax, were also major contributors to the fatal outcome. Acute toxic cardiomyopathy was identified in several cases, reflecting the multiorgan impact of severe poisoning. Children under 2 years were significantly more likely to be exposed to caustic agents and organophosphate insecticides (Fisher's exact test, $p = 0.03$). Furthermore, there was a strong association between type of toxic agent and organ involvement, with organophosphate exposure predominantly linked to neurological and pulmonary complications ($\chi^2 = 12.7$, $p = 0.005$).
Conclusion: Accidental poisoning predominantly affected children under 5 years, with organophosphate insecticides and caustic agents being the most frequent toxicants. Fatalities were strongly associated with neurological and pulmonary complications, reflecting the multiorgan impact of severe poisoning. Despite a decline in deaths after 2006, rural children remain disproportionately affected, highlighting the need for targeted prevention strategies.

DAY 3: POSTERS, ABSTRACTS 167-231

167. Acute Grayanotoxin Intoxication Due to Rhododendron Flower Consumption in Himalayan Region of Western Nepal

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Background: Rhododendron flowers from Nepal's Himalayas contain grayanotoxin, and the flowers are often used in alternative medicine. However, it can cause severe side effects like vomiting, hypotension, and bradyarrythmias.
Hypothesis or Research Question: What are the manifestations of acute grayanotoxin intoxication due to consumption of rhododendron flowers?
Methods: This is a case report discussing an event of acute grayanotoxin intoxication in Himalayan region of western Nepal. A 26-year-old male presented with epigastric pain, dizziness, vomiting, generalized weakness, and blurring of vision 2 hours after consuming around 20 dried petals of rhododendron flowers grown at high-altitude (2800 meters) of western Nepal. He had no known comorbidities. In the emergency department, he had severe dizziness, low blood pressure of 90/62 mm Hg, heart rate of 45 beats per minute, and oxygen saturation of 92% at room air.
Results: Routine laboratory examinations were unremarkable. Toxicological analysis could not be performed due to unavailability in resource-limited Himalayan region.

Electrocardiogram showed sinus bradycardia. Cardiac markers were negative. With a positive history of flower consumption and no known established cause for the symptoms, acute grayanotoxin intoxication was suspected. He received supportive treatment with atropine, proton pump inhibitor, IV fluid, oxygen, and ondansetron. His condition gradually improved, and he was discharged after 48 hours with stable vitals and complete recovery. Follow-up examinations showed stable vitals and no symptoms.
Conclusion: Plant products are used as alternative remedies, alternative food sources and delicacies in many Himalayan regions of Nepal. Nepal harbours more than 33 species of rhododendron varying in sizes and colors. Grayanotoxins form in plants of family Ericaceae including certain rhododendron species depending on geography, altitude and environmental factors. Ingestion of petals result in acute intoxication leading to nausea, vomiting, hypotension, bradycardia, blurring of vision and dizziness. Grayanotoxins bind to sodium channels in the cell membrane, preventing their inactivation leaving excitable tissues, including nerve and muscle cells, in a state of depolarisation, facilitating entry of calcium into the cells. The range of adverse effects by this toxin on the cardiovascular system include systemic hypotension, bradyarrhythmias, and atrioventricular block. Treatment by atropine and IV fluids is typically sufficient. Being a rare condition with potential disastrous complications, clinicians should consider this in patients presenting with unexplained hypotension and bradycardia and a history of consumption of plant products. Community awareness is vital in areas that frequently use plant products for medicinal purposes.

168. Carbon Monoxide Poisoning Among Children in Pennsylvania and Delaware From 2020-2024: A Cross-Sectional Study

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Background: Carbon monoxide (CO) poisoning uniquely affects children due to biological causes and behavioral reasons: children have higher minute ventilation, higher metabolic rates, and typically spend more time in residential settings than adults. Carbon monoxide exposure is highly influenced by environmental factors and as such, detecting epidemiological trends in CO poisoning assists in targeting preventative interventions to reduce rates of exposure to dangerous levels of CO among children.
Hypothesis or Research Question: What is the crude incidence of CO poisoning cases among children and what is the severity of medical outcomes in Pennsylvania (PA) and Delaware (DE) from January 2020 through December 2024?
Methods: Cases were identified via the National Poison Database System (NPDS), and discreet fields from a blinded dataset were analyzed. Cases from PA and DE reported to PA's poison centers for individuals younger than 20 years old with a substance coded as "carbon monoxide" (generic code: 106000) from January 2020 to December 2024 were included. Cases later deemed to be non-exposures were excluded from the analysis. Crude incidence rates were estimated using 2019-2023 American Community Survey census data.
Results: From 2020 to 2024, PA's poison centers recorded 786 cases of pediatric carbon monoxide poisoning from PA and DE. The overall crude incidence rate of poison center CO-related cases was 4.8 per 100,000 person-years. These rates were highest among infants <1 year of age (16.9 per 100,000 person-years) and lowest among children 15-19 years in age (2.7 per 100,000 person-years). Cases primarily occurred in the winter (N=289, 37%) and fall (N=199, 25%). Moreover, most cases (N=742, 94%) reported the exposure site as a residential setting. Of cases with medical outcomes determined (N=435, 55%), there was a spectrum of severity with 364 cases with no/minor effects, 63 cases with moderate/major effects, and eight cases with fatal outcomes. Of the cases with reported mortality, five (62%) had reported co-exposures with cyanide or house fire smoke inhalation. In contrast, among non-fatal cases, carbon monoxide poisoning rarely co-occurred with house fires or cyanide poisoning, including cases with documented mild/moderate effects (N=2 of 364, 0.5%) and moderate/major effects (N=1 of 63, 2%).
Conclusion: Eight child deaths and 786 pediatric CO poisoning cases were reported to Pennsylvania and Delaware poison centers in five years, indicating a continued need for injury prevention. Infants are disproportionately affected by CO poisoning. Residential spaces represent a key setting for targeted interventions to reduce these poisoning rates.

169. Significantly Elevated False Positive Digoxin Assay and Cost Savings of Timely Identification

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Background: False positive digoxin assays may occur secondary to endogenous digoxin-like immunoreactive substances (DLIS). Serum elevations in DLIS develop in liver disease, preeclampsia, and uremia. Typically, these false positives result in a digoxin concentration less than 6 ng/ml.
Hypothesis or Research Question: False positive digoxin assays with concentrations >6 ng/mL can occur.

Methods: This is a single patient case report. A 97-year-old man with hypertension, paroxysmal atrial fibrillation, and stage V chronic kidney disease presented to the emergency department from an assisted living facility for reported dyspnea. He arrived bradycardic, hypotensive, and in respiratory distress. He was stabilized with bilevel positive pressure ventilation (BiPAP). An EKG demonstrated atrial fibrillation with a ventricular rate of 50 beats per minute. Due to his initial EKG findings and an unclear medication history, a digoxin level was sent, which came back elevated at 19.5 ng/mL (normal range: 0.7 ng/mL – 2.0 ng/mL). Notable laboratory testing included a blood urea nitrogen of 125 mg/dL, creatinine of 10 mg/dL, potassium of 6.8 meq/L, lactic acid of 8.8 mmol/L and an AST/ALT over 1000 IU/L. He was given a dose of atropine with improvement of his heart rate. The administration of digoxin immune fab was withheld after discussion of the case with medical toxicology, and a repeat digoxin level drawn two hours later was 0.2 ng/mL. Goals of care were discussed with the patient's daughter who opted for comfort care. The patient was admitted to inpatient hospice and expired six days later.
Results: In this case, the initial digoxin concentration obtained was suspected to be false positive given improvements in hemodynamics with atropine alone and a later medication review with patient's nursing facility. However, the concentration was high enough for treatment to be indicated regardless of the timing of digoxin ingestion and higher than previously reported false positives from DLIS making the decision to withhold treatment difficult. After review of the case by a medical toxicology, supportive care was continued and digoxin antibody Fab was not given. By appropriately withholding digoxin immune Fab in this case, the hospital saved ~$69,432.60. We considered whether an incorrect sample had been tested however no patient was concurrently being treated for digoxin toxicity in our hospital system.
Conclusion: False positive digoxin concentrations that would necessitate antidotal therapy can occur. Antidotal treatment should be guided by patient history, physical examination, and a clinical course consistent with digoxin toxicity.

170. Acute Poisoning by Chemicals in Spain. Results of the Spanish Toxic Surveillance System (STSS) – 2024

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Background: The Spanish Toxic Surveillance System (STSS), established in 1999 by the Ministry of Health in collaboration with a group of Clinical Toxicologists from public hospital Emergency Departments (EDs), aims to record cases of acute chemical poisoning to assess exposure risks under current EU regulations. This report presents the 2024 results of the Program.
Methods: Participating hospitals report all cases of poisoning caused by household, agricultural, or industrial chemicals treated in their EDs. Data are collected through an online questionnaire accessible 24/7 via the FETOC website using an encrypted system. The records are regularly downloaded into a database (FileMaker 9.0®) for analysis, allowing the preparation of an annual report submitted to the Ministry of Health.
Results: In 2024, the Program recorded 1,300 cases from 20 hospitals, covering an estimated population of 10 million. The median patient age was 41 ± 23 years, with an even sex distribution (624 men, 676 women). Patients under 16 years represented 16% of the total. Domestic accidents were the most frequent cause (70%), followed by occupational exposures (12%) and suicide attempts (10%) ($p < 0.05$). The main toxic agents involved were toxic gases (30%), caustics (26%), irritant gases (17%), solvents (10%), detergents (7%), and pesticides (4%). The primary routes of exposure were respiratory (50%) and oral (35%), while ocular (12%) and cutaneous (7%) exposures were less frequent ($p < 0.05$). At admission, 77% of patients were symptomatic, presenting mainly with digestive (28%), respiratory (26%), neurological (20%), or ocular (13%) symptoms, most of them mild. Treatment was provided in 76% of cases, mostly symptomatic (56%). Antidotes were administered in 30%: oxygen in 281 CO exposures (9 of them combined with hydroxocobalamin), and ethanol or fomepizole in two methanol poisonings. No specific antidotes were used for pesticide exposures, except for vitamin K in three rodenticide cases. Only 13% of patients required hospitalization beyond 24 hours, and 3% (41 patients) were admitted to the ICU. The overall mortality rate was 0.69%, including 5 suicides (4 by hydrochloric acid and 1 by bleach) and 4 domestic accidents involving CO/HCN inhalation during house fires.
Conclusion: Acute chemical poisonings in Spain are generally low-risk events, mostly due to domestic accidents involving toxic gases (CO) and caustic cleaning products. The STSS electronic platform, which enables multicentric and continuous data collection from a representative network of EDs, proves to be a valuable tool for ongoing national toxicosurveillance of chemical incidents.

171. 7-Hydroxymitragynine Overdose in a Pediatric Patient

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Background: Kratom products are increasingly popular in the United States with widespread commercial availability both

online and in smoke shops. Mitragynine is the major active alkaloid in kratom, and its metabolite 7-hydroxymitragynine (7-OH) has emerged as a separate substance of concern as manufacturers develop concentrated 7-OH products. In vitro studies suggest that while mitragynine and 7-OH demonstrate variable mu-opioid receptor activity, they are distinct from classical opioids and may impart less risk of respiratory depression. To our knowledge, this is the first documented case of 7-OH overdose in a pediatric patient, highlighting the potential severity of toxicity of these products

Hypothesis or Research Question: Clinically significant respiratory depression can occur after acute, single-substance pediatric ingestion of 7-hydroxymitragynine.

Methods: This is a single patient chart review at a tertiary care center. Basic labs and imaging were obtained, and a comprehensive drug panel was performed by liquid chromatography with tandem Quad-Time of Flight (QTOF) mass spectrometer.

Results: A 2-year-old male with no previous medical history presented to the emergency department (ED) by ambulance for altered mental status. He developed somnolence and respiratory depression requiring endotracheal intubation in the ED. Initial work-up included a computed tomography of the head and cervical spine, chest X-Ray, complete blood count with differential, complete metabolic panel and high sensitivity troponins, which were all normal. History obtained from mom revealed that she had used kratom products for years to treat chronic pain and had a prior history of opioid dependence. She began using 7-OH products over recent months and noticed two 30 mg 7-OH capsules missing from her purse that was located at the scene where the patient became symptomatic. A comprehensive urine drug panel performed by QTOF detected both 7-OH and mitragynine. The other presumptive compounds detected included rocuronium, methylprednisolone, and trans-3-hydroxycotinine. The patient was extubated within 24 hours of exposure and returned to neurologic baseline within several days.

Conclusion: Although kratom-associated ED presentations typically involve withdrawal or polysubstance use, overdose resulting in opioid toxicity are less frequently reported. Mitragynine and 7-OH are considered partial mu-opioid receptor agonists with relatively less beta-arrestin activity, characteristics that have been thought to confer a lower risk of respiratory depression compared with traditional opioids; however, this case highlights that clinically significant respiratory compromise can still occur. Pediatric patients may be uniquely vulnerable due to smaller body mass relative to product concentration and potentially increased sensitivity of the blood-brain barrier to central opioid effects.

172. CB1-Receptor Antagonist Selonabant Blocks and Reverses THC Intoxication in Healthy Adults: A Phase II Randomized, Controlled Trial

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Background: Widespread use and increasing tetrahydrocannabinol (THC) concentration of cannabis products have increased emergency department visits due to acute cannabis-induced toxicity, including acute cannabinoid intoxication (ACI). This study assessed the potential of cannabinoid receptor type 1 (CB_1) antagonist selonabant for treatment of acute THC intoxication in healthy adults.

Hypothesis or Research Question: To determine the efficacy of selonabant to reverse acute cannabis-induced toxicity.

Methods: This was a randomized, double-blind, placebo-controlled study of single oral doses of selonabant or placebo administered 1 hour after THC oral doses of 21, 30 or 40 mg, followed by an open-label phase, where selonabant was orally co-administered with 40 mg or 60 mg THC. Primary outcomes were THC-related effects on visual analogue scales (VAS) for feeling high and alertness, and objective measures of postural stability and heart rate, analyzed using a linear mixed effects model.

Results: Forty-nine participants were enrolled in the delayed dosing phase and 20 were enrolled in the open-label co-administration phase. Delayed selonabant dosing effectively reversed THC effects on VAS "Feeling High" (-80.2%, 95% CI: -89.4%, -63.0%, $p<0.0001$), body sway (-32.4%, 95% CI: -46.1%, -15.3%, $p=0.001$) and heart rate (-8.1 bpm, 95% CI: -14.4, -1.9 bpm, $p=0.013$), and increased VAS "Alertness" (11.3 mm, 95% CI: 4.4, 18.3 mm, $p=0.002$) compared to placebo in participants dosed with 21 or 30 mg THC (Figure 1). Co-administration of selonabant blocked the effects of 40 and 60 mg THC, demonstrating protection from THC-induced toxicity in this clinical model of ACI.

Conclusion: Results of this study support further development of selonabant for treatment of acute cannabis-induced toxicity.

173. Ceramic Hip Replacements Risk Systemic Cobalt Toxicity Following Revision Surgery

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Background: Total hip arthroplasty is one of the most common orthopedic procedures with roughly half a million primary surgeries in the US annually. After primary hip replacement, many patients require revision surgery within three decades. Currently, the main prosthetic material options include metal-on-metal, metal-on-polyethylene, ceramic-on-polyethylene, or ceramic-on-ceramic. The metal-on-metal approach has largely been abandoned due to cases of systemic cobalt/chromium toxicity in the setting of abrasive wear and tear and possible electrochemical corrosion. Ceramic prostheses are generally regarded as safe, however they carry the risk of fracture. If fracture occurs, residual ceramic particles predispose patients to metallosis and systemic metal toxicity following eventual revision surgeries involving metal implants due to abrasive forces within the joint space.
Hypothesis or Research Question: Revision hip surgery following ceramic fracture may result in severe systemic cobalt toxicity.
Methods: This study reviews the chart of a single patient. A 45-year-old woman with history of congenital hip dysplasia status post hip replacement at age 23 experienced progressive hip pain prompting revision surgery at age 44. At that time, the ceramic liner was found to be fractured. A cobalt-chromium dual-mobility liner and femoral head were placed. Four months later, she experienced vertigo, hair loss, and weight loss, followed by bilateral sensorineural hearing loss refractory to corticosteroid treatment. Five months after the revision, her plasma cobalt concentration was found to be markedly elevated at 991 ng/mL (reference range <0.9 ng/mL).
Results: The patient underwent repeat revision surgery roughly one week later. At that time, significant metallosis was noted behind the liner, which was removed and replaced with a polyethylene liner and ceramic head. Her plasma cobalt concentration decreased to 130 ng/mL following surgery. Her whole blood cobalt concentration at that time was 537 ng/mL (reference range <3 ng/mL). The patient then underwent chelation with two courses of succimer and one course of intravenous N-acetylcysteine. Her plasma cobalt concentrations and whole blood cobalt concentrations downtrended over the next six months to 14.2 ng/mL and 18.4 ng/mL, respectively. The patient also underwent hyperbaric oxygen treatments for her ototoxicity, and she experienced partial improvement in hearing acuity. She never experienced classic cobalt toxicity signs or symptoms such as cardiotoxicity, acute thyroid dysfunction, or paresthesias.
Conclusion: Revision hip surgeries utilizing cobalt prostheses in the setting of prior fractured ceramic prostheses can risk severe systemic cobalt toxicity requiring extensive therapies.

174. Carbapenem Therapy for Valproic Acid Toxicity

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Background: Carbapenems lower serum valproic acid (VPA) concentrations in patients with therapeutic VPA use. The safety and efficacy of this effect is not well studied in VPA toxicity.
Hypothesis or Research Question: We hypothesize that carbapenem therapy (CBT) can be safely used to lower the serum concentration of VPA in toxicity
Methods: This is an IRB exempt retrospective observational study from state poison control center and a regional academic medical center data of all VPA exposures called to the state poison center between January 1, 2024 – September 30, 2025, regardless of age. Use of CBT to treat VPA toxicity was considered on a patient-by-patient basis in consultation with a medical toxicologist. Cases were excluded if the highest serum VPA concentration was ≤100 mcg/mL, if seizures were present, or if the patient had a seizure disorder. Risk and benefit were considered when there was co-ingestion of a substance that could cause seizures. Outcomes studied were a decrease in VPA concentration after CBT, adverse effects from CBT (seizures or allergic reactions), and extracorporeal treatments.
Results: Three hundred twenty-three patients were evaluated by the poison center for VPA exposure. Seven patients received CBT for VPA toxicity. Six of these patients had subsequent decreasing VPA concentrations after CBT. In one patient we were unable to determine if their VPA concentration increased by 3 mcg/mL or decreased since time of CBT was not documented. Six of the patients had VPA concentrations that were below 100 mcg/mL within 18 hours of starting CBT. One patient did not have VPA concentrations

trended until below 100 mcg/mL. Six patients did not have seizures after CBT, including 2 patients with a history of seizures. One patient with a history of seizures had seizures prior to CBT, continued to have seizures after CBT. Two patients required extracorporeal replacement therapy, which was started concurrently with CBT due to encephalopathy and hyperammonemia.
Conclusion: All but one patient had a post-CBT VPA concentration that was lower than their pre-CBT VPA concentration. Reduction of supratherapeutic VPA concentrations by CBT was similar to the reported reduction in therapeutic VPA concentrations with carbapenems used for antimicrobial effects. In our series of 7 patients, 6 patients did not have any reported adverse effects. Two patients received extracorporeal treatment, this was due to therapeutic intervention and not due to CBT failure.

