Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Risk prediction of hepatotoxicity in paracetamol poisoning

Wong A, Graudins A. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1317349:

Context

Paracetamol (acetaminophen) poisoning is the most common cause of acute liver failure in the developed world. A paracetamol treatment nomogram has been used for over four decades to help determine whether patients will develop hepatotoxicity without acetylcysteine treatment, and thus indicates those needing treatment. Despite this, a small proportion of patients still develop hepatotoxicity. More accurate risk predictors would be useful to increase the early detection of patients with the potential to develop hepatotoxicity despite acetylcysteine treatment. Similarly, there would be benefit in early identification of those with a low likelihood of developing hepatotoxicity, as this group may be safely treated with an abbreviated acetylcysteine regimen.

Aim

To review the current literature related to risk prediction tools that can be used to identify patients at increased risk of hepatotoxicity.

Methods

A systematic literature review was conducted using the search terms: "paracetamol" OR "acetaminophen" AND "overdose" OR "toxicity" OR "risk prediction rules" OR "hepatotoxicity"

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OR "psi parameter" OR "multiplication product" OR "half-life" OR "prothrombin time" OR "AST/ALT (aspartate transaminase/alanine transaminase)2 OR "dose" OR "biomarkers" OR "nomogram". The search was limited to human studies without language restrictions, of Medline (1946 to May 2016), PubMed and EMBASE. Original articles pertaining to the theme were identified from January 1974 to May 2016. Of the 13,975 articles identified, 60 were relevant to the review.

Paracetamol treatment nomograms

Paracetamol treatment nomograms have been used for decades to help decide the need for acetylcysteine, but rarely used to determine the risk of hepatotoxicity with treatment.

Reported paracetamol dose and concentration

A dose ingestion >12 g or serum paracetamol concentration above the treatment thresholds on the paracetamol nomogram are associated with a greater risk of hepatotoxicity.

Paracetamol elimination half-life

Patients with more severe hepatotoxicity are more likely to have a longer paracetamol elimination half-life. While median elimination half-life increases in those developing hepatotoxicity, there is wide variation in half-life, making this an insensitive parameter to use as a negative risk prediction tool.

Prothrombin time (PT)

An initially normal PT is associated with a lower risk of developing hepatotoxicity, but cannot be used alone to identify patients not requiring acetylcysteine treatment.

Hepatic aminotransferase activity

A normal ALT activity on presentation is associated with a high negative predictive value of hepatotoxicity following paracetamol-poisoning.

Psi parameter

The psi parameter takes into account the time from ingestion, the serum paracetamol concentration and the time to initiation of acetylcysteine. A hepatotoxicity risk nomogram based on this parameter may be easier to use, but is limited to acute ingestions.

Paracetamol-aminotransferase multiplication product

If a hepatotoxicity risk nomogram is not available, an alternate strategy may be to use the paracetamol-aminotransferase product (<1500 low risk, 1500–10,000 low to moderate risk, >10,000 mg/L × IU/L high risk) to define liver injury risk. Serial blood tests can be performed if patients present prior to 8 h post-overdose for ultimate specificity, or a single blood test can be taken if presenting more than 8 h post-overdose. Patients receiving acetylcysteine within 8 h of their overdose, with a product less than 10,000 mg/L × IU/L have a low likelihood of developing hepatotoxicity. Any clinical trials of intensified treatment (e.g., higher dose) to prevent fulminant hepatic failure might potentially use a product of >10,000 mg/L × IU/L as a criterion for inclusion. The paracetamol-aminotransferase product <1500 mg/L × IU/L may also identify those suitable for an abbreviated acetylcysteine regimen.

Newer biomarkers

These show promise in the early identification of patients with a higher risk of developing hepatic injury. Point of care devices measuring paracetamol adducts need further trials.

Conclusions

Risk prediction tools can stratify those that are more likely to develop hepatotoxicity. Currently, the paracetamol-aminotransferase multiplication product may be such a tool. Novel biomarkers show promise but need further validation and greater clinical availability. These tools may help inform clinical trials on modified acetylcysteine regimens.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1317349

Resolution of cannabis hyperemesis syndrome with topical capsaicin in the emergency department: a case series

Dezieck L, Hafez Z, Conicella A, Blohm E, O'Connor MJ, Schwarz ES, Mullins ME. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1324166:

Background

Cannabinoid hyperemesis syndrome (CHS) is characterized by symptoms of cyclic abdominal pain, nausea, and vomiting in the setting of prolonged cannabis use. The transient receptor potential vanilloid 1 (TRPV1) receptor may be involved in this syndrome. Topical capsaicin is a proposed treatment for CHS; it binds TRPV1 with high specificity, impairing substance P signaling in the area postrema and nucleus tractus solitarius via overstimulation of TRPV1. This may explain its apparent antiemetic effect in this syndrome.

