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Increase in hand sanitiser use during Covid-19 – compliance and safety

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Background: Covid-19 resulted in a sharp increase in the use of, demand and supply for alcohol-based hand sanitisers. A dramatic increase in calls to the NSW Poisons Information Centre (PIC) regarding hand sanitiser exposures prompted this investigation into increased risks of hand sanitisers.

Methods: This prospective observational study aims to evaluate hand sanitiser products resulting in calls to the NSW PIC from April to July 2020. Photos and extra information of products including brand, alcohol type and percent, bottle size, formulation, country of manufacture, amount ingested and symptoms were obtained during normal NSWPIC operation. Follow-up phone calls were made following caller’s permission to determine outcome of exposures. Two specialists in poisons information critically reviewed all images for compliance. First step determined whether the products classify as therapeutic goods or cosmetic goods in accordance with therapeutic good regulations. Second determined appropriateness of labelling and packaging respectively against its category.

Results: 309 images were received from callers for 124 separate hand sanitisers. Review of images revealed 105 products (84.7%) classified as cosmetic goods, 17 made claims that classify them as therapeutic goods, of which 14 did not comply with regulations. NSWPIC reported these 14 products to the TGA and prompted relevant regulatory bodies and industry representatives. Only 3 of 124 products had ARTG number on the packaging. 18 products had packaging similar to drink/beverage containers or cosmetics. Community members reported concerns of inappropriate packaging for another 15 products. There was a 2.2-times increase in calls to NSWPIC regarding hand-sanitisers from January to July 2020 (1095 cases) when compared to the same period in 2019 (504 cases). Most patients were children under 5 years old and had minor illness. No death was observed in our patients.

Discussion: A significant number of hand sanitiser products in this study were misclassified by the manufactures and had inappropriate containers and labelling. Safety measures must be critically taken in timely manner to achieve safe hand sanitiser use. Poisons Information Centre has played an important role enacting prompt data collections and public health interventions leading to modification of the regulations and recommendations.

Enhancing surveillance for fentanyl and analogues misrepresented as other drugs in NSW

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Background: Surveillance for harms from fentanyl and analogues is a priority for NSW Health’s Centre for Alcohol and Other Drugs. In October 2020 the...
Prescription, Recreational and Illicit Substance Evaluation (PRISE) Program detected three cases of unintended fentanyl and acetylfentanyl exposures, prompting enhanced surveillance activities and a public health response.

Methods: Following the sentinel cases in October 2020 the PRISE program expanded the threshold for testing suspected fentanyl exposures, accepting cases that may not otherwise have been sufficiently severe or unusual enough to warrant testing under the program. Enhanced surveillance of emergency department (ED) data was undertaken using expanded search terms and increased search frequency, and data from NSW Ambulance acquired. The case finding harnessed existing collaborations with NSW Health Pathology Forensic & Analytical Science Service, NSW Poisons Information Centre, clinical toxicology services, the Ministry’s Public Health Rapid ED Surveillance System, doctors and nurses, local hospital laboratories, the Sydney Uniting Medically Supervised Injecting Centre, NSW Ambulance, and NSW Police.

Results: From October 2020 to February 2021, there were 15 confirmed cases of fentanyl and analogue exposures in people intending to use another drug. There were two different exposure scenarios: heroin for injection, often described as purple; and white powder insufflation, thought to be either cocaine, methamphetamine or ketamine. There were an additional 16 notifications, 8 of which were excluded following testing, and another 8 likely cases though samples were not available for testing. Supported by an expert panel, a clinical alert and multiple public drug alerts were disseminated. This was paired with local health service engagement and checks of naloxone supply.

Discussion: Fentanyl and analogues are circulating in NSW and given their potency, increase the risk of overdose, particularly among opioid naïve people who take it unexpectedly. Continued surveillance is vital in directing efforts in the form of harm reduction messaging and clinical awareness; and to highlight priority locations and populations for programs such as Take Home Naloxone. The case finding highlighted the need to utilise multiple information sources to understand a cluster including people who may refuse transport to hospital or discharge from the ED against medical advice.

Cardiac glycoside poisoning – 3 cases of accidental ingestion of presumed foxglove

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Introduction: Plant ingestion is an uncommon cause of severe poisoning in Australia. We describe three cases of accidental probable foxglove ingestion with cardiac toxicity.

Case summaries: An 80 year old male made tea from a ‘sage’ plant. He developed severe vomiting and bradycardia. On his electrocardiogram he had second degree heart block and widespread ST segment depression. His serum potassium was 5.8 mmol/L and serum digoxin >10 mcg/L. After 2 vials of anti-digoxin-Fab he reverted to first degree heart block but ST segment depression persisted. He did not tolerate activated charcoal. 24 h later he developed 4 s sinus pauses and received another vial of anti-digoxin-Fab, reverting to first degree heart block with ST segment depression. Another vial of anti-digoxin-Fab was given 48 h later for recurrence of sinus pauses. Cardiotoxicity resolved after 10 days. Digoxin concentrations measured at three laboratories by different assays confirmed non-digoxin cardenolide exposure demonstrated by the poor relationship between results from variable cross reactivity.

Two 56 year old female international tourists consumed leaves from a plant they thought was edible. Neither had a cardiac history. Hours later they developed severe vomiting. The first patient felt pre-syncopal with pulse 40–50 beats/minute, second degree heart block, global ST depression, serum potassium 5.1 mmol/L and serum digoxin 3.9 mcg/L. The second patient had first degree heart block with global ST segment depression, serum potassium 4.2 mmol/L and serum digoxin 2.89 mcg/L. Both received 1 vial anti-digoxin-Fab. The first patient reverted to first degree heart block, but there was minimal improvement for the second. Both received 2 doses activated charcoal on day 2. The first patient received another 2 vials over the course of the admission for recurrent vomiting and worsening bradycardia. Both discharged against advice prior to resolution of toxicity on day 3. In each case the
plants were identified by non-botanists as being Foxglove.

Brief summary of discussion: These cases show severe and persistent cardiotoxicity following accidental Foxglove exposures which responded partially to anti-digoxin-Fab. Blood testing using different assays identified the exposure to be non-digoxin in origin in one case.

Of mushrooms not so magic – suspected magic mushroom capsule contamination with bacterial endotoxin resulting in systemic inflammatory response and intensive care admission

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Introduction: Novel oral ingested synthetic or natural drugs have the potential to cause significant harm through either contamination or via off-target effects due to unknown ingredients. Clinicians should be aware of the potential for critical illness. We present two cases of severe shock/systemic inflammatory response syndrome (SIRS) requiring vasopressor support following the ingestion of one capsule containing powdered magic mushroom.

Case Summary: A 32-year-old female (patient 1) and 40-year-old male (patient 2) presented to hospital after ingestion of presumed magic mushroom within a home manufactured capsule. The intent was recreational use using ‘micro-dosing’ for a euphoric but not psychedelic experience. Neither patient had used the capsules before.

Patient 1 presented with severe nausea and muscle cramping within 30 min of ingestion, requiring morphine. During the following hours she developed shock with tachycardia, hypotension, lactataemia, fever and delirium. She was treated with noradrenaline (norepinephrine) and antibiotics with resolution within 2 days.

