

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

2015 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 33rd Annual Report
Mowry JB, Spyker DA, Brooks DE, Zimmerman A, Schauben JL. Clin Toxicol 2016; 54: 924-1109.

Introduction

This is the 33rd Annual Report of the American Association of Poison Control Centers' (AAPCC) National Poison Data System (NPDS). As of 1 January 2015, 55 of the nation's poison centers (PCs) uploaded case data automatically to NPDS. The upload interval was 9.52 [7.40, 13.6] (median [25%, 75%]) minutes, creating a near real-time national exposure and information database and surveillance system.

Methods

We analyzed the case data tabulating specific indices from NPDS. The methodology was similar to that of previous years. Where changes were introduced, the differences are identified. Poison center cases with medical outcomes of death were evaluated by a team of medical and clinical toxicologist reviewers using an ordinal scale of 1-6 to assess the Relative Contribution to Fatality (RCF) of the exposure.

Results

In 2015, 2,792,130 closed encounters were logged by NPDS: 2,168,371 human exposures, 55,516 animal exposures, 560,467 information calls, 7657 human confirmed nonexposures,

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and 119 animal confirmed nonexposures. US PCs also made 2,695,699 follow-up calls in 2015. Total encounters showed a 3.42% decline from 2014, while health care facility (HCF) human exposure cases increased by 5.09% from 2014. All information calls decreased by 15.5% but HCF information calls increased 2.67%, and while medication identification requests (Drug ID) decreased 31.7%, human exposures reported to US PCs were essentially flat, increasing by 0.149%. Human exposures with less serious outcomes have decreased 2.95% per year since 2008 while those with more serious outcomes (moderate, major or death) have increased by 4.34% per year since 2000. The top 5 substance classes most frequently involved in all human exposures were analgesics (11.1%), household cleaning substances (7.54%), cosmetics/personal care products (7.41%), sedatives/hypnotics/ antipsychotics (5.83%), and antidepressants (4.58%). Sedative/Hypnotics/Antipsychotics exposures as a class increased the most rapidly (2597 calls (11.4%)/year) over the last 14 years for cases showing more serious outcomes. The top 5 most common exposures in children age 5 years or less were cosmetics/personal care products (13.6%), household cleaning substances (11.2%), analgesics (9.12%), foreign bodies/toys/miscellaneous (6.45%), and topical preparations (5.33%). Drug identification requests comprised 35.0% of all information calls. NPDS documented 1831 human exposures resulting in death with 1371 human fatalities judged related (RCF of 1-Undoubtedly responsible, 2-Probably responsible, or 3-Contributory).

Conclusions

These data support the continued value of PC expertise and need for specialized medical toxicology information to manage more serious exposures, despite a decrease in calls involving less serious exposures. Unintentional and intentional exposures continue to be a significant cause of morbidity and mortality in the US. The near real-time, always current status of NPDS represents a national public health resource to collect and monitor US exposure cases and information calls. The continuing mission of NPDS is to provide a nationwide infrastructure for surveillance for all types of exposures (e.g., foreign body, viral, bacterial, venomous, chemical agent, or commercial product), the identification of events of public health significance, resilience, response and situational awareness tracking. NPDS is a model system for the real-time surveillance of national and global public health.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1245421>

What can clinicians learn from therapeutic studies about the treatment of acute oral methotrexate poisoning?

Chan BS, Dawson AH, Buckley NA. Clin Toxicol 2017; 55: 88-96.

Context

Methotrexate (MTX) is an anti-folate drug that has been utilized in both malignant and chronic inflammatory conditions. Doctors are often concerned with a potential adverse outcome when managing patients with acute oral MTX poisoning given its potential for serious adverse reactions at therapeutic doses. However, there is surprisingly little data from acute poisoning cases and more data from the therapeutic use of high-dose MTX.

Objectives

To review pharmacokinetic and pharmacological properties of MTX and systematically review series of acute MTX poisonings and therapeutic studies on high-dose MTX that provide pharmacokinetic or clinical data.

