Acute toxicity profile of tolperisone in overdose: observational poison centre-based study

Introduction
Tolperisone is a centrally acting muscle relaxant that acts by blocking voltage-gated sodium and calcium channels. There is a lack of information on the clinical features of tolperisone poisoning in the literature. The aim of this study was to investigate the demographics, circumstances and clinical features of acute overdoses with tolperisone.

Methods
An observational study of acute overdoses of tolperisone, either alone or in combination with one non-steroidal anti-inflammatory drug in a dose range not expected to cause central nervous system effects, in adults and children (< 16 years), reported to our poison centre between 1995 and 2013.
Results

75 cases were included: 51 females (68%) and 24 males (32%); 45 adults (60%) and 30 children (40%). Six adults (13%) and 17 children (57%) remained asymptomatic, and mild symptoms were seen in 25 adults (56%) and 10 children (33%). There were nine adults (20%) with moderate symptoms, and five adults (11%) and three children (10%) with severe symptoms. Signs and symptoms predominantly involved the central nervous system: somnolence, coma, seizures and agitation. Furthermore, some severe cardiovascular and respiratory signs and symptoms were reported. The minimal dose for seizures and severe symptoms in adults was 1500 mg. In 11 cases the latency between the ingestion and the onset of symptoms was known and was reported to be 0.5–1.5 h.

Conclusions

The acute overdose of tolperisone may be life-threatening, with a rapid onset of severe neurological, respiratory and cardiovascular symptoms. With alternative muscle relaxants available, indications for tolperisone should be rigorously evaluated.

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Scorpion-related cardiomyopathy: clinical characteristics, pathophysiology, and treatment


Context

Scorpion envenomation is a threat to more than 2 billion people worldwide with an annual sting number exceeding one million. Acute heart failure presenting as cardiogenic shock or pulmonary edema, or both is the most severe presentation of scorpion envenomation accounting for 0.27% lethality rate.

Objective

The purpose of this review is to characterize the scorpion-related cardiomyopathy, clarify its pathophysiological mechanisms, and describe potentially useful treatments in this particular context.

Methods

We searched major databases on observational or interventional studies (whether clinical or experimental) on the cardiorespiratory consequences of scorpion envenomation and their treatment. No limit of age or language was imposed. A critical appraisal of the literature was conducted in order to provide a pathophysiological scheme that reconciles reported patterns of cardiovascular toxicity and hypotheses and assumptions made so far.

Results

Early cardiovascular dysfunction is related to the so-called "vascular phase" of scorpion envenomation, which is related to a profound catecholamine-related vasoconstriction leading to a sharp increase in left ventricular (LV) afterload, thereby impeding LV emptying, and increasing LV filling pressure. Following this vascular phase, a myocardial phase occurs, characterized by a striking alteration in LV contractility (myocardial stunning), low cardiac output, and hypotensive state. The right ventricle involvement is symmetric to that of LV with a profound and reversible alteration in right ventricular performance. This phase is unique in that it is reversible spontaneously or under inotropic treatment. Scorpion myocardiopathy combines the features of takotsubo myocardiopathy (or stress myocardiopathy) which is linked to a massive release in catecholamines leading to myocardial ischemia through coronary vasomotor abnormalities (epicardial coronary spasm
and/or increase in coronary microvascular resistance). Treatment of pulmonary edema due
to scorpion envenomation follows the same principles as those applied for the treatment of
cardiogenic pulmonary edema in general: this begins with oxygen supplementation targeting
an oxygen saturation of 92% or more, by oxygen mask, continuous positive airway
pressure, noninvasive ventilation, or conventional mechanical ventilation. Dobutamine
effectively improves hemodynamic parameters and may reduce mortality in severe scorpion
envenomation.

**Conclusion**
Scorpion cardiomyopathy is characterized by a marked and reversible alteration in
biventricular performance. Supportive treatment relying on ventilatory support and
dobutamine infusion is a bridge toward recovery in the majority of patients.

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**Comparison of lisdexamfetamine and dextroamphetamine exposures reported to U.S. poison centers**


**Context**
Lisdexamfetamine is a pro-drug stimulant that requires the enzymatic hydrolysis of lysine
from dexamphetamine for pharmacologic effects. There is limited information comparing
non-therapeutic lisdexamfetamine and dextroamphetamine exposures.

**Objective**
The objective was to compare lisdexamfetamine exposures with dextroamphetamine/
amphetamine extended release and dextroamphetamine/amphetamine immediate release.

**Methods**
A retrospective observational case series of single-substance exposures to lisdexamfetamine,
dextroamphetamine/amphetamine extended release, or dextroamphetamine/amphetamine
immediate release reported to the National Poison Data System from 2007 to 2012 was
performed. Data were analyzed for demographics, reason, clinical effects, management site,
and outcomes.