Patient 2 developed symptoms of severe vomiting and diarrhoea within 4 h of ingestion, but did not present to hospital for 36 h. On presentation he was also shocked, with tachycardia, hypotension and an acute kidney injury. He was treated with noradrenaline (norepinephrine) and antibiotics for 24 h.

Both patients had raised inflammatory markers, with procalcitonin concentrations of >45 microgram/L (N < 0.05 microgram/L) and CRPs >200 mg/L (N < 5).

All cultures were negative. Forensic testing of each patient’s serum and urine revealed no evidence of any significant pharmacological or synthetic drug adulterants.

Discussion: Neither patient developed a typical mushroom toxidrome. Although we cannot exclude culture-negative septic shock, the cases, including the rapid recovery, are remarkably similar to those of endotoxemia poisoning from contaminated intravenous drug use and compounded pharmacy products (1,2).

Endotoxin, i.e. the lipopolysaccharide (LPS) outer membrane structure of gram-negative bacteria, induces an acute inflammatory response, similar to early septic shock (3). Although the oral route makes LPS poisoning less likely, as LPS does not usually cross the gut membrane, it is possible that the other ingredients within the capsule might have increased permeability and hence systemic toxicity. Both patients had severe gastrointestinal symptoms/signs at the onset of their illness.

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Pharmacovigilance for counterfeit alprazolam in NSW

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Background: Alprazolam is a highly desired benzodiazepine for non-prescribed use and was restricted in Australia to Schedule 8 in 2014. Decreased use
and harms were initially reported, however international reports of counterfeit alprazolam and shifts in availability of alprazolam require vigilance. We sought to describe the extent and characteristics of counterfeit alprazolam in Poisons Information Centre calls, patients in the NSW public health system and police seizures.

Methods: Cases of counterfeit alprazolam resulted in public drug warnings and clinician alerts in December 2019 and July 2020 by NSW Health. The Therapeutic Goods Administration issued a Safety Advisory in June 2020.

During July-December 2020, NSW-based callers to the Poisons Information Centre and NSW Health clinicians were asked to provide additional information about alprazolam exposures, including product appearance.

Data sources extracted included: NSW Poisons Information Centre database (2015–20), NSW Ministry of Health suspected counterfeit alprazolam notifications register (2020), NSW Health Pathology Forensic & Analytical Science Service Illicit Drug Analysis Unit (2017–20).

Results: Unique PIC exposure calls involving alprazolam increased over 2015–20: 274, 311, 272, 311, 340, 506. The average monthly count approximately doubled from May 2020.

During the period of enhanced surveillance, there were 91 reports received and 46 confirmed as counterfeit (13 from product analysis and remainder by product appearance).

Analysis of tablets identified (in order of frequency): etizolam, flualprazolam, clonazolam, alprazolam, flubromazolam.

Increases in counterfeit alprazolam detections from police seizures were noted from 2019.

Discussion: We observed increasing poisonings involving alprazolam in 2020 which coincides with increasing detections of counterfeit alprazolam in NSW from multiple data sources. Clinicians can incorporate routine questions about product appearance into discussions involving non-prescribed use for enhanced pharmacovigilance. A detailed review of the impacts that COVID-19 and alprazolam restrictions is needed to manage non-prescribed benzodiazepine use.

Case report of caustic ingestion with delayed presentation

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Introduction: Both accidental and intentional ingestion of household cleaning products can potentially cause significant caustic injury. The injury can involve airway, gastrointestinal and respiratory systems making patient management challenging and even more so if it is a delayed presentation.

Case Summary: A 53-year-old male was brought in by ambulance 28 h after an intentional ingestion of 1 litre of Domestos bleach, 10 g of paracetamol and 100 mg of temazepam. On arrival to the Emergency department, he was hypoxic, tachypnoeic and drooling. Nasendoscopy revealed chemical burns to his hypopharynx without vocal cord oedema. He was subsequently intubated for respiratory failure due to chemical pneumonitis. He was managed with high dose steroid for caustic ingestion and Acetylcysteine infusion for paracetamol overdose. Endoscopy on day 3 after ingestion revealed inflammation of the proximal oesophagus, gastro-oesophageal junction and stomach, and confirmed significant sloughing and burns to the hypopharynx. He was extubated on day 4 post ingestion but had a persistent oxygen requirement on high flow nasal oxygen and ongoing thickened secretions. His swallow was impaired due to burns to the hypopharynx and required nasogastric feeding. He will require long term respiratory and gastroenterology follow up for his caustic injuries.

Discussion: The management of this caustic injury was challenging in a number of ways. This included indications and timing of airway intervention, severity of chemical pneumonitis, and risk of gastrointestinal perforation due to delay in presentation.

Ambulance service enquiries to a poison information centre: a retrospective series

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Background: There is a paucity of information describing the nature of ambulance service enquiries to a Poison Information Centre (PIC). This study was
undertaken to characterise how ambulance providers use the PIC service and to better understand the interaction, in order to improve resource utilisation and patient care.

Methods: This was a retrospective review of Queensland Poison Information Centre (QPIC) data via the Queensland Health CHIRPs Dashboard and the Pharmhos database from January 2020 to December 2020. The data included patient demographics, mode of exposure, substances involved, and advice provided.

Results: There were 1522 calls from the Queensland Ambulance Service over the study period, including 191 recalls. This represents approximately 5% of Queensland exposure calls received by QPIC. Females were involved in 679 (51%) cases. Patients aged 0–4 years comprised 369 (28%) of cases with 727 (55%) regarding exposures in adults over 20 years. There were 550 (41%) intentional cases and 752 (57%) unintentional exposures. There were 1748 substances identified in the calls. Quetiapine 57 (3.2%), alcohol 46 (2.6%), paracetamol 45 (2.5%) and ibuprofen 44 (2.5%) were the four most common agents involved in exposures. The main information 1091/1522 (71%) provided by QPIC included giving exposure specific advice while paramedics were en-route to the patient and risk-stratifying on scene, to determine if transport is recommended.

Conclusion: Poison Information Centres are a valuable resource for the first responders of the ambulance service. Further research and understanding of how best to support this frontline service would result in improved patient service and experience as well as potentially significant cost savings to the health sector.

Unusual clinical manifestation of mushroom poisoning: a case report

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Introduction: Fever is rarely described in mushroom poisoning in Australia. This case report describes fever and hypotension as predominant features following potential Chlorophyllum poisoning.

Case: 2 patients of the same family, 22 M and 48 M presented with vomiting and diarrhoea 5h after ingestion of mushrooms obtained from their backyard in regional NSW. The mushrooms were white topped with grey gills and white stalks, consumed raw or lightly grilled. The 22 M had a bite of the cap whereas the 48 M consumed 5 whole mushrooms. On presentation, both were mildly hypotensive with SBP 90-100. Blood tests obtained 7h after consumption demonstrated a neutrophilia, hyperlactataemia up to 3.5, mild renal impairment and normal liver functions in both patients. Approximately 10h after consumption, both patients developed a febrile illness with temperature >38 degrees, diaphoresis and worsening hypotension despite 4–5 L of crystalloids each. The 48 year old required an ICU admission for vasopressors. The degree of hypotension was inconsistent with degree of gastrointestinal fluid loss. Other causes such as sepsis were excluded. Both patients recovered with good supportive care, and discharged 24–48h later. Mycologists identified the mushroom as likely Chlorophyllum molybdites based on images. The mushroom were unavailable for analysis.

