

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Treatment of cocaine cardiovascular toxicity: a systematic review

Richards JR, Garber D, Laurin EG, Albertson TE, Derlet RW, Amsterdam EA, Olson KR, Ramoska EA, Lange RA. Clin Toxicol 2016; online early: doi: 10.3109/15563650.2016.1142090:

Introduction

Cocaine abuse is a major worldwide health problem. Patients with acute cocaine toxicity presenting to the emergency department may require urgent treatment for tachycardia, dysrhythmia, hypertension, and coronary vasospasm, leading to pathological sequelae such as acute coronary syndrome, stroke, and death.

Objective

The objective of this study is to review the current evidence for pharmacological treatment of cardiovascular toxicity resulting from cocaine abuse.

Methods

MEDLINE, PsycINFO, Database of Abstracts of Reviews of Effects (DARE), OpenGrey, Google Scholar, and the Cochrane Library were searched from inception to November 2015. Articles on pharmacological treatment involving human subjects and cocaine were selected and

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reviewed. Evidence was graded using Oxford Centre for Evidence-Based Medicine guidelines. Treatment recommendations were compared to current American College of Cardiology/American Heart Association guidelines. Special attention was given to adverse drug events or treatment failure. The search resulted in 2376 articles with 120 eligible involving 2358 human subjects.

Benzodiazepines and other GABA-active agents

There were five high-quality (CEBM Level I/II) studies, three retrospective (Level III), and 25 case series/reports (Level IV/V) supporting the use of benzodiazepines and other GABA-active agents in 234 subjects with eight treatment failures. Benzodiazepines may not always effectively mitigate tachycardia, hypertension, and vasospasm from cocaine toxicity.

Calcium channel blockers

There were seven Level I/II, one Level III, and seven Level IV/V studies involving 107 subjects and one treatment failure. Calcium channel blockers may decrease hypertension and coronary vasospasm, but not necessarily tachycardia.

Nitric oxide-mediated vasodilators

There were six Level I/II, one Level III, and 25 Level IV/V studies conducted in 246 subjects with 11 treatment failures and two adverse drug events. Nitroglycerin may lead to severe hypotension and reflex tachycardia.

Alpha-adrenoceptor blocking drugs

There were two Level I studies and three case reports. Alpha-1 blockers may improve hypertension and vasospasm, but not tachycardia, although evidence is limited.

Alpha-2-adrenoceptor agonists

There were two high-quality studies and one case report detailing the successful use of dexmedetomidine.

Beta-blockers and β/α -blockers

There were nine Level I/II, seven Level III, and 34 Level IV/V studies of β -blockers, with 1744 subjects, seven adverse drug events, and three treatment failures. No adverse events were reported for use of combined β/α -blockers such as labetalol and carvedilol, which were effective in attenuating both hypertension and tachycardia.

Antipsychotics

Seven Level I/II studies, three Level III studies, and seven Level IV/V case series and reports involving 168 subjects have been published. Antipsychotics may improve agitation and psychosis, but with inconsistent reduction in tachycardia and hypertension and risk of extrapyramidal adverse effects.

Other agents

There was only one high level study of morphine, which reversed cocaine-induced coronary vasoconstriction but increased heart rate. Other agents reviewed included lidocaine, sodium bicarbonate, amiodarone, procainamide, propofol, intravenous lipid emulsion, propofol, and ketamine.

Conclusions

High-quality evidence for pharmacological treatment of cocaine cardiovascular toxicity is limited but can guide acute management of associated tachycardia, dysrhythmia, hypertension, and coronary vasospasm. Future randomized prospective trials are needed to evaluate new agents and further define optimal treatment of cocaine-toxic patients.

Full text available from: <http://dx.doi.org/10.3109/15563650.2016.1142090>

754 exposures to reed diffusers reported to the United Kingdom National Poisons Information Service 2010–2014

Panchal B, Eddleston M, Thomas SHL, Thompson JP, Vale JA. Clin Toxicol 2016; online early: doi: 10.3109/15563650.2016.1140772:

Objective

The objective of this study is to review the reported toxicity of reed diffuser fragrance liquid which, in addition to essential oils, commonly contains glycol ethers but other ingredients and/or alternatives are 3-methoxy-3-methyl-1-butanol, petroleum distillates, ethanol and isopropanol.

Methods

We analysed retrospectively enquiries to the United Kingdom National Poisons Information Service between 1 January 2010 and 31 December 2014.

Results

754 patients were exposed to reed diffusers; the majority (n = 712) were children < 5 years. Ingestion was the most common route of exposure (706 of 754 patients) and involved the liquid alone (n = 570), water beads alone (n = 84), sucking on the reeds (n = 31) or ingesting the liquid and water beads (n = 21). The reported amount of fragrance liquid ingested was known in only 76 of 591 cases (12.9%), with a median (IQR) volume of 20.0 (IQR = 10–40) mL. The WHO/IPCS/EC/EAPCCT Poisoning Severity Score (PSS) was known in 702 of 706 sole ingestions: in 574 (81.3%), the PSS was 0 (asymptomatic); in 117 (16.6%) patients, the PSS was 1 (minor toxicity); in 11 (1.6%), the PSS was 2 (moderate toxicity); there were no patients with features graded PSS 3 (severe toxicity). Significantly (p = 0.008) more patients became symptomatic (PSS 1 and PSS 2) following the ingestion of a reed diffuser containing 3-methoxy-3-methyl-1-butanol than propylene glycol monobutyl ether, though there was no significant difference when compared with those containing dipropylene glycol monomethyl ether (p = 0.181). The most common features following ingestion of fragrance liquid were nausea and vomiting (n = 53), coughing (n = 17) and CNS depression (n = 9). Seven patients suffered eye exposure alone: two developed eye pain and four conjunctivitis. Dermal exposure alone was reported in six patients, two of whom developed skin irritation.