Purpose

We describe a series of thirteen cases of suspected cannabis hyperemesis syndrome treated with capsaicin in the emergency departments of two academic medical centers.

Methods

A query of the electronic health record at both centers identified thirteen patients with documented daily cannabis use and symptoms consistent with CHS who were administered topical capsaicin cream for symptom management.

Results

All 13 patients experienced symptom relief after administration of capsaicin cream.

Conclusion

Topical capsaicin was associated with improvement in symptoms of CHS after other treatments failed.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1324166

Drug-induced hyperlactatemia

Blohm E, Lai J, Neavyn M. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1317348:

Background

Hyperlactatemia is common in critically ill patients and has a variety of etiologies. Medication toxicity remains an uncommon cause that providers often fail to recognize. In this article, we review several medications that cause hyperlactatemia in either therapeutic or supratherapeutic dosing. When known, the incidence, mortality, pathophysiology, and treatment options are discussed.

Methods

We performed a literature search using PUBMED and Google Scholar for English language articles published after 1980 regarding medication induced hyperlactatemia and its management. Our search string resulted in 798 articles of which 138 articles met inclusion criteria and were relevant to the topic of our review.

Conclusions

Hyperlactatemia is a relatively rare but life-threatening toxicity of various medication classes. Discontinuation of the drug is always advised, and some toxicities are subject to specific antidotal treatment. If there is no apparent medical cause for hyperlactatemia (sepsis, hypotension, hypoxia), clinicians should consider a toxicological etiology.

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Association of renal function and symptoms with mortality in star fruit (*Averrhoa carambola*) intoxication

Chua C-B, Sun C-K, Tsui H-W, Yang P-J, Lee K-H, Hsu C-W, Tsai I-T. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1314490:

Background

Star fruit (SF) is a commonly available fruit produced and eaten in tropical and subtropical countries. Since 1993, various reports have described neurotoxicity after eating SF, but this clinical condition remains unfamiliar. We aimed to describe this clinical entity, the role of renal dysfunction in this disorder, treatment strategies, and prognosis of patients with SF intoxication.

Methods

We conducted a search of PubMed and Google Scholar databases from 1993 to 2016. We included reports describing patients with a clear history of SF ingestion with acute symptoms. We described the demographic characteristics, reported SF intake, treatments used, and outcomes.

Results

We reviewed totally 126 patients (male:female = 1.5:1) from 33 articles with mean age 54.4 ± 11 (range: 30–84). The most common symptom was hiccups (65%), whereas confusion and seizure were the most common symptoms associated with mortality (42% and 61%, respectively). Pre-intoxication renal function also affected mortality. While there was no mortality in patients with normal renal function (NRF), the mortality of patients among reported cases with chronic renal insufficiency and end-stage renal disease undergoing dialysis were 36% and 27%, respectively. With the inclusion of patients reported to have NRF, the overall mortality was 24%. Consistently, the number of SF consumed was substantially higher in the patients with NRF than those with renal functional impairment. The most common treatment strategy was hemodialysis (59%).

Conclusions

Patients with impaired renal function were at higher risks of SF intoxication. Severe neurologic symptoms mandate immediate medical intervention because of the association between their occurrence and high mortalities. Toxin removal through dialysis, rather than symptomatic relief, seems to be beneficial to patient survival. Early and continuous dialysis appears to alleviate severe symptoms and prevent symptom rebounds.

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Toxicity resulting from exposure to oven cleaners as reported to the UK National Poisons Information Service (NPIS) from 2009 to 2015

Day RC, Bradberry SM, Sandilands EA, Thomas SHL, Thompson JP, Vale JA. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1306070:

Introduction

Oven cleaning products contain corrosive substances, typically sodium or potassium hydroxide.

Objective

To determine the reported toxicity from exposure to oven cleaning products.

Methods

Telephone enquiries to the UK National Poisons Information Service regarding oven cleaning products were analysed retrospectively for the period January 2009 to December 2015.

