

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Hypoglycemia and lactic acidosis outperform King's College criteria for predicting death or transplant in acetaminophen toxic patients

Levine M, Stellpflug SJ, Pizon AF, Peak DA, Villano J, Wiegand T, Dib C, Thomas SH. Clin Toxicol 2018; online early: doi: doi: 10.1080/15563650.2017.1420193:

Importance

Acetaminophen toxicity is common and is characterized by hepatic failure. In cases that are not improving with standard medical therapy with N-acetylcysteine, some patients may require hepatic transplant. While there are various criteria to predict patients who might benefit from transplant, the King's College criteria remain one of the most widely used. However, the King's College criteria have several limitations and do not incorporate glucose, an important marker of hepatic function.

Objective

The primary objective of this study is to compare the presence of hypoglycemia, coagulopathy, and metabolic acidosis with the King's College criteria for predicting a composite endpoint of death or transplant.

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Design

This study is a retrospective cohort study of adult patients admitted with a discharge diagnosis of acetaminophen-induced liver failure.

Setting

The patients were admitted at one of six university-affiliated teaching hospitals in the United States.

Results

A total of 334 subjects were identified who met inclusion criteria. Fifty-one subjects (15.3%) met the composite endpoint of death or transplant. Ninety-six (28.7%) subjects met the King's College criteria for transplant. The presence of hypoglycemia increased the odds of reaching the composite endpoint by 3.39-fold. This model performed better than the King's College criteria (pseudo R^2 for the area under the curve of 0.93 vs. 0.20 for the King's College criteria).

Conclusions

The combination of hypoglycemia, coagulopathy, and lactic acidosis performed better than the King's College criteria for predicting death or transplant.

Full text available from: <https://doi.org/10.1080/15563650.2017.1420193>

Anti-colchicine Fab fragments prevent lethal colchicine toxicity in a porcine model: a pharmacokinetic and clinical study

Eddleston M, Fabresse N, Thompson A, Al Abdulla I, Gregson R, King T, Astier A, Baud FJ, Clutton RE, Alvarez J-C. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2017.1422510:

Background

Colchicine poisoning is commonly lethal. Colchicine-specific Fab fragments increase rat urinary colchicine clearance and have been associated with a good outcome in one patient. We aimed to develop a porcine model of colchicine toxicity to study the pharmacokinetics and efficacy of ovine Fab.

Methods

A Göttingen minipig critical care model was established and serial blood samples taken for colchicine and Fab pharmacokinetics, clinical chemistry, and haematology. Animals were euthanised when the mean arterial pressure fell below 45 mmHg without response to vasopressor, or at study completion.

Results

Initial studies indicated that oral dosing produced variable pharmacokinetics and time-to-euthanasia. By contrast, intravenous infusion of 0.25 mg/kg colchicine over 1 h produced reproducible pharmacokinetics (AUC_{0-20} 343 [SD = 21] $\mu\text{g/L/h}$), acute multi-organ injury, and cardiotoxicity requiring euthanasia a mean of 22.5 (SD = 3.2) h after dosing. A full-neutralising equimolar Fab dose given 6 h after the infusion (50% first hour, 50% next 6 h [to reduce renal-loss of unbound Fab]) produced a 7.35-fold increase in plasma colchicine (AUC_{0-20} 2,522 [SD = 14] $\mu\text{g/L/h}$), and removed all free plasma colchicine, but did not prevent toxicity (euthanasia at 29.1 [SD = 3.4] h). Earlier administration over 1 h of the full-neutralising dose, 1 or 3 h after the colchicine, produced a 12.9-fold (AUC_{0-20} 4,433 [SD = 607] $\mu\text{g/L/h}$) and 6.0-fold (AUC_{0-20} 2,047 [SD = 51] $\mu\text{g/L/h}$) increase in plasma colchicine, respectively, absence of free plasma colchicine until 20 h, and survival to study end without marked cardiotoxicity.

