Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

The management of ventricular dysrhythmia in aconite poisoning

Coulson JM, Caparrotta TM, Thompson JP. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1291944:

Introduction

Aconite poisoning is relatively rare but is frequently complicated by ventricular dysrhythmias, which may be fatal.

Molecular basis of aconite alkaloid ventricular arrhythmogenicity

Aconite exerts its toxic effects due to the presence of an admixture of alkaloids present in all parts of the plant. The major target of these aconite alkaloids is the fast voltage-gates sodium channel, where they cause persistent activation. This blockade of the channel in the activated state promotes automaticity within the ventricular myocardium and the generation of ventricular arrhythmias.

Aconitine-induced arrhythmias

Aconite alkaloids are known to cause many different types of disturbance of heart rhythm. However, this focused review specifically looks at ventricular rhythm disturbances, namely ventricular ectopy, ventricular tachycardia, torsades des pointes and ventricular fibrillation.

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Objective

The objective of this review was to identify the outcome of anti-dysrhythmic strategies from animal studies and case reports in humans in order to guide the management of ventricular dysrhythmias in aconite poisoning in humans.

Methods

A review of the literature in English was conducted in PubMed and Google Scholar from 1966 to July 2016 using the search terms "aconite/aconitine"; "aconite/aconitine"+ "poisoning" and "aconite/aconitine"+"dysrhythmia". 168 human case-reports and case-series were identified by these searches, of which 103 were rejected if exposure to aconite did not result in ventricular dysrhythmias, if it was uncertain as to whether aconite had been ingested, if other agents were co-ingested, if there was insufficient information to determine the type of treatments administered or if there was insufficient information to determine outcome. Thus, 65 case reports of probable aconite poisoning that resulted in ventricular dysrhythmias were identified.

Toxicokinetic data in aconite poisoning

Data were only available in three papers; the presence of ventricular rhythm disturbances directly correlated with the concentration of aconite alkaloids in the plasma.

Management

54 of 65 cases developed ventricular tachycardia, six developed torsades des pointes, 15 patients developed ventricular fibrillation, 10 developed ventricular ectopics and one developed a broad complex tachycardia not otherwise specified; each dysrhythmia was regarded as separate and patients may have had more than one dysrhythmia. 10 patients died, giving a mortality of 15%. In total, 147 treatments were administered to 65 patients. 46 of the interventions were assessed by the authors as having been associated with successful restoration of sinus rhythm. Flecainide administration was accompanied by dysrhythmia termination in six of seven cases. Mexiletine was connected with correcting dysrhythmias in 3 of 3 cases. Procainamide administration was associated with return to sinus rhythm in 2 of 2 cases. Prolonged cardio-pulmonary resuscitation was administered to 15 patients where it was associated with a return to sinus rhythm in nine of these. Amiodarone was linked to success in correcting dysrhythmias in 11 of 20 cases. Cardiopulmonary bypass use was associated with a return to sinus rhythm in four out of six cases. Epinephrine was documented as being employed on four occasions, and was associated with a restoration of sinus rhythm on two of these. Magnesium sulphate administration was accompanied by dysrhythmia termination in two of nine cases. Direct cardioversion was associated with a return of sinus rhythm in 5 of 30 cases. However, it is not certain whether the drug treatment influenced the course of the dysrhythmia.

Conclusions

Based on the evidence available from human case reports, flecainaide or amiodarone appear to be more associated with a return to sinus rhythm than lidocaine and/or cardioversion, although it is not established whether the administration of treatment caused reversion to normal sinus rhythm. The potential beneficial effects of amiodarone were not observed in animal studies. This may be due to intra-species differences between ion channels or relate to the wider cardiovascular toxicity of aconite that extends beyond arrhythmias. Prolonged cardiopulmonary resuscitation and cardiopulmonary bypass should be considered as an integral part of good clinical care as "time-buying" strategies to allow the body to excrete the toxic alkaloids. There may also be a role for mexiletine, procainamide and magnesium sulphate.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1291944

A systematic review of the evidence for acute tolerance to alcohol – the "Mellanby effect"

Holland MG, Ferner RE. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1296576:

Objective

To review the evidence for "the Mellanby effect", that is, whether the response to a given blood alcohol concentration (BAC) is more marked when BAC is rising than at the same concentration when BAC is falling.