Discussion: New toxic mushroom species continue to be identified, with approximately 800 new species being identified worldwide yearly (1). Chlorophyllum molybdites is a gastrointestinal irritant that causes nausea, vomiting, diarrhea, and abdominal pain (2). The concentration of toxin is highest in the cap (3). It usually causes a benign gastrointestinal illness, but hypovolemic shock is described in case reports (2,3). Fever is not a typical feature. Difference in toxicity have been observed, due to variations in the mushroom age, climate, substrate where the mushroom was growing, and genetic differences between populations (3). Case reports describing fevers with mushroom poisonings have been reported in other countries in the context of hyperprocalcitoninemia and pancytopenic mushroom poisoning (4–6). This case illustrates the importance of species identification in clinical management. We often rely from data from overseas, but Australian data is necessary to confirm that such extrapolations are applicable.

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A retrospective case series on the toxicity of orphenadrine

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Objective: Case reports in published literature describe severe toxicity from acute orphenadrine poisonings, whereas anecdotally, in Australia, a less severe spectrum of toxicity is observed. This case series aims to describe the effects of orphenadrine in overdose, including the frequency and severity of anticholinergic, neurological and cardiovascular toxicity.

Methods: All cases of orphenadrine poisoning that presented to a healthcare facility over 7 years were identified from the NSW PIC database using a key word search ‘orphenadrine’, from 1st January 2014 to 1st January 2021. Cases that presented to a healthcare facility were shortlisted. Preliminary data was collected from the PIC database. Patient demographics, dose ingested, and clinical features on presentation were analysed.

Results: 46 cases were identified. The median age was 32 years, with a range from 3 to 72 years. 54% were females. Based on the known doses in 39 patients, the median dose ingested was 770 mg, with a mean of 1647 mg. 4 patients potentially ingested up to 10 g. The most common symptom was drowsiness, with 6 patients having a GCS <8. 3 of these had ingested up to 10 g. All except 1 patient with a GCS <8 had coingested other sedatives. Other common symptoms were tachycardia, followed by agitation and hallucinations. Only 2 patients were intubated for persistently low GCS. No patient had significant cardiotoxicity. 2 patients had fluid responsive hypotension. No arrhythmias or ECG abnormalities were recorded on presentation. 26% of patients were asymptomatic. Of the 12 patients without coingestants, 8 were asymptomatic with the highest dose in this cohort of 850 mg.

Discussion: Orphenadrine in Australia is available as orphenadrine citrate in the form of Norflex (uncoated tablet containing 100 mg orphenadrine citrate, extended release) or Norgesic (orphenadrine citrate 35 mg and paracetamol 450 mg, immediate release). The potentially lethal dose is 2–3 g in adults, 600–800 mg in children, with death occurring in 3–5 h due to respiratory insufficiency or cardiotoxic effects. Multiple case reports describe severe toxicity with ventricular arrhythmias, sustained seizures and anticholinergic delirium with severe agitation. In this case series, it was observed that the spectrum of toxicity was mild – moderate, with a low risk of cardiotoxicity, despite potentially large doses ingested.

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Paediatric clonidine overdose: a retrospective analysis at a tertiary regional hospital

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Background: The frequency of clonidine prescribing and exposures in children has increased in recent years. We aimed to describe the effects and treatment of paediatric clonidine exposures.

Methods: This was a six year (2014–2020) retrospective review of paediatric clonidine overdoses. Demographic information, clinical effects, treatment, complications, length of stay (LOS) and disposition were extracted from a prospective database or medical records. Abnormal heart rate (HR) and blood pressure (BP) were defined according to the NSW Health paediatric observation charts: blue, yellow and red zones defined by age and clinical emergency response systems, in which yellow zone activates a clinical review and red zone activates a rapid response.
Results: There were 111 cases and 60 (23, 0–6 y, 1, 7–11 y, 36, 12–17 y) admitted to hospital and 23 (8, 0–6 y and 15, 12–17 y) admitted to intensive care unit (ICU). Median LOS for 0–6 y inpatients was 21 h (interquartile range [IQR]:13–27 h) and for 12–17 y inpatients 33 h (IQR: 20–91 h). 101 patients had at least one abnormal observation [76 (68%) HR, 77 (69%) BP , 50 (45%) low GCS]. Median GCS was 14 (3–15), with four having a lowest GCS <9. 31 (28%) patients had a HR in the yellow zone and 13 (12%) in the red zone. 31 patients had a BP in the yellow zone and 23 (21%) in the red zone. There were 41 aged 0–6 y, 18 female and all accidental with a median dose of 9 mcg/kg (4–125 mcg/kg). 23 were admitted to hospital and 8 to ICU. Intravenous fluids were administered to 15. Three were treated with naloxone with no obvious improvement. There were nine patients aged 7–11 y; 4 females and all accidental. One patient was admitted to the ward. There were 61 aged 12–17 y, all deliberate self-poisonings. Thirty-six were admitted to hospital, and 15 to ICU. Twelve received intravenous fluids, 2 received atropine and one was intubated to facilitate retrieval.

Discussion: Paediatric clonidine overdoses commonly present with bradycardia and hypotension, but only one patient required intubation, and the remainder only observation and intravenous fluids. Presentations in the 7–11 age group are of concern highlighting the continual need for education about safe administration and storage.

Disclosure of Interest Statement: Nil to disclose.

Survival following ecmo-cpr in a massive nortriptyline overdose

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Introduction: Extracorporeal Membrane Oxygenation (ECMO) is an emerging treatment in the management of critically poisoned patients. There are few cases of survival following the use of ECMO during prolonged cardiac arrest.

Case report: We report a 26-year-old (80 kg) woman that ingested 2500 mg of nortriptyline in a suicide attempt. On ambulance arrival (60 min after ingestion) she was initially rousable, but rapidly deteriorated into cardiac arrest. Mechanical cardiopulmonary resuscitation was immediately commenced with a Corpuls device. In the field she received 4 mg adrenaline, 500 mmol bicarbonate and 6 shocks without return of spontaneous circulation. She arrived at a tertiary emergency department (ED) 60 min following her arrest. The ECMO team had been mobilised by ambulance services and were present in the ED on patient arrival.

Her arrival ABG showed a pH of 7.45, with a pCO2 of 61, pO2 of 86, a lactate of 9.3 and a potassium of 3.3. She received a further 150 mmol bicarbonate, 100 mg lidocaine, 20 mmol potassium chloride and an adrenaline infusion at 50 mcg/hr.

She was cannulated and commenced on ECMO 30 min post arrival (total downtime 90 min). The patient rapidly stabilised and was transferred to the intensive care unit. She was decannulated day 2 and extubated day 5. Following extubation it was noted she had left upper limb weakness and MRI confirmed a bilateral basal ganglia hypoxic ischaemic injury. She was discharged home after a one-month admission and had an excellent neurological recovery.