Conclusions

Colchicine-specific Fab given early, in equimolar dose, bound colchicine, eliciting its movement into the blood, and preventing severe toxicity. Clinical studies are now needed to determine how soon this antidote must be given to work in human poisoning.

Full text available from: <https://doi.org/10.1080/15563650.2017.1422510>

Accidental pharmacological poisonings in young children: population-based study in three settings

Bell JC, Bentley JP, Downie C, Cairns R, Buckley NA, Katelaris A, Pearson S-A, Nassar N. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2017.1422509:

Introduction

Pharmacological poisonings in young children are avoidable. Previous studies report calls to poisons centres, presentations to emergency departments (ED) or hospital admissions. There are limited data assessing concurrent management of poisonings across all three settings. We aimed to describe accidental pharmacological poisonings in young children across our Poisons Information Centre (PIC), EDs and hospitals.

Methods

A population-based study in New South Wales, Australia, of PIC calls, ED presentations and hospital admissions for accidental pharmacological poisoning in children aged <5 years, 2007–2013. We examined trends, medicines responsible and subsequent management. Medicines were coded using ICD10-AM diagnosis codes (T36-50).

Results

Over 2007-2013, pharmacological poisonings accounted for 67,816 PIC calls, 7739 ED presentations and 2082 admissions. Rates (per 10,000 children) of PIC calls declined from 220 to 178; ED presentations were stable (~22–24), with a decrease in emergency cases offset by an increase in semi- or non-urgent presentations; hospital admissions declined (8–5). Most PIC calls related to "non-opioid analgesics" (25%), and "topical agents" (18%). Nearly every day, one child aged <5 years was admitted to hospital for poisoning. "Benzodiazepines", "other and unspecified antidepressants", "uncategorised antihypertensives", and "4-aminophenol derivatives" accounted for over one-third of all admissions. Most PIC calls (90%) were advised to stay home, 6% referred to hospital. One-quarter of ED presentations resulted in admission.

Conclusions

Poisonings reported to PIC and hospitals declined, however, non-urgent ED presentations increased. Strategies to reduce therapeutic errors and access to medicines, and education campaigns to improve Poisons Centre call rates to prevent unnecessary ED presentations are needed.

Full text available from: <https://doi.org/10.1080/15563650.2017.1422509>

Trends in dextromethorphan cough and cold products: 2000–2015 National Poison Data System intentional abuse exposure calls

Karami S, Major JM, Calderon S, McAninch JK. Clin Toxicol 2017; online early: doi: [10.1080/15563650.2017.1416124](https://doi.org/10.1080/15563650.2017.1416124):

Context

Recent restrictions in access to and availability of dextromethorphan (DXM) cough and cold medications may correlate with changes in abuse exposures.

Objective

To extend and update existing knowledge about DXM abuse, we describe recent trends and patterns of calls to poison control centers involving DXM abuse, by demographics, geography, common brands, and medical outcomes.

Methods

We utilized data from the National Poison Data System (NPDS) maintained by the American Association of Poison Control Centers (AAPCC), which captures data on calls to U.S. poison centers on a near real-time basis. We analyzed demographic, geographic, brand and medical outcome data for single-substance DXM cough and cold product intentional abuse exposure calls in multiple age groups reported to NPDS from 2000 to 2015.

Results

The annual rate of single-substance DXM intentional abuse calls tripled from 2000 to 2006 and subsequently plateaued from 2006 to 2015. The highest abuse call rate was observed among adolescents 14–17 years old, where the mean annual number of calls was 1761 per year, corresponding to an annual rate of 103.6 calls per million population. From 2006 to 2015, the rate for single-substance DXM abuse calls among adolescents 14–17 years decreased by 56.3%, from 143.8 to 80.9 calls per million population.

Conclusion

DXM intentional abuse exposure call rates have declined among adolescents 14–17 years, since their peak in 2006. The observed decline in DXM abuse call rates corresponds to a period of growing public health efforts to curtail the abuse of over-the-counter (OTC) DXM containing products, particularly among adolescents. Further evaluation of state-level sales and abuse trends among adolescents would be valuable to better understand how restricted availability of OTC DXM cough and cold products and other efforts may affect abuse rates.