Results

There were 796 enquiries relating to 780 patients. Ninety-six percent of the products involved in the reported exposures contained sodium hydroxide and/or potassium hydroxide. Ingestion alone (*n* = 285) or skin contact alone (*n* = 208) accounted for the majority of cases; inhalation alone (*n* = 101), eye contact alone (*n* = 97), and multiple routes of exposure (*n* = 89) accounted for the remainder. Ninety-five percent of patients exposed by inhalation, 94% exposed dermally and 85% reporting eye exposure, developed features of toxicity. Patients exposed by multiple routes developed symptoms in 70% of cases. Only 103 of the 285 patients ingested oven cleaner directly, whereas 182 patients ingested food they considered to have been contaminated with oven cleaner. In 100 of the 103 direct ingestions where the features and World Health Organisation/International Programme on Chemical Safety/European Commission/European Association of Poison Centres and Clinical Toxicologists Poisoning Severity Score were known, 56 reported symptoms which were minor in 51 cases. The most common features following ingestion were vomiting (*n* = 26), abdominal pain (*n* = 22) or pharyngitis (*n* = 15). Skin burns (*n* = 91) predominantly involving the hands or arms, occurred in 44% of dermal exposures. Following inhalation, patients frequently developed respiratory features (*n* = 52) including coughing and chest pain/tightness. Eye pain (*n* = 43) and conjunctivitis (*n* = 33) commonly occurred following ocular exposure.

Conclusions

Most (71%) patients exposed to an oven cleaner irrespective of the route of exposure developed features of toxicity, though in most cases only minor features developed; moderate or severe features ensued in ~4%. Those patients exposed dermally, ophthalmically or by inhalation developed features more frequently (≥85%) than those who ingested a product directly (56%).

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1306070

Unintentional ingestion of *Cordyceps* fungus-infected cicada nymphs causing ibotenic acid poisoning in Southern Vietnam

Doan UV, Mendez Rojas B, Kirby R. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1319066:

Background

Cordyceps fungus found in infected cicada nymphs ("cicada flowers") is utilized in traditional Chinese medicine. Cordyceps fungus toxicity in humans has not been previously reported. We report 60 cases of apparent Cordyceps poisoning in Southern Vietnam.

Methods

We retrospectively collected demographic and clinical data from the medical records (21 cases) and by telephone interview (39 cases) of patients admitted to seven hospitals in Southern Vietnam following ingestion of cicada flowers between 2008 and 2015. We also determined the species of Cordyceps present in the cicada flowers and performed a partial chemical analysis of the fungus.

Results

Sixty cases of toxic effects following ingestion of cicada flowers were documented. Symptom onset occurred within 60 minutes following ingestion. Symptoms included dizziness, vomiting, salivation, mydriasis, jaw stiffness, urinary retention, seizures, agitated delirium, hallucinations, somnolence and coma. None of the patients suffered liver or kidney injury. There was one fatality. The Cordyceps fungus involved in these poisoning was identified as *Ophiocordyceps heteropoda*. The presence of ibotenic acid was confirmed, but musimol and muscarine were absent.

Conclusions

Cicada infected with *Ophiocordyceps heteropoda* in Vietnam contain ibotenic acid and are associated with a clinical syndrome consistent with its effects.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1319066

Medication organizers (pill minders) increase the risk for unintentional pediatric ingestions

Wang GS, Hoppe JA, Brou L, Heard KJ. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1321117:

Objective

Medication organizers may help improve medication compliance; however, they may increase the risk of having an unintentional pediatric exposure. The objective of this study was to measure the association between a pediatric emergency department (ED) visit for an unintentional pharmaceutical ingestion and the use of a medication organizer in the household.

Methods

This was a cross-sectional case control study at a tertiary care children's hospital ED. Cases included subjects <6 years of age who were evaluated in the ED for an unintentional pharmaceutical ingestion. The control group presented to the ED for a non-injury complaint and was matched using age and sex.

Results

The unadjusted odds ratio (OR) of risk for unintentional pharmaceutical ingestion with use of a medication organizer was 2.0 (95% CI, 1.3, 2.9). After adjusting for the presence of prescription medications in the home, the OR of risk for ingestion remained statistically significant at 1.8 (95% CI, 1.1, 2.7). The child obtained the exposure medication from the medication organizer in 63% of cases where a medication organizer was present in the home. Cases were more likely to have knowledge of, and previous contact with poison control centers (PCC) than non-injury controls. Overall, a large number of caregivers (36%) did not have any knowledge of PCC. There were also differences in smoking and use of seat belts between cases and controls.

Conclusions

The use of medication organizers may be a risk factor for unintentional pediatric pharmaceutical ingestions, even when controlling for the use of prescription medications in the home. Further research is needed to evaluate the specific role of medication organizers, and subsequently, improve prevention strategies.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1321117

Pharmacokinetics of concentrated naloxone nasal spray over first 30 minutes post-dosing: analysis of suitability for opioid overdose reversal

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CHEMICAL WARFARE, BIOLOGICAL WARFARE AND RIOT CONTROL AGENTS

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