Nortriptyline levels peaked at 906 mcg/L 16.5 h following ingestion.

Discussion: Resuscitation for cardiac arrest due to cardiotoxic drugs should be prolonged, partnered with aggressive supportive cares including ECMO where available. These patients should be primarily taken to centres able to provide ECMO where possible, and if within a reasonable time frame. ECMO-CPR has been used in MI and should be considered in appropriate toxicological cardiac arrests.

Overdose and off-label prescribing in patients with borderline personality disorder: a retrospective series

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Background: Borderline personality disorder (BPD) is common with a suicide rate 50 times higher than the general population. Despite limited evidence of effectiveness in BPD, psychotropic medications are often prescribed. We aimed to investigate deliberate self-poisonings in patients with BPD in an effort to identify avenues of potential risk-mitigation in this group.

Methods: This was a retrospective observational series of patients with BPD presenting to the Princess
Alexandra Hospital following an overdose. The unit’s database was searched for all patients presenting with deliberate self-poisoning over a two-year period (January 2019 to December 2020). Those with a documented history of BPD were included in the study. Medical records were reviewed to determine baseline characteristics, details of overdose, clinical features, treatment and disposition.

Results: There were 608 presentations in 366 people (76.9% female). The median age was 28 years (range 16–75 years). There were 242 representations in 101 people (median representations 1, range 1–20).

The majority (331[90.4%]) of patients were prescribed at least one psychotropic medication, with 118(35.6%) being prescribed three or more different psychotropic agents. Of the total prescribed psychotropics, 513/1450(35.4%) were for off-label indications. The majority of agents (859/1486[57.8%]) taken in overdose were prescribed. Of these, 573/859(66.7%) were psychotropics, of which 211/573(36.8%) were for off-label indications. Severe toxicity was present in 99 cases with either coma (GCS <9) or hypotension (systolic BP <90 mmHg). There were 23 (3.8%) intubations resulting in ICU admissions. The commonest agents in severe toxicity were quetiapine (44[44%]), diazepam (30[30%]) and pregabalin (14[14%]). The median length of stay was 15.1 h (IQR 8.1–23.1 h). There were 87 (14.3%) mental health admissions.

Discussion: Psychotropic polypharmacy is common in patients with BPD and many of these are prescribed for off-label indications. Prescription medications are commonly taken in overdose. Quetiapine is over-represented both in off-label prescriptions and associated harm from overdose in this group.

Accidental 5-hydroxytryptophan (5-htp) overdose causing hippocampal ischaemia

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Introduction: 5-hydroxytryptophan (5-HTP), the precursor to serotonin, is a non-prescription nutritional supplement available for the treatment of depression. It readily crosses the blood brain barrier. We report the first case of hippocampal injury as a result of accidental 5-HTP overdose.

Case summary: A previously well 44-year-old male was brought to emergency department (ED) with a 3 h history of confusion and word finding difficulty. He reported an inadvertent intake of 800 mg of 5-HTP supplement, instead of the intended 80 mg dose. His history was only otherwise notable for methamphetamine use 3 days prior. He was alert but disorientated (GCS-14) to time and place and clinically dehydrated. His neurological examination was otherwise unremarkable, with no signs of serotonin toxicity. Initial investigations were consistent with a respiratory acidosis and urine drug screen was positive for methamphetamines. He had no abnormalities on his chest x-ray or ECG. His short term memory continued to be significantly impaired over the following days. MRI brain revealed extensive symmetrical restricted diffusion bilaterally in the hippocampi (body and head regions), suggestive of ischaemia. A repeat MRI 6 days later showed persistent T2/FLAIR signal in the same regions, with diffusion restriction consistent with the temporal evolution of an ischaemic insult. Nine days after admission, formal neuropsychology testing demonstrated a reduction in verbal and non-verbal memory domains, in keeping with the pathological changes seen on neuroimaging. Throughout the course of his admission cognitive function, including short and long-term memory, slowly improved. The patient was discharged after 9 days in hospital, and had adequately recovered to return to work in 30 days.

Brief summary of discussion: 5-HTP has been shown to increase catecholamine (dopamine, norepinephrine, and epinephrine) turnover in animal studies (1), but can also result in catecholamine depletion (2). The latter has been reported to result in cerebral ischaemia (3), with particular risk of hippocampal injury as an area of high metabolic activity. Increased susceptibility to neuronal death in the hippocampus following cerebral ischaemia has also been reported with other centrally acting adrenergic drugs, such as methamphetamines (4,5) and cocaine (6,7). We propose that catecholamine depletion, due to excess 5-HTP, resulted in hippocampal neurotoxicity.

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Prescription, recreational and illicit substance evaluation (prise) program: the 2-year journey

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Background: The Prescription, Recreational and Illicit Substance Evaluation (PRISE) Program is a collaborative network between NSW Ministry of Health, NSW Poisons Information Centre (PIC) and associated toxicology units, and NSW Health Pathology Forensic & Analytical Science Service. This state-wide program facilitates specialised toxicological testing of clinical samples from people presenting to NSW Health facilities with suspected drug related toxicity that is severe and atypical.

Methods: This study aims to review the PRISE Program’s operations and outputs from July 2018 to September 2020.

Results: There were 241 cases notified, of which 208 cases (86.3%) proceeded to testing. Most cases were young (median age: 24.8; IQR: 20–28.5 years) and one third (78 cases, 32.4%) attended music festivals (median age: 21.2; IQR: 19.6–22.9 years). The largest proportion of notifications was from toxicology units (95 cases, 39%) and 34 cases (14%) were informed by NSW PIC. There were an increased number of cases tested in October 2019 to March 2020 (16 cases/month) when compared with the overall period (9 cases/month) due to the music festival season and formal establishment of the PRISE team in September 2019. Tested cases declined during initial COVID-19 pandemic restrictions (4 cases/month in April to June 2020). A triage scale for testing urgency was initiated during this period. Toxicology results demonstrated common and emerging substances in NSW which included but were not limited to the first clinical detection of carfentanil in NSW, illicit fentanyl and acetylfentanyl, 2,4-dinitrophenol and pentobarbitone. The PRISE toxicology testing from 8 distinct events led to the release of 15 alerts. The PRISE Program’s outputs were pivotal in developing a management strategy for NSW music festivals, surveillance for counterfeit alprazolam, prompt notifications and collaborations with other regulatory bodies.

Discussion: The PRISE Program has successfully detected a significant number of recreational and illicit substances in patients presenting to emergency departments with severe drug toxicity in NSW, and has informed clinical management and catalysed public health interventions. Despite the COVID-19 pandemic challenges, the PRISE Program has continued its compelling operations with ongoing support from all relevant organisations. The next step is to incorporate its streamlined process into existing workflows.

Hand sanitiser exposure in New South Wales: from data to interventions

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Background: The use of hand sanitiser has been enthusiastically promoted following the COVID-19 pandemic. However, adverse health effects from ingestion, including fatalities, have been reported in many countries.