Full text available from: <https://doi.org/10.1080/15563650.2017.1416124>

Assessing the public health impact of using poison center data for public health surveillance

Wang A, Law R, Lyons R, Choudhary E, Wolkin A, Schier J. Clin Toxicol 2017; online early: doi: [10.1080/15563650.2017.1413194](https://doi.org/10.1080/15563650.2017.1413194):

Context

The National Poison Data System (NPDS) is a database and surveillance system for US poison centers (PCs) call data. The Centers for Disease Control and Prevention (CDC) and American Association of Poison Control Centers (AAPCC) use NPDS to identify incidents of potential public health significance. State health departments are notified by CDC of incidents identified by NPDS to be of potential public health significance. Our objective was to describe the public health impact of CDC's notifications and the use of NPDS data for surveillance.

Methods

We described how NPDS data informed three public health responses: the Deepwater Horizon incident, national exposures to laundry detergent pods, and national exposures to e-cigarettes. Additionally, we extracted survey results of state epidemiologists regarding NPDS incident notification follow-up from 1 January 2015 to 31 December 2016 to assess current public health application of NPDS data using Epi Info 7.2 and analyzed data using SAS 9.3. We assessed whether state health departments were aware of incidents before notification, what actions were taken, and whether CDC notifications contributed to actions.

Discussion

NPDS data provided evidence for industry changes to improve laundry detergent pod containers safety and highlighted the need to regulate e-cigarette sale and manufacturing. NPDS data were used to improve situational awareness during the 2010 Deepwater Horizon oil spill. Of 59 health departments and PCs who responded to CDC notifications about anomalies (response rate = 49.2%), 27 (46%) reported no previous awareness of the incident, and 20 (34%) said that notifications contributed to public health action. Conclusions: Monitoring NPDS data for anomalies can identify emerging public health threats and provide evidence-based science to support public health action and policy changes.

Full text available from: <https://doi.org/10.1080/15563650.2017.1413194>

Serum neuron-specific enolase levels at presentation and long-term neurological sequelae after acute charcoal burning-induced carbon monoxide poisoning

Moon JM, Chun BJ, Lee SD, Jung EJ. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1415347:

Objective

This study aimed to investigate whether clinical parameters and serum neuron-specific enolase (NSE) levels measured at emergency department (ED) presentation help stratify the risk of acute or delayed persistent severe neurological sequelae after acute carbon monoxide (CO) poisoning induced by charcoal burning.

Methods

This retrospective study included 236 patients who suffered from CO poisoning. Demographic information, serum NSE levels measured in the ED, treatment, clinical course, and long-term neurological outcomes were recorded.

Results

The median serum NSE level at presentation was 15.5 (10.9–22.7) ng/mL. No differences were observed in the duration of CO exposure; the initial Glasgow Coma Scale (GCS) score; the levels of arterial HCO_3^- , white blood cells (WBCs), C-reactive protein (CRP) or troponin I; or the frequency of abnormal diffusion-weighted imaging finding at presentation among the groups with different serum NSE levels at presentation. The incidences of acute and delayed persistent neurologic sequelae assessed at 22.3 months after acute charcoal CO poisoning were 5.1% and 8.5%, respectively. No difference in the NSE level was observed between patients stratified according to long-term neurological status. According to the multinomial logistic regression analysis, age, serum CRP levels and the initial GCS score were risk factors for the two types of persistent severe neurological sequelae, whereas troponin I levels were associated only with the acute persistent severe neurological sequelae. However, the adjusted NSE level was not a risk factor for any persistent neurological sequelae.

Conclusions

Serum NSE levels at presentation were not correlated with the risk of acute or delayed persistent neurological sequelae. Further studies with blood sampling at optimal time points and serial measurements should be conducted. Age, initial GCS score, and CRP levels may be risk factors for persistent severe neurological sequelae.