Methods

We systematically searched the databases EMBASE, Medline, and Scopus up to and including December 2016 using text words "tolerance", "ascending", "descending" or "Mellanby" with Medline term "exp *alcohol/" or "exp *drinking behavior/" or equivalent. Articles were identified for further examination by title or abstract; full text articles were retained for analysis if they dealt with acute (within dose) alcohol tolerance in human subjects and provided quantitative data on both the ascending and descending parts of the BAC–time curve. Reference lists of identified works were scanned for other potentially relevant material. We extracted and analyzed data on the subjective and objective assessment of alcohol effects.

Results

We identified and screened 386 unique articles, of which 127 full-text articles were assessed; one provided no qualitative results, 62 involved no human study, 25 did not consider acute tolerance within dose, and 13 failed to provide data on both ascending and descending BAC. We extracted data from the 26 remaining articles. The studies were highly heterogeneous. Most were small, examining a total of 770 subjects, of whom 564 received alcohol and were analyzed in groups of median size 10 (range 5–38), sometimes subdivided on the basis of drinking or family history. Subjects were often young white men. Doses of alcohol and rates of administration differed. Performance was assessed by at least 26 different methods, some of which measured many variables. We examined only results of studies which compared results for a given alcohol concentration (C) measured on the ascending limb (C_{up}) and the descending limb (C_{down}) of the BAC-time curve, whether in paired or parallel-group studies. When subjects were given alcohol in more than one session, we considered results from the first session only. Rating at C_{down} was better than at C_{up} for some measures, as expected if the Mellanby effect were operating. For example, subjects rated themselves less intoxicated on the descending limb than at the same concentration on the ascending limb in 12/13 trials including 229 subjects that gave statistically significant results. In 9 trials with a total of 139 subjects, mean difference could be calculated; weighted for study size, it was 29% [range 24–74%]. Willingness to drive was significantly greater in 4 of 6 studies including a total of 105 subjects; weighted mean difference increased by 207% [range 79–300%]. By contrast, measure of driving ability in three groups of a total of 200 trials in 57 subjects showed worse performance by a weighted mean of 96% [range 3–566%]. In three trials that tested inhibitory control (cued go or no-go response times), weighted mean performance was 30% [range 14–65%] worse on the descending limb.

Conclusions

The "Mellanby effect" has been demonstrated for subjective intoxication and willingness to drive, both of which are more affected at a stated ethanol concentration when BAC is rising than at the same concentration when BAC is falling. By contrast, objective measures of skills necessary for safe driving, such as response to inhibitory cues and skills measured on driving simulators, were generally worse on the descending part of the BAC-time curve for the same BAC.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1296576

Analytical confirmation of synthetic cannabinoids in a cohort of 179 presentations with acute recreational drug toxicity to an Emergency Department in London, UK in the first half of 2015

Abouchedid R, Hudson S, Thurtle N, Yamamoto T, Ho JH, Bailey G, Wood M, Sadones N, Stove CP, Dines A, Archer JRH, Wood DM, Dargan PI. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1287373:

Context

Synthetic cannabinoid receptor agonists are the largest group of new psychoactive substances reported in the last decade; in this study we investigated how commonly these drugs are found in patients presenting to the Emergency Department with acute recreational drug toxicity.

Methods

We conducted an observational cohort study enrolling consecutive adult patients presenting to an Emergency Department (ED) in London (UK) January–July 2015 (6 months) with acute recreational drug toxicity. Residual serum obtained from a serum sample taken as part of routine clinical care was analyzed using high-resolution accurate mass-spectrometry with liquid-chromatography (HRAM-LCMSMS). Minimum clinical data were obtained from ED medical records.