Methods: We aim to report data on calls made about hand sanitiser to NSW Poisons Information Centre (PIC) from 1 January to 31 July 2019 and the same period in 2020 including relevant interventions. We examined electronic records of ‘exposure’ pertaining to either ‘hand sanitiser’, ‘ethanol(non-beverage)’,
‘isopropanol’ or ‘methylated spirits’ coded under ‘description’ field.

Results: Compared to 1 January –31 July 2019, the number of calls from 1 January –31 July 2020 concerning alcohol-based hand sanitiser increased by 117% (from 504 to 1095). The rise began in February 2020 with an increase of 92.7%. The peak difference was in June 2020 when there were 2.8 times as many calls as in 2019. Most cases in both years were in 1 to 4 years old (2020: 641 cases, 58.5%; 2019: 299 cases, 59.2%). There were 111 older children (5 to 14 years) reported in 2020 (53 cases, 47.7% occurred in school), which was an increase of 316% when compared with 24 cases with similar age in 2019 (5 cases, 20.8% occurred in school). Two patients (4 and 6 years old) required ICU admissions due to significant alcohol intoxication (blood ethanol levels 200 mg/dL and 189 mg/dL respectively) but both were discharged uneventfully. Both patients reported liking the smell and the taste of hand sanitisers. Fourteen out of 124 products exposed were clearly non-compliant with labelling and packaging legislation (based on 309 photos submitted to PIC by the callers). NSW PIC notified the NSW Ministry of Health and other relevant regulatory agencies about the increases, exposures at school, and noncompliant products when they were detected, and further actions were subsequently taken by responsible agencies. We also trained NSW PIC Staff and developed a protocol for standardised recommendations, additional data collection, and instituted follow-up calls to determine outcomes.

Discussion: NSW PIC provided timely management advice, performed data monitoring and led the public health interventions concerning increased hand sanitiser exposures that followed the COVID-19 pandemic in NSW. This report highlights the role of PICs to alleviate individual harm and to prevent negative impacts to community secondary to toxic exposures.

Ibuprofen overdose causes minor effects but dose-related hyperlactataemia

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Objective: Ibuprofen is a commonly ingested drug for deliberate self-poisoning due to its ready availability. Like most non-steroidal anti-inflammatory drugs it does not appear to be associated with major toxicity, but there are reports of metabolic acidosis with massive doses. We aimed to investigate the range of toxicity of ibuprofen overdoses.

Methods: All presentations of ibuprofen overdose to two tertiary toxicology services from 2008 to 2018 were identified from prospective databases. Only patients ingesting >800mg were included. The following data was extracted: demographics, co-ingestants, complications (coma [GCS <9], acute kidney injury, seizure), treatments and outcomes (length of stay [LOS], intensive care [ICU] admission, death). In additional, medical records were retrieved for the additional information: gastrointestinal symptoms (nausea, vomiting or abdominal pain) and blood gas results.

Results: There were 1267 ibuprofen overdoses, 901 females (71%), with a median age of 25 y (interquartile range [IQR]: 20–35). The commonest co-ingestants were paracetamol, alcohol, codeine, benzodiazepines, selective serotonin reuptake inhibitors and atypical antipsychotics (mainly quetiapine). Median ingested dose was 3200 mg (IQR: 2000–6000 mg). The median LOS was 13 h (IQR 5.3–20 h) and 26 patients (2.1%) were admitted to ICU. There were 179 ibuprofen alone overdoses with 134 females (78%); median age 20 y (IQR: 18 to 25 y). The median ingested dose was 4400 mg (IQR: 2400–7200 mg; range: 800–36,000 mg), which was significantly different to those co-ingesting other medications (median 2800 mg, p < 0.0001). Gastrointestinal symptoms were commonest, with 42 patients (23%) having nausea, 16 (9%) vomiting and 41 (23%) having abdominal pain. No patients had seizures, acute kidney injury or died in the ibuprofen alone group. Blood gases were available for 66 patients with a median pH of 7.37 (7.35–7.40) and a median lactate of 1.5 (1.0–1.9). There was a significant correlation between dose and lactate (r^2 = 0.31; p < 0.0001), but not between dose and pH in this range of doses.

Conclusions: Ibuprofen overdose causes minor gastrointestinal effects in the majority of cases. Increasing doses are associated with increased hyperlactataemia, which may explain cases of severe metabolic acidosis with greatly increased lactate concentrations in rare massive ingestions.

Tranexamic acid overdose – should we be concerned?

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Objective: Tranexamic acid is a synthetic lysine analog, developed in 1962, which is widely used to reduce bleeding via its antifibrinolytic effects. Recently, there has been an increase in tranexamic acid prescriptions, with 73761 Pharmaceutical Benefits Scheme prescriptions in 2015 compared to 23253 in 2003. Despite this long history of use, little is known about the effects of tranexamic acid in overdose. We aimed to investigate the frequency and outcomes of tranexamic acid overdose.

Methods: All presentations of tranexamic acid overdose to two tertiary toxicology services, Hunter Area Toxicology Service (HATS) and Princess Alexandra Toxicology Service (PATS), from 2014 to 2021 were identified from prospective databases. Cases reported to NSW and QLD Poisons Information Centres (PIC) during this period were also included. Medical records or call records from identified cases were retrieved and reviewed.

Results: During this period, there were 70 calls to NSW PIC and 12 calls to Queensland PIC for tranexamic deliberate self-poisoning. Calls increased from 5 per year to 12 per year in NSW. A total of 16 cases were identified, seven from tertiary toxicology units and nine cases referred to a clinical toxicologist via the Poison Information Centres. All overdoses were in females, median age 20 y (12 to 46 y), with a median dose of 25 g (5 to 195 g).

Common co-ingestants included paracetamol, selective serotonin reuptake inhibitors, non-steroidal anti-inflammatory drugs, benzodiazepines and antihistamines. No effects were reported in three patients, mild sedation (GCS13-14) in four and gastrointestinal symptoms in eight patients. Tranexamic acid was likely to be the cause of effects in 2/4 patients with sedation and 3 of 8 patients with gastrointestinal effects. Complications from co-ingestants included toxic paracetamol levels requiring acetylcysteine, anti-cholinergic toxicity, serotonin toxicity and QT prolongation. One patient was treated with prophylactic enoxaparin, although there were no thrombotic complications. No patients experienced seizure. No patients were admitted to ICU or died.

Conclusion: Tranexamic acid overdoses are uncommon, but increasing, and do not appear to cause significant complications. Large overdoses can cause mild sedation and gastrointestinal symptoms.

Reverse takotsubo cardiomyopathy precipitated by chronic cocaine and cannabis use

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Introduction: Reverse Takotsubo cardiomyopathy (rTTC) is a variant of TTC characterised by basal hypokinesis and apical hyperkinesis with an acute coronary syndrome-like presentation in the absence of obstructive coronary artery disease. Drug-induced causes are described.