Full text available from: <https://doi.org/10.1080/15563650.2017.1415347>

Analysis of the development and progression of carbon monoxide poisoning–related acute kidney injury according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria

Kim Y-J, Sohn CH, Seo D-W, Oh BJ, Lim KS, Chang JW, Kim WY. Clin Toxicol 2018; online early: doi: 10.1080/15563650.2018.1424890:

Context

Acute kidney injury (AKI) can occur after carbon monoxide (CO) intoxication; however, limited data are available. This study aimed to evaluate the prognostic value of the development and progression of AKI in patients with acute CO poisoning.

Materials and methods

We conducted a retrospective cohort study using a prospective registry of CO poisoning between January 2010 and December 2015. AKI was defined and classified according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria. Multivariate logistic regression analysis was conducted to determine the association between AKI and adverse outcomes, defined as neurological deficits at discharge or 28-day mortality.

Results

A total of 661 patients were evaluated. According to KDIGO criteria, 114 patients (17.2%) had AKI (initial: stage 1, 70.2%; stage 2, 26.3%; stage 3, 3.5%) on admission and 119 (18.0%) finally developed AKI during their hospital stay (maximum: stage 1, 68.9%; stage 2, 23.5%; stage 3, 7.6%). Almost all patients (99.2%) were diagnosed as having their highest KDIGO stage within three days (median, one day). AKI development was associated with adverse outcomes (odds ratio (OR) 17.53, 95% confidence interval 45.00–77.14). Both initial and maximum AKI stages demonstrated a stepwise increase of adjusted OR for adverse outcomes. AKI stage progression occurred in 8.4% of patients with AKI and was an independent factor for adverse outcomes.

Conclusion

CO poisoning- related AKI occurred in 18% and was mostly detected within one day after CO intoxication. The development and progression of AKI had a strong association with adverse outcomes and deserve further prospective investigation.

Full text available from: <https://doi.org/10.1080/15563650.2018.1424890>

Evaluation of relationship between coronary artery status evaluated by coronary computed tomography angiography and development of cardiomyopathy in carbon monoxide poisoned patients with myocardial injury: a prospective observational study

Cha YS, Kim H, Lee Y, Kwon W, Son J-W, Youk H, Kim HI, Kim OH, Park KH, Cha K-C, Lee KH, Hwang SO. Clin Toxicol 2018; 56: 30-6.

Objectives

Whether coronary artery changes are a main mechanism in the development of carbon monoxide (CO)-induced cardiomyopathy remains unknown. We investigated the effects of coronary artery stenosis on the presence or patterns of cardiomyopathy in CO-poisoned patients with myocardial injury defined as elevation of troponin I.

Materials and methods

This prospective observational study collected data from consecutive patients who were diagnosed with CO poisoning and myocardial injury during the 24-month study period. Transthoracic echocardiography (TTE) and coronary computed tomography angiography (CCTA) were performed to evaluate cardiac function and coronary artery status.

Results

TTE and CCTA were performed in 32 consecutive patients. The observed echocardiographic patterns included non-cardiomyopathy (59.4%), left ventricular global dysfunction (25%), Takotsubo cardiomyopathy (6.3%), and cardiomyopathy matching the distribution of the left anterior descending (LAD) artery (9.4%). Four patients had more than moderate stenosis, while stenoses of the LAD, left circumflex, and right coronary arteries were observed in two (6.3%), three (9.4%), and zero patients, respectively. Patients with coronary artery stenosis did not develop cardiomyopathy except for one patient; this patient also did not have regional wall motion abnormalities (RWMA) matched with the stenosis territory.

Conclusions

Because there was no difference in coronary artery stenosis according to the presence or patterns of CO-induced cardiomyopathy, coronary artery stenosis is not the main mechanism for the development of CO-induced cardiomyopathy. Thus, the evaluation of coronary arteries is not necessary in all patients with CO-induced cardiomyopathy unless there is RWMA consistent with ischemic changes in electrocardiograms and elevated troponin I levels.

Full text available from: <https://doi.org/10.1080/15563650.2017.1337910>

Baclofen in gamma-hydroxybutyrate withdrawal: patterns of use and online availability

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