Methods

An Embase (1974–October 2016) and Medline (1946–October 2016) search was performed by combining "MTX" and "overdose/poison" or "MTX" and "toxicity" or "MTX" and "high-dose MTX" or "MTX" and "bioavailability" or "pharmacokinetics"; 25, 135, 109 and 365 articles were found, respectively, after duplicates were removed. There were 15 papers that

provided clinical data on acute ingestion and toxicity that occurred with low-dose administration. Eighteen papers were on high-dose MTX (>1 g per m^2 body surface area) used as a single chemotherapy agent which provided pharmacokinetic or clinical data on MTX toxicity. Thirty papers were reviewed to determine the toxic dose, pharmacokinetics, risk factors, clinical symptoms and management of acute MTX toxicity. Given the limited acute poisoning data, a retrospective audit was performed through the consultant records of the New South Wales Poisons Information Centre from April 2004 to July 2015 to examine the clinical syndrome and toxicity of acute oral MTX poisoning.

Pharmacokinetics

Reduced MTX bioavailability is a result of saturable absorption. Although maximal bioavailable absorption occurs at a dose of ~ 15 mg m^{-2} , splitting the dose increases bioavailability. MTX clearance is proportional to renal function.

Acute toxicity

Oncologists prescribe doses up to 12 g m^{-2} of MTX. Patients treated with an intravenous dose of MTX <1 g m^{-2} do not require folinic acid rescue. MTX toxicity correlates better with duration and extent of exposure than peak serum concentration.

Acute oral poisoning

Acute oral MTX poisoning in 177 patients did not report any severe toxicity. In the New South Wales Poisons Information Centre audit data (2004-2015), 51 cases of acute MTX poisoning were reported, of which 15 were accidental paediatric ingestions. The median reported paediatric ingestion was 50 mg (IQR: 10–100; range: 10–150) with a median age of 2 years (IQR: 2–2; range: 1–4). Of the 36 patients with acute deliberate MTX poisoning, median age and dose were 47 years (IQR: 31–62; range: 10–85) and 325 mg (IQR: 85–500; range: 40–1000), respectively. Of the 19 patients who had serum MTX concentrations measured, all were significantly below the concentrations used in oncology and the folinic acid rescue nomogram line and no patient reported adverse sequelae.

Management of acute oral poisoning

Due to the low bioavailability of MTX, treatment is not necessary for single ingestions. Oral folinic acid may be used to lower the bioavailability further with large ingestions >1 g m^{-2} . Oral followed by intravenous folinic acid may be used in patients with staggered ingestion >36 h or patients with acute overdose and renal impairment ($\text{eGFR} <45$ mL/min/ 1.73 m^2).

Conclusions

As a consequence of saturable absorption MTXs bioavailability is so low that neither accidental paediatric MTX ingestion nor acute deliberate MTX overdose causes toxicity. An acute oral overdose will not provide a bioavailable dose even close to 1 g m^{-2} of parenteral MTX. Hence, no treatment is required in acute ingestion unless the patient has renal failure or staggered ingestion. There is also no need to monitor MTX concentrations in acute oral MTX poisoning.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271126>

The toxicology of zinc chloride smoke producing bombs and screens

El Idrissi A, van Berkel L, Bonekamp NE, Dalemans DJZ, van der Heyden MAG. Clin Toxicol 2017; online early:

doi: 10.1080/15563650.2016.1271125:

Context

Zinc chloride (ZnCl_2)-based smoke bombs and screens are in use since the Second World War (1939–1945). Many case descriptions on ZnCl_2 smoke inhalation incidents appeared since 1945.

Objective

We provide a comprehensive overview of the clinical symptoms and underlying pathophysiology due to exposure to fumes from ZnCl₂ smoke producing bombs. In addition, we give a historical overview of treatment regimens and their outcomes.