Case summary: A 31 year old man with ten years of cocaine and cannabis dependence presented with three hours of severe left temporal headache and vomiting which began whilst smoking cannabis, several hours after smoking methamphetamine and using intranasal cocaine. Examination revealed pulse 91 beats/minute (bpm), blood pressure 163/107 mmHg, respiratory rate 24 breaths/min, temperature 36.5° Celsius, oxygen saturations 100% and Glasgow Coma Scale 15. There were no features of heart failure. He was agitated, diaphoretic, pupils 4 mm bilaterally and reactive, with lower limb hyperreflexia and sustained ankle clonus. Computed tomography of the brain and angiography excluded intracranial haemorrhage, ischaemia and dissection. His headache resolved with opioid analgesia. Two hours post presentation he became tachycardic (160 bpm) with tonic extension of all four limbs and deviation of eyes superiorly then left, resolving with 5 mg IV midazolam. Venous blood gas showed a mixed respiratory and metabolic acidosis with lactate 16.0 mmol/L, consistent with a drug induced seizure. He subsequently developed cardiogenic shock (BP 82/49 mmHg), with antero-inferior ST segment depression on electrocardiogram and troponin-T rise to 126 ng/L. Coronary angiogram demonstrated normal coronary arteries. Transthoracic echocardiogram demonstrated severe impairment of left ventricular (LV) systolic function with ejection fraction 15–20% and hypokinesis sparing the apex. Dobutamine and levosimendan infusions were initiated. Thyrotoxicosis, nutritional, vasculitic and viral screens were negative. Cardiac MRI demonstrated mildly dilated LV, severe impairment of function with dilated and hypocontractile basal segments, hyperintense on T2-weighted imaging consistent with myocardial oedema and rTTC. Candesartan and bisoprolol
were commenced and he was discharged to drug and alcohol rehabilitation.

Brief summary of discussion: Proposed triggers of rTTC include coronary artery spasm, catecholamine cardiotoxicity, intracranial haemorrhage, general anaesthesia, serotonin syndrome and sympathomimetic use. Management is largely supportive. In this case, multiple insults led to catecholamine cardiotoxicity resulting in rTTC. Severe headache, cannabis hyperemesis and cocaine and methamphetamine induced serotonin toxicity culminated in a drug-induced seizure resulting in this unusual presentation.

Intentional drug overdose and illicit drug use during admission to hospital: a potentially preventable cause of morbidity

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Background: Intentional drug overdoses and illicit drug use during hospital admission are potentially preventable causes of morbidity. To our knowledge there is no published data on the frequency of this problem or clinical outcomes in these patients.

Objectives: Our aim was to describe all cases of intentional overdose and illicit drug use by inpatients over the past two years at a major teaching hospital.

Methods: Royal Prince Alfred Hospital is a quaternary referral centre with 80,000 annual admissions. A retrospective audit was undertaken of all inpatients identified as taking an overdose of legal drugs or using illicit drugs during their admission between February 2019 and 2021. Cases were identified from the toxicology database, mortality and morbidity meeting and clinician recall. Data was collected from electronic medical records. Descriptive analysis was undertaken.

Results: 27 cases were identified. Median age was 42 years (IQR 30–50), 56% were female, 78% had a substance use disorder, 93% had one or more psychiatric co-morbidities, 59% were admitted under drug and alcohol or toxicology service and most were on a medical ward (48%) or in ED (30%) at the time of drug use. The majority (74%) had presented with drug overdose, intoxication or suicidal ideation. Around half (48%) of the 42 substances used were illicit (mainly heroin and methamphetamine), 36% were prescribed (mainly anti-hypertensives and antidepressants), 12% over-the-counter (antihistamines and paracetamol) and 5% alcohol. 44% of patients consumed medications that they brought to hospital and kept in their possession and 56% acquired them during admission (from visitors, hospital pharmacy, purchased on the “street”). Management involved activated charcoal (19%), naloxone (11%), N-acetylcysteine (7%), intubation (15%), cardiac monitoring (41%) and intensive care (26%). Improperly disposed sharps were found in 19%. There were no deaths.

Discussion: Inpatient drug use occurs infrequently but presents a significant risk of harm to patients and staff and increases inpatient service utilisation. Enquiry about medications in their possession as well as mandating bag search for all patients in ED presenting with suicidal ideation, intoxication or drug overdose may be appropriate. Encouraging incident reporting will help to capture the full spectrum of the problem.

Beware stimulants, strong men and superhuman strength causing permanent neurological disability

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Introduction: We present two cases of stimulant use (Cocaine and MDMA) in of young males resulting in lactataemia, rhabdomyolysis, compartment syndrome or muscular injury and neurological injury.

Case summary:

Case 1: A male in his thirties presented with agitation, fever, tachycardia, and metabolic acidosis in the context of cocaine and ecstasy use. He required substantial physical and chemical restraint on a warm day. He was treated with sedation, intubation, paralysis, and cooling. Despite this, his CK continued to rise. He developed bilateral erythema, with tense compartments in both anterior shins and poor perfusion distally. Bilateral fasciotomies were performed demonstrating non-viable anterior compartments. While he recovered from his acute drug intoxication, he has been left with significant disability and morbidity due to this with bilateral foot drop.
Case 2: A male in his twenties was brought in by ambulance in an agitated and combative state after cocaine use requiring substantial prehospital restraint and sedation. He was critically unwell with fever, tachycardia, GCS 11, hypotension, tachypnoea, with a life-threatening metabolic acidosis, acute kidney injury and hyperkalaemia. He was treated with cooling, sedation, and paralysis. His CK peaked at 194,200. He was noted to have globally firm compartments around both shoulder girdles with swelling without evidence of compartment syndrome. The patient described significant bilateral shoulder girdle pain, weakness and altered sensation. His stay was complicated by coronary vasospasm and bilateral cerebellar haemorrhages.

Discussion: Compartment syndrome and rhabdomyolysis are described complications of drug exposure, particularly stimulant agents or in cases of “long lie”. One case series described a cluster of cases of synthetic cathinone agents resulting in compartment syndrome. Physicians need to be mindful of assessing compartments in intubated and ventilated patients. We believe both patients’ large muscle bulk contributed to limited compartment space, and consequent vulnerability to rhabdomyolysis, and their behaviour prior to sedation and containment resulted in large muscle activation and damage.

Conclusion: A rise in CK, evidence of firm compartments, metabolic acidosis with a known exposure to stimulants and on scene agitation and distress should prompt the clinician to serially assess all compartments to minimise the likelihood of long-term neurological sequelae.

Two cases of chlorpyrifos ingestion: a single centre experience

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Introduction: Chlorpyrifos is an organophosphorus agent (OP) which is rapidly absorbed from the small intestine with high lipid solubility and a long half-life resulting in extended toxicity. We present two cases of chlorpyrifos ingestion with prolonged cholinergic symptoms and intermediate syndrome.

Case 1: A male in his 70s presented after ingesting 1.5 litres of chlorpyrifos “Fortune 500” Cockroach Bait. He had evidence of hypertension, tachycardia, reduced consciousness and periods of apnoea. Other than diarrhoea there were no other cholinergic features. He was treated with early atropinisation and intubated due to nicotinic features. An atropine infusion was required for 15 days as attempts at weaning resulted in hypotension and respiratory secretions. He developed neuromuscular weakness consistent with intermediate syndrome. His ICU stay was complicated by multiorgan failure secondary to aspiration and respiratory sepsis. The patient died on day 17 of his admission.