Results

18 (10%) of the 179 patient samples were positive for synthetic cannabinoid receptor agonists. The most common was 5F AKB-48 (13 samples, concentration 50–7600 pg/ml), followed by 5F PB-22 (7, 30–400 pg/mL), MDMB-CHMICA (7, 80–8000 pg/mL), AB-CHMINACA (3, 50–1800 pg/mL), Cumyl 5F-PINACA (1, 800 pg/mL) and BB-22 (1, 60 pg/mL). Only 9/18 (50%) in whom synthetic cannabinoid receptor agonists were detected self-reported synthetic cannabinoid receptor agonist use. The most common clinical features were seizures and agitation, both recorded in four (22%) individuals. Fourteen patients (78%) were discharged from the ED, one of the four admitted to hospital was admitted to critical care.

Conclusions

Synthetic cannabinoid receptor agonists were found in 10% of this cohort with acute recreational drug toxicity but self-reported in only half of these. This suggests that presentations to the ED with acute synthetic cannabinoid receptor agonist toxicity may be more common than reported.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1287373

Self-reported cocaine use is not associated with elevations in high-sensitivity troponin I

Jordan CD, Korley FK, Stolbach AI. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1285404:

Objective

High-sensitivity troponin (hsTn) assays detect 10 times lower concentrations of cardiac troponin than conventional assays. We examined the effects of self-reported cocaine use to determine whether those with acute cocaine use being evaluated for ACS are more likely to have elevated hsTnI than those nonusers being evaluated for ACS.

Methods

We conducted a sub-analysis of a prospective cohort of ED patients evaluated for acute coronary syndrome. Recent cocaine use was determined by structured patient interviews. High-sensitivity troponin (Abbott) and conventional troponin I (Abbott, cTnI) were measured on samples drawn at presentation. Urine toxicology screen for cocaine metabolite was obtained at the discretion of treating clinicians.

Results

Of 1862 patients enrolled, 444 reported prior cocaine use and 99 reported cocaine use within the preceding month. Median hsTn in patients with last cocaine use within 24 h, 2–7 days, 1 week–1 month, >1 month, and no prior cocaine use were: 9 (IQR: 3–17) ng/L, 6 (IQR: 3–24.3) ng/L, 6 (IQR: 3–89.5) ng/L, 3 (IQR: 3–18.5) ng/L and 3 (IQR: 3–17) ng/L, respectively. Urine toxicology assays (UTox) for cocaine were performed in 640 (34.4%) patients. The median hsTn for those who were UTox+, UTox – and those without a UTox were: 9 ng/L (IQR: 3–48.5), 9?ng/L (IQR: 3–40) and 3 ng/L (IQR: 3–12), respectively. There were no differences in the prevalence of new troponin elevations (hsTn >99th percentile but cTnI <99th percentile) in those with recent cocaine use compared to those without recent cocaine use.

Conclusions

In this first investigation of hsTn in patients with self-reported recent cocaine use, we have determined that hsTn does not lead to an increase in the prevalence of troponin elevation in cocaine users.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1285404

Pediatric ingestion of vilazodone compared to other selective serotonin reuptake inhibitor medications

Russell JL, Spiller HA, Chounthirath T, Casavant MJ. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1287375:

Background

Unintentional ingestion of selective serotonin reuptake inhibitor (SSRI) medications is common amongst children <6 years of age. Current evidence-based management guidelines are based on a low incidence of significant medical outcomes in these children.

Objective

To describe and compare outcomes of pediatric exposures to vilazodone with other SSRIs.

Methods

A retrospective observational case series analysis of both single and polysubstance SSRI exposures amongst children <6 years old reported to the National Poison Data System (NPDS).

Results

11,384 SSRI exposures in children <6 years of age reported to NPDS between January 2012 and June 2016 were assessed. Vilazodone only accounted for 5.9% of all exposures, but resulted in the highest proportion of health care facility admission compared to other SSRIs, both in single substance (165 of 531 (31.1%); OR 9.0 [7.3–11.2]) and polysubstance (57 of 107 (53.3%); OR 4.1 [2.7–6.2]) exposures. Children exposed to vilazodone also have higher odds of experiencing a major or moderate outcome in single (134 of 531 (25.2%); OR 20.5 [15.5–27.1]) and polysubstance (37 of 107 (35.6%); OR 5.9 [3.7–9.0]) exposures compared to other SSRIs. Several severe clinical outcomes, such as seizure and coma, were more common among the vilazodone exposures.