**Results**
There were 23,553 exposures: lisdexamfetamine (7,113), dextroamphetamine/amphetamine extended release (6,245), and dextroamphetamine/amphetamine immediate release (10,195). The most frequent clinical effects observed for lisdexamfetamine, dextroamphetamine/amphetamine extended release, and dextroamphetamine/amphetamine immediate release were agitation (19.8%, 21.7%, and 25.1%, respectively) and tachycardia (19.2%, 22.8%, and 23.9%, respectively). The reason was most often exploratory (93.4%) in children < 6 years and therapeutic error (65.6%) in children aged 6-12 years. In adolescents and adults most common reasons were suicide attempts (28.4%) followed by abuse (19.5%) and therapeutic errors (18.8%). Overall, 61.6% of cases were managed in a health care facility, with the majority treated in the emergency department only. The majority of cases (76.0%) experienced no or minor effects. More serious outcomes (moderate/major/death) occurred in 21.2% of lisdexamfetamine, 24.7% of dextroamphetamine/amphetamine extended release, and 25.5% of dextroamphetamine/amphetamine immediate release. There were 4 deaths (1 dextroamphetamine/amphetamine extended release and 3 dextroamphetamine/amphetamine immediate release). In patients aged 6 years and more, abuse/misuse was more frequently reported for dextro-
amphetamine/amphetamine immediate release (32.5%) and dextroamphetamine/amphetamine extended release (23.0%) than that for lisdexamfetamine (13.5%). The odds of abuse/misuse was 2.3 (95% confidence interval [CI]: 2.0-2.4) times higher for dextroamphetamine/amphetamine immediate release than that for lisdexamfetamine and dextroamphetamine/amphetamine extended release combined; the odds of dextroamphetamine/amphetamine extended release abuse/misuse was 1.9 (95% CI: 1.7-2.2) times higher than lisdexamfetamine. In 2011, the number of lisdexamfetamine abuse/misuse cases exceeded dextroamphetamine/amphetamine extended release by approximately 26% and plateaued in 2012, but was significantly lower (approximately 75%) than dextroamphetamine/amphetamine immediate release.

Conclusions
Toxic effects were similar for all three drugs. Although the majority of cases were treated at health care facilities, the majority of patients experienced no effects or minor toxicity. Serious outcomes occurred in approximately 21% of lisdexamfetamine and 25% of dextroamphetamine/amphetamine extended release and dextroamphetamine/amphetamine immediate release. Lisdexamfetamine may have less abuse potential, especially compared with the immediate-release dextroamphetamine/amphetamine formulation.

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2-Methyl-4-chlorophenoxyacetic acid and bromoxynil herbicide death

Case report
We report a fatal case of a 37 year old gentleman who ingested a MCPA/bromoxynil co-formulation herbicide. Although clinically well on initial examination, our patient declined dramatically over his 18 h admission with increasing CO₂ production, hyperthermia and metabolic derangement to eventually die from cardiac asystole 20 h post ingestion. Two hours after ingestion the MCPA concentration was 83.9 µg/mL and bromoxynil concentration was 137 µg/mL.

Discussion
The patients' mechanism of death appeared to be uncoupling of oxidative phosphorylation, excess CO₂ production and hyperthermia. There is limited knowledge on the acute toxicity of these herbicides, in particular bromoxynil, and this case highlights the relentless progression of severe toxicity in humans.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1030025

Fab fragments of ovine antibody to colchicine enhance its clearance in the rat

Context
Colchicine is an anti-inflammatory alkaloid used for the treatment of acute gout, but has a narrow therapeutic index. Colchicine overdoses are relatively rare, but have high mortality requiring rapid treatment.

**Objective**
To evaluate the ability of a newly available ovine fragment antigen-binding (Fab) antibody to colchicine (ColchiFab™) to protect rats against renal and other injury 24 h after colchicine ingestion.

**Materials and methods**
Rats were gavaged with colchicine (5 mg/kg), then 2 h later injected intraperitoneally with 5 ml of sterile saline, or Fab anti-colchicine, a newly available ovine antibody to colchicine. Samples of blood were taken at 1, 2, 5 and 24 h after gavage, and urine was collected from 5 to 24 h after gavage. Concentrations of colchicine in tissue, blood and urine were measured by liquid chromatography/mass spectrometry, concentrations of Fab anti-colchicine, urinary neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 or KIM-1 by enzyme-linked immunosorbent assay or ELISA, while concentrations of creatine kinase and creatinine (Cr) were measured enzymatically.