Methodology

We performed a literature search on Medline, Scopus and Google Scholar databases using combinations of the following search terms "smoke bomb", "smoke screen", "ZnCl₂", "intoxication", "poisoning", "case report", "HE smoke", "hexachloroethane smoke", "smoke inhalation" and "white smoke". We retrieved additional reports based on the primary hits. We collected 30 case reports from the last seven decades encompassing 376 patients, 23 of whom died. Of all the patient descriptions, 31 were of sufficient detail for prudent analysis.

Results and conclusions

Intoxication with clinical signs mainly took place in war situations and in military and fire emergency training sessions in enclosed spaces. Symptoms follow a biphasic course mainly characterised by dyspnoea, coughing and lacrimation, related to irritation of the airways in the first six hours, followed by reappearance of early signs complemented with inflammation related signs and tachycardia from 24 h onwards. Acute respiratory stress syndrome developed in severely affected individuals. Chest radiographs did not always correspond with clinical symptoms. Common therapy comprises corticosteroids, antibiotics and supplemental oxygen or positive pressure ventilation in 64% of the cases. Of the 31 patients included, eight died, three had permanent lung damage and 15 showed complete recovery, whereas in five patients outcome was not reported. Early signs likely relate to caustic reactions in the airway lining, whereas inhaled ZnCl₂ particles may trigger an inflammatory response and associated delayed fibrotic lung damage. Smoke bomb poisoning is a potentially lethal condition that can occur in large cohorts of victims simultaneously.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271125>

Acute salicylate poisoning: risk factors for severe outcome

Shively RM, Hoffman RS, Manini AF. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2016.1271127:

Context

Salicylate poisoning remains a significant public health threat with more than 20,000 exposures reported annually in the United States.

Objective

We aimed to establish early predictors of severe in-hospital outcomes in Emergency Department patients presenting with acute salicylate poisoning.

Methods

This was a secondary data analysis of adult salicylate overdoses from a prospective cohort study of acute drug overdoses at two urban university teaching hospitals from 2009 to 2013. Patients were included based on confirmed salicylate ingestion and enrolled consecutively. Demographics, clinical parameters, treatment and disposition were collected from the medical record. Severe outcome was defined as a composite occurrence of acidemia (pH <7.3 or bicarbonate <16 mEq/L), hemodialysis, and/or death.

Results

Out of 1997 overdoses screened, 48 patients met inclusion/exclusion criteria. Patient characteristics were 43.8% male, median age 32 (range 18–87), mean initial salicylate concentration 28.1 mg/dL (SD 26.6), and 20.8% classified as severe outcome. Univariate analysis indicated that age, respiratory rate, lactate, coma, and the presence of co-ingestions

were significantly associated with severe outcome, while initial salicylate concentration alone had no association. However, when adjusted for salicylate concentration, only age (OR 1.13; 95% CI 1.02–1.26) and respiratory rate (OR 1.29; 95% CI 1.02–1.63) were independent predictors. Additionally, lactate showed excellent test characteristics to predict severe outcome, with an optimal cutpoint of 2.25 mmol/L (78% sensitivity, 67% specificity).

Conclusions

In adult Emergency Department patients with acute salicylate poisoning, independent predictors of severe outcome were older age and increased respiratory rate, as well as initial serum lactate, while initial salicylate concentration alone was not predictive.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271127>

***In-vivo* evidence of nephrotoxicity and altered hepatic function in rats following administration of diglycolic acid, a metabolite of diethylene glycol**

Robinson CN, Latimer B, Abreo F, Broussard K, McMartin KE. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2016.1271128:

Context

Diglycolic acid (DGA) is one of the two primary metabolites of diethylene glycol (DEG). DEG is an industrial solvent that has been implicated in mass poisonings resulting from product misuse in the United States and worldwide, with the hallmark toxicity being acute kidney injury, hepatotoxicity, encephalopathy and peripheral neuropathy. Our laboratory has generated in-vitro evidence suggesting that DGA is the metabolite responsible for the proximal tubule necrosis and decreased kidney function observed following DEG ingestion. Furthermore, we have shown that DGA specifically accumulates in kidney tissues (100× higher than peak blood concentrations) following DEG administration.