Conclusions

Exposure to vilazodone in this age group results in an increased rate of hospitalization as well as more severe clinical effects as compared to other SSRIs. Current evidence-based SSRI exposure management guidelines may not be appropriate for the management of vilazodone ingestion in this age group.

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Lipid emulsion improves survival in animal models of local anesthetic toxicity: a meta-analysis

Fettiplace MR, McCabe DJ. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1288911:

Introduction

The Lipid Emulsion Therapy workgroup, organized by the American Academy of Clinical Toxicology, recently conducted a systematic review, which subjectively evaluated lipid emulsion as a treatment for local anesthetic toxicity. We re-extracted data and conducted a meta-analysis of survival in animal models.

Methods

We extracted survival data from 26 publications and conducted a random-effect meta-analysis based on odds ratio weighted by inverse variance. We assessed the benefit of lipid emulsion as an independent variable in resuscitative models (16 studies). We measured Cochran's Q for heterogeneity and I^2 to determine variance contributed by heterogeneity. Finally, we conducted a funnel plot analysis and Egger's test to assess for publication bias in studies.

Results

Lipid emulsion reduced the odds of death in resuscitative models (OR =0.24; 95%CI: 0.1–0.56, p = .0012). Heterogeneity analysis indicated a homogenous distribution. Funnel plot analysis did not indicate publication bias in experimental models.

Discussion

Meta-analysis of animal data supports the use of lipid emulsion (in combination with other resuscitative measures) for the treatment of local anesthetic toxicity, specifically from bupivacaine. Our conclusion differed from the original review. Analysis of outliers reinforced the need for good life support measures (securement of airway and chest compressions) along with prompt treatment with lipid.

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Accuracy of the paracetamol-aminotransferase multiplication product to predict hepatotoxicity in modified-release paracetamol overdose

Wong A, Sivilotti MLA, Graudins A. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1290253:

Context

The paracetamol-aminotransferase multiplication product (APAP x ALT) is a risk predictor of hepatotoxicity that is somewhat independent of time and type of ingestion. However, its accuracy following ingestion of modified-release formulations is not known, as the product has been derived and validated after immediate-release paracetamol overdoses.

Objective
The aim of this retrospective cohort study was to evaluate the accuracy of the multiplication product to predict hepatotoxicity in a cohort of patients with modified-release paracetamol overdose.

Methods
We assessed all patients with modified-release paracetamol overdose presenting to our hospital network from October 2009 to July 2016. Ingestion of a modified-release formulation was identified by patient self-report or retrieval of the original container. Hepatotoxicity was defined as peak alanine aminotransferase ≥1000 IU/L, and acute liver injury (ALI) as a doubling of baseline ALT to more than 50 IU/L.

Results
Of 1989 paracetamol overdose presentations, we identified 73 modified-release paracetamol exposures treated with acetylcysteine. Five patients developed hepatotoxicity, including one who received acetylcysteine within eight hours of an acute ingestion. No patient with an initial multiplication product <10,000 mg/L × IU/L developed hepatotoxicity (sensitivity 100% [95%CI 48%, 100%], specificity 97% [90%, 100%]). Specificity fell to 54% (95%CI: 34, 59%) at a product cut-off point <1500 mg/L × IU/L. When calculated within eight hours of ingestion, mild elevations of the multiplication product fell quickly on repeat testing in patients without ALI or hepatotoxicity.

Conclusions
In modified-release paracetamol overdose treated with acetylcysteine, the paracetamol-aminotransferase multiplication product demonstrated similar accuracy and temporal profile to previous reports involving mostly immediate-release formulations. Above a cut-point of 10,000 mg/L × IU/L, it was very strongly associated with the development of acute liver injury and hepatotoxicity, especially when calculated more than eight hours post-ingestion. When below 1500 mg/L × IU/L the likelihood of developing hepatotoxicity was very low. Persistently high serial multiplication product calculations were associated with the greatest risk of hepatotoxicity.