Case 2: A female in her 50s presented after ingesting 300mls of Chlorpyrifos Cockroach Bait. She had evidence of diarrhoea, vomiting, tachycardia and hypertension. She had no other cholinergic features but developed progressive drowsiness and respiratory failure. She was treated with both atropine and pralidoxime. Her ICU stay was complicated by paroxysmal atrial fibrillation, evidence of multiple cerebral infarcts on MRI, aspiration, deranged liver function, autonomic dysfunction, intermediate syndrome requiring tracheostomy and prolonged cholinergic syndromes. She had a protracted hospital stay, with significant neurocognitive sequelae.

Discussion: Both patients demonstrate the significant morbidity and mortality associated with chlorpyrifos ingestions. Both of their courses were complicated by sepsis, haemodynamic lability and the need for protracted atropinisation likely due to the fat solubility and redistribution of chlorpyrifos over time. Both patients demonstrated a mixture of nicotinic and muscarinic effects (hypertension and tachycardia). One patient was treated with pralidoxime. The evidence for the use of pralidoxime remains unclear in this cohort.

Conclusion: Despite legislation regulating chlorpyrifos availability, the agent is still circulating in the community and causes significant morbidity and mortality. Clinicians should be aware of the likely need for ongoing protracted atropinisation. Pralidoxime should be discussed with toxicology services due to its toxic effects and limited evidence base.

Hand sanitiser ingestions have increased in young children during the COVID-19 pandemic

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Background: Since the beginning of the COVID-19 global pandemic, the Australian Government has promoted the use of hand sanitisers, soaps and disinfectants to reduce transmission of the SARS-CoV-2 virus. We hypothesised that there was an increase in unintentional oral exposures to topical antiseptics (including hand sanitisers) during the COVID-19 Pandemic, particularly in young children.

Methods: This retrospective observational study utilised data from a search of the Victorian Poisons Information Centre (VPIC) database, for the period between February 1, 2019 and January 31, 2021. We looked at exposures by ingestion only. Calls from interstate, recalls of a single exposure or ‘query’ calls where no exposure has occurred, were excluded. Substances of interest included topical antiseptics (including hand sanitisers), bleach and other cleaners, disinfectants and high percentage ethanol products. We analysed exposure data in young children (<5 years old).

Results: There was an increase in the number of calls relating to topical antiseptic ingestion in children under five. 812 calls (5.4% of all exposures in this age group) were received between February and December 2020 (inclusive) compared to 347 (2.3%) in 2019. There was a 3.1-fold increase in antiseptic ingestion calls during the first Victorian COVID-19 wave beginning in March 2020, compared with the same period in 2019. During the peak of the second COVID-19 wave in September, VPIC received 76 calls relating to topical antiseptics, a 2.5-fold increase. However, the data does not suggest a significant increase in harm, with only 17 cases (2.1% of ingestions) where a child had minor symptoms at the time of the call in 2020, compared to 19 (5.5%) in 2019. No children had moderate or severe symptoms. These findings were also reflected in older children and adults, with 268 reported ingestions in 2020 (1.3% of calls in this age range) compared to 100 (0.5%) in 2019. Trends regarding paediatric ingestion with other disinfectants, bleaches and cleaners were less clear.

Discussion: Hand sanitiser ingestions by young children increased during the COVID-19 pandemic and were more prevalent during periods of increased COVID-19 cases. However, there was no evidence of increased harm.

A retrospective, descriptive case series of the clinical and haemodynamic consequences of overdose with amlodipine and angiotensin receptor blocking (ARB) or angiotensin converting enzyme inhibitor (ACE-I) drug combination medication

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Objective: To detail our experience on the clinical course of overdose with amlodipine in combination with an angiotensin receptor blocker (ARB) or angiotensin converting enzyme inhibitor (ACE-I).

Background: Amlodipine and an ACE-I or ARB are commonly co-prescribed in the management of hypertension, often in a fixed-dose combination tablet. Case reports describe significant haemodynamic compromise when this combination is taken in overdose – some cases have resulted in significant vasodilatory shock refractory to vasopressors. To date, there are no case series describing this poisoning.

Methods: We searched all referrals to the Monash Health Toxicology Service from August 2013 to September 2019 to identify overdoses involving amlodipine with an ARB/ACE-I. We excluded overdoses which involved other cardio-active or sedative medications or presented greater than 8-hours post-ingestion. We analysed their haemodynamic, biochemical and therapeutic sequelae.

Results: Nine cases fulfilling these criteria were identified: Median age 20 years (IQR: 17–53 yrs. Range: 14–79 yrs). Five patients ingested fixed-dose combination tablets. The median defined daily dose (DDD) equivalent ingested was 12 (IQR). The median drop in systolic blood pressure from presentation to lowest initial recorded BP was 26 mmHg (IQR 6.5–49.8). The median time to lowest initial BP was four hours (IQR 3.1–6.1). All patients demonstrated hypotension within nine hours of their overdose, and 90% within 6h. One patient required no medical intervention; five patients required intravenous fluid therapy; one patient was given a single bolus of metaraminol; and two patients required more than one vasopressor over the course of multi-day Intensive Care Unit admissions. These two patients were aged 20 and 79 years, and ingested 28 and 8 DDDs, respectively. In both cases, vasopressor therapy was commenced within 4h of ingestion. There were no deaths.

Conclusion: In our case series, hypotension was apparent within 9h of ingestion and most overdoses of amlodipine and a renin-angiotensin system blocker responded to intravenous fluid therapy alone. However,
persistent and refractory hypotension requiring intensive care and vasopressor therapy may develop. This may be more likely in the elderly or after large-dose ingestions.

**A case of methoxyflurane use disorder**

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Introduction: Methoxyflurane is an inhaled anaesthetic commonly used as an analgesic for the short term relief of pain associated with trauma. Reports of methoxyflurane use disorder are limited. However toxicity has been described.

Case summary: A 28 year old female patient with a history of severe and frequent cluster headaches presented to a general practitioner (GP) for a repeat prescription of methoxyflurane (Penthrax inhaler). Her headaches were resistant to other medications including triptans, verapamil, lithium, protypsin and she had unsuccessfully trialled a nerve block. Two years previously a neurologist had prescribed a trial of methoxyflurane up to three times per week for 6–12 months. However use was recorded in hospital records prior to this.

The GP noted disparity between the patient’s reported frequency of use and prescribing records. Dispensing records were reviewed and indicated she had been seeing multiple prescribers. Further investigations were performed. Renal and hepatic function were normal but unexplained vision disturbances were noted. Use ceased when pregnancy was confirmed at 6 weeks. However use was recorded in hospital records prior to this.

The frequency of headaches decreased and is currently managed with paracetamol and oxycodone.

Discussion: Methoxyflurane is a fluorinated hydrocarbon anaesthetic. It is indicated in Australia for emergency relief of pain associated with trauma. It is a Schedule 4 agent and is available on the Pharmaceutical Benefits Scheme under Prescriber’s bag supply.

