Current Awareness in Clinical Toxicology

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CONTENTS

CURRENT AWARENESS PAPERS OF THE MONTH

The efficacy and adverse effects of dicobalt edetate in cyanide poisoning

Marrs TC, Thompson JP. Clin Toxicol 2016; online early:
doi: 10.1080/15563650.2016.1186804:

Introduction

Dicobalt edetate is one of a number of cobalt compounds that have been studied in the treatment of cyanide poisoning, their efficacy being based upon the fact that cyanide combines with cobalt to form relatively non-toxic complexes. Inorganic cobalt salts are quite toxic (cyanide and cobalt antagonise one another's toxicity) and complexes such as dicobalt edetate were studied with the aim of identifying compounds that were less acutely toxic, but which retained the antidotal properties of cobalt salts. The proprietary preparation, Kelocyanor™, contains free cobalt and glucose as well as dicobalt edetate.

Objective

The aim of this study was to evaluate the published evidence for the efficacy and adverse effects of dicobalt edetate.

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Methods

A Pubmed search was undertaken for the period 1961–September 2015. The search terms were "dicobalt edetate", "cobalt edetate" and "Kelocyanor", which produced 24 relevant citations. A review of the references in four relevant books (*L'intoxication cyanhydrique et son traitement, Clinical and Experimental Toxicology of Cyanides, Antidotes for Poisoning by Cyanide and Antidotes*) produced three further relevant papers, making a total of 27 papers.

Efficacy of dicobalt edetate

There is evidence from animal pharmacodynamic studies that dicobalt edetate is an effective cyanide antidote in experimental animals. Some 39 cases of human poisoning treated with dicobalt edetate have been reported, but in only nine cases were blood cyanide concentrations measured, although administration of dicobalt edetate procured survival in four of the seven patients with concentrations in the lethal range (>3.0 mg/L). It is unlikely that death in any of the adequately documented fatal cases was attributable to treatment failure with dicobalt edetate, as it is probable that they all had suffered anoxic brain damage before treatment could be initiated. Furthermore, in one case, acute gold toxicity contributed substantially to death.

Adverse effects of dicobalt edetate

Adverse effects reported have included hypertension, tachycardia, nausea, retrosternal pain, sweating, palpebral, facial and laryngeal oedema, vomiting, urticaria and/or a feeling of impending doom. Such effects appear to be more prevalent where the antidote has been administered without evidence of substantial systemic poisoning or where other antidotes have been used which might have been expected also to combine with cyanide. Although the adverse effects observed were doubtless unpleasant, and some were severe, no fatal reactions were found.

Conclusions

Dicobalt edetate is an effective cyanide antidote when given to patients with systemic cyanide poisoning, but it has the potential to give rise to adverse reactions, particularly when administered in the absence of intoxication.

Full text available from: http://dx.doi.org/10.1080/15563650.2016.1186804

Inhalational mercury toxicity from artisanal gold extraction reported to the Oregon poison center, 2002–2015

Noble MJ, Decker SL, Horowitz BZ. Clin Toxicol 2016; online early: doi: 10.1080/15563650.2016.1199029:

Context

Mercury exposure has been described among small-scale gold mining communities in developing countries, but reports of inhalational mercury toxicity among home gold extractors in the US remain uncommon.

Objective

We sought to identify inhalational mercury exposures and toxicity among artisanal gold extractors.

Methods

This is an observational case series of a single Poison Center database from 2002–2015. We review all cases of "mercury" or "mercury inhalation" exposures, with detailed description of a recent representative case.

Results

Nine cases were reported, with patients' ages ranging 32–81 years. Eight (89%) patients were male. Seven of eight (88%) patients with acute exposures reported pulmonary symptoms consistent with mercury vapor inhalation such as dyspnea and cough; two (29%) patients had severe toxicity requiring intubation. Four of six (67%) patients had markedly elevated whole blood mercury concentrations up to 346 mcg/L; each received a different chelation regimen. Four (44%) patients used methamphetamines at the time of their exposure. The case report describes a patient with elevated mercury concentrations who required intubation for hypoxic respiratory failure. He received chelation therapy based on chelator availability, with decreasing 24-hour urine mercury concentrations. The house where he was exposed remains uninhabitable from elevated ambient mercury vapor concentrations.

Conclusion

Artisanal gold extraction may be associated with inhalational mercury toxicity, including elevated blood mercury concentrations and acute hypoxic lung injury requiring intubation.