175. Tox Case of the Month

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Background: Toxicology education is necessary in emergency medicine (EM) training, but exposure to real-world tox cases may be sporadic. Asynchronous, case-based learning provides an opportunity to engage residents, strengthen pattern recognition, and reinforce clinical reasoning longitudinally beyond formal rotations, all without disrupting clinical responsibilities.
Hypothesis or Research Question: Will EM residents participate in and engage with voluntary, monthly, email-based toxicology case challenges?
Methods: This is a prospective, observational study involving 52 trainees across two EM residency programs: 46 EM MD/DO Residents and six EM PA Residents.
A monthly case-based email curriculum was implemented, supplemented by weekly "clue" emails to sustain engagement and reinforce learning. Cases were developed and reviewed by toxicology trained faculty, then distributed electronically. Participants submitted diagnostic answers, received individualized feedback, and accumulated points tracked on a leaderboard to promote gamification. Engagement metrics were collected throughout the intervention period.
Results: During the first three months (three cases), a total of 34 submissions were received from 20 unique participants. Some residents resubmitted corrected answers after receiving additional clues. Measures of engagement, including number of submissions, time to first response, and time to correct response, all improved after the first month. The number of unique first-time responders remained stable over the three-month period.
Conclusion: EM residents demonstrated engagement with asynchronous, case-based toxicology education. These findings support the effectiveness of low-effort, email-based toxicology education as a means of promoting continuous learning within EM residency programs.

176. Infusion of N-Acetylcysteine Causing Laboratory Artifact Mimicking Diabetic Ketoacidosis

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Background: N-Acetylcysteine (NAC) is one of the most commonly used antidotes in toxicology. In vitro studies have shown NAC produces aberrations in laboratory measurement of glucose, creatinine, and bilirubin, but NAC infusion has not previously been associated with laboratory artifact that mimics diabetic ketoacidosis.
Hypothesis or Research Question: Obtaining blood samples for laboratory analysis from the same limb that is recieving an infusion of NAC may lead to false measurement of hyperglycemia and elevated anion gap.
Methods: This was a single patient chart review. A 40-year-old non-diabetic woman presented with a repeated supratherapeutic acetaminophen ingestion. On presentation to a community hospital, AST was 5259 IU/L, bilirubin was 3.2 mg/dl, and INR was 1.8. She was initiated on NAC and transferred to a tertiary care hospital. She continued to receive NAC infusion at 12.5 mg/kg/hr and laboratory studies were serially obtained.
Results: Initial laboratory studies were: sodium of 139 mEq/L, chloride 103 mEq/L, potassium 3.5 mEq/L, and CO2 of 20 mEq/L, yielding an anion gap of 16 (Na-Cl-CO2). Glucose concentration was 121 mg/dL, and baseline creatinine was 1.32 mg/dL. Testing 6 hours later revealed sodium 141 mEq/L, potassium 2.8 mEq/L, chloride 98 mEq/L, CO2 21 mEq/L, glucose 282 mg/dL, creatinine 0.31mg/dL, and anion gap 22. The rising glucose and anion gap raised concern that the patient was inexplicably developing diabetic ketoacidosis. Repeat labs were obtained again in 4 hours, now with the sodium 144 mEq/L, potassium 3.2 mEq/L, chloride 94 mEq/L, CO2 19 mEq/L, glucose 433 mg/dL, creatinine <0.2 mg/dL, and anion gap 33. Lactate was undetectable, but beta-hydroxybutyrate was >6.0 mmol, obtained by beta-hydroxybutyrate dehydrogenase assay. The pattern suggested contamination with exogenous intravenous fluids. It was discovered that although the patient's nurse was properly wasting 3 mL of blood, the samples were obtained from an intravenous catheter on the same arm into which the NAC was infusing. When samples were taken from a vein in the contralateral arm, sodium was 136 mEq/L, potassium 3.6 mEq/L, chloride 105 mEq/L, CO2 21 mEq/L, glucose 102 mg/dL, creatinine 1.14 mg/dL,

and anion gap 9. Serial testing demonstrated that obtaining blood samples from the arm opposite the NAC infusion eliminated the laboratory artifact.
Conclusion: NAC infusion may cause laboratory results falsely indicating diabetic ketoacidosis if blood samples are obtained from the limb receiving NAC infusion.

177. We Need a Bigger Kitchen Sink: The Rise of Methylene Blue and Hydroxocobalamin in Calcium Channel Blocker Toxicity

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Background: Calcium channel blocker (CCB) overdoses are among the most lethal prescription medication exposures reported to the American Association of Poison Control Centers, primarily due to severe cardiovascular collapse characterized by decreased cardiac inotropy and chronotropy, along with profound vasoplegia. In cases of vasoplegic shock refractory to traditional high-dose vasopressor therapy, methylene blue and hydroxocobalamin have emerged as non-adrenergic adjunct treatments, although national utilization trends have not been well described.
Hypothesis or Research Question: National trends in the use of methylene blue and hydroxocobalamin as adjunct therapy to vasopressors in CCB overdose were evaluated over a ten-year period.
Methods: This is a retrospective review using data from the National Poison Data System (NPDS). Aggregate data from intentional CCB overdoses reported between 2015 and 2024 was analyzed for medical outcomes and the use of methylene blue and hydroxocobalamin in addition to vasopressor therapy. Descriptive statistics were used to characterize national trends in coded treatments and outcomes in NPDS over the ten-year period.
Results: The number of intentional CCB overdoses reported to poison centers increased from n=2,519 in 2015 to n=3,309 in 2024, with a total of 30,662 cases over the ten-year period. Reported fatalities increased from n=118 (4.7% of all intentional CCB overdoses) in 2015 to n=166 cases (5%) in 2024. Vasopressor use increased from n=629 (25%) in 2015 to n=1,015 (30.7%) in 2024, and in this subset there were corresponding increases in the use of methylene blue and hydroxocobalamin from n=25 (4%) to n=180 (17.7%) and n=1 (0.2%) to n=50 (4.9%), respectively.
Conclusion: In this ten-year review, there was an increased use of methylene blue and hydroxocobalamin as adjunctive therapies to vasopressors in intentional CCB overdoses reported to NPDS. The growing adoption of these treatments is notable given their distinct mechanisms in targeting nitric oxide–mediated vasodilation in vasoplegic shock. However, evidence supporting their routine use remains limited to case reports and small case series. Further research is needed to clarify their effect on clinical outcomes and better define their role in treating refractory vasoplegia in CCB poisoning.

178. T-1 Weighted Basal Ganglia Hyperintensities Secondary to Chronic Dietary Supplement

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Background: Basal ganglia damage is often associated with a constellation of parkinsonian-like symptoms. Etiologies vary and include primary neurodegenerative, hepatic dysfunction, ischemia, and toxin mediated. The latter include carbon monoxide, copper, methanol, kratom and manganese. Manganism can present with cognitive abnormalities such as deficits in attention and memory; classically, it is associated with extrapyramidal movement abnormalities including bradykinesia, rigidity, postural instability and gait disturbance.
Hypothesis or Research Question: Chronic use of over-the-counter dietary supplements can result in hyperdensities on MRI due to manganese deposition.
Methods: This is a case report of a 77-year-old female with a history of ethanol-induced cirrhosis who was referred to our outpatient medical toxicology clinic for an elevated blood manganese concentration. Neurology had previously evaluated her for mild cognitive impairment and workup was notable for an MRI demonstrating T1-hyperintensity in the basal ganglia along with an elevated whole blood manganese concentration. Other labs including heavy metal screen for mercury, arsenic, lead and copper were below detection limits, and her autoimmune workup was negative. Further history revealed she was taking an over-the-counter dietary supplement: Ligaplex II, which contained 35 mg of manganese (1,522% of the recommended daily value) for approximately 20 years. Her physical exam displayed mild stooped posturing with decreased arm swing. No other significant parkinsonian features were noted. She was given dietary guidance on high manganese containing foods. Manganese supplements are high risk in cirrhosis patients as manganese is excreted primarily via the bile and can gradually accumulate. Risk of chelation therapy with Na_2Ca-EDTA outweighed potential benefits and para-aminosalicylic acid was contraindicated due to her liver disease.
Results: Manganese concentrations were obtained via quantitative inductively coupled plasma-mass spectrometry (ICP-MS) with an initial whole blood concentration of 27.2 μg/L (Reference range 4.2 - 16.5 μg/L). Her neurologist instructed her to discontinue her supplement and her

manganese concentration down-trended to 18.4 μg/L prior to outpatient toxicology evaluation.
MRI-dementia protocol showed T1 hyperintensity of the bilateral basal ganglia most prominently involving the globus pallidus.
Conclusion: Chronic dietary supplements with elevated manganese content can result in mild cognitive symptoms and bilateral basal ganglia lesions without significant parkinsonism symptoms.

179. Transgender and Nonbinary Representation and Reasons for Calling to One Poison Center

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Background: Transgender and nonbinary people experience high rates of stigma and discrimination, and the number of self-poisonings among transgender people worldwide has increased over the past several years. A lack of data regarding healthcare utilization (and reason for utilization) is a barrier to studying the unique risks populations may face.
Hypothesis or Research Question: Transgender and nonbinary patients are under-identified in poison center calls and have a higher proportion of self-harm ingestions compared to an age-matched cohort of cisgender-assumed patients.
Methods: This was a retrospective, observational study using a regional poison center database from 2011 through September 2025. We queried free-text notes for terms suggesting that the patient was transgender (e.g., 'FTM', 'female to male', 'nonbinary', etc). We identified an age-matched control group to compare the age and reasons for exposure between cases and controls. This analysis was restricted to ages 13-65 years, with no limit on the number of controls. We also described the proportion of our database's transgender cases with transgender population data published by the UCLA Williams Institute.
Results: There were 685 transgender patients and 214,529 controls. Of the transgender patients, there were 275 transgender women (40.1%), 299 transgender men (43.6%), 80 nonbinary (11.7%), and 31 that could not be determined (4.5%). In the control group, the sex was 59.3% female, 40.5% male, and 0.01% unknown. Average age was 23 years in the transgender group and 34 years in control group. In the transgender group, 'Intentional – suspected suicidal' was the reason in 76.9% of calls, versus 17.9% of calls in the control group. There were fewer cases of misuse (1.6% vs 22.6%), therapeutic error (6.7% vs 18.3%), and abuse (3.8% vs 6.5%) in the transgender group (all $p < 0.01$). Our database had a low proportion of transgender patients. In 2016, 0.19% of 13-17-year-olds in our database were transgender, whereas in the same age group, approximately 1.1% were transgender in the state. In 2023, this changed to 0.84% transgender cases compared to approximately 3.4% transgender in this age group in the state. This was consistent across all age groups.
Conclusion: Our transgender cohort was younger than the age-matched cohort, had a higher proportion of suicidal ingestions, and had a lower proportion of abuse ingestions. Identified transgender patients were significantly underrepresented relative to our state's population, possibly indicating under-reporting, under-coding, or under-utilization of the center. Limitations include being a single center study and use of a poison center database.

180. Pediatric Alpha-2 Adrenergic Receptor Agonist Ingestions Reported to the National Poison Data System From 2013-2023

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Background: Alpha-2 adrenergic agonists are frequently implicated in exploratory and intentional pediatric ingestions and carry risks of cardiovascular and neurologic toxicities. Our objective was to identify the demographics, outcomes, and treatments used for pediatric exposures to alpha-2 adrenergic agonists reported to Poison Centers.
Methods: We performed a retrospective review of data obtained from the National Poison Data System (NPDS) for cases of single-agent exposures to alpha-2 adrenergic agonists in patients age ≤ 19 years for whom a Poison Center was consulted between 01/01/2013 and 12/31/2023. Fatality narratives were reviewed individually.
Results: 75,309 cases were identified, of which 1793 (2.4%) reported a major clinical effect. Intentional exposures accounted for 14,466 cases (19.2%) and were most common in patients age 13-19 years. 10,883 (14.5%) patients were admitted to an ICU, of which 2,489 were classified as having minor or no clinical effects.
The most common effects reported were mild CNS depression (26,366 cases, 35.0%), bradycardia (13,864 cases, 18.4%), and hypotension (8,287 cases, 11.0%). Patients 13-19 years old developed bradycardia (35.8% vs. 13.2%) and hypotension (21.69% vs. 7.8%) more often than patients < 13 years old, but had similar rates of major effects (2.9% vs. 2.2%). Other notable effects included coma (813 cases, 1.1%), respiratory depression (1,556 cases, 2.1%), respiratory arrest (61 cases, 0.1%), heart block (10 cases, < 0.1%), QTc prolongation (101 cases, 0.1%), and QRS prolongation

(17 cases, < 0.1%). Four cases (< 0.1%) resulted in cardiac arrest. The most-commonly administered treatments were IV fluids (16,582 cases, 22.0%), naloxone (5,660 cases, 7.5%), and single-dose activated charcoal (3,915 cases, 5.2%). Additional notable treatments included atropine (1,826 cases, 2.4%), vasopressors (573 cases, 0.8%), cardioversion (1 case, < 0.1%), pacing (4 cases, < 0.1%), intubation (1,272 cases, 1.7%), and CPR (39 cases, 0.1%).
There was 1 fatality, an intentional ingestion in a 19-year-old patient who was found down with severe metabolic acidosis and shock and subsequently suffered cardiac arrest.
Conclusion: In this multi-year national dataset, alpha-2 adrenergic receptor agonist exposures were primarily associated with cardiovascular and neurologic effects. Exposures were infrequently associated with severe effects and rarely required critical care treatments. Of patients admitted to an ICU, 22.9% were reported as having either minor or no clinical effects, suggesting an opportunity for cost-savings and improved resource utilization. Bradycardia and hypotension occurred more often in patients 13 and older, likely due to higher rates of intentional and higher-dose ingestions. These patients may be more likely to require cardiac monitoring and/or admission.

181. Amlodipine Overdose With Refractory Vasoplegia Treated With VA-ECMO

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Background: Amlodipine is a dihydropyridine calcium channel blocker which can result in life threatening cardiogenic and vasoplegic shock. Rescue options in refractory vasoplegic shock are limited and the role of VA-ECMO is not well defined.
Hypothesis or Research Question: VA-ECMO may still have a role in the vasoplegic shock present in severe amlodipine overdose as an adjunct to traditional therapies such as escalating vasopressors, methylene blue and hydroxocobalamin.
Methods: This is a single patient chart review of a 45 year-old female who initially presented to an outside hospital after intentional overdose on amlodipine, lisinopril, ibuprofen and trazodone and was ultimately transferred to a tertiary care center for ECMO cannulation. The patients chart was reviewed for vital signs, medication therapy, clinical course and diagnostic data.
Results: Patient was initially treated at an outside hospital, where she was initiated on Levophed, vasopressin and Neosynephrine to max doses of 2mcg/kg/min, 0.03 U/min and 3mcg/kg/min respectively. Despite increasing vasopressor requirements, intralipid and methylene blue, patient demonstrated persistent hemodynamic instability (BP 70/35 mmHg, HR of 99) . The patient was discussed with our tertiary referral center for higher level of care and transferred to an ECMO-capable ICU. On arrival to our ICU, bedside ECHO demonstrated hyper-dynamic physiology but persistent shock (BP 82/40 mmHg). The patient had escalating vasopressor requirements despite methylene blue and the decision was made to cannulate. After cannulation, hemodynamics improved with de-escalation of vasopressor requirements. The following day, the patient had a transient episode of worsening status the following day that improved after 5 g of hydroxocobalamin. Patient continued to progress with decannulation on hospital day 3.
Conclusion: VA-ECMO was utilized in this case despite lack of evidence of cariogenic shock with improvement in clinical status in conjunction with vasopressor therapy, methylene blue and hydroxocobalamin. This suggests benefit to VA-ECMO in severe amlodipine overdoses even in the absence of cardiogenic shock.

182. Evaluation of Vasopressor Volume Administration in Amlodipine Poisoned Patients

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Background: Amlodipine is a dihydropyridine calcium channel antagonist targeting L-type calcium channels that can result in vasoplegic shock in the overdose setting. Treatment of refractory vasoplegic shock can include high-dose vasopressors in addition to other infusions. Diluents in continuous infusions may be an unrecognized source of significant amounts of volume in patients already at risk for difficult-to-manage volume status. Large volumes of fluid administration, as a result, may cause iatrogenic injury. We sought to evaluate the volume of vasopressors administered to critically ill amlodipine-poisoned patients and calculate the difference in volume when applying our institution's most concentrated vasopressor guidelines.
Hypothesis or Research Question: Amlodipine-poisoned patients receive a significant volume of fluid from vasopressors during their initial resuscitation period.
Methods: We reviewed 25 years of our poison center records and searched for amlodipine poisoning cases with reported ingestions ≥ 400 mg who did not report co-ingestion of cardioactive substances (such as bupropion, tricyclic antidepressants,

alpha-2 agonists, and Vaughan Williams Class 1, 2, 3, 4, or 5 anti-dysrhythmics). The rate and concentrations of all vasopressors administered were recorded and used to calculate the total volume administered. Patients were excluded if they did not have 24 hours of vasopressor data. The vasopressor volume administered to each patient was summed from the start of the first vasopressor to 24 hours. We then applied our institution's most concentrated vasopressor guidelines to calculate the difference in fluid volume had these protocols been initiated during their initial resuscitation period.
Results: Eleven patients met initial inclusion criteria, of which four were excluded due to death or early initiation of V-A ECMO. The mean(±SD) volume of fluids related to vasopressors was 3433 (±2774) mL. Using concentrated vasopressors would have theoretically resulted in a mean 1.0 liter less fluid volume, a mean reduction of 32% (SD±0.15). Three of the seven patients developed radiologic evidence of pulmonary edema, all of whom had an ejection fraction (EF) > 55%, suggesting fluid overload. The largest volume difference in administered fluid volume compared to our most concentrated guidelines in a single patient was 2.6 liters (L); this patient later developed oliguria and pulmonary edema.
Conclusion: Patients with amlodipine-induced vasoplegic shock receive large amounts of fluid in the form of vasopressors. Adopting a concentrated vasopressor policy for amlodipine-poisoned patients may limit the amount of fluid administered during their initial resuscitation period. Limitations include the theoretical outcome of these calculations.

183. Refractory Lithium Toxicity Due to Persistent Gastrointestinal Reservoir Necessitating Serial Hemodialysis: Missed by Radiology and Identified by an Emergency Medicine Physician

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Background: Lithium toxicity is a clinical syndrome resulting from elevated serum lithium concentrations. Due to lithium's narrow therapeutic index and prolonged elimination half-life, toxicity can occur even with levels just above the therapeutic range. Because lithium is neither metabolized nor protein-bound, its bioavailability approaches 100%, and it is rapidly absorbed, allowing large ingestions to produce severe, life-threatening toxicity. Renal dysfunction, sustained-release formulations, and large gastrointestinal reservoirs can further delay elimination and worsen clinical severity. This case report describes the intensive, multimodal management required for massive lithium overdose, integrating gastrointestinal decontamination and extracorporeal modalities such as intermittent hemodialysis and continuous renal replacement therapy.
Hypothesis or Research Question: Massive lithium ingestions with a large gastrointestinal (GI) burden require early recognition and aggressive treatment with GI decontamination and multiple sessions of hemodialysis.
Methods: This is a case report of a 44-year-old female who presented to the Emergency Department intubated and unresponsive. On CT imaging, the Emergency Department team identified radio-opaque tablets in the stomach that were missed by radiology. They obtained a serum lithium level and consulted toxicology. The neurologic exam was notable for lower extremity hyperreflexia and myoclonus, consistent with lithium toxicity. Activated charcoal was administered for possible co-ingestions, and whole bowel irrigation was initiated. Initial serum lithium level resulted at 2.19 mEq/L, However, the markedly large gastrointestinal reservoir raised concern for continued absorption and further elevation of serum lithium levels, prompting the urgent initiation of hemodialysis after discussion with toxicology and nephrology.
Results: The patient had continued signs of neurotoxicity with persistently elevated lithium levels, which peaked on day three at 4.10 mEq/L. Over the course of 7 days, the patient received 6 sessions of intermittent hemodialysis as well as continuous renal replacement therapy at night after HD during the day. Ultimately, extracorporeal removal was terminated after day 5 hemodialysis session, the patient was extubated on day 7, and initially had some confusion but then returned to her neurologic baseline. Whole bowel irrigation continued for 5 days and was stopped on day 5.
Conclusion: Massive lithium ingestions may result in altered pharmacokinetics necessitating a multimodal approach involving frequent laboratory monitoring after hemodialysis sessions, whole-bowel irrigation, and continued extracorporeal removal. Clinicians need to have a high index of suspicion and review abdominal imaging for radioopaque foreign bodies and pills.