Objective

To examine renal and hepatic accumulation and dysfunction following direct administration of DGA in-vivo. We hypothesize that administration of DGA will result in renal and hepatic DGA accumulation, as well as proximal tubular necrosis and liver injury.

Materials and methods

Adult male Wistar rats were divided into three groups dosed with 0, 100 or 300 mg/kg DGA via single oral gavage. Urine was collected every 6–12 h and blood, kidneys and liver were removed upon sacrifice at 48 h post-dosing for analysis.

Results

DGA accumulated significantly in both kidney and liver tissue only at 300 mg DGA/kg. DGA concentrations in the kidneys and liver correlated with renal and hepatic injury, respectively. Histopathological and clinical chemistry analysis revealed that DGA-treated animals exhibited moderate liver fatty accumulation and marked renal injury, again only at 300 mg/kg.

Discussion

DGA-induced kidney injury demonstrated a steep dose response threshold, where severe damage occurred only in animals given 300 mg/kg DGA, while no toxicity was observed at 100 mg/kg.

Conclusion

These results provide evidence for in-vivo toxicity following direct administration of DGA, a metabolite of DEG. The steep dose–response threshold for toxicity suggests mechanistically that there is likely a saturable step that results in DGA accumulation in target organs.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271128>

Toxicity from automotive screenwashes reported to the United Kingdom National Poisons Information Service (NPIS) from 2012 to 2015

Day R, Eddleston M, Thomas SHL, Thompson JP, Bradberry SM, Vale JA. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2016.1271130:

Background

Automotive screenwashes commonly contain ethylene glycol, methanol, and/or isopropanol; ethanol is also included in many formulations. The concentrations and combinations of each constituent vary considerably between the products. This study was undertaken to investigate the toxicity of automotive screenwashes as reported by telephone to the United Kingdom National Poisons Information Service (NPIS).

Methods

Enquiries to the NPIS relating to automotive screenwashes were analyzed retrospectively for the period January 2012 to December 2015.

Results

There were 295 enquiries involving 255 individual exposures. The majority ($n = 241$, 94.5%) of exposures involved ingestion and 14 of these also involved other routes. Six cases were due to skin contact alone, three to inhalation alone, three to eye contact alone, one to ear exposure alone and another occurred from inhalation and skin contact. Children below 5 years of age accounted for 26% of all ingestions. The identity (and therefore composition) of the screenwash was known with certainty in 124 of 241 ingestions and included methanol in 106 formulations, isopropanol in 72, ethylene glycol in 38, and ethanol in 104. The World Health Organisation/International Programme on Chemical Safety/European Commission/European Association of Poison Centres and Clinical Toxicologists Poisoning Severity Score was known in 235 of 241 cases of ingestion; most patients were asymptomatic ($n = 169$, 71.9%), but 59 (25.1%) developed minor (PSS 1), six (2.6%) moderate (PSS 2), and one patient severe (PSS 3) features; this patient later died. Nausea ($n = 10$), vomiting ($n = 11$), abdominal pain ($n = 10$), metabolic acidosis ($n = 8$) and raised anion gap ($n = 8$) were reported most commonly after ingestion.

Conclusions

Most patients (71.9%) ingesting automotive screenwash did not develop features. The implication is that the amount of screenwash ingested was very small. Skin and eye exposure produced either no features or only minor toxicity.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271130>

New evidence for oxetorone toxicity

Deguigne M, Bruneau C, Touré A, Turcant A, Le Roux G. Clin Toxicol 2017; 55: 142-6.

Context

Oxetorone is a serotonin antagonist antimigraine drug but literature relating to its toxic properties is poor. The aim of this study is to describe the toxicological profile of oxetorone and to highlight any relationship between clinical and analytical findings.