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Estimating the impact of adopting the revised United Kingdom acetaminophen treatment nomogram in the U.S. population

Levine M, Stellpflug S, Pizon AF, Traub S, Vohra R, Wiegand T, Traub N, Tashman D, Desai S, Chang J, Nathwani D, Thomas S. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1291945:

Background
Acetaminophen toxicity is common in clinical practice. In recent years, several European countries have lowered the treatment threshold, which has resulted in increased number of patients being treated at a questionable clinical benefit.

Objective
The primary objective of this study is to estimate the cost and associated burden to the United States (U.S.) healthcare system, if such a change were adopted in the U.S.

Methods
This study is a retrospective review of all patients age 14 years or older who were admitted to one of eight different hospitals located throughout the U.S. with acetaminophen exposures during a five and a half year span, encompassing from 1 January 2008 to 30 June 2013. Those patients who would be treated with the revised nomogram, but not the current nomogram were included. The cost of such treatment was extrapolated to a national level.

Results

139 subjects were identified who would be treated with the revised nomogram, but not the current nomogram. Extrapolating these numbers nationally, an additional 4507 (95%CI 3641–8751) Americans would be treated annually for acetaminophen toxicity. The cost of lowering the treatment threshold is estimated to be $45 million (95%CI 36,400,000–87,500,000) annually.

Conclusions

Adopting the revised treatment threshold in the U.S. would result in a significant cost, yet provide an unclear clinical benefit.

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A preliminary study in the alterations of mitochondrial respiration in patients with carbon monoxide poisoning measured in blood cells

Jang DH, Kelly M, Hardy K, Lambert DS, Shofer FS, Eckmann DM. Clin Toxicol 2017; online early: doi: 10.1080/15563650.2017.1288912:

Objectives

Carbon monoxide (CO) is a colorless and odorless gas responsible for poisoning mortality and morbidity in the United States. At this time, there is no reliable method to predict the severity of poisoning or clinical prognosis following CO exposure. Whole blood cells, such as peripheral blood mononuclear cells (PBMCs) and platelets, have been explored for their potential use to act as sensitive biomarkers for mitochondrial dysfunction which may have a role in CO poisoning.

Design

The objective of this study was to measure mitochondrial respiration using intact cells obtained from patients exposed to CO as a potential biomarker for mitochondrial inhibition with results that can be obtained in a time frame useful for guiding clinical care. This was a prospective, observational pilot study performed from July 2015 to July 2016 at a single academic tertiary care center that is the location of the region's only multi chamber hyperbaric.

Measurements

Clinical characteristics, patient demographics, mitochondrial respiration and outcomes were recorded.

Main results

There were 7 patients enrolled with a mean COHb level 26.8 ± 10 and with a mean lactate of 1.1 ± 0.4 mmol/L. All 7 CO exposures were related to heat generators used during winter months with two deaths. There was a positive correlation between maximal respiration and COHb levels with both high maximal respiration and high spare respiratory capacity correlating with a high COHb level. There was a subset of PBMCs (n = 4) that were analyzed for Complex IV (cytochrome c oxidase) activity.

Conclusions

In this pilot study, measurements can be performed in an appropriate timeline for clinical care with potential to serve as a prognostic marker. Further work is necessary to develop high-resolution respirometry as a clinical tool for assessing the severity of illness and guiding therapy.

Full text available from: http://dx.doi.org/10.1080/15563650.2017.1288912

Carbamate insecticides target human melatonin receptors

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General

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Analytical toxicology

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Analytical confirmation of synthetic cannabinoids in a cohort of 179 presentations with acute recreational drug toxicity to an Emergency Department in London, UK in the first half of 2015.
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CHEMICAL WARFARE, BIOLOGICAL WARFARE AND RIOT CONTROL AGENTS

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