184. The Kratom Leaf Falls Far From the Tree: 7-Hydroxymitragynine Abuse Potential

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Background: There is increasing availability of concentrated kratom-derived products containing the alkaloid 7-hydroxymitragynine (7-OH), a potent μ-opioid receptor agonist. We describe a case of opioid withdrawal secondary to the use of 7-OH.
Hypothesis or Research Question: Use of 7-hydroxymitragynine is addictive, and cessation can produce an opioid withdrawal syndrome.

Methods: Single-patient chart review.
Results: An 18-year-old male was brought to the hospital by his parents for worsening apathy and concerns regarding substance use. He presented with a Clinical Opiate Withdrawal Scale (COWS) score of 18. He reported insufflating or ingesting 7-OH products purchased from local gas stations and estimated daily use of 150–200 mg for approximately six weeks. Initial management included clonidine and hydroxyzine. He was subsequently started on buprenorphine/naloxone, with withdrawal symptoms and cravings stabilizing at 4 mg twice daily. The patient was admitted to inpatient psychiatry for depressive symptoms and was ultimately discharged with chemical-dependency follow-up.
Conclusion: Concentrated 7-hydroxymitragynine products appear to have substantially higher addictive and abuse potential than kratom or mitragynine-only preparations. 7-OH exhibits approximately five-fold greater affinity for the μ-opioid receptor compared with mitragynine. Animal studies demonstrate enhanced reinforcement, the development of cross-tolerance to morphine, and self-administration of high-dose 7-OH. These findings are consistent with significant reinforcing effects. Compared with kratom, which has been widely used in the United States for over a decade, there is considerably less published literature specifically addressing 7-OH toxicity, use patterns, and withdrawal. Only three case reports describe 7-OH withdrawal treated with buprenorphine, yet a growing number of clinicians report encountering 7-OH products in practice, and regulatory agencies have issued warnings regarding their risk. While kratom use and dependence are now commonly seen in clinical settings, clinicians may be less familiar with concentrated kratom alkaloids, including 7-OH, particularly when synthesized or purified outside traditional botanical preparations. Increased clinical vigilance is warranted given the potency, accessibility, and rising prevalence of 7-OH products, and clinicians should consider buprenorphine treatment when opioid withdrawal is identified.

185. Paradichlorobenzene (PDB) Exposure Causing Hemolytic Anemia in a Child With Sickle Cell Trait

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Background: Paradichlorobenzene (PDB) exposure is usually well-tolerated. Toxicity is typically neurologic and occasionally cutaneous. There are scattered reports of ingestion causing hemolytic anemia, including in a child with sickle-cell trait.
Hypothesis or Research Question: Exposure to PDB leads to hemolytic anemia potentiated by underlying hemoglobinopathies.
Methods: This is a single-patient case report. A 14-month-old boy with no known history presented due to lethargy, black stool, and mothball remnants in his stool. Mother reported that she had placed 10 mothballs that she purchased from a large retailer locally 2 weeks prior. There was no witnessed ingestion. The patient presented with pallor and a room air saturation of 85%, without response to supplemental oxygen.
Results: A picture of the mothball package was obtained, listing the contents as 99.5% paradichlorobenzene. Labs showed hemoglobin 5.6 g/dL, haptoglobin <10 mg/dL, lactate dehydrogenase 535 u/L, reticulocyte count 4.9%, G6PD was normal (although assessed during a hemolytic episode), and Coombs test was negative. Schistocytes were demonstrated on manual differential. The patient tested positive for sickle cell trait. Methemoglobin levels peaked at 5.9%, with minimal clinical response to methylene blue. They received two units of packed red blood cells and were discharged with hematology follow-up. This case report is limited due to a lack of confirmatory testing and a lack of follow-up.
Conclusion: Hemolytic anemia, typically associated with naphthalene mothballs, should also be considered as a potential toxicity from PDB mothballs, especially if the patient has an underlying hemoglobinopathy.

186. An Analysis of Toxicology Consults Without Evidence of Intoxication: Re-Visiting No Underlying Toxicological Syndrome

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Background: In 2004 Leiken et al. described a set of patients presenting to an outpatient clinic with concern for adverse effects from a toxicologic exposure without objective findings or other evidence of any toxicologic effect. This was an initial report of a broad group of patients who received a medical toxicology consultation but did not have findings consistent with a toxicologic effect. These patients, and the reason they seek toxicologic evaluation, are of interest not only because of the patient factors described in the original study, but also because other factors beyond the patient's control contribute to their referral.
Hypothesis or Research Question: Certain agents and factors affecting cases referred for medical toxicology consultation are more likely to be associated with no underlying toxicologic concern or the development of signs/symptoms.

Methods: Using the Toxicology Investigators Consortium (ToxIC) Core Registry we identified cases evaluated by a medical toxicologist between December 2010 and June 2024 that either did not have any signs or symptoms (S/S), or if they did, they were found to be unrelated to any exposure by the evaluating toxicologist. We performed qualitative statistical analysis that sorted the cases by category and generated a list of agents that had the highest/lowest rate of cases either being either without signs or symptoms or, if present, were unlikely related to an exposure. A regularized regression technique, elastic net, was performed to explore additional potential relationships that might predict non-toxicological exposures.
Results: Of a total of 17,052 cases, 14,336 cases were found to have exposures that were without S/S and 2,716 were unrelated to the exposure. The most common agent classes without any S/S, when normalized, were rodenticides, foreign objects, metals, anticoagulants, and GI medications. Of those that were unlikely to have caused the S/S, plants/fungi, herbicides, insecticides, metals, and foreign objects were the most common agents. These cases were also delineated into sets of data that were pediatric or outpatient vs. inpatient and analyses repeated. The exploratory analysis suggested factors such as outpatient evaluations, unusual or uncommon agents, and reporting certain clinical symptoms such as agitation were associated with consults later determined to be non-toxic.
Conclusion: Our study demonstrates that in a significant number of cases referred for medical toxicology consultation no underlying toxicology syndrome exists. Certain agents and factors are associated with this phenomenon including plants/fungi, rodenticides, and outpatient evaluations.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

187. Which Residency Programs Produce the Most Medical Toxicologists?

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Background: Medical toxicology (MT) is a growing medical specialty. Fewer than 1000 physicians have ever become board certified (BC) in MT. Of over 250 emergency medicine residency programs in the United States, currently only 32 have associated MT fellowship programs. There is limited information regarding residency training programs of BC medical toxicologists, and this data can be useful in targeting engagement with trainees considering fellowship.
Hypothesis or Research Question: Which residency programs have produced the most medical toxicologists? Are medical toxicologists more likely to graduate from residency programs that have an affiliated MT fellowship?
Methods: We reviewed the American College of Medical Toxicology (ACMT) membership database to assess for trends in residency programs attended by medical toxicologists. We included all BC medical toxicologists and MT fellow-in-training (FIT) members for whom residency program information could be determined either by self-report data or by information publicly available online. Those who graduated from residency programs not currently participating in the United States National Resident Match Program (NRMP) or whose residency graduation information was not available were excluded. We then compared those who attended residency programs with an affiliated MT fellowship program to those who attended residency programs without an affiliated MT fellowship program. We restricted analysis to those whose fellowship completion date is 2000 or later. Descriptive statistics were performed using Microsoft Excel.
Results: Records for 689 ACMT BC medical toxicologist and MT FIT active members were reviewed. 448 met inclusion criteria and were included in the analysis. 147 distinct residency programs were represented – among these residency programs, 30 (20.4%) are associated with a MT fellowship program (representing 30 of 32 total ACGME-accredited MT fellowships). 209 (46.7%) individuals graduated from a residency associated with a MT fellowship. All of the 10 residency programs that were attended by the highest number of medical toxicologists were affiliated with a MT fellowship program: University of Massachusetts Chan Medical School (n=28), Lehigh Valley Health Network (n=19), University of Pittsburgh Medical Center (n=14), University of California Davis (n=13), New York University (NYU)/Bellevue (n=13), Cook County Hospital (n=11), Denver Health (n=10), Harvard Affiliated Emergency Medicine Residency (n=10), Barnes-Jewish Hospital/Washington University (n=9), and Jefferson Health/Jefferson Einstein Philadelphia Hospital (n=9).
Conclusion: Residency programs with an affiliated MT fellowship program have trained almost 50% of all medical toxicologists since 2000. Efforts to expand toxicology presence at training sites without a MT fellowship program may increase the pool of BC medical toxicologists from other locations across the country.

188. Serotonin Syndrome Following One Dose of Duloxetine Despite Negative Pharmacogenomic Testing

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Background: Serotonin syndrome primarily results from overdose or exposure to multiple serotonergic agents; it is rare after a single dose of one serotonergic medication. Pharmacogenomic testing can be considered when there is no history of overdose or medication interactions; however, the reliability and external validity of different pharmacogenomic testing platforms to identify clinically significant effects has not been extensively studied.
Hypothesis or Research Question: Serotonin syndrome from one dose of a serotonergic agent is possible, and pharmacogenomic testing may not reliably predict an individual's clinical response to certain medications.
Methods: This is a single patient case report. A 30-year-old male with a past medical history of depression presented to the emergency department with reports of "feeling unwell" after taking one 30 mg dose of duloxetine prior to presentation. Initial examination revealed an alert, ill-appearing male with diaphoresis, mydriasis, tachycardia, increased muscular tone, hyperreflexia and inducible lower extremity clonus. Electrocardiogram demonstrated sinus tachycardia at 152 with QRS duration of 117 milliseconds and corrected QT interval of 449 milliseconds. He reported no other serotonergic agent exposure. Laboratory studies revealed a lactate of 3.2 mmol/L, but otherwise were unremarkable. He was treated with intravenous fluids and benzodiazepines. The patient reported he had a similar reaction to a single dose of escitalopram one year prior, following which he had pharmacogenomic testing from a third-party company (GeneSight®). Repeat institutional pharmacogenomic testing with OneOme® was obtained during this hospitalization.
Results: Clinically, the patient met Hunter Criteria for serotonin syndrome following one 30 mg dose of duloxetine. Initial GeneSight® testing listed duloxetine without significant gene-drug interaction; however, the report did identify a genetic allele polymorphism within the HTR2A gene possibly leading to an increased risk of adverse drug reactions to selective serotonin reuptake inhibitors. OneOme® testing reported his HTR2A gene as normal variant. It was noted that the specific genotype location was different in the GeneSight® test versus the OneOme® test. Both panels reported no cytochrome P450 polymorphisms that would contribute to an increased risk for serotonin toxicity. The patient had gradual improvement in his serotonergic symptoms and ultimately was discharged on hospital day four without sequelae.
Conclusion: Providers should be aware that serotonin syndrome can develop from a single therapeutic dose of a serotonergic medication. Additionally, the external validity and reliability of pharmacogenomic tests are not well-established, and providers should primarily rely upon clinical judgement when selecting the appropriate medications for a patient.

189. Early Trainee Associations: Medical School Reported by American College of Medical Toxicology Full Members and Fellows-in-Training Between 1980 and 2025

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Background: Medical toxicology in the United States has grown significantly over the past 75 years. In 1993, the American Board of Medical Specialties officially recognized medical toxicology as a specialty and the American College of Medical Toxicology (ACMT) was formed. Fellowships were present for decades prior to gaining formal accreditation in 2000 by the Accreditation Council for Graduate Medical Education (ACGME). There is a paucity of information regarding the evolution of ACMT membership in relation to undergraduate medical training institutions and chronological trends in the setting of the above major milestones.
Hypothesis or Research Question: Are medical schools with affiliated medical toxicology fellowships associated with more medical toxicology graduates?
Methods: A retrospective database review was performed of all ACMT members who were classified as full members or fellows-in-training who self-reported medical school with either fellowship program name or graduation year, serving as inclusionary criteria. Exclusionary criteria included subjects who were not full and fellow-in-training members and did not report medical school. This study was exempt from informed consent. Wherever possible, incomplete data was obtained from internet searches based on publicly available data. Descriptive statistics were performed using Microsoft Excel.
Results: 685 respondents reported medical school and fellowship program; 569 reported medical school and fellowship graduation year. Prior 2000, 75 members reported both medical school and fellowship graduation year. The earliest

year reported was 1980. Starting in 2000, 494 members reported both medical school and fellowship graduation year. The ten medical schools that prepared the most medical toxicologists for residency accounted for 16.96% of ACMT members. 1.44% of medical toxicologists graduated from the ten medical schools associated with the least number of toxicologists registered with ACMT. 27.4% of medical toxicologists graduated from a medical school affiliated with a ACGME approved medical toxicology fellowship; 72.6% graduated from a medical school with no affiliated fellowship.

Conclusion: In the past 25 years, the growth and recognition of medical toxicology has been remarkable. There is wide variability in undergraduate medical training institutions among toxicologists, and most toxicologists graduate from a medical school without an affiliated medical toxicology fellowship. Further examination of this data in conjunction with data regarding regional presence of medical toxicologists may assist in targeting recruitment efforts to increase visibility at medical schools and foster a future generation of medical toxicologists.

190. When ECMO Is a No Go: Impella as an Alternative for ECMO Failure in Bupropion Overdose

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Background: Bupropion is an aminoketone antidepressant that primarily inhibits norepinephrine and dopamine reuptake. In massive ingestion it can cause cardiac dysrhythmias and cardiovascular collapse. Venoarterial extracorporeal membrane oxygenation (VA ECMO) has been used in several cases in the literature as a bridge to cardiac recovery. To the best of our knowledge, there are no cases in the literature of Impella 5.5 being used as mechanical cardiac support in bupropion overdose.

Hypothesis or Research Question: Impella 5.5 can be used as a bridge to cardiac recovery in select patients with cardiogenic shock resulting from bupropion overdose.

Methods: This is a single patient case report. A 23-year-old male presented approximately eight hours after an intentional ingestion of 100 tablets of 300 mg bupropion XL and 100 tablets of 0.2 mg clonidine. He developed persistent hypotension (nadir blood pressure of 75/51 mmHg) despite multiple vasopressors and QRS widening (peak QRS of 186 ms) refractory to sodium bicarbonate boluses and infusion. Cardiac arrest due to ventricular tachycardia occurred approximately 23 hours post ingestion requiring advanced cardiac life support, including defibrillation. 30 hours post-ingestion an echocardiogram showed an ejection fraction of less than 20% and the patient underwent VA ECMO and intra-aortic balloon pump (IABP) implantation. At approximately 60 hours post ingestion, a bupropion level was found to be 723.4 ng/mL and the hydroxybupropion metabolite 5707 ng/mL. Complications from VA ECMO arose including clotting of the circuit and concern for limb ischemia. The patient required continued mechanical circulatory support, therefore Impella 5.5 was placed as a bridge to cardiac recovery with VA ECMO decannulation and IABP removal on day three post ingestion.

Results: After Impella 5.5 placement, he was weaned down to dobutamine 5 mcg/kg/min and levophed infusion range 2-8 mcg/min over the next 24 hours. On day six post ingestion, echocardiography was performed showing cardiac recovery with normal biventricular function and ejection fraction of 55-60%, therefore Impella was removed. He was weaned off all vasoactive medications and cardiac function was restored by day seven. He was following commands and extubated on day 10 post-ingestion.

Conclusion: In select cases of severe cardiogenic shock due to toxic ingestion, Impella 5.5 can be utilized to deescalate from VA ECMO and provide continued mechanical circulatory support as bridge to recovery.

191. Hydroxocobalamin Use in Non-Cyanide Poisoned Patients: A Retrospective Analysis of the ToxIC Core Registry

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Background: Hydroxocobalamin is an established antidote for cyanide poisoning reportedly administered in patients presenting with refractory vasodilatory shock, in part due

to its scavenging of nitric oxide and hydrogen sulfide. Despite growing interest, limited clinical evidence exists to guide its use in poisoned patients with non-cyanogenic exposures.

Hypothesis or Research Question: In what context are non-cyanide poisoned patients at centers with medical toxicologists receiving therapeutic hydroxocobalamin?

Methods: This was an analysis of data from the Toxicology Investigators Consortium (ToxIC) Core Registry who received therapeutic hydroxocobalamin from September 2010 to January 2025. Exclusion criteria included: patients with no documentation of administered vasopressors, exposures consistent with cyanide or smoke-related poisoning (e.g., carbon monoxide, sodium nitroprusside, acetonitrile, and sodium azide), and if the bedside medical toxicologist deemed the signs and symptoms were uncertain/unlikely to be due to a toxic exposure. Demographics, substances, co-administered therapies, vasopressor utilization, and clinical outcomes were summarized.

Results: Among 151 hydroxocobalamin-treated cases identified, 19 met inclusion criteria. 63% were female (12/19) with a median age of 38 years. All patients received vasopressors; 16/19 (84%) had a documented systolic blood pressure under 80 mmHg. Two (11%) exposures involved ethanol alone. Of the remaining 17 cases, eight (47%) were single-substance ingestions and nine (53%) were poly-ingestions. Amlodipine was the most frequent exposure, present in nine (53%) non-ethanol cases; four (50%) single-substance exposures were amlodipine ingestions. Other common exposures included angiotensin-converting enzyme inhibitors and angiotensin receptor blockers (six patients) and non-dihydropyridine calcium channel antagonists (three patients). Adjunctive therapies were common: two received atropine, seven received calcium, 10 received high-dose insulin euglycemia therapy, six received intralipid emulsion, and 10 received methylene blue. Many patients received multiple vasopressors: angiotensin II (37%), epinephrine (74%), dobutamine (5%), norepinephrine (89%), phenylephrine (42%), and vasopressin (74%). One case documented therapeutic use of vasopressors but not specific agents. Mortality was 47%, and in one case clinical outcome was unknown.

Conclusion: Hydroxocobalamin is being used in a subset of hypotensive poisoned patients in the ToxIC Core Registry, most commonly in severe calcium channel blocker toxicity. These patients experienced high mortality. Limitations of this study include potential misclassification bias and missing data for those given hydroxocobalamin, and the inability to account for nationwide hydroxocobalamin shortages.

ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

192. "Antabuse" and Misuse: Disulfiram as an Antidote to a Chemist's Intentional Overdose With Acetonitrile

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Background: Acetonitrile is an organic solvent used in cosmetics, chemical industries, and laboratories. The toxic effects of acetonitrile are attributable to the hepatic metabolism into cyanide via CYP2E1. There is evidence that disulfiram, a CYP2E1 inhibitor, reduces cyanide formation in acetonitrile poisoning.

Hypothesis or Research Question: In a case of intentional acetonitrile poisoning, we postulate that disulfiram significantly reduced cyanide production.

Methods: This case report is from a single-patient chart review of a 46-year-old female who presented after intentional ingestion of acetonitrile. She endorsed taking an estimated 10mL "sip" of 100% concentration acetonitrile and presented to the Emergency Department approximately 60 minutes post-ingestion (PI). She was asymptomatic, and her physical exam was unremarkable with the following vital signs: temperature 98.6°F, heart rate 84, respiratory rate 16, and blood pressure 102/59. Preliminary laboratory results and electrocardiogram were unrevealing, including undetectable ethanol, aspirin, and acetaminophen levels. Initial lactate was 1.2 mmol/L. She was admitted to the ICU for further management. The decision was made to use disulfiram to reduce cyanide formation.