A 3 mL dose of methoxyflurane provides analgesia for approximately 20–30 min with continuous use and for up to 1 h with intermittent use. Recommended daily maximum is 6 mL, and weekly maximum is 15 mL. Administration on consecutive days is not recommended.

Evidence supporting the use of methoxyflurane for headache is lacking. Evidence of harm associated with repeated use of methoxyflurane for both anaesthetic and analgesic purposes, at recommended and supratherapeutic dosing, is available. Reported toxicity includes hepatitis, diffuse, bilateral renal cortical calcification, calcium oxalate retinopathy and subacute fluorosis. Adverse effects during early pregnancy are not reported.

Despite extensive use, the patient exhibited no apparent signs of toxicity. However, vision disturbance may be due to calcium oxalate deposition in the retina.

**Lurasidone mono-ingestion overdoses: a case series with minimal toxicity**

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Objective: Lurasidone is a second-generation antipsychotic agent used to treat schizophrenia. It has few reported side-effects, less weight gain than other similar agents and little effect on the electrocardiogram (ECG). Currently, data are lacking regarding toxicity after lurasidone mono-ingestion overdoses. We describe clinical and electrocardiographic features in a series of lurasidone mono-ingestion overdoses.

Method: Retrospective case series of self-reported ingestion of lurasidone identified from referrals made to the Monash Health toxicology service between 2016 and 2019.

Results: Six patients with mono-ingestion overdose of lurasidone were identified. Sixty-six percent (n = 4) were female, median age: 31 years (range: 16–44 years), median reported ingested dose 820 mg (range: 300–2000 mg). Median lowest GCS 14 occurred at 2.6 h (GCS range 13 to 15; time range 2–9.9 h) post-ingestion. Hypotension was observed in two patients (median SBP 103 mmHg, responsive to fluid). Transient tachycardia was observed in one patient (median 103 bpm, range: 85–112). Median heart rate for all patients was 78 bpm (range: 50–112). No patients displayed anti-cholinergic signs. There were no reports of mydriasis, urinary retention or delirium.

A total of 13 ECGs were recorded from 1.2 to 15 h post-ingestion. Median number of ECGs per patient was two. Median QTc (Fridericia) was 425 msec (range: 360–475 msec). When measured QT-heart rate pairs were plotted on the QT-nomogram, only one just crossed the nomogram line. Pre-overdose ECGs were available for three patients. QT-intervals were similar to post-overdose ECGs. There was no evidence of any other conduction or rhythm disturbances.
patients were admitted to the toxicology observation unit. Median hospital length of stay was 13.2 h (range: 6.2–17.1 h). There were no complications or deaths in this cohort.

Conclusion: In this small cohort, isolated lurasidone overdose resulted in mild sedation and negligible ECG interval effects.

We did not observe any significant anticholinergic signs seen after overdose with other atypical antipsychotics, such as quetiapine and olanzapine. All patients exhibited signs of sedation by nine hours post-ingestion. Based on this data, asymptomatic patients at 10 h may be suitable for discharge. Larger case series would assist in further determining features of toxicity after lurasidone mono-overdoses.

An unexpected case of fatal QAC ingestion

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Introduction: Benzalkonium Chloride (BZK) is a quaternary ammonia cationic (QAC) detergent used for disinfection. Accepted evidence suggests severe injury is rare unless the concentration is greater than 7.5%. In this case an 89-year-old inadvertently ingested 200 mL of BZK 5% leading to a fatal outcome.

Case summary: An 89-year-old male living in a nursing home, was found leaning over his bathroom sink coughing and salivating, unusually unable to say what had occurred. A bottle of a cleaning product containing BZK had inadvertently been left in his room. It is found half empty, spray nozzle and lid missing. At 1439, a nurse contacts the Poisons Information Centre (PIC) stating 10–15 mL of dilute product had been ingested, describing the patient as asymptomatic. Between 1540 and 1645 he deteriorates, persistently coughing, dysphagic, with impaired breathing and haemoptysis prompting ambulance retrieval. At 1800, the Emergency Department calls PIC reporting he has haematemesis, is unable to lie flat and ingested 200 mL of BZK. He is subsequently intubated. Endoscopy finds excessive bleeding and friability of necrotic appearing mucosa. At 0420, he develops ventricular tachycardia followed by cardiac asystole.

Brief Summary of discussion: This is a case of an unexpected death due to a BZK ingestion. BZK disrupts lipid bilayers, the effects of ingestion include mucosal and tissue irritation, and gastritis, however, more serious adverse reactions including corrosive injury, circulatory failure and death can occur. The risk of toxicity was thought to be related to concentration as exposure to products more than 7.5% are known to cause corrosive injury. This case demonstrates concentration of product and total dose ingested must be considered together to make an appropriate risk assessment. Thorough history taking is vital to ascertain specific patient risk factors including current medications, previous medical history, dilution factors, volume ingested, and symptoms exhibited which, when considered as a whole, contribute to seeking early medical attention and may prevent ongoing harm. In humans, an oral dose of BZK 100–400 mg/kg is thought to be fatal. This patient ingested approximately 139 mg/kg, lack of symptom recognition and assumptions around size of ingestion, dilution of the product all contributed to delayed access to definitive care and a fatal outcome.

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Fatal Chironex Fleckeri in the far North Queensland – who is the expert

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Introduction: Chironex Fleckeri has a 25–30 cm diameter bell and 10–12 tentacles of more than 2 m in length. It produces potent, rapid-acting venom affected by geographical location causing severe localized and systemic effects that are life-threatening in humans (1–3). The CSL™ produce box jellyfish antivenom (4-BJAV). In envenomed patient’s death may occur within 10–20 min of contact with tentacles (5–6).
Case summary: A 17 M was stung whilst fishing in chest deep water near Bamaga, Queensland, in February 2021. He collapsed on shore, tentacles still attached. He was driven 20 min without CPR to Bamaga Hospital (7). CPR commenced on arrival (16:40, 30 min post collapse). ROSC occurred after 25 min of ALS and 1 vial 20,000 units BJAV given at 16:50. Following discussion with a PIC Toxicologist, another vial of BJAV and 20 mmol magnesium were administered. VBG showed severe metabolic and respiratory acidosis. He was resuscitated, intubated and transported to Townsville ICU. CT brain on arrival demonstrated diffuse hypoxic brain injury he was pronounced brain dead on Day 7.

Discussion: This is a tragic case of severe envenomation by Chironex Fleckeri. The benefit of BJAV and magnesium with cardiac arrest isn't validated but is recommended (5). There have been 6 reported deaths since 2000, the last being a 6 M who was stung in 2007 in NT (8). These stings often occur in under resourced settings with minimal staff and limited BJAV reserves. As seen here, staff in these centres may have more experience in managing these presentations than the toxicologists whose expert advice is sought. Treatment consists of prolonged resuscitation and administration of BJAV. Evidence on the best management of these stings is limited, with expert advice based on case reports and lab cell and animal studies. Uncertainties surrounding the appropriate dose of BJAV are multi-factorial and the recommended dose controversial. One study suggested that BJAV is dose and time dependent, with a higher dose required than that recommended and prolonged supportive care in terms of CPR to achieve cell survival in the presence of venom (3).

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