Full text available from: http://dx.doi.org/10.1080/15563650.2016.1199029

Pesticide-related poison center exposures in children and adolescents aged ≤19 years in Texas, 2000–2013

Trueblood AB, Forrester MB, Han D, Shipp EM, Cizmas LH. Clin Toxicol 2016; online early: doi: 10.1080/15563650.2016.1201676:

Context

Although national poison center data show that pesticides were the 8th most commonly reported substance category (3.27%) for children aged ≤5 years in 2014, there is limited information on childhood and adolescent pesticide exposures.

Objective

This study assessed pesticide-related poison center exposures in children and adolescents aged ≤19 years from 2000–2013 in Texas to characterize the potential burden of pesticides.

Materials and methods

Pesticide-related poison center exposures among children and adolescents aged ≤19 years reported to Texas poison centers were identified. The distribution of exposures was estimated by gender, age category, medical outcome, management site, exposure route, and pesticide category.

Results

From 2000 to 2013, there were 61,147 pesticide-related poison center exposures in children and adolescents aged ≤19 years. The prevalence was highest among males at 864.24 per 100,000 population. The prevalence of unintentional exposures was highest among children aged ≤5 years at 2310.69 per 100,000 population, whereas the prevalence of intentional exposures was highest among adolescents aged 13–19 years at 13.82 per 100,000 population. A majority of medical outcomes reported were classified as having no effect (30.24%) and not followed, but minimal clinical effects possible (42.74%). Of all the exposures, 81.24% were managed on site. However, 57% of intentional exposures were referred to or treated at a health-care facility. The most common routes of exposure were ingestion (80.83%) and dermal (17.21%). The most common pesticide categories included rodenticides (30.02%), pyrethrins/pyrethroids (20.69%), and other and unspecified insecticides (18.14%).

Discussion

The study found differences in the frequency of exposures by intent for sex and age categories, and identified the most common medical outcomes, management site, exposure route, and pesticide category.

Conclusion

Through characterizing pesticide-related poison center exposures, future interventions can be designed to address groups with higher prevalence of exposure.

Full text available from: http://dx.doi.org/10.1080/15563650.2016.1201676

Absorption of salicylate powders versus tablets following overdose: a poison center observational study

Rose SR, Cumpston KL, Kim J, Difranco D, Wills BK. Clin Toxicol 2016; online early: doi: 10.1080/15563650.2016.1204549:

Background

Salicylate absorption following overdose of aspirin (ASA) tablet formulations can be prolonged for greater than 24 h. Accordingly, serial serum concentrations are typically recommended to guide treatment. However, there are little published data on absorption following ingestion of powder ASA formulations, and it is not known if delayed ASA absorption occurs following overdose of powder formulations. The objective of this study is to compare the absorption characteristics of powder and tablet formulations of ASA in patients reported to a single poison center.

Methods

Electronic records from an accredited poison center were searched for single substance acute or acute on chronic ingestions of ASA in powder form between 1 January 2002 and 31 January 2014. An identical search for ingestions of ASA tablet products between 1 January 2012 and 31 December 2013 was undertaken as the comparator group. Other inclusion criteria were age >12 years, documented time of ingestion, treatment in a health care facility within nine hours of ingestion and at least two detectable serum salicylate concentrations.

Results

16 of 25 powder and 22 of 49 tablet cases met inclusion criteria for analysis. Repeat serum salicylate concentrations following ingestion of tablets increased or insignificantly changed in 11 of 22 (50%) cases, and median serum salicylate concentrations in followed cases remained elevated for up to 12 h in some cases. In comparison, serum salicylate concentrations following powder ingestions declined in 15 of 16 (94%) cases. One patient, who ingested a powder product, underwent hemodialysis pursuant to an initial serum salicylate concentration of 96 mg/dL.

Conclusions

In contrast to persistent concentrations following overdose of tablets, the majority of serum salicylate concentrations declined following ingestion of powder formulations. In this small study population, these findings suggest that prolonged absorption is unlikely following ingestions of ASA powders.

Full text available from: http://dx.doi.org/10.1080/15563650.2016.1204549

Glutathione, glutathione-related enzymes, and oxidative stress in individuals with subacute occupational exposure to lead

Dobrakowski M, Pawlas N, Hudziec E, Kozłowska A, Mikołajczyk A, Birkner E, Kasperczyk S. Environ Toxicol Pharmacol 2016; 45: 235-40.

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Is exposure to Agent Orange a risk factor for hepatocellular cancer?—A single-center retrospective study in the U.S. veteran population

Krishnamurthy P, Hazratjee N, Opris D, Agrawal S, Markert R. J Gastrointest Oncol 2016; 7: 426-32.

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TOXICOLOGY

General

Litovitz T, Benson BE, Smolinske S.
webPOISONCONTROL: can poison control be automated?
Am J Emerg Med 2016; online early: doi: 10.1016/j.ajem.2016.06.018:

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INDEX

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