Results: 500mg of disulfiram was given daily for a total of 5 doses, starting approximately 6 hours PI. The presenting cyanide level was 4.0 mcg/mL, drawn 4.5 hours PI. At 6 hours PI, her blood pressure decreased to 80/32 despite administration of two liters of crystalloid fluid, and the patient was started on a norepinephrine infusion. Lactate peaked at 2.3 mmol/L 8 hours PI. One dose of hydroxocobalamin 5g IV was administered 12 hours PI, and norepinephrine was discontinued shortly thereafter. Subsequent cyanide levels measured at approximately 25 and 39 hours PI were 0.21 mcg/mL and 0.13 mcg/mL, respectively. Cyanide concentrations of greater than 3mcg/mL can be associated with death. At 4 hours PI, our patient was above a "lethal level" with anticipation of continued elevation. De Paepe et al. reported a similar case in which disulfiram was administered on day seven, after multiple doses of hydroxocobalamin and sodium thiosulfate. In Rzodkiewicz et al., there was persistent hypotension and lactic acidosis despite 40g of hydroxocobalamin, and five days of disulfiram was started on day four with rapid improvement. In our case, disulfiram was

given early with prompt clinical improvement, decreasing cyanide levels, and minimal hydroxocobalamin and vasopressor requirements.
Conclusion: Early administration of disulfiram in a potentially fatal acetonitrile overdose was followed by rapid clinical improvement with minimal vasopressor and hydroxocobalamin requirements.

193. New Onset Diabetic Ketoacidosis Precipitated by a Supratherapeutic Clozapine Level: A Case Report

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Background: Clozapine is an atypical antipsychotic that antagonizes dopaminergic, cholinergic, adrenergic, histaminergic, and serotonergic receptors. Development of metabolic abnormalities with clozapine use is common, from interference with glucose homeostasis and insulin signaling pathways. Diabetic ketoacidosis (DKA) is a rare, but reported, severe potential adverse outcome. Few data exist linking the serum concentration of clozapine to this outcome.
Hypothesis or Research Question: Diabetic ketoacidosis is more likely to develop in the setting of a supratherapeutic serum concentration of clozapine.
Methods: This is a single case report performed by retrospective chart review.
Results: A 35-year-old female with a medical history of treatment-resistant schizophrenia presented to the Emergency Department with shortness of breath. Her schizophrenia had been well controlled on clozapine, paliperidone palmitate, and aripiprazole for nine months. Her initial vitals were: heart rate 136 bpm, respiratory rate 40/min, blood pressure 130/90 mm Hg, and pulse oximetry 94% on room air. She was noted to be ill-appearing, lethargic, and to have Kussmaul respirations. Her blood glucose was 1,178 mg/dL, her anion gap was 37, and her pH was 6.98. She was diagnosed with diabetic ketoacidosis and was admitted for further management. She had no prior history of diabetes, and her most recent HbA1c three months prior to hospitalization was 5.7%. A serum clozapine concentration drawn at an outpatient appointment five days prior to her presentation was supratherapeutic, at 653 ng/mL (reference range 350-600 ng/mL). At that time, she was reporting no adverse symptoms. However, two days later, she developed polyuria and changes in mental status. The only modification to her medication regimen during the preceding nine months was a gradual up-titration of clozapine. However, the increase to her most recent dose had been completed seven weeks prior to hospital presentation. She had seven documented clozapine levels within the therapeutic range during that time, performed once weekly. While hospitalized, she was treated with IV insulin, dextrose, and crystalloid until her clinical and laboratory parameters normalized. She was transitioned to subcutaneous insulin, diagnosed with type II diabetes, and discharged on metformin, insulin, and clozapine at a lower dose. Her HbA1c later that month was 12.7%, and she continues to require insulin.
Conclusion: Clozapine is an atypical antipsychotic with a well-documented risk of metabolic side effects. This case demonstrates that a supratherapeutic serum clozapine concentration may precipitate rapid onset of DKA and induce onset of long-term insulin resistance.

194. Squash-Induced Methemoglobinemia in a Pediatric End-Stage Renal Disease Patient With Undiagnosed G6PD Deficiency

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Background: Methemoglobinemia is a condition where iron within hemoglobin is oxidized from ferrous (Fe^{2+}) to ferric (Fe^{3+}), resulting in decreased oxygen-carrying capacity. Although most commonly associated with pharmaceutical or chemical exposures, unusual triggers can present diagnostic challenges. Methylene blue relies on NADPH-dependent reduction to function and is renally cleared. It may be ineffective, or even harmful, in glucose-6-phosphate dehydrogenase (G6PD) deficient patients.
Hypothesis or Research Question: Clinically significant methemoglobinemia in a medically complex 5-year-old can result from uncommon oxidant exposures not typically recognized in standard pediatric risk assessments. Concurrent G6PD-deficiency and end-stage renal disease (ESRD) can complicate methylene blue administration.
Methods: This is a single-patient chart review. A 5-year-old boy with asphyxiating thoracic dystrophy, restrictive lung disease, ESRD on dialysis, and chronic hypertension presented from dialysis clinic with acute hypoxemia (SpO_2 78–80%). Hypoxemia was refractory to escalating respiratory support, including non-breather, BiPAP, mechanical ventilation, and inhaled nitrous oxide. Arterial and venous blood gases showed a PaO_2 of 536 mmHg and a PvO_2 of 229 mmHg despite SpO2 of 78%. Co-oximetry demonstrated 9% methemoglobinemia. Sulfhemoglobin was not obtainable. Family reported no new medications or change in routine; no other dialysis patients experienced symptoms. Family reported the patient had a new ingestion of multiple servings of homemade squash the two days prior to presentation.
Results: He received 1 mg/kg methylene blue and high-dose vitamin C with in minimal, transient response in methemoglobin percentage. However, his hemoglobin decreased

from 10 g/dL to 6.5 g/dL with no obvious bleeding, elevated lactate dehydrogenase, and low haptoglobin. Hemolysis and lack of response to methylene blue suggested undiagnosed G6PD deficiency. He required blood transfusions on Days 1 and 2. G6PD activity later resulted at <5% of normal.
Conclusion: Naturally-occurring vegetable nitrates convert to nitrites over time and with improper storage. They are an unusual methemoglobinemia trigger more commonly seen in infants and rarely reported in older children or adults. In this patient, ingestion of stored squash was the likely trigger. Pre-existing restrictive lung disease allowed a low methemoglobin percentage to produce severe hypoxemia; undiagnosed severe G6PD deficiency amplified oxidative vulnerability, predisposing him to hemolysis from both the initial oxidant insult and from methylene blue administration. ESRD further complicated management, as methylene blue is not cleared by renal replacement therapies and prolonged exposure to unmetabolized methylene blue may have exacerbated this patient's oxidative stress, worsening hemolysis and slowing recovery. Limitations include being a single-patient case report and lack of squash nitrite concentrations.

195. Massive Bupropion Ingestion Causing Transient Left Ventricular Dysfunction and Global Hypokinesis Confirmed With Serial Echocardiography

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Background: Bupropion overdose is associated with high mortality due to recurrent seizures, life-threatening cardiac dysrhythmias, and cardiogenic shock.
Hypothesis or Research Question: Massive bupropion ingestion can cause cardiogenic shock, with significantly reduced ejection fraction and global left ventricular hypokinesis, which can be confirmed with echocardiography.
Methods: This is a single-patient chart review. A 55-year-old female with a history of anxiety, depression, fibromyalgia, and a prior ejection fraction of 55-60% on transthoracic echocardiogram presented to the emergency room after being found unresponsive and bradypneic. She had been drinking alcohol heavily and taking multiple of her prescribed medications, which include bupropion. Naloxone was given with no response, so she was intubated for airway protection. Computed tomography of the abdomen and pelvis showed multiple pills in the gastric lumen. An electrocardiogram (ECG) initially showed sinus tachycardia at 100 beats/min, with a QRS interval of 96ms and a QTc interval of 431ms. After several hours following presentation, the patient became hypotensive, and a repeat ECG showed a left bundle branch morphology with a QRS interval of 190ms and a QTc interval of 618ms. A sodium bicarbonate intravenous bolus and multiple inotropic and vasopressor medications did not improve the patient's hemodynamics. A cardiology-performed bedside echocardiogram showed severely reduced ejection fraction of approximately 20-25% and global hypokinesis. Soon after, the patient had a cardiac arrest, CPR was initiated, and venoarterial extracorporeal oxygenation (VA-ECMO) cannulation was performed.
Results: Transthoracic echocardiogram (TTE) performed 15 hours after VA ECMO cannulation, while the patient was receiving four inotropic and vasopressor medications, showed a reduced ejection fraction at 35-40%. ECG showed a QRS of 107 and a QTc of 567. Over the next two days, inotropes and vasopressors were weaned. She was decannulated after 4 days on VA-ECMO. A repeat TTE done 2 days after decannulation showed a hyperdynamic left ventricle with an ejection fraction of 70-75% and normal wall motion. An ECG during this time showed a QRS of 77ms and a QTc of 491ms. Although the patient had a prolonged hospital course due to delirium, she was ultimately discharged without any neurologic deficits. Her initial bupropion level resulted at 15,390 ng/mL (ref 50-100) and 4-hydroxybupropion level at 7,479.9 ng/mL (ref 600-2,000).
Conclusion: Massive bupropion ingestions may result in severe cardiopulmonary collapse. During the course of illness, bedside and transthoracic echocardiography, along with correlative electrocardiography, can be helpful in hemodynamic monitoring.

196. The Quantitative Efficacy of Plasmapheresis in a Pediatric Case of Amlodipine Poisoning

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Background: Amlodipine is a dihydropyridine calcium channel antagonist (CCB) that can cause vasodilatory shock at large doses. The high protein-binding and lipophilicity of CCBs suggest potential for drug removal via plasmapheresis; however, evidence supporting its efficacy remains sparse and largely anecdotal.
Hypothesis or Research Question: What is the efficacy of plasmapheresis for drug removal in amlodipine poisoning?
Methods: A 15-year-old male presented 12 hours after intentional ingestion of up to 135 tablets of 5 mg amlodipine and up to 30 tablets of 100 mg losartan along with indeterminate amounts of pediatric cough syrup, isopropyl alcohol,

and amoxicillin-clavulanate. He developed profound vasodilatory shock initially managed with high doses of six vasoactive agents (calcium gluconate, norepinephrine, epinephrine, vasopressin, angiotensin II, and methylene blue), high-dose insulin, hydrocortisone, and therapeutic hypothermia to 35°C. A single session of plasmapheresis around 36 hours post-ingestion exchanged 1.5 plasma volumes with fresh frozen plasma. Plasma amlodipine levels were quantified pre-, immediately post-, and six hours post-plasmapheresis and from the removed plasma. Amlodipine concentrations were quantified by high-performance liquid chromatography/tandem mass spectrometry.

Results: Pre-, immediately post-, and six hours post-plasmapheresis serum concentrations were 160, 170, and 150 ng/mL, respectively, all within the 20% range of error for the laboratory test. Amlodipine removed in the plasmapheresis fluid had a concentration of 220 ng/mL, equating to just over one mg of drug removed (< 1% of presumed ingested amount). Using a simplified, first-order pharmacokinetic model, endogenous metabolism would be predicted to eliminate 19 mg amlodipine in the same time period. Around eight hours post-plasmapheresis, the patient's hypothermia goal was decreased from 35°C to 33°C. Within 24 hours of plasmapheresis, angiotensin II and methylene blue were discontinued while doses of epinephrine and norepinephrine were halved. Within 48 hours of plasmapheresis, vasopressin was discontinued. The patient did not require extracorporeal membrane oxygenation (ECMO).

Conclusion: Plasmapheresis is ineffective for drug removal in the setting of amlodipine poisoning; however, the patient demonstrated clinical improvement within 48 hours of procedure completion. It is difficult to discern whether this was attributable to plasmapheresis via an alternative mechanism, the augmented vasoconstriction from deeper hypothermia, the concurrent metabolism of losartan which boasts a far shorter half-life, or simply allowing more time for endogenous drug metabolism. Importantly, this case addresses a confounder from a prior report, as plasmapheresis was performed independently rather than in tandem with an ECMO circuit allowing for a more direct assessment of drug removal by plasmapheresis.

197. Purple Haze - Visual Disturbances From an Ivabradine Overdose

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Background: Ivabradine has traditionally been used in the management of patients with congestive heart failure, but has recently been used off-label to treat syndromes causing inappropriate tachycardia such as postural orthostatic tachycardia syndrome (POTS). However, data on toxicity is very limited and the extent of toxicity can vary greatly at similar doses. Additionally, visual side effects are known but not commonly reported and may serve as a diagnostic aid in suspected ivabradine ingestion.

Hypothesis or Research Question: Intentional Ivabradine overdose led to symptomatic bradycardia and visual disturbances in a young, healthy patient.

Methods: This was a single patient chart review of a nineteen year old female with history of POTS and Ehlers-Danlos who intentionally ingested 292.5mg of Ivabradine which she had been prescribed for POTS. She presented to the ED the same day complaining of fatigue, exertional chest pain, and near syncopal episodes. She also reported visual disturbances which included seeing purple streaks around moving objects which she had not experienced when taking her therapeutic dose. Ocular exam and visual acuity were otherwise normal. She was bradycardic to 35 but normotensive and HR did not respond to 0.5mg atropine. Labs were notable for initial mild transaminase elevation and magnesium of 1.8. Multiple ECG's showed sinus bradycardia with early repolarization. She was admitted to the intensive care unit for cardiac monitoring and supportive care. The following day, she remained overall stable with only mild improvement in HR to 50s, and was ultimately transferred to the floor. She was discharged on hospital day 3 still slightly bradycardic to 50-60 with improvements in symptoms. Her ivabradine was discontinued.

Results: This patient developed symptomatic bradycardia without evidence of shock as a result of intentional ivabradine ingestion of 292.5mg. She did not require aggressive supportive care and improved without targeted intervention. Other case reports, however, have described profound toxicity from ingestions of similar or smaller quantities of ivabradine, including a case in which another 19 year old female ingested only 150-225mg of ivabradine and required transvenous pacing. Furthermore, the presence of visual phenomena has been rarely reported in cases of overdose despite being a known side effect of the drug.

Conclusion: This case adds to the limited number of reported cases of ivabradine overdose. When considering other reported cases, it demonstrates that similar doses of ivabradine may produce wide variations in severity of cardiotoxicity. It also highlights the potential value of visual phenomena as a diagnostic aid in patients with suspected ivabradine overdose.

198. A Case of Capecitabine Overdose and the Quest for Uridine Triacetate

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Background: Capecitabine is a chemotherapeutic antimetabolite that is the precursor to 5- fluorouracil. While overdoses are rare, clinical manifestations can range from primary gastroenteritis and bone marrow suppression to dysrhythmias, cardiogenic shock, encephalopathy and coma. Uridine triacetate is the antidote for 5-fluorouracil or capecitabine overdose but is rarely stocked by hospitals. We present a case of intentional capecitabine overdose and our process to obtain uridine triacetate.
Methods: Information for this case report was obtained by patient interview and chart review.
Results: A 75-year-old male with history of colorectal cancer in remission presented to an emergency department two hours after an acute intentional overdose of 56 g of capecitabine in addition to gabapentin and trazodone approximately five hours prior to arrival. He had one episode of vomiting and complained of headache. Vital signs were within normal limits. Laboratory evaluation demonstrated leukocytosis (12.4 K/μL), hypokalemia (3.4 mmol/L) and elevated creatinine (1.27 mg/dL). Acetaminophen, salicylate, and alcohol concentrations were undetectable. EKG was unremarkable. Toxicology was consulted and recommended transfer to our tertiary care facility for procurement and administration of uridine triacetate. Toxicology had been previously involved in decisions to not stock uridine triacetate and to have a procurement process due to the rarity, cost, and 96-hour initiation recommendation. We contacted the inpatient pharmacist who notified hospital administration. The attending toxicologist verbally confirmed need for uridine triacetate, and the pharmacist placed an urgent order to the institution's preferred vendor, Cardinal Health. After the order was confirmed, the medication was delivered via courier from seven hours away by ground. The full recommended course of uridine triacetate (10g every six hours for five days) was administered without development of additional symptoms. Labs were drawn daily for eight days. White blood cell count remained stable with nadir at 4.0 K/μL. Hypokalemia resolved and creatinine peaked at 1.47mg/dL on day two and normalized by day three. Labs two weeks post-ingestion were normal, and the patient was asymptomatic at outpatient appointment one month after discharge.
Conclusion: Despite a large overdose of capecitabine, uridine triacetate was initiated within 16 hours of ingestion, and the patient had minimal effects and no leukopenia. While literature suggests that uridine tractate is effective if started within 96 hours of exposure, hospitals may not stock this due to cost and rarity of overdose events. This case highlights that this is a reasonable approach, provided there is a process in place for rapid procurement.

199. Poisoning Due to Alternative Therapy With Kambo Frog Cutaneous Secretions (Phyllomedusa Bicolor)

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Background: Kambo is used as an alternative medicine therapy purported to treat metabolic and oncological conditions. In this alternative therapy, superficial circular burns are created on the skin, and a small amount of frog secretion (resin) is applied directly to the open wounds. The secretion is obtained by gently scraping the skin secretions from the legs and back of the frog.
Hypothesis or Research Question: Systemic toxicity from the bioactive components of Kambo frog extract exacerbated the metabolic decompensation.
Methods: We present a case report of a 40-year-old woman from Oaxaca, México, with hypothyroidism and type 2 diabetes, treated with levothyroxine, metformin, dapagliflozin, and insulin glargine. The patient consulted a naturopath for metabolic dysregulation and was administered Kambo extract via inhalation and topically on the back of her neck. One hour after the topical application, the patient developed palpitations, abdominal pain, myalgias, arthralgia, decreased muscle strength, and episodes of hallucinations; 8 hours later, tachypnea, aphasia, gait disturbances, and generalized weakness developed. She was admitted to the emergency room with hypotension and diabetic ketoacidosis. On physical examination, dry oral mucosa, a 5-second capillary refill, and decreased muscle strength were identified. In the laboratory test, acute kidney injury was observed. No abnormalities were found on the electrocardiogram. The initial treatment consisted of intravenous (IV) fluids; those who failed to respond to fluids required 0.3 mcg/kg/min norepinephrine for 48 hours, along with insulin therapy to address glycemic dyscontrol. At 72 hours, clinical improvement was observed, and she was discharged.
Results: After exposure to the secretion of P. bicolor, the patient developed gastrointestinal, musculoskeletal, and neurological complications. These manifestations were consistent with the systemic effects of vasoactive and neuroactive peptides (phyllocaerulein, phyllomedusin, phyllokinin, and sauvagine), which can induce gastrointestinal hypermotility, vasodilation, and central nervous system excitation. Fluid loss together with renal dysfunction led to metabolic

decontrol and hemodynamic instability, requiring vasopressor support to maintain adequate tissue perfusion.
Conclusion: Using kambo extract as an "alternative" therapy can cause serious metabolic disturbances and hemodynamic instability. There is no scientific evidence supporting its effectiveness, and its use may pose significant health risks.