Materials and methods

This is a retrospective and observational study of cases exposure to oxetorone, reported to the Angers Poison and Toxicovigilance Centre between January 2002 and May 2016. Severity was assessed using the Poisoning Severity Score (PSS). Cases where data were incomplete, where oxetorone was deemed not accountable, where clinical signs were linked

mainly to a co-ingested drug or where the plasma concentration of oxetorone was negative were all excluded.

Results

We included 43 cases of exposure, 31 of whom were suicide attempts. The assumed ingested dose (60–3600 mg) was correlated to severity ($r_s = 0.45$, $p = 0.01$). Symptoms of moderate severity (PSS2 = drowsiness, hypertonia, myosis, convulsions, arterial hypotension, QRS widening, QTc prolongation) were observed following ingestion of more than 600 mg of oxetorone (median dose = 1200 mg) and severe symptoms (PSS 3 = coma, convulsions, QTc prolongation, QRS widening, ventricular tachycardia, arterial hypotension, cardiogenic shock) were observed starting from 1800 mg (median dose = 2700 mg). In four cases, a secondary worsening of symptoms 10–48 h following ingestion was observed. Plasma oxetorone was measured in four patients. Severe symptoms were observed in the event of a concentration over 0.3 mg/L and the highest measured serum oxetorone level was delayed by 20–48 h following the ingestion for two cases.

Conclusions

Several clinical and paraclinical parameters strongly point towards membrane-stabilising properties of the molecule and the risk of a delayed occurrence of symptoms or a secondary worsening.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1267358>

The impact of an international initiative on exposures to liquid laundry detergent capsules reported to the United Kingdom National Poisons Information Service between 2008 and 2015

Day R, Eddleston M, Thomas SHL, Thompson JP, Vale JA. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2016.1267359:

Introduction

Although the majority of those exposed to liquid laundry detergent capsules remain asymptomatic or suffer only minor clinical features after exposure, a small proportion develop central nervous system depression, stridor, pulmonary aspiration and/or airway burns following ingestion or conjunctivitis and corneal ulceration following eye exposure. As a consequence, the International Association for Soaps, Detergents and Maintenance Products (AISE) established a Product Stewardship Programme in Europe, requiring that safety measures be implemented to reduce the visibility of, and restrict access to, these detergent capsules by small children. Implementation occurred in the United Kingdom over several months during the first half of 2013.

Objective

This study investigated whether the AISE Programme had an impact on the number and severity of exposures reported to the United Kingdom National Poisons Information Service.

Methods

Telephone enquiries to the National Poisons Information Service relating to liquid laundry detergent capsules were analysed for the period January 2008 to December 2015.

Results

While there was a significant difference ($p = 0.0002$) between the mean number of annual exposures (469.4) reported between 2008 and 2012 and the mean number reported between 2014 and 2015 (403.5), the number of exposures was decreasing steadily prior to implementation of the Programme in 2013, which did not impact this fall from 2013 onwards. In addition, the number of exposures per million units sold was not impacted by

the Programme. There was no significant difference ($p = 0.68$) between the mean number of exposures (11.8) with PSS ≥ 2 reported between 2008 and 2012 and the mean number (13.0) reported between 2014 and 2015. Although there was a 28.7% decrease between 2010–2012 and 2014–2015 in the number of exposures with PSS ≥ 2 per million units sold, this decrease was not statistically significant ($p = 0.18$).

Conclusion

There is no evidence that the Product Stewardship Programme had a beneficial impact on the number of exposures reported to the National Poisons Information Service or their severity.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1267359>

Lead intoxication due to ayurvedic medications as a cause of abdominal pain in adults

Mehta V, Midha V, Mahajan R, Narang V, Wander P, Sood R, Sood A. Clin Toxicol 2017; 55: 97-101.

Background

Though a majority of cases of lead intoxication come from occupational exposures, traditional and folk remedies have also been reported to contain toxic amounts of lead. We present a large series of patients with lead poisoning due to intake of Ayurvedic medicines, all of whom presented with unexplained abdominal pain.