Conclusions

The majority of patients in our study developed no features or only minor symptoms following ingestion of reed diffuser fragrance liquid.

Full text available from: <http://dx.doi.org/10.3109/15563650.2016.1140772>

Neurotoxicity in Russell's viper (*Daboia russelii*) envenoming in Sri Lanka: a clinical and neurophysiological study

Silva A, Maduwage K, Sedgwick M, Pilapitiya S, Weerawansa P, Dahanayaka NJ, Buckley NA, Siribaddana S, Isbister GK. Clin Toxicol 2016; online early: doi: 10.3109/15563650.2016.1143556:

Context

Russell's viper is more medically important than any other Asian snake, due to number of envenoming's and fatalities. Russell's viper populations in South India and Sri Lanka (*Daboia russelii*) cause unique neuromuscular paralysis not seen in other Russell's vipers.

Objective

To investigate the time course and severity of neuromuscular dysfunction in definite

Russell's viper bites, including antivenom response.

Methodology

We prospectively enrolled all patients (>16 years) presenting with Russell's viper bites over 14 months. Cases were confirmed by snake identification and/or enzyme immunoassay. All patients had serial neurological examinations and in some, single fibre electromyography (sfEMG) of the orbicularis oculi was performed.

Results

245 definite Russell's viper bite patients (median age: 41 years; 171 males) presented a median 2.5 h (interquartile range: 1.75-4.0 h) post-bite. All but one had local envenoming and 199 (78%) had systemic envenoming: coagulopathy in 166 (68%), neurotoxicity in 130 (53%), and oliguria in 19 (8%). Neurotoxicity was characterised by ptosis (100%), blurred vision (93%), and ophthalmoplegia (90%) with weak extraocular movements, strabismus, and diplopia. Neurotoxicity developed within 8 h post-bite in all patients. No bulbar, respiratory or limb muscle weakness occurred. Neurotoxicity was associated with bites by larger snakes ($p < 0.0001$) and higher peak serum venom concentrations ($p = 0.0025$). Antivenom immediately decreased unbound venom in blood. Of 52 patients without neurotoxicity when they received antivenom, 31 developed neurotoxicity. sfEMG in 27 patients with neurotoxicity and 23 without had slightly elevated median jitter on day 1 compared to 29 normal subjects but normalised thereafter. Neurological features resolved in 80% of patients by day 3 with ptosis and weak eye movements resolving last. No clinical or neurophysiological abnormality was detected at 6 weeks or 6 months.

Conclusion

Sri Lankan Russell's viper envenoming causes mild neuromuscular dysfunction with no long-term effects. Indian polyvalent antivenom effectively binds free venom in blood but does not reverse neurotoxicity.

Full text available from: <http://dx.doi.org/10.3109/15563650.2016.1143556>

Trends in analgesic exposures reported to Texas Poison Centers following increased regulation of hydrocodone

Haynes A, Kleinschmidt K, Forrester MB, Young A. Clin Toxicol 2016; online early: doi: 10.3109/15563650.2016.1148720:

Context

In October 2014, the Drug Enforcement Administration reclassified hydrocodone to schedule II, increasing regulations on use. The impact of rescheduling hydrocodone on opioid exposures is unclear, especially in states with special restrictions required for prescribing schedule II agents.

Objective

To assess whether changes in exposures to prescription opioid analgesics and heroin as reported to poison centers occurred in the 6 months after hydrocodone rescheduling. We hypothesized that hydrocodone exposures would decrease, while less tightly regulated opioids, such as codeine and tramadol, would increase.

Materials and methods

This study compares opioid analgesic exposures reported to Texas Poison Centers before and after this change in a state that requires special prescription pads for Schedule II agents. Cases included all opioid analgesic exposures reported to a statewide poison center network, comparing exposures from 6 months before to 6 months after heightened regulations. Specific opioids with large changes in reported exposures were further

characterized by patient age and exposure intent.

Results

Hydrocodone exposures decreased from 1567 to 1135 (28%, $p = 0.00017$), decreasing for all ages. Codeine exposures increased significantly from 189 to 522 (176%, $p = 0.00014$), including a 263% increase for age >20 years. Codeine misuse increased 443% and adverse drug events 327%. Oxycodone exposures increased from 134 to 189 (39%, $p = 0.0143$), increasing only among patients age >20 years. Reported heroin exposures increased from 156 to 179 (15%, $p = 0.2286$) and tramadol from 666 to 708 (6%, $p = 0.0193$). Other opioid exposures changed little or had limited reports.

Discussion

The increased regulation of hydrocodone was followed temporally by a decrease in reported hydrocodone exposures, but also increases in codeine, oxycodone and tramadol exposures. This may reflect a shift in prescribing practices, changes in street availability of hydrocodone or decreased drug diversion.

Conclusion

The increased regulation was temporally associated with decreased hydrocodone exposures reported to Texas Poison Centers.

Full text available from: <http://dx.doi.org/10.3109/15563650.2016.1148720>

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Systemic cobalt toxicity from total hip arthroplasties. Review of a rare condition Part 1 - history, mechanism, measurements, and pathophysiology

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Systemic cobalt toxicity from total hip arthroplasties. Review of a rare condition Part 2. Measurement, risk factors, and step-wise approach to treatment

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TOXICOLOGY

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