200. Generative Artificial Intelligence and Human Diagnostic Accuracy for Toxicology-Related Clinical Pathologic Cases

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Background: The North American Congress of Clinical Toxicology hosts an Academic Congress of Medical Toxicology Clinical Pathologic Case Presentation Competition (CPC) each year, in which real medical cases are presented. A case discussant leads a structured diagnostic discussion, concluding with their presumptive diagnosis. Artificial intelligence (AI) tools like ChatGPT are increasingly used for clinical decision support, but the diagnostic performance of generative large language models in complex toxicology cases has not been characterized.
Hypothesis or Research Question: How does the diagnostic accuracy of a generative AI system (ChatGPT-5) compare with that of medical toxicologists for toxicology-related CPCs?
Methods: This is a retrospective study using historical CPC cases. In total, 42 cases were analyzed from 2010–2019 and 2021–2024. Prompts were standardized and input into ChatGPT-5 for either a single best diagnosis or a ranked top ten differential under two settings: "Thinking" and "Auto." Partially correct AI responses were excluded from analysis, accounting for the smaller denominators in some results.
Results: Human case discussants correctly diagnosed 23 (54.8%) cases. ChatGPT-5 set to "Thinking" mode correctly diagnosed 22 (52.4%) cases when asked to name a single best diagnosis, with a mean generation time of 90.8 seconds (range: 27–271).
When asked for a top ten differential in "Thinking" mode (n=41), ChatGPT-5 correctly identified the diagnosis as its most likely diagnosis in 20 (48.8%) cases. Under the same conditions, ChatGPT-5 correctly identified the diagnosis in its top three most likely diagnoses in 23 (56.1%) cases, in its top five most likely diagnoses in 28 (68.3%) cases, and in its top ten most likely diagnoses in 31 (75.6%) cases. ChatGPT-5 generated a top ten differential with a mean generation time of 135.9 seconds (range: 42–347). When set to "Auto" mode and asked for a top ten differential (n=39), ChatGPT-5 correctly identified the diagnosis as its most likely diagnosis in six (15.4%) cases, in its top three most likely diagnoses in 11 (28.2%) cases, in its top five most likely diagnoses in 13 (33.3%) cases, and in its top ten most likely diagnoses in 17 (43.6%) cases.
Conclusion: ChatGPT-5 performed well, with accuracy approaching that of medical toxicologists. We conclude that the use of generative AI tools such as ChatGPT may be clinically useful for differential diagnosis generation. Because performance depends on how clinicians prompt and apply these tools, results may vary widely and should be evaluated critically. Prompting a broad differential and optimizing model settings may improve diagnostic accuracy.

201. Takotsubo Cardiomyopathy Following Intentional Clonidine Overdose: A Rare Manifestation of a Common Toxic Exposure

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Background: Clonidine toxicity typically presents with central nervous system depression, bradycardia, and hypotension. At higher doses, clonidine loses alpha-2 receptor selectivity, resulting in paradoxical alpha-1 activation and catecholamine surge. This pathophysiology may predispose patients to type II myocardial injury and stress-induced cardiomyopathy. Takotsubo cardiomyopathy is an uncommon sequela of toxic exposures and has been rarely associated with clonidine ingestion.
Hypothesis or Research Question: Clonidine overdose can cause Takotsubo cardiomyopathy in adults through catecholamine-mediated myocardial stunning.
Methods: This is a single-patient case report from the Oregon Poison Center and an affiliated tertiary care medical center. Clinical data were obtained through chart review, as well as documentation from the poison center. Laboratory, electrocardiographic, and echocardiographic findings were reviewed to characterize the cardiac effects of clonidine ingestion.
Results: A 20-year-old female with no cardiac history presented approximately four hours after intentionally ingesting 71 tablets of 0.1 mg clonidine. On arrival in the emergency department, she was lethargic with a heart rate of 30 bpm and systolic blood pressure of 100 mmHg. After administration of 1 mg atropine, her heart rate increased to 108 bpm with a systolic blood pressure of 150 mmHg. Laboratory evaluation in the ED demonstrated an elevated troponin at 222 ng/L that peaked at 624 ng/L. Her electrocardiograms revealed sinus bradycardia and QTc prolongation of 521

ms. During her admission a transthoracic echocardiogram identified hypokinesis of the basal segments of the left ventricle along with left ventricular systolic thickening, consistent with Takotsubo cardiomyopathy. The patient was treated with supportive care, including intravenous fluids and atropine. Vasopressors were not required. Over the next 48 hours, her heart rate and blood pressure normalized, and the patient was ultimately discharged in stable condition.
Conclusion: This case represents a rare instance of Takotsubo cardiomyopathy following clonidine overdose in an adult patient. While pediatric cases have been described, adult presentations are exceedingly uncommon. The proposed mechanism involves catecholamine-mediated myocardial stunning secondary to alpha-adrenergic activation. Clinicians should maintain a high index of suspicion for cardiac complications, which could present from clonidine toxicity, particularly when troponin elevation or ECG abnormalities are present. Recognition of this potential complication may influence both diagnostic workup and monitoring in clonidine overdose patients.

202. Where Did Your Chatbot Train? - Do AI Platforms Provide Clinical Recommendations Consistent With Medical Toxicology Practice?

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Background: With the use of artificial intelligence (AI) becoming increasingly prominent today, individuals in healthcare are turning to these platforms to assist in medical decision making. Medical toxicology is a complex field, with which other healthcare professionals may be unfamiliar, making management of poisoned patients more difficult. Healthcare professionals may turn to AI systems to assist with management of poisoned patients if medical toxicologists are not easily accessible for consultation.
Hypothesis or Research Question: When given prompts for triage, diagnostic, and therapeutic toxicologic scenarios, will various AI platforms generate appropriate clinical recommendations?
Methods: This was a qualitative study to discern the utility of common AI platforms as a reference in clinical practice. Three popular AI platforms were chosen: Google Gemini, ChatGPT and OpenEvidence. Six prompts were created to be run through each AI platform. The prompts were chosen by a group of medical toxicologists and divided into three sections: triage, diagnostic and therapeutic. A prompt with a straightforward answer that medical toxicologists have come to a consensus on and a prompt that is currently controversial within the toxicology community were chosen for each section. These were entered into each AI platform twice, separated by two weeks, to see if answers produced varied. References used to generate answers were also reviewed.
Results: Almost all platforms generated similar recommendations for each prompt. However, OpenEvidence provided responses related to referral dose and observation period for asymptomatic, unintentional bupropion exposure that medical toxicologists would consider inaccurate or at least controversial. After the two week interval, OpenEvidence and Gemini produced the same response as on the initial run. However, ChatGPT now produced a response that quickly disappeared and provided information for the suicide hotline. References were assessed as each answer was generated. Google Gemini and ChatGPT most frequently used open-source online website resources while OpenEvidence cited published papers to support its response.
Conclusion: AI platforms, despite their variation in references, produced clinical recommendations that might be accepted by medical toxicologists as standard practice with the prompts provided. Future research could include more controversial topics in medical toxicology and/or more complex patient scenarios.

203. Massive Ondansetron Ingestion Causing Seizures, Myoclonus, and QTc Prolongation in a Toddler

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Background: Massive pediatric ondansetron ingestions are rare, and reported cases describe significant neurologic and cardiac toxicity. Recognition is essential because the presentation may mimic other toxidromes or co-ingestants.
Hypothesis or Research Question: Can isolated massive ondansetron ingestion cause severe neurologic toxicity and QTc prolongation in toddlers?
Methods: This is a single-patient retrospective case report. Clinical data were obtained from emergency medical services (EMS) documentation, electronic medical records, laboratory results, imaging, and toxicology consultation notes. The objective was to characterize the clinical manifestations, diagnostic evaluation, and management of a toddler following a large ondansetron ingestion.
Results: A thirty-one-month-old previously healthy child was brought by EMS for altered mental status after being found with a sibling near a box of medications intended for a neighbor. En route, the child developed seizure-like activity and received one milligram intravenous midazolam. On arrival, vital signs were blood pressure 123/86 mmHg, heart rate 130 beats/min, respiratory rate 30 breaths/min, oxygen saturation 93% on room air, and temperature 98 °F. The patient was confused, non-verbal, and intermittently demonstrated

whole-body involuntary muscle contractions. Laboratory studies, including electrolytes, liver function tests, creatine kinase, and acetaminophen and salicylate levels, were unremarkable. Imaging—including head computed tomography and chest and abdominal radiographs—was normal. An initial urine drug screen returned positive for cocaine; however, a repeat screen was negative. When the child's father brought the medication box, it contained ondansetron four-milligram tablets (bottle of one hundred twenty with one hundred ten tablets missing), amlodipine five-milligram tablets, empagliflozin twenty-five-milligram tablets, and lisinopril/hydrochlorothiazide twenty/twenty-five-milligram tablets. Only ondansetron tablets were missing. The sibling was asymptomatic. The patient's rigidity and myoclonus worsened despite diphenhydramine administration, progressing to back-to-back generalized clonic seizures. Concern for recurrent seizures and airway compromise led to endotracheal intubation, followed by sedation with midazolam, rocuronium, and propofol, and loading with levetiracetam. Serial electrocardiograms showed QTc prolongation from 360 ms to 444 ms, consistent with ondansetron-related hERG potassium channel blockade. The patient was transferred to a pediatric intensive care unit in stable condition and was successfully extubated after twenty-four hours.

Conclusion: Massive ondansetron ingestion can produce significant neurologic toxicity, including seizures, myoclonus, and altered mental status, as well as QTc prolongation in toddlers. Clinicians should consider ondansetron overdose in unexplained pediatric neurologic presentations, particularly when access to large quantities is possible.

204. Supratherapeutic Injection of Semiglutide in the Face

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Background: Semaglutide is medication used in the treatment of obesity and diabetes. It is structurally similar to the hormone glucagon-like peptide-1 (GLP-1) and acts as an agonist at the GLP -1 receptor. Initiation of subcutaneous semaglutide usually involves weekly injections of 0.25 milligrams with maximum therapeutic dosing of 2.4 mg once per week after a gradual dose escalation. The most common side effects of therapeutic use of a GLP-1 agonist are nausea, vomiting, diarrhea, and abdominal pain. There have been few reports of overdose of GLP-1 agonists to date. We report a case of an unintentional supratherapeutic injection of semaglutide into a patient's face after a medication error.

Methods: This is a case report of a 55-year-old female with a history of hypertension who presented to the emergency department (ED) with nausea and vomiting 30 minutes after an accidental injection of semaglutide 10 mg subcutaneously into her forehead. The patient had presented to an aesthetic clinic for a planned botulinum toxin injection; however, the provider mistakenly administered semaglutide. After recognizing the error, the aesthetician initiated a D5W infusion and transferred the patient to the ED. Serial symptom assessments, glucose and vital sign measurements were performed.

Results: On arrival, her initial glucose was 115 mg/dL. During her ED stay her vital signs were: HR 69–87 bpm, BP 158–202/86–102 mmHg, and glucose ranged from 92–120 mg/dL both on and then off D5W. She continued to have intractable vomiting despite receiving IV doses of glycopyrrolate 100 mcg, metoclopramide 10 mg, and ondansetron 4 mg. Due to insurance considerations, she was transferred to another facility, where she remained hospitalized for two additional days for intractable vomiting. CT abdomen/pelvis demonstrated possible colitis, with no evidence of pancreatitis, free air, or free fluid. Serum potassium ranged from 3.3–3.8 mmol/L. Glucose monitoring every 6 hours ranged from 100 – 120 mg/dL. During her admission she had her potassium repleted; received maintenance non-dextrose containing IV fluids; and was treated symptomatically with prochlorperazine, metoclopramide, and ondansetron. She had no documented episodes of hypoglycemia. Symptoms gradually resolved with supportive care, and she was discharged in stable condition on hospital day 3.

Conclusion: Accidental subcutaneous administration of a supratherapeutic dose of semaglutide can result in significant gastrointestinal toxicity, including persistent nausea and vomiting. This case underscores the importance of strict medication verification and storage protocols in aesthetic practice environments to prevent such errors.

205. Evaluation of CYP2C19 Utilization in an Academic Health System: A 5-Year Retrospective Analysis

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Background: Cytochrome P450 2C19 (CYP2C19**)** is an enzyme that metabolizes approximately 10% of common drugs. Notably, those with CYP2C19 loss-of-function (LOF) alleles are unable to bioactivate clopidogrel, one of the most prescribed drugs in the U.S. In these cases, P2Y12 inhibitors, like ticagrelor, are recommended. Due to high genetic polymorphism, differences in enzyme activity can create adverse effects; yet, routine CYP2C19 testing is uncommon.

Hypothesis or Research Question: CYP2C19 genetic testing is utilized when prescribing high risk medications, such as clopidogrel, to inform medication selection.
Methods: This retrospective observational study examined CYP2C19 testing trends and patient outcomes at a single academic hospital from May 1, 2020, to May 1, 2025. Inclusion criteria were adult patients with LOF alleles associated with the poor metabolizer phenotype (*2/*2, *2/*3, *3/*3, *2/*8) within our health system. Patient demographics, ordering reason, ordering service, medication involved, medication changes, documented decision-making, and adverse outcomes were extracted. Data was analyzed using Microsoft Excel and IBM SPSS.
Results: Twenty-two cases were identified. The mean age was 70.4 years (SD= 16.8), and 63.6% were male. The most common racial/ethnic backgrounds were Caucasian (36.4%) and East/Southeast Asian (27.3%). The majority (86.4%) of tests were ordered by neurology as part of stroke evaluation, with the remainder (13.6%) ordered by transplant. Orders originated similarly from the emergency department, medical/surgical floor, and intensive care (seven from each). The median time to test result was 7.7 days (IQR= 6.1–8.8). Prior to the CYP2C19 result, 11 patients started clopidogrel and three started ticagrelor. Following the CYP2C19 result, five (45.5%) were switched from clopidogrel to ticagrelor. One additional switch occurred pre-result for unclear reasons. Among the remaining clopidogrel patients, two were discontinued (one due to bleeding). One patient had clopidogrel maintained following a therapeutic platelet function study. Two were lost to follow-up. No patients started on clopidogrel post-test developed a subsequent stroke, though four patients already on chronic clopidogrel presented with new strokes. Of the three patients tested pre-transplant, two were started on voriconazole and both developed adverse effects (QT prolongation, dysphagia, transaminitis) and were switched to alternative antifungals.
Conclusion: CYP2C19 testing patterns were uneven, with neurology ordering the majority despite clopidogrel's use in other specialties. Test results did not universally affect decision-making, though most patients with abnormal results received alternative agents. Pharmacogenomics is valuable when drug activity depends on metabolism and may inform medication selection.

206. Biopsy Proven Cobalt Cardiomyopathy Secondary to Prosthetic Hip Temporized With N-Acetylcysteine Chelation

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Background: Cobalt is a heavy metal found in industry and older prosthetic joints which is known to cause cardiomyopathy in toxicity. N-Acetylcysteine may function as a chelator for cobalt when removal from exposure is not able to be quickly obtained.
Hypothesis or Research Question: N-Acetylcysteine will effectively decrease serum cobalt concentrations while awaiting definitive management with prosthetic hip removal.
Methods: This is a single-patient chart review. A 64-year-old woman with a history of hyperlipidemia, chronic kidney disease, and hip replacement performed in the late 1990s presented to the hospital due to shortness of breath. Her initial evaluation was significant for cardiomegaly on chest x-ray, and a new depressed left ventricular ejection fraction of 17% on echocardiogram. She was admitted to the hospital with new-onset heart failure. Cardiac MRI demonstrated dilated cardiomyopathy with severe global hypokinesis and biventricular dilation. Cardiac catheterization excluded ischemic cardiomyopathy. Given her history of an older prosthetic hip, cobalt induced cardiomyopathy was considered. A serum cobalt concentration was obtained and resulted at 360.3 mcg/L. Due to her significantly depressed cardiac function, she was deemed too high risk to undergo surgical removal of her cobalt orthopedic implant.
Results: One month following initial diagnosis of heart failure, the patient was evaluated in clinic by Medical Toxicology Based on a high suspicion for cobalt toxicity, she was started on N-Acetylcysteine 600mg twice daily for the following 45 days. She underwent a cardiac biopsy for definitive diagnosis which showed myocyte hypertrophy, moderate focal interstitial fibrosis, and abnormal mitochondrial forms consistent with cobalt cardiotoxicity. After completing N-Acetylcysteine course, the patient's repeat serum cobalt decreased to 278.2 mcg/L. A repeat echocardiogram was performed at that time which revealed an improving left ventricular ejection fraction of 27%. The patient also reported symptomatic improvement. Given the patient's improving clinical course while on N-Acetylcysteine without any significant side effects, she was continued on N-Acetylcysteine until surgery was able to be performed for cobalt hip removal.
Conclusion: N-Acetylcysteine is a safe and effective chelator for cobalt toxicity and may be associated with improved cardiac function.

207. Navigating a National Shortage: Trends in Hydroxocobalamin Utilization

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Background: Hydroxocobalamin is the first-line treatment for cyanide poisoning and is also used off-label for vasoplegia. In 2024, a national shortage prompted strict

prioritization and implementation of conservation measures, including reserving use for cyanide poisoning, sequestering inventory to emergency departments (ED) and central pharmacy, and deploying legacy medication alternative (LMA) alerts to encourage alternative use of methylene blue for vasoplegia.

Hypothesis or Research Question: Following the implementation of conservation strategies, how did hydroxocobalamin utilization patterns change during the national shortage at a large academic medical center?

Methods: This retrospective cohort analysis was conducted at an 874-bed Level 1 trauma center. The pre-shortage period was defined as February 26th to November 20th, 2024, and the shortage period as November 21st, 2024 to August 15th, 2025. These dates corresponded to the activation and removal of the healthcare system's LMA alert. Patients were identified via dispensing records for hydroxocobalamin. Descriptive statistics were used to compare utilization before and during the shortage, including location, indication, and prior use of alternative therapies.

Results: Hydroxocobalamin use declined during the shortage (46 vs. 132 dispenses). Nearly half of all administrations during the shortage occurred in the operating room (OR) (22/46, 48%), predominantly for cardiac surgeries such as heart transplantation (5/22, 23%) and ventricular assist device implantation (4/22, 18%), with additional use in liver transplantation (3/22, 14%). Most OR doses (91%) were override pulls without an associated provider placed medication order. Intensive care unit use decreased during the shortage (20 vs. 85 dispenses), while ED utilization remained stable (4 vs. 4 dispenses). All ED dispenses were for cyanide poisoning. Vasoplegia was the predominant use indication both before (128/132, 97%) and during (42/46, 91%) the shortage. Use of methylene blue prior to hydroxocobalamin increased during the shortage (17/42, 41%) compared to before (33/128, 26%). However, 'Contraindicated Drug Combination' alerts flagged for possible risk of serotonin syndrome when methylene blue was ordered with other serotonergic agents. Among the 25 patients who did not receive methylene blue prior to hydroxocobalamin during the shortage, it was documented to be withheld due to this concern in 3 cases.

Conclusion: Overall, the conservation strategies instituted at our facility were unsuccessful in preserving hydroxocobalamin use exclusively for cyanide poisoning. In future shortages, emphasis should be placed on improved provider education, particularly OR staff, and enhanced clinical decision support including the integration of automated dispensing cabinet alerts to optimize antidote allocation and encourage appropriate use of alternative therapies.

208. Wrong Drug: A Case of DRESS Syndrome Following a Pharmacy Dispensing Error

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Background: Lamotrigine is an antiepileptic drug known to cause serious dermatologic reactions, therefore it is typically started at a low dose and titrated over a period of time to reach the desired dose. Pharmacy medication errors can lead to patients being exposed to inappropriate drugs in incorrect dosing patterns, which may cause significant adverse events.

Hypothesis or Research Question: A pharmacy error leading to a patient taking high doses of lamotrigine can cause serious complications such as drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome.

Methods: This is a single patient case report. A 40-year-old female with history of hypertension was prescribed a refill of 200mg labetalol twice daily and began the medication as prescribed. Fourteen days later she developed symptoms of fever and maculopapular rash, presenting to the emergency department (ED) on three separate occasions over 10 days as well as to a dermatologist for biopsy. During the third ED visit it was recognized that the "labetalol" pills were 200 mg lamotrigine tablets. In the ED, clinical features and laboratory testing were concerning for DRESS syndrome. Lamotrigine was discontinued, and the patient was admitted for treatment with prednisone and N-acetylcysteine.