Methodology

This was a retrospective, observational case series from a tertiary care center in India. The charts of patients who underwent blood lead level (BLL) testing as a part of workup for unexplained abdominal pain between 2005 and 2013 were reviewed. The patients with lead intoxication (BLLs >25 $\mu\text{g/dl}$) were identified and demographics, history, possible risk factors, clinical presentation and investigations were reviewed. Treatment details, duration, time to symptomatic recovery, laboratory follow-up and adverse events during therapy were recorded.

Results

BLLs were tested in 786 patients with unexplained abdominal pain and high levels were identified in 75 (9.5%) patients, of which a majority (73 patients, 9.3%) had history of Ayurvedic medication intake and only two had occupational exposure. Five randomly chosen Ayurvedic medications were analyzed and lead levels were impermissibly high (14–34,950 ppm) in all of them. Besides pain in abdomen, other presenting complaints were constipation, hypertension, neurological symptoms and acute kidney injury. Anemia and abnormal liver biochemical tests were observed in all the 73 patients. Discontinuing the Ayurvedic medicines and chelation with d-penicillamine led to improvement in symptoms and reduction in BLLs in all patients within 3–4 months.

Conclusion

The patients presenting with severe recurrent abdominal pain, anemia and history of use of Ayurvedic medicines should be evaluated for lead toxicity. Early diagnosis in such cases can prevent unnecessary investigations and interventions, and permits early commencement of the treatment.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1259474>

11 analytically confirmed cases of mexedrone use among polydrug users

Roberts L, Ford L, Patel N, Vale JA, Bradberry SM. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2016.1271424:

Introduction

Mexedrone, 3-methoxy-2-(methylamino)-1-(4-methylphenyl)propan-1-one, is the alpha-methoxy-derivative of mephedrone (4-methyl-N-methyl cathinone). Mexedrone inhibits the re-uptake of serotonin and dopamine in a dose-dependent manner and has affinity for serotonin and dopamine membrane transporters and receptors (5-HT₂ and D₂ receptors), producing sympathomimetic effects similar to amfetamines. To date there are no published clinical reports on mexedrone use that are analytically confirmed.

Objective

To characterise the features of mexedrone use in patients who presented to our hospital after using a variety of psychoactive substances including mexedrone, with analytical confirmation in each case.

Methods

This is an observational case series. Urine toxicological screening using ultra-performance liquid chromatography with tandem mass spectrometry and exact mass time of flight was employed in all patients.

Results

A total of 305 cases were screened and mexedrone was identified in 11 urine samples. Agitation was the most common presenting feature in 10 of 11 patients. This was marked to the extent of aggression in some cases, with six patients requiring sedation and/or physical restraint. Delusions and hallucinations, often with paranoia, were observed in three cases with a prominent supernatural/demonic theme. None of these individuals had a history of psychosis. Seven of 11 patients were tachycardic >100 bpm. The median length of stay was 20 hours (range 2-77; IQR 4-33). Mexedrone *alone* is only likely to have been responsible for these clinical features in 2 cases; in two others mexedrone was found in high concentration along with substantial amounts of other stimulants. In 7 other cases other stimulants detected more likely explained the features. However, comprehensive analytical data enabled us to identify the full complement of agents contributing to the clinical presentation.

Conclusions

Agitation was the predominant clinical feature in this case series and was often accompanied by a sinus tachycardia; mexedrone was primarily responsible in 2 patients but contributed substantially in two others. Patients typically recovered fully within 24 hours, unless they required sedation.

Full text available from: <http://dx.doi.org/10.1080/15563650.2016.1271424>

Outcomes from massive paracetamol overdose: a retrospective observational study

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Abstract and full text available from: <http://dx.doi.org/10.1016/j.ajem.2016.12.002>

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Pharmacokinetic properties and human use characteristics of an FDA-approved intranasal naloxone product for the treatment of opioid overdose

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Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute carbon monoxide poisoning

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TOXICOLOGY

General

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MANAGEMENT

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