Results: At the third ED visit, the patient was noted to have a diffuse maculopapular rash encompassing > 50% of total body surface area without skin sloughing or mucosal involvement. Laboratory results were notable for leukopenia (1.8k cells/μL) with 48.4% neutrophils (880 cells/μL) and 9.3% eosinophils (170 cells/μL), AST 249 U/L, and ALT 203 U/L. No signs of pancreatic, lung, cardiac, muscle, or renal impairment were noted. Serum lamotrigine level was 4.2 μg/mL, confirming exposure. The patient was hospitalized for five days, with peaking transaminases on day three (AST 473 U/L and ALT 379 U/L) before downtrending. Her leukopenia resolved and she was discharged with a three-week taper of prednisone. Her skin biopsy taken prior to hospitalization was reported to be consistent with DRESS syndrome. The pharmacy error was reported to the dispensing pharmacy as well as the state education department.

Conclusion: This case demonstrates the potential for serious adverse drug effects due to pharmacy errors which may be difficult to recognize, with this case requiring four healthcare visits to diagnose leading to delay in discontinuation of the offending agent. Clinicians need to remain vigilant for

adverse drug reactions as a cause of unusual presentations, particularly after medication refills even in the absence of changes to a medication regimen.

209. Mechanical Capture and Hemodynamic Effect of Electrical Pacing in Cardiovascular Medication Poisoning

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Background: Electrical pacing is an established therapy for refractory unstable bradycardia from medical causes, but pacing failures have been reported in cardiovascular medication poisoning. The role of electrical pacing in this setting is not well described.
Hypothesis or Research Question: In cardiovascular medication poisoning-associated bradycardia, how frequently does electrical pacing 1) achieve mechanical capture and 2) improve hemodynamics?
Methods: This is a retrospective chart review of cases from one United States regional poison center. Included patients were >18-years-old with bradycardia secondary to suspected cardiovascular medication poisoning who received transcutaneous (TCP) or transvenous pacing (TVP) between 1/1/2014—12/31/2024. We excluded patients with pre-existing pacemakers, non-toxicologic etiologies of bradycardia, or missing data. The primary outcome was mechanical capture with TVP, defined as post-pacing heart rate >60 beats-per-minute or explicit documentation of paced rhythm or capture. Secondary outcomes were mechanical capture with TCP and post-pacing improvement in hemodynamics, defined as greater systolic or mean arterial blood pressures without increased hemodynamic support (vasopressors, inotropes, or mechanical support), or reduced hemodynamic support. Patients that improved hemodynamically with TVP vs. those that did not were compared for differences in demographics, exposures, and clinical features (e.g. initial heart rate, serum lactate, blood glucose, and bradydysrhythmias) by univariate testing.
Results: Of 39 cases screened, 20 were included: fifteen (75%) female, median age 64 (IQR 55–75) years. Fourteen (70%) developed bradydysrhythmias, the most common (five [25%] patients) being complete atrioventricular block. Fourteen (70%) received TVP, and 11 (55%) received TCP, with five (25%) receiving both. TVP achieved capture in 14 (100%, 95% CI 77–100%) cases and hemodynamic improvement in nine (64%, 95% CI 35–87%). TCP achieved capture in six (55%, 95% CI 23–83%) cases and hemodynamic improvement in three (27%, 95% CI 6–61%). Additional treatments between pre- and post-TVP hemodynamic assessments included calcium, lipid emulsion, sodium bicarbonate, glucagon, and hydrocortisone. We did not identify statistically significant differences in demographics, exposures, or clinical features in those that improved hemodynamically after TVP vs. those that that did not (p=NS).
Conclusion: Transvenous pacing resulted in frequent mechanical capture but inconsistent hemodynamic improvement in this retrospective observational study. Limitations include small sample size reducing ability to detect differences between those that did vs. did not improve with transvenous pacing, and that patients received multiple interventions between pre- and post-pacing hemodynamic assessments, which may have biased towards greater rates of hemodynamic improvement.

210. Left Ventricular Ejection Fraction in Patients With Shock After Negative Inotrope or Vasodilator Overdose

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Background: Left ventricular ejection fraction (LVEF) is a key metric of systolic function in drug overdose-associated shock. However, limited information exists on LVEF associations with mechanisms of hemodynamically significant drugs in overdose.
Hypothesis or Research Question: Do drug effects on LVEF in overdose align with pharmacological mechanisms?
Methods: We conducted a retrospective chart review of patients >18 years old receiving hemodynamic support (vasopressors, inotropes, or mechanical circulatory support) after suspected acute drug overdose involving drugs with either negative inotropic or vasodilatory properties who received transthoracic echocardiography (TTE) at our institution, 2013—2023. Patients with missing LVEF data, unknown drug of overdose, alternative etiologies of shock (e.g. sepsis), known prior systolic dysfunction (LVEF < 50%), cardiac arrest prior to TTE, or co-ingestion of both negative inotropes and vasodilators, were excluded. The outcomes were LVEF values and phenotypes: normal LVEF 50-70%, reduced <50%, and hyperdynamic >70%. Based on pharmacological mechanisms and prior literature, drugs were classified

as negative inotropes or vasodilators. Drugs with multiple potential mechanisms were assigned the predominant theorized effect of the drug class (e.g. all beta blockers were classified as negative inotropes). Systolic function was compared between the two drug classes via Wilcox rank-sum or Fisher exact testing, as applicable. Discordance from expected systolic function was defined as hyperdynamic LVEF in negative inotrope overdose or reduced LVEF in vasodilator overdose.
Results: After exclusions, 31 patients were included for analysis (median age 50.0 [IQR 38.0—55.5] years, 52% female, median LVEF 65.0% (IQR 56.0—75.0%). Median LVEF was 61.0% (IQR 56.0—71.0%) in negative inotrope overdoses vs. 71.5% (66.5—75.0%) in vasodilator overdoses (p=0.28). The LVEF phenotype was normal in 18 (58%) patients, reduced in three (10%), and hyperdynamic in 10 (32%).
Three of 25 (12%) patients with negative inotrope overdoses had reduced LVEF, and seven (28%) had hyperdynamic LVEF (discordance 28% [95% CI 12–50%]). Of six patients with vasodilator overdoses, three (50%) had hyperdynamic LVEF, and none had reduced LVEF (discordance 0% [95% CI 0–46%]). Phenotype distributions were not statistically different between negative inotrope and vasodilator overdoses (p=0.66).
Conclusion: In this small cohort of patients with drug overdose-associated shock, systolic function was frequently but not uniformly consistent with expected drug effects. These **results** illustrate the complexity of evaluating systolic function in overdose-associated shock and the need for further investigations. Limitations include small sample size, potential confounding via drug doses or structural cardiac abnormalities, and varying degrees of shock and inotrope support during TTE.

211. Survival Following Extracorporeal Membrane Oxygenation in Xenobiotic Exposures: Age Stratified Analysis of the National Poison Data System

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Background: Extracorporeal Membrane Oxygenation (ECMO) has shown promise in treating refractory shock and respiratory failure related to acute poisoning. ECMO survival rates are lower in elderly patients and consensus guidelines recommend individualized decisions to initiate ECMO in this population; however, pathophysiologic factors specific to acute poisoning could potentially mitigate age-related discrepancies in survival following ECMO.
Hypothesis or Research Question: Do survival rates following the initiation of ECMO for the treatment of acutely poisoned patients differ by age?
Methods: We conducted a retrospective, observational study using data reported to the National Poison Data System (NPDS). We queried the NPDS to identify all xenobiotic exposures in patients of all ages who underwent ECMO between January 2010 and December 2022. Cases were excluded if they missed age/survival outcomes, were confirmed to be a nontoxic exposure/non-exposure, or a majority of signs/symptoms were deemed "not related." Included cases were age-stratified into decades of life. Descriptive statistics were calculated, and survival proportions across age groups were compared using a chi-square test with a significance level of $\alpha = 0.05$.
Results: A total of 949 patients were identified with 189 excluded for missing data or non-toxicologic classification. After excluding an additional 14 cases of patients aged 70-79 years and 80-89 years for low counts, 746 patients were included in the final analysis. Overall, 384 (51.8%) were female, the median age was 26 years (IQR 17-42, range 0-69), and the aggregate survival rate was 41.3%. Age-stratified survival rates were 32/58 (55.2%), 91/211 (43.1%), 64/150 (42.7%), 44/119 (37.0%), 33/87 (37.0%), 29/73 (39.7%) and 15/48 (31.8%) for 0-9 years, 10-19 years, 20-29 years, 30-39 years, 40-49 years, 50-59 years and 60-69 years, respectively. While there was a trend towards decreased survival with increasing age, there was no significant difference in survival between the age groups ($X^2 = 8.4$, $p = 0.209$).
Conclusion: While we observed an overall trend towards decreased survival with increasing age, the survival rate of patients undergoing ECMO for severe poisoning did not differ significantly when stratified by decade of life. In fact, survival exceeded 30% even in patients over 60 years of age. This study is limited by its retrospective, observational design, and the inherent limitations of poison center data. Moreover, voluntary reporting results in under-capture of cases. Additional research is needed to further examine the role and potential benefits of ECMO in severely poisoned patients with specific attention to the various classes of toxins and other clinical factors.

212. ECMO for the Management of Severe Hydroxychloroquine-Induced Cardiotoxicity

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Background: Chloroquine and hydroxychloroquine are 4-aminoquinolones derivates of quinine which is derived from the Peruvian Cinchona tree with chloroquine historically used

as an antimalarial agent, although studies showing levels of high human toxicity led to the development of hydroxychloroquine as a less toxic metabolite.[1] Here we discuss a case report of an individual after overdose on hydroxychloroquine managed with sodium bicarbonate, lidocaine, vasopressors and ultimately ECMO.

Hypothesis or Research Question: ECMO can be utilized as an effective therapy for arrhythmia refractory to hydroxychloroquine-induced cardiotoxicity.

Methods: Here we present a single case report of a 32-year-old man who had overdosed on hydroxychloroquine, alprazolam and methocarbamol, requiring VA-ECMO for support through his cardiotoxicity.

Results: A 32-year-old man presented to the emergency department after being found unresponsive with suspected overdose on hydroxychloroquine, alprazolam and methocarbamol. He had become hypotensive, and bradypneic requiring intubation. His electrocardiogram showed sinus tachycardia with a QRS of 146ms and QTc of 639ms. Despite boluses of intravenous sodium bicarbonate, he remained hypotensive with a brief cardiac arrest with return of spontaneous circulation, requiring multiple vasopressors, and was started on an isotonic bicarbonate infusion. The toxicology service was consulted, and the patient was transferred to the intensive care unit. The patient continued to be hemodynamically unstable with QRS/QTc prolongation transiently responsive to boluses of sodium bicarbonate while on sodium bicarbonate and lidocaine infusions. Given refractory cardiogenic shock and arrhythmias, decision was made to cannulate for VA-ECMO. Point-of-care cardiac ultrasound confirmed global hypokinesis. Early after transition to VA-ECMO, he had multiple episodes of monomorphic ventricular tachycardia ultimately resolving with transvenous pacer placement. Urine drug liquid chromatography qTOF testing was positive for hydroxychloroquine, alprazolam, and methocarbamol, confirming initial suspicion for hydroxychloroquine-induced cardiotoxicity. He was de-cannulated from VA-ECMO on hospital day 4 with full recovery of biventricular failure. He was transferred out of the ICU on day 19 and discharged on an involuntary psychiatric commitment on day 29.

Conclusion: With hydroxychloroquine toxicity, mortality rates are estimated between 10-30%. Hydroxychloroquine can inhibit myocardial sodium and potassium channels in a dose-dependent fashion manifesting as prolonged QRS, and QTc intervals with risk for life-threatening dysrhythmias. Traditional therapies aimed at treatment of the sodium channel blockade secondary to hydroxychloroquine in this case were ineffective requiring ECMO and trans-venous pacer placement with subsequent recovery as the patient's acute toxicity resolved. ECMO was an efficacious therapy in this patient in the setting of severe hydroxychloroquine cardiotoxicity refractory to sodium bicarbonate, lidocaine, and vasopressor therapies.

213. Frequency of Clinically Significant Volume Overload in Cases of Suspected Poisoning Treated With High-Dose Insulin

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Background: High-dose insulin euglycemia (HIE) therapy is used to treat cardiovascular collapse from poisoning, improving hemodynamic stability via inotropic and metabolic effects. Although volume overload is a known complication of HIE, this is underreported in existing literature, which primarily focuses on electrolyte abnormalities and vasodilation.

Hypothesis or Research Question: How common is clinically significant volume overload seen with HIE use?

Methods: We conducted a retrospective chart review of cases from a single regional poison center in which HIE was administered for suspected poisoning between January 2019—December 2024. We included patients >18 years old receiving HIE (> 0.5 units/kg/hour). We excluded patients with a history of end stage renal disease (ESRD), those who were thought to have a non-toxicologic etiology of symptoms, and those who developed volume overload prior to HIE. Patient demographics, exposure substance class, past medical history, and clinical outcomes were extracted from poison center records. The primary outcome was volume overload, defined as any of the following occurring after HIE infusion: mechanical ventilation for respiratory failure, non-invasive positive pressure ventilation, continuous infusion of diuretics, continuous renal replacement therapy (CRRT) or hemodialysis not performed for toxin removal, veno-venous extracorporeal membrane oxygenation (VV ECMO), and abdominal compartment syndrome. Cases where therapy indications were unclear were reviewed by a second investigator for adjudication. Results are reported descriptively; a 95% confidence interval (CI) was calculated for the frequency of volume overload.

Results: Of 74 patients screened, 15 were excluded, and 59 were analyzed. The median age was 59 (IQR 43—68) years, and 35 (59%) were female. Four (7%) had a history of heart failure, and no patients had documented chronic kidney disease.

Seventeen (29%, 95% CI 18—42%) patients developed clinically significant volume overload: three patients (5%) required mechanical ventilation for respiratory failure, two (3%) failed extubation, five (8%) received diuretic

infusions, eight (14%) received CRRT, two (3%) patients received hemodialysis, and one (2%) received VV ECMO. No patients developed abdominal compartment syndrome.
Conclusion: Clinically significant complications of volume overload appear relatively common with HIE. Limitations to this study include the absence of a comparator group, absence of data on insulin concentrations, and the fact that some therapies like CRRT and intubations may have had multifactorial indications. However, this study is strengthened by focusing on clinically meaningful manifestations of volume overload. Further investigations are warranted to reduce rates of volume overload in this critically ill patient population.

214. Toxic Exposures to Novel Anti-Diabetic Agents: Insights From the ToxIC Core Registry

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Background: The toxicological profiles of newer anti-diabetic agents are not well defined. Understanding adverse effects and management across anti-diabetics is essential to guide clinicians faced with acute presentations.
Hypothesis or Research Question: Toxic exposures to novel anti-diabetic agents would result in class-specific adverse effects.
Methods: This is a case series from the ToxIC Core Registry. The ToxIC Core Registry includes patients that receive a formal consultation by a medical toxicology physician at participating sites. All cases involving a primary exposure to novel anti-diabetic agents between 2010 and 2024 were included, specifically Glucagon-Like Peptide-1 (GLP-1) receptor agonists, Dipeptidyl Peptidase-4 (DPP-4) inhibitors, and Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors. Descriptive statistics were performed to summarize demographics, frequency of implicated anti-diabetic agents, adverse outcomes, management, and mortality. Comparative analyses were conducted among newer drug classes to evaluate for significance differences.
Results: A total of 33 patients with a novel anti-diabetic drug exposure were identified, of whom 19 (58%) were female, with a median (IQR) age of 40 (25 – 58) years. Reported race included 10 (31%) non-Hispanic White patients, and 8 (24%) Hispanic. Novel therapies included 15 (46%) GLP-1 receptor agonists, 12 (36%) DPP-4 inhibitors, and 6 (18%) SGLT2 inhibitors. Overall, 14 (42%) patients developed hypoglycemia, occurring in 6 (40%) patients exposed to GLP-1 receptor agonists, 6 (60%) to DPP-4 inhibitors, and 2 (33%) to SGLT2 inhibitors. Hypotension (SBP <80 mmHg), respiratory depression, and acute kidney injury occurred exclusively in patients exposed to SGLT2 inhibitors, affecting 33%, 17%, and 17% of this group, respectively. Anion gap metabolic acidosis was observed in 12% of the cohort, predominantly among SGLT2 exposures. One case of rhabdomyolysis occurred following exposure to DPP-4 inhibitor. Glucagon and octreotide were administered in 6% and 15% of cases, respectively. Continuous renal replacement therapy (CRRT) and intubation were required in 3% and 6% of cases, while none required vasopressors or hemodialysis. Notably 18% of patients were admitted to the ICU, with 83% of those patients surviving to hospital discharge. Mortality occurred in 10% of patients, all of whom had exposures to either SGLT2 or DPP-4 inhibitors. Among these fatalities, extracorporeal membrane oxygenation (ECMO) was attempted in 33% without recovery. These findings are limited by a small sample size and by some missing data. Additionally, cases may not be representative as more severe presentations may be overrepresented in the registry.
Conclusion: This study highlights toxicological patterns across novel anti-diabetic agents. Further studies are warranted to better define the toxicological profiles of newer anti-diabetic agents.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

215. You Had Me at Physostigmine: Reversing A Pediatric Cyclobenzaprine Overdose

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Background: Cyclobenzaprine is a centrally acting muscle relaxant with structural similarity to tricyclic antidepressants (TCAs) and can cause significant anticholinergic toxicity and dysrhythmias in overdose. Physostigmine, a reversible cholinesterase inhibitor, is effective in reversing central anticholinergic symptoms. Concern exists of a potential risk of bradydysrthmias and seizures, specifically when patients display signs of cardiotoxicity, for using physostigmine in TCA overdoses. Pediatric cyclobenzaprine overdose is infrequently reported, and despite its utility, treatment with physostigmine remains uncommon.
Hypothesis or Research Question: Physostigmine can safely and effectively reverse the anticholinergic toxidrome in a pediatric cyclobenzaprine overdose.
Methods: This single-patient retrospective chart review of a 14-year-old female with a history of ADHD and asthma who presented with altered mental status (AMS) after ingesting an unknown amount of her grandmother's 10 mg cyclobenzaprine tablets. She was drowsy but arousable, confused, tachycardic at 120 BPM, with dry mucous membranes, warm to

the touch, and had significant urinary retention (1.5 liters by bladder sonography), consistent with an anticholinergic toxidrome. Initial labs were unremarkable, and acetaminophen, salicylate, and ethanol were negative. Electrocardiogram showed sinus tachycardia with a QRS of 68 ms and a QTc of 441 ms. There were no signs of sodium channel blockade, such as a prominent terminal R-wave in aVR.

Results: Medical toxicology was consulted and recommended intravenous fluids and, given the anticholinergic toxidrome, physostigmine. There were initial conversations with the primary team regarding the potential concern of physostigmine-precipitated seizures, but due to its safety profile and low concern in the context of a cyclobenzaprine overdose, the decision was made to proceed with administration. She received a total of 1 mg IV, in 0.5 mg increments, which improved her mental status, urinary retention, and other anticholinergic symptoms. After rapid improvement in her mental status, she was able to tolerate activated charcoal. She was observed, and her mental status fully resolved. She did not develop any seizure activity, require additional doses or further workup, and was discharged home in good condition.

Conclusion: This case highlights the importance of promptly recognizing an anticholinergic toxidrome, particularly in pediatric AMS cases. Pediatric overdoses involving cyclobenzaprine are a rare occurrence that warrants early identification of its potential anticholinergic and cardiotoxic effects. Physostigmine administration can quickly and effectively reverse central anticholinergic symptoms. In pediatric AMS cases where an anticholinergic toxidrome is suspected, using physostigmine can help prevent extensive emergency and inpatient workups, including neuroimaging or lumbar punctures.

216. Characteristics of Pediatric GLP-1 Agonist Exposures Reported to a Single Poison Center

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Background: Glucagon-like peptide-1 (GLP-1) receptor-agonists were initially approved for the treatment of type 2 diabetes mellitus but are now increasingly prescribed for their weight loss benefits. As these medications become more common in households, unintentional and exploratory exposures among younger children may rise. Limited literature describes the clinical presentation or recommended management of pediatric GLP-1 agonist exposures.

Hypothesis or Research Question: Among pediatric GLP-1 agonist exposures reported to a single poison center, what clinical effects were observed, and what management recommendations were provided?

Methods: Single poison center retrospective chart review of all cases involving GLP-1 agonists reported between January 2021 and October 2025. Cases were included if the patient was ≤18 years old and there was a single-substance exposure to a GLP-1 agonist. Variables included demographic data, exposure circumstances, substance, clinical effects, laboratory results, and poison center recommendations. Descriptive statistics were used to characterize the cohort.

Results: Twenty-two cases were identified between January 2021 and October 2025. Calls increased each year, with one call in 2021, two in 2022, four in 2023, and seven in both 2024 and 2025. There were 16 exposures to semaglutide, four to tirzepatide, and two to dulaglutide. There was an even distribution between male (10/22, 45%) and female (12/22, 55%) patients. Patients ranged in age from 4 months to 18 years (median age, 10.5 years). Most commonly, the reported exposure was unintentional (14/22, 64%), but there was one case of intentional self-harm in a 17-year-old male. Three cases (14%) involved teenagers accidentally administering incorrect doses. Twelve of the patients (55%) were not evaluated in a healthcare facility and were instructed to monitor for the development of symptoms at home. No patients who were evaluated at a healthcare facility were noted to be hypoglycemic. The most common clinical effects across exposures were nausea and vomiting, seen in 10 patients (45%). Of the 10 patients evaluated in the emergency department, antiemetics were recommended for five patients (50%). One 17-year-old patient required admission for intractable vomiting and a lactic acidosis of 5.7.

Conclusion: Pediatric GLP-1 agonist exposures are rare but are still an area of potential concern, as their use has become more widespread. In this cohort, most exposures were unintentional and resulted in mild gastrointestinal symptoms, which were managed supportively. Hypoglycemia and severe toxicity were not seen. Continued surveillance is warranted as prescribing expands and off-label adolescent use grows, particularly for weight management.

217. Descriptive Analysis of Pediatric Hypothermia Reported to U.S. Poison Centers

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Background: Hypothermia in the pediatric population is a potentially life-threatening condition that may manifest from environmental exposure and toxicological causes. Children have distinct vulnerabilities due to higher surface-area-to-body-mass ratio, lower glycogen reserves, and

developmental behaviors such as exploratory ingestion. The etiology regarding hypothermia in relation to pediatric ingestions is currently unknown.

Hypothesis or Research Question: This study was conducted to evaluate the age distribution, substance associations, clinical manifestations, and therapeutic interventions in pediatric cases presenting to US poison centers in which hypothermia was a clinical finding.

Methods: This was a retrospective review of hypothermia cases reported to U.S. poison centers. Data were extracted from the National Poison Data System (2005–2024) and included single substance exposures among patients 19 years old or younger. Age, associated substances, clinical effects, therapies, and reason for exposure were descriptively analyzed.

Results: A total of 4,878 cases were identified. Adolescents aged 13–19 years accounted for the largest proportion (42.3%), followed by children aged 0-2 years (32.3%); children aged 6–12 years made up 10.4% of cases. Males comprised 51.2% of the cases. The most common associated exposures were ethanol (8.5%), marijuana edibles and marijuana dried plant (7.6%), and clonidine (7.5%). Other notable substances included ibuprofen (2.3%), baclofen (2.1%), atypical antipsychotics (2.0%), and benzodiazepines (1.3%). Unintentional exposures accounted for 38.4% of exposures while suspected suicide attempts accounted for 18.9%. Ninety five percent of suspected suicide attempts occurred in adolescents. Other reasons included abuse/misuse (12.0%), adverse drug reactions (4.3%), and therapeutic errors (4.1%). One in four cases (24.5%) required intubation. Other commonly reported effects included drowsiness/lethargy (26.0%), tachycardia (23.8%), vomiting (21.0%), and bradycardia (20.7%).

Conclusion: Hypothermia in pediatric patients reported to U.S. poison centers was commonly associated with sedatives including ethanol, clonidine and marijuana. Adolescents represent the largest affected group and ethanol was the most common ingested substance associated with hypothermia. The clinical picture was seldom limited to hypothermia, with frequent co-occurrence of CNS depression and cardiac effects, necessitating advanced supportive care in nearly one-quarter of patients. Beyond unintentional consequences, suspected suicide attempt was the second most common reason for hypothermia. Safe storage of alcohol, prescription medications, and THC-containing edibles, particularly those resembling candy or snacks, is essential to reducing unintentional exposures in young children. For adolescents, who accounted for the largest group affected and demonstrated a substantial proportion of intentional exposures, prevention must also emphasize early identification of mental health concerns and access to appropriate behavioral-health resources.

218. A Wandering Embolus: Sequelae of Gel-Foam Renal Angiomyolipoma Embolization

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Background: Gel-foam products are commonly used in interventional radiology as temporary embolic agents during acute hemorrhage. These proprietary formulas are primarily composed of gelatin and water. They are designed to promote hemostasis by temporarily occluding the vasculature. While considered safe, intraoperative embolization is an uncommon complication defined by gel-foam fragment migration and systemic embolization. Management is largely supportive, with outcomes ranging from mild discomfort to hypoxemia and even death.

Hypothesis or Research Question: Angiomyolipomas (AML) are largely vascular tumors prone to hemorrhage, high-flow shunts, and arteriovenous connections. This permits the rapid systemic embolization of gel-foam fragments into local venous drainage and systemic circulation. These biologic products serve as both mediators for concerning occlusion and inflammation.

Methods: This is a single patient chart review. A 30-year-old female, G2P1001 at 30 weeks of gestation, presented to labor and delivery for embolization of bilateral renal angiomyolipomas (AML). Embolization of her estrogen-receptive AMLs was pursued due to the high likelihood of rupture with life-threatening postpartum hemorrhage, and her preoperative period was complicated by a small right spontaneous pneumothorax requiring a pigtail catheter placement. During her intraoperative renal angiogram, a "huge mass with near complete embolization was performed after superselection of the feeding artery with embosphere and Gelfoam. During embolization, a large draining shunt was identified." She then developed hypoxemia, tachycardia, and respiratory distress. A bedside echocardiogram was performed, noting right ventricular dilatation with increased right ventricular systolic pressure. She was placed on a high-flow nasal cannula (HFNC) at 30 liters, 70% FiO2. Computerized tomography angiography with IV contrast was performed and showed bilateral ground glass opacities and consolidations concerning for pulmonary embolism, multifocal pneumonia, or acute respiratory distress syndrome.

Results: Imaging, hemodynamic monitoring, and clinical presentation supported a diagnosis of pulmonary gel-foam embolus. Safety data sheets and existing case studies were reviewed, which suggest the severity of hemodynamic compromise is determined by the degree of pulmonary arterial obstruction and resulting hypoxemia. Supportive care was optimized using high-flow oxygen therapy, therapeutic

low-molecular-weight heparin, and pulse-dose dexamethasone to address suspected hemostatic pneumonitis. Importantly, gel-foam hemostatic materials undergo complete resorption within approximately 4–6 weeks, which may contribute to clinical improvement as embolic burden diminishes over time.
Conclusion: Gel-foam embolization presents with inflammation-mediated and mechanical obstructive patterns. Recognition of this complication is important, and treatment with supportive care is the mainstay of management.

219. An Uncommon Cause of Caustic Injury: Inner Components of an Alkaline AA Battery

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Background: While button battery ingestions have been well documented in the literature, reports of mucosal damage from exposure to cylindrical batteries are limited. This is largely because the mucosa is protected from the battery's alkaline inner material, preventing caustic injury.
Hypothesis or Research Question: Alkaline AA batteries can be a serious health hazard when the outer casing is compromised.
Methods: This is a single patient case report. A two-year-old male was brought to a local emergency department two hours after he was found chewing on an alkaline AA battery. The negative end of the battery was missing, and the internal components were exposed. The mother believed a portion of the battery had been swallowed. He had one episode of non-bloody emesis prior to hospital arrival. In the emergency department, the patient did not require oxygen, had multiple areas of ulceration on the oral mucosa, and was drooling blood-tinged saliva. An X-ray showed faint radiopaque foreign objects in the stomach. Over time, the patient had increased difficulty tolerating his secretions and developed mild stridor, prompting intubation for airway protection.
Results: After intubation the patient had a transnasal fiberoptic laryngoscopy performed, demonstrating superficial appearing ulcerations involving the hard and soft palate with peripheral eschar and uvular edema. Pharyngeal collapse and the endotracheal tube made further evaluation difficult. He received dexamethasone 4 mg given the findings. Endoscopy performed the next morning showed pinpoint erythematous lesions in the body of the stomach with no injury to the esophagus. The patient was extubated later that afternoon with improvement in uvular edema on repeat laryngoscopy. The patient was discharged on hospital day two with a proton pump inhibitor.
Conclusion: Cylindrical alkaline AA batteries contain a combination of zinc, potassium hydroxide, and manganese dioxide. Our case demonstrates that a compromised AA battery, even without full ingestion, can pose a significant risk of injury. Clinicians should be aware of this uncommon mechanism of exposure.

220. Severe Ibuprofen Poisoning With Multisystem Organ Failure, Refractory Shock, and Refractory Hyperlactatemia Successfully Treated With Therapeutic Plasma Exchange

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Background: There are few reports of supermassive ibuprofen overdose resulting in multisystem organ failure with severe acidosis and hyperlactatemia, and fewer reports of patients surviving such severe poisoning. While enhanced elimination has been proposed for these extreme situations, there is limited clinical data to guide this.
Hypothesis or Research Question: Plasma exchange may enhance elimination in ibuprofen poisoning.
Methods: This is a single patient case report. A 16-year-old boy with history of depression and substance use overdosed on three bottles of ibuprofen and was taken to a community emergency department. At presentation, he was alert, with heart rate 122 bpm, blood pressure 132/70 mmHg, and otherwise asymptomatic. Initial labs were all within normal range. Serum concentrations of acetaminophen, salicylate, and ethanol were undetectable, and urine drug screening was positive only for cannabinoids. He had decline in level of consciousness requiring intubation and was transferred to the pediatric intensive care unit of an academic children's hospital.
Results: On arrival, venous pH was 7.16 (7.33-7.43) with lactic acid 10.0 mmol/L (1.0-1.4 mmol/L), bicarbonate 17.5 mmol/L (22-26 mmol/L), and blood pressure 80/46 mmHg. Hemodynamics and subsequent blood gas values progressively worsened despite increasing interventions, including sodium bicarbonate boluses and continuous infusion, norepinephrine infusion (0.3 mcg/kg/min), epinephrine infusion (0.3 mcg/kg/min), vasopressin infusion (2 milli-units/kg/min), calcium chloride infusion, and colloid administration, to pH 7.02 and lactic acid greater than 17.0 mmol/L. Given refractory shock, veno-arterial extracorporeal membrane oxygenation (ECMO) was initiated. Three hours after ECMO initiation, vasopressor requirements had not improved and lactic acid levels remained above detection threshold, and continuous veno-venous hemodiafiltration (CVVHD) was initiated. Two hours after CVVHD initiation, vasopressor

requirements and lactic acid levels had not improved, and therapeutic plasma exchange (TPE) was initiated with 50% albumin (100g in 2L) and 50% plasma. One hour after TPE initiation, lactic acid level dropped to 16.9 mmol/L and continued to decline. Ninety minutes after initiating TPE, vasopressor requirements decreased and continued to decrease. Serum ibuprofen concentrations obtained prior to initiating TPE and after TPE were 620 and 200 mcg/mL, respectively. Patient was ultimately discharged to home on hospital day 35.
Conclusion: While severe ibuprofen poisoning is rare, it is associated with high morbidity and mortality. Despite being primarily renally cleared, ibuprofen and its metabolites exhibit extremely high levels of protein binding and are not effectively cleared by hemodialysis. Plasma exchange should be considered in cases of extreme ibuprofen poisoning.

221. Trends in Pediatric Toxin-Induced Seizures reported to the ToxIC Core Registry

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Background: Toxicological exposures that cause seizures in pediatric patients vary widely in both causative agents and management strategies. This study aimed to characterize the exposures associated with toxin-induced seizures in pediatric patients.
Methods: The Toxicology Investigators Consortium (ToxIC) Core Registry contains over 110,000 cases of patients evaluated at the bedside by participating medical toxicologists. The current analysis included pediatric patients (17 and younger) who experienced a seizure between January 1, 2012 and May 31, 2025. Descriptive statistics were used to identify the most common agents and to summarize key clinical outcomes.
Results: Of the 24965 pediatric cases in the ToxIC Core Registry during the time period, 1309 involved suspected toxin-related seizures (5.2%). Patients ranged from 2 to 17 years, and 5 patients (0.4%) had a prior history of seizures. The most common exposure agent classes were antidepressants (N=582; 44.5%) and anticholinergic/antihistamine agents (N=233; 17.7%), with bupropion (N=364; 27.8%) and diphenhydramine (N=199; 15.2%) being the most frequent specific agents. The most common toxidrome was anticholinergic syndrome (N=249; 19.0%). Primary reasons for the consultation were intentional pharmaceutical/nonpharmaceutical exposures (N=1125; 85.9%) and unintentional pharmaceutical/nonpharmaceutical exposures (N=144; 11.0%). Among the intentional ingestions, 63.8% (N=836) were self-harm attempts.
Benzodiazepines were administered in 68.8% (N=901) of patients, anticonvulsants in 9.8% (N=128), propofol in 4.0% (N=52), and phenobarbital in 0.9% (N=12). Over half (N=717; 54.8%) were admitted to the intensive care unit, and 26.8% (N=351) were intubated. Among intubated patients, antidepressants (N=128; 36.5%) were the most common exposures, with bupropion accounting for the majority (N=126; 35.9%). There were 11 deaths (0.8%), and 36.4% (N=4) of these deaths were associated with diphenhydramine exposure. Younger pediatric patients ≤10 years old (N=151; 11.5%) had fewer intubations (N=25; 16.5%) and deaths (N=1; 0.7%). Exposures in younger patients were attributed to psychoactive agents (N=36; 23.8%), with the majority of these being cannabinoid products (N=22; 61.1%). Older pediatric patients between 11–17 years (N=1158; 88.5%) had higher rates of intentional exposures with self-harm attempt (N=831; 71.8%). More severe outcomes occurred in these patients, with intubation in 28.2% (N=326) and 0.8% died (N=10). There were no significant differences in treatment choices among the two age groups.
Conclusion: Toxin induced seizures in pediatric patients were most commonly associated with antidepressants and anticholinergic agents, particularly bupropion. Older patients (ages 11-17 years) experienced more severe outcomes, likely related to a higher frequency of intentional self-harm exposures, highlighting the importance of age specific prevention and management strategies.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

222. Severe Hypermagnesemia and Cardiac Arrest Following Accidental Milk of Magnesia Administration in an Infant

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Background: Hypermagnesemia is rare in infants because of efficient renal excretion and limited exposure to magnesium-containing products. Toxicity typically arises in the context of renal impairment or excessive magnesium ingestion. Milk of Magnesia, an over-the-counter laxative, is not recommended for infants and is rarely associated with life-threatening toxicity. We describe a case of profound hypermagnesemia resulting in cardiac arrest in a previously healthy infant after unintentional administration of Milk of Magnesia.

Hypothesis or Research Question: Ingestion of Milk of Magnesia by an infant without underlying renal disease can result in severe hypermagnesemia and cardiac arrest.
Methods: This is a single-patient case review. A previously healthy seven-month-old girl presented to the emergency department with one day of vomiting and diarrhea. During evaluation, she developed bradycardia, poor perfusion, and pulseless electrical activity cardiac arrest. Cardiopulmonary resuscitation was performed for 18 minutes, and three doses of epinephrine were administered before return of spontaneous circulation. The initial history, limited by a language barrier, revealed no known ingestion. The serum magnesium concentration was unmeasurably high. She was admitted to the pediatric intensive care unit for post–cardiac arrest care, including targeted temperature management, mechanical ventilation, electrolyte correction, and seizure prophylaxis with levetiracetam and lacosamide.
Results: Admission laboratory studies showed severe hypermagnesemia (>10 mg/dL), hypocalcemia (Ca 6.8 mg/dL), hypernatremia (Na 160 mmol/L), and acute kidney injury (SCr 1.48 mg/dL). With supportive management, magnesium levels decreased gradually (10.2 to 2.9 mg/dL over several days). The patient developed recurrent seizures, and magnetic resonance imaging demonstrated hypoxic–ischemic encephalopathy. Following extubation, she exhibited persistent spasticity, neuroirritability, and profound neurologic impairment. She remained hemodynamically stable but continued to have significant feeding difficulties, absent visual tracking or voice response, and minimal responsiveness to external stimuli. Subsequent history obtained through an interpreter revealed that caregivers had substituted Milk of Magnesia for infant formula for approximately two days before presentation, misunderstanding its purpose and safety. A long-term neurorehabilitation plan was initiated.
Conclusion: Accidental administration of Milk of Magnesia in infants can cause profound hypermagnesemia leading to cardiac arrest and irreversible neurologic injury, even in the absence of renal dysfunction. This case emphasizes the need for culturally and linguistically appropriate caregiver education and highlights hypermagnesemia as a reversible but potentially fatal cause of pediatric cardiac arrest.

223. NO Laughing Matter: Nitrous Oxide Use in California From 2014-2024

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Background: Nitrous oxide is a gas used as a medical anesthetic agent and food propellant. Abuse typically involves inhalation from pressurized cartridges, colloquially known as "whip-its". Recreational use is associated with euphoria as well as lightheadedness, confusion, dysrhythmias, seizures, and thermal skin injury. Long-term use can result in myeloneuropathy and megaloblastic anemia from functional vitamin B12 deficiency, which can become chronic if abuse is not stopped and supplemental vitamin B12 not started, though the most effective dosing regimen has not been determined.
Hypothesis or Research Question: What variations exist in vitamin B12 treatment recommendations for patients with nitrous oxide toxicity in California poison control centers?
Methods: This is a retrospective study of patients with nitrous oxide exposures from the California Poison Control System January 1, 2014 to December 31, 2024. All patients with reported nitrous oxide exposures were included. Poison Center electronic medical records were abstracted using a pre-defined abstraction sheet. Results were analyzed using descriptive statistics in Microsoft Excel (Microsoft Corp., Redmond, WA, USA). Chronic exposures were defined by reported use longer than one month.
Results: In total, 221 patients were identified: 143 males and 78 females. years. The mean age was 28.5 years (n=218; 2-73 years). Most exposures were recreational use (77%). Other reasons included suspected suicide (6%), industrial use (6%), and occupational exposure (3%). Eighty (36%) were chronic use, whereas 46 (21%) were acute cases; the remainder were not defined in the documentation.
The most common symptoms included altered mental status (31%), peripheral neuropathy (25%), ataxia (15%), and motor weakness (12%). MRI results were documented for 15 patients, and 5 (33%) had typical myelopathic findings. Sixteen patients had anemia, 3 (19%) with documented macrocytic anemia (19%).
Vitamin B12 supplementation was recommended in 83 (37%) cases, though only 60 (27%) were documented having received it. In total, nine different vitamin B12 regimens were recommended, and adjunctive methionine was recommended in eight cases. No follow up data were available regarding treatment adherence or response.
Conclusion: Nitrous oxide toxicity was uncommonly reported to the Poison Center, with more chronic than acute users. A minority were symptomatic and fewer had characteristic imaging findings. Appropriate treatment for chronic toxicity was recommended in 37% of cases, with widely heterogeneous dosing recommendations. No outcomes were otherwise available due to lack of longer-term follow-up data.
While cessation and vitamin B12 supplementation remain the cornerstones of treatment, further research is needed to determine the best vitamin B12 regimen.

224. Isolated Clonidine Ingestions in Pediatric Patients Reported to the Toxicology Investigators Consortium Core Registry

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Background: Clonidine is a centrally acting α_2-adrenergic receptor agonist often used to treat attention-deficit hyperactivity disorder. Because of its availability, it is often implicated in exploratory and in intentional overdoses in the pediatric population.
Hypothesis or Research Question: What are the demographics, clinical features, treatments, and outcomes of pediatric clonidine ingestions reported to the American College of Medical Toxicology's Toxicology Investigators Consortium (ToxIC) Core Registry?
Methods: This was a review of prospectively collected de-identified patient information reported to the ToxIC core registry by medical toxicologists providing bedside care for poisoned patients between January 1, 2012 and July 1, 2025. Pediatric patients less than 18 years old with clonidine ingestions, whether intentional or unintentional, were included. Data regarding patient demographics, clinical features, antidote administration, other interventions, and outcomes were reviewed.
Results: There were 443 cases entered into ToxIC during the study period. The median age was 6 years, range 2 – 17 years. Females accounted for 222 (50.1%) patients. The median age of 251 (56.7%) children with unintentional ingestions was 3 years (range 2 – 17 years), with 95 (37.8%) female patients. The most common clinical manifestation was central nervous system (CNS) depression, which was observed in 200 (79.7%) cases. Bradycardia was reported in 95 (37.8%) patients. Hypotension was present in 62 (24.7%) cases. Bradypnea was seen in 20 (8%) patients. No patients had QRS widening or QT prolongation on electrocardiogram (ECG). There were no deaths. Naloxone was administered to 91 (36.3%) patients. Intravenous fluid resuscitation was required in 97 (38.6) cases. Endotracheal intubation with mechanical ventilation was performed in 34 (13.5%) cases. Vasopressors were administered to seven (2.8%) patients. Of the 192 (43.3%) cases that were considered intentional overdoses, the median age was 14 years (range 3 – 17 years). Females accounted for 127 (66.1%) cases. CNS depression was observed in 130 (67.7%) cases. Bradycardia was reported in 117 (60.9%) patients. Hypotension was present in 43 (22.4%) cases. Bradypnea was seen in 6 (3.1%) patients. There was one patient with QRS widening but no cases on QT prolongation on ECG. There were no deaths. Naloxone was administered to 50 (26%) patients. Intravenous fluid resuscitation was required in 84 (43.8%) cases. Endotracheal intubation with mechanical ventilation was performed in 15 (7.8%) cases. Vasopressors were administered to 12 (6.3%) patients.
Conclusion: In this study, pediatric clonidine overdoses were characterized primarily by CNS depression and bradycardia. Hypotension was observed less commonly and typically responded to intravenous fluids.
ToxIC: This research was performed by the ACMT Toxicology Investigators Consortium

225. Linezolid Toxicity Monitoring in the Setting of Intermittent Hemodialysis Versus Continuous Renal Replacement Therapy

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Background: We report a novel case of trending serial serum linezolid concentrations in a patient receiving sequential hemodialysis and continuous renal replacement therapy (CRRT).
Hypothesis or Research Question: Linezolid should theoretically clear faster with intermittent hemodialysis (IHD) than CRRT, but does theory reflect clinical practice in a patient with serial linezolid concentrations receiving both IHD and CRRT?
Methods: This is a single patient chart review case report. A 59-year-old male with history of metastatic colon cancer status post sigmoid resection with recurrence and hepatic metastases status post living donor liver transplant twice admitted for acute on chronic rejection and management of a recurrent infection. He presented to the transplant ICU (TICU) from a long-term acute care facility with worsening mentation, with his work-up demonstrating kidney injury (Cr 1.6 - 2.0 mg/dL), thrombocytopenia (39 x 109/L), hyperlactatemia of 7.3 mmol/L, and elevated linezolid level of 36.2 mg/L. The patient had been treated with linezolid continuously for approximately 125 days. On arrival to the TICU, he was intubated for airway protection, and he underwent intermittent hemodialysis (IHD) for 11 hours. Pre-IHD linezolid and lactate levels were 29.1 mg/L and 11.5 mMol/L respectively, and levels were trended throughout dialysis.
Results: Post-IHD linezolid was 2.7 mg/L and lactate was 6.5 mMol/L. Following cessation of IHD, linezolid rebounded slightly to 3.0 mg/L, however lactate levels

increased dramatically. At this point, continuous renal replacement therapy (CRRT) was initiated. Although linezolid levels did decrease with CRRT, the rate of clearance was significantly slower. At this point, it seemed unlikely that linezolid toxicity was the primary driver of his presentation, and other processes were investigated. BAL cultures were concerning for Stenotrophomonas. Unfortunately, the patient's hospitalization was complicated by recurrent infections from various opportunistic infections culminating in his death.
Conclusion: Linezolid removal is more rapid with IHD than CRRT. There was an unexpected rebound after stopping hemodialysis given its low volume of distribution. There is an association with linezolid toxicity and hyperlacticaemia, but concomitant conditions are confounding factors.

226. Fatal Vasopressin Overdose Resulting in Refractory Cardiac Arrest: A Case Report

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Background: Vasopressin is a potent vasoconstrictor and antidiuretic hormone. Reports of vasopressin overdose and its management are exceedingly rare.
Hypothesis or Research Question: What is the clinical presentation and outcome of a patient with a massive vasopressin overdose?
Methods: This is a single patient chart review. A 67-year-old female suffered from an iatrogenic error while receiving vasopressin when her pump was mis-programmed and inadvertently administered 100mL of a 40 Unit/100mL solution over one hour. This led to the patient receiving an infusion rate of 0.66 units/minute, well above the typical rate of 0.01 - 0.04 units/minute. Shortly after, the patient complained of chest pain and shortness of breath before becoming pulseless with a narrow, then sinusoidal rhythm on telemetry. The patient was managed with standard ACLS protocols except for epinephrine, which was held due to concern for coronary vasospasm and vasoconstriction. The patient was also given amiodarone when her telemetry rhythm showed ventricular tachycardia. Further interventions included a nitroglycerin bolus in an attempt to counteract the vasoconstriction from the vasopressin. Additionally, a single bolus of lipid emulsion therapy was given with no reported effect. Resuscitative efforts were continued for approximately 45 minutes but unfortunately were futile and the patient expired. A subsequent autopsy listed vasopressin in the cause of death list.
Results: To date, very limited literature exists describing complications associated with large iatrogenic intravenous doses of vasopressin. Cases of cardiovascular complications, including bradycardia and cardiac arrest, have been reported in gynecologic procedures though in these cases, events were transient and responded to aggressive supportive measures. In this case, a massive vasopressin overdose caused acute hemodynamic changes leading to cardiac arrest with multiple arrythmias, including narrow-complex tachycardia, a sinusoidal rhythm, and wide-complex tachycardia. Standard ACLS protocols (excluding epinephrine), a nitroglycerin bolus, and lipid emulsion therapy were unsuccessful in resuscitating the patient.
Conclusion: Vasopressin overdose can cause hemodynamic instability, arrythmias, and cardiac arrest that may be refractory to standard treatment modalities. Awareness of the serious nature of this potential iatrogenic complication and strategies to mitigate dosage errors are essential.

227. Clinical Effects of Ranolazine Overdose: A Retrospective Poison Control Center Study

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Background: Cardiac disease is a major source of morbidity and mortality in the US, and angina pectoris in the context of coronary artery disease is associated with increased healthcare utilization. Initially approved in 2006 for treatment of refractory angina, ranolazine has rarely been reported as causing life-threatening events.
Hypothesis or Research Question: What are the typical manifestations and outcomes of supratherapeutic ranolazine ingestions?
Methods: This is a retrospective study of cases called into a regional poison control center who were evaluated in a healthcare facility after a supratherapeutic ingestion of ranolazine. A search of Toxicall® was performed of all cases with exposure to ranolazine from January 2006 until December 2024. Each case was reviewed for coded effects, therapies provided, and outcomes. The narrative was also reviewed to ensure capture of all clinically relevant data.
Results: A total of 34 cases of supratherapeutic ingestion of ranolazine evaluated in a healthcare facility were identified. Most ingestions occurred in patients over the age of 40 (73.5%), were unintentional (74.7%), and had coingested medications (64.7%). The most common clinical effects were hypotension and mild CNS depression (n=7

for each, 20.6 %). There were only five instances of QTc prolongation in our cohort (14.7%).The majority (n=24, 70.8%) had symptoms lasting less than eight hours. Most cases (n = 19, 55.9%) were coded as no effect, followed by moderate effect (n = 12, 32.4%). There were no cases coded as a major effect. There was only one death in our cohort attributed to ranolazine, an asystole cardiac arrest which occurred in a female in her 60s four hours after arrival. The only coingested medication was acetaminophen (initial level 469 µg/mL) and all other labs drawn at the time of arrival were normal. The available literature on ranolazine overdose only includes scattered case reports. Most of these reports indicate effects including hypotension, seizure, significantly prolonged QT intervals, and two occurrences of dysrhythmia. In our cohort, there were no instances of ventricular tachydysrhythmia and only a single seizure episode. Hypotension and QT prolongation did occur, however our data suggests that most patients only required supportive care prior to medical clearance.

Conclusion: Most cases of supratherapeutic ranolazine ingestion have no effect, but QT prolongation, hypotension, and seizures may occur. Patients with small, asymptomatic unintentional overdoses of ranolazine can be managed conservatively and dispositioned after a brief period of observation.

228. Clinical Case: Severe Acute Liver Failure Due to Combined Paracetamol and Glufosinate Poisoning Treated With Intermittent Hemodialysis, Plasma Exchange, High-Dose NAC, and Intensive Supportive Care

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Background: Combined intentional ingestion of Paracetamol and Glufosinate is rare. Severe Paracetamol poisoning can lead to acute liver failure with high mortality risk, which may be further aggravated when co-ingested with Glufosinate. Intermittent hemodialysis (IHD) facilitates elimination of Paracetamol and partially removes Glufosinate. Plasma exchange (PEX) serves as an adjunctive therapy for acute liver failure by removing endogenous and exogenous toxins, supporting failing hepatic function, and providing time for hepatic recovery. We report a case of severe acute liver failure due to combined Paracetamol and Glufosinate poisoning treated successfully with IHD, PEX, high-dose N-acetylcysteine (NAC), and intensive supportive care.

Hypothesis or Research Question: In cases of severe acute liver failure caused by combined paracetamol and glufosinate poisoning, can a multimodal treatment strategy incorporating intermittent hemodialysis, plasma exchange, and high-dose N-acetylcysteine effectively reverse hepatic injury and improve clinical outcomes?

Methods: Data were obtained through caregiver interview, direct clinical observation during hospitalization, and retrospective review of medical records.

Results: A 21-year-old male with a history of prior suicide attempt ingested 120 tablets of 500 mg Paracetamol in a suicide attempt, subsequently developing multiple episodes of vomiting with brown-red gastric content, dark urine, and abdominal pain. Twenty-four hours later, he ingested approximately 200 mL of Glufosinate herbicide, after which he experienced burning pain in the oropharynx and vomiting of green-brown material. Upon admission, he was conscious. Laboratory findings included AST/ALT >7000 U/L, ammonia 135 µmol/L, total/direct bilirubin 73.4/30.6 µmol/L, PT 21%, INR 3.26, and platelets 56 G/L. Urine tests were positive for Paracetamol (TLC method) and Glufosinate. The patient underwent plasma exchange combined with intermittent hemodialysis (IHD), high-dose intravenous NAC, and supportive management including fluid therapy, electrolyte correction, proton pump inhibitor, antiemetics, and 12-hour monitoring of blood gases and coagulation. The first IHD session was initiated six hours after admission, followed by the first PEX session, a second IHD session, and two additional PEX procedures. After approximately 76 hours of treatment, AST/ALT decreased to 72/392 U/L; ammonia decreased to 48 µmol/L; total/direct bilirubin decreased to 53.1/19.4 µmol/L; PT improved to 56%, and INR decreased to 1.51. The patient remained conscious, showed marked clinical and biochemical recovery, and was subsequently transferred for psychiatric management.

Conclusion: Severe acute liver failure due to combined Paracetamol and Glufosinate poisoning can be successfully managed using a multimodal approach including intermittent hemodialysis, plasma exchange, high-dose NAC, and intensive supportive care.

229. Descriptive Analysis of Race and Gender Breakdown in the Population of Current and Trainee United States Medical Toxicologists

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Background: Increased diversity is associated with positive outcomes in healthcare systems such as improved patient outcomes, more effective team communication, and improved financial performance. Prior analyses demonstrate a mismatch between demographic characteristics of US physicians and those of the US population as a whole. There are, however, no published data regarding the current demographic profile of US-based medical toxicologists.
Hypothesis or Research Question: What are the demographic characteristics of current US-based medical toxicologists?
Methods: This is a descriptive, cross-sectional analysis of self-reported demographic data from American College of Medical Toxicology (ACMT) members. We included full, associate, emeritus, and fellow-in-training (FIT) members based in the United States. International, affiliate, resident, and student members were excluded. Missing or ambiguous responses were categorized as "unknown." Descriptive statistics were generated to characterize the composition of the medical toxicology workforce overall and within subgroups. All data were analyzed using Microsoft Excel.
Results: Full, Associate, and Emeritus ACMT members were 67.5% (420/624) male, 31.7% (198/624) female, and 0.2% (1/624) gender-nonconforming; 0.8% (5/624) did not respond or indicated "prefer not to answer." Of these, 65.4% (408/624) identified as White, 11.5% (72/624) Asian, 4% (25/624) Hispanic/Latino, 1.3% (8/624) Black or African, 0.5% (3/624) Middle Eastern or North African, 0.5% (3/624) Native Hawaiian, 0.2% (1/624) American Indian, and 3.2% (20/624) Multi-racial; 8.3% (52/624) preferred not to answer and 5.2% (32/624) did not respond or indicated "unknown." Among FIT members, 51.4% (56/109) were male and 48.6% (53/109) were female. 67.9% (74/109) identified as White, 10.1% (11/109) Asian, 4.6% (5/109) Black or African, 2.8% (3/109) Hispanic/Latino, 0.9% (1/109) Asian, Native Hawaiian or Other Pacific Islander, 0.9% (1/109) Middle Eastern or North African, and 3.7% (4/109) Multi-racial; 5.5% (6/109) preferred not to answer and 3.7% (4/109) did not respond. Among Full, Associate, Emeritus, and FIT members, birth decade ranged from the 1920s to 1990s. There is an inverse association between the percentage of members identifying as female and age.
Conclusion: This study describes the current demographic makeup of US-based toxicologists and apparent demographic shifts over time. Given previously established benefits of increased physician diversity, further research should be pursued supporting the goal of increased diversity in medical toxicology.

230. Disparities in Emergency Department Use of Urine Toxicology Screening

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Background: Prior studies demonstrate that discretionary urine drug screen (UDS) testing varies significantly by patient race, ethnicity, and gender across multiple clinical settings, including trauma, burns, and obstetrics. These disparities decrease with implementation of standardized testing protocols, suggesting that protocolization may mitigate bias. In the emergency department (ED), certain presentations such as ST-segment elevation myocardial infarction (STEMI) and Stroke alerts trigger protocolized activation pathways, whereas similar but non-activated complaints (e.g., chest pain, dizziness) rely on clinician discretion. It is unclear whether demographic disparities in UDS use described in trauma care also exist between protocolized and non-protocolized ED presentations or whether related resource use differs similarly.
Hypothesis or Research Question: Do racial or gender-based disparities in UDS ordering and related resource use differ between non-protocolized ED complaints (chest pain, dizziness) and protocolized activations (STEMI, Stroke)?
Methods: This is a retrospective chart review of 350 ED encounters at a single urban academic medical center. Encounters were grouped as chest pain vs. STEMI and dizziness vs. Stroke. Extracted data included demographics, UDS ordering, UDS results, and consultations (social work, psychiatry, emergency psychiatry [EPAT], and toxicology). Analyses used chi-square or Fisher's exact tests for categorical variables and independent t-tests for continuous variables, reported as means with standard deviations.

Results: UDS ordering differed significantly by demographic characteristics. Males were more likely than females to receive a UDS (27.7% vs. 16.6%; p = 0.012). Patients who received a UDS were younger (57.1 ± 14.8 years) than those who did not (63.9 ± 16.0 years; p < 0.001). Racial differences were observed, with a higher proportion of Black/African American patients in the UDS group (64.0%) compared with the non-UDS group (44.4%), and a lower proportion of White patients in the UDS group (22.7% vs. 52.4%). Resource utilization also differed. Inpatient psychiatry consultations were more frequent in the UDS group (9.3% vs. 2.5%). EPAT and toxicology consultations were also more common among UDS patients (both 4.0% vs. 0.4%). Social work consultations occurred more often in neurological presentations than cardiac presentations (31.2% vs. 19.7%; p = 0.023). Among STEMI patients specifically, social work involvement was higher compared with non-STEMI encounters (42.7% vs. 17.0%; p < 0.001).
Conclusion: UDS ordering in the ED demonstrates significant demographic disparities, with younger, male, and Black/African American patients more likely to undergo testing. Differences in consultation patterns between protocolized and non-protocolized presentations suggest that discretionary testing may contribute to inequitable care. Standardized testing pathways may help reduce these disparities.

231. Implementation of a Pharmacist-Led Toxicology Support System at Our Tertiary Care Hospital.

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Background: Japan's Poison Information Centers provide essential toxicology information focused primarily on generalized poisoning information rather than individualized patient management. Given these functional differences from poison centers, hospital-based pharmacists in Japan play a key role in toxicology support. This report describes the implementation of a pharmacist-led toxicology support system at our tertiary care hospital.
Hypothesis or Research Question: We hypothesize that trained hospital pharmacists can effectively assist in the management of toxicologic patients.
Methods: Pharmacists at our medical center developed a hospital-based toxicology support framework centered on a poison hotline. Information was collected (from the Japan Poison Information Center, international literature, and cases from our facility) and organized into an electronic "Toxicology Notebook." A printed version of the notebook was used in daily practice by pharmacists and Emergency Department (ED) physicians to provide immediate, evidence-based consultation. An antidote stock list and standardized management guidelines were established, including dosing adjustments based on pharmacokinetic factors (e.g., renal impairment) as well as considerations for potential drug interactions. Educational sessions were provided for hospital pharmacists, and additional lectures were conducted for paramedic and nursing students.
Results: A hospital-based poison hotline was established in May 2022 and is operated by one trained pharmacist during weekday hours (8:30 a.m.–5:15 p.m.). During nights and weekends, general on-duty pharmacists handle toxicology-related inquiries due to staffing limitations, and establishing 24/7 coverage remains a future challenge. From May 2022 through October 2025, the hotline has managed a total of 30 calls, primarily at night and from Nurse Practitioners working in the ED. A notable case involved real-time guidance on the indication and dosing of N-acetylcysteine for acetaminophen poisoning. Pharmacists used the Toxicology Notebook to provide information on toxic agents, medications, and treatment recommendations to support early clinical decision-making. A toxicology drill was conducted involving hospital staff and First Responders, promoting interprofessional collaboration and enhancing institutional toxicology preparedness.
Conclusion: Implementation of this pharmacist-led toxicology support system improved access to real-time toxicology information, standardized antidote management, and expanded educational activities within and outside the hospital. This model may enhance patient care for toxicologic emergencies, particularly in regions where poison center resources are limited. We plan to track hotline consultations and clinical outcomes to better assess the effectiveness of this system.

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