**Results**
Colchicine equilibrated rapidly throughout the body and increased serum creatine kinase. Fab anti-colchicine also rapidly redistributed to the blood and remained at high concentrations over 24 h. Fab anti-colchicine caused a rapid 7.1-fold increase in serum colchicine level, followed by excretion of both colchicine and Fab anti-colchicine through the urine. This was associated with the accumulation of colchicine in the kidney, a reversal of colchicine-induced diarrhoea, and increasing urinary NGAL level; from 168 ± 48 to 477 ± 255 ng/mmol Cr [mean ± standard deviation or SD].

**Discussion**
Fab anti-colchicine greatly increased the clearance of colchicine, although increasing NGAL level suggested the presence of mild kidney damage.

**Conclusion**
These data suggest clinical utility for Fab anti-colchicine in the treatment of colchicine overdose.

Full text available from: [http://dx.doi.org/10.3109/15563650.2015.1030026](http://dx.doi.org/10.3109/15563650.2015.1030026)

**Hospital outcomes and economic costs from poisoning cases in Illinois**

**Krajewski AK, Friedman LS. Clin Toxicol  2015; online early: doi: 10.3109/15563650.2015.1030677:**

**Context**
Since 2009, poisonings have been the leading cause of fatal injuries in the United States (US) and remain a continuing public health issue. Because of the varying definitions for what constitutes a poisoning case, there are inconsistencies in the annual number of cases reported among national health surveys.

**Objectives**
The main objective of this study was to describe poisonings treated in Illinois hospitals by type of exposure, as well as to detail demographic characteristics, acute outcomes, and general cost estimates for those exposed to poisoning. We also compared a broad definition for poisoning used in our analysis with the definitions used by four national health surveys in order to assess the adequacy of various definitions in capturing poisonings for surveillance.
**Material and methods**

We conducted a comprehensive analysis of outpatients and inpatients treated in Illinois hospitals in 2010 using the Illinois hospital database. Age-adjusted incidence rates were calculated.

**Results**

In Illinois, 425,491 patients were treated in hospitals for poisoning in 2010, of whom 222,339 were inpatients. The age-adjusted incidence rate was 3,189 per 100,000 persons, with an average length of stay among inpatients of 5.5 days. The cumulative hospital charges were $7.9 billion.

**Discussion and conclusion**

The definitions used in national surveys miss 60–90% of poisoning cases. Poisoning is the leading cause of fatal injuries in the U.S., but as this study shows broadening the definition for poisoning may provide a more accurate representation of the direct and indirect effects of poisoning in the US.

Full text available from: [http://dx.doi.org/10.3109/15563650.2015.1030677](http://dx.doi.org/10.3109/15563650.2015.1030677)

**Initiation of a medical toxicology consult service at a tertiary care children's hospital**


Currently, only 10% of board-certified medical toxicologists are pediatricians. Yet over half of poison center calls involve children < 6 years, poisoning continues to be a common pediatric diagnosis and bedside toxicology consultation is not common at children's hospitals. In collaboration with executive staff from Department of Pediatrics and Emergency Medicine, regional poison center, and our toxicology fellowship, we established a toxicology consulting service at our tertiary-care children's hospital. There were 139 consultations, and the service generated 13 consultations in the first month; median of 11 consultations per month thereafter (range 8–16). The service increased pediatric cases seen by the fellowship program from 30 to 94. The transition to a consult service required a culture change. Historically, call center advice was the mainstay of consulting practice and the medical staff was not accustomed to the availability of bedside medical toxicology consultations. However, after promotion of the service and full attending and fellowship coverage, consultations increased. In collaboration with toxicologists from different departments, a consultation service can be rapidly established. The service filled a clinical need that was disproportionately utilized for high acuity patients, immediately utilized by the medical staff and provided a robust pediatric population for the toxicology fellowship.

Full text available from: [http://dx.doi.org/10.3109/15563650.2015.1013196](http://dx.doi.org/10.3109/15563650.2015.1013196)

**Global incidence of rhabdomyolysis after cooked seafood consumption (Haff disease)**


**Context**

Haff disease is a syndrome of myalgia and rhabdomyolysis that occurs after consuming cooked seafood.
Objectives
(1) To identify the most common seafood vectors of Haff disease worldwide. (2) To describe and to compare the most commonly recurring clinical and laboratory manifestations of Haff disease. (3) To compare the Haff disease toxidrome with other similar toxidromes.

Methods
Internet search engines were queried with the keywords, and selected articles were stratified by reporting Old World or New World nations. Continuous variables were reported as means with standard deviations; categorical values were reported as proportions.

Results
Over 1,000 cases of Haff disease were initially described in Eastern Europe and Sweden during and following the ingestion of several species of cooked freshwater fish including burbot, pike, freshwater eel, and whitefish. More recent case reports followed consumption of cooked freshwater pomfret and boiled crayfish in China, and cooked or raw boxfish in Japan. There were 29 case reports of Haff disease in the United States with most following consumption of buffalo fish, crayfish, or Atlantic salmon.

Conclusion
The consumption of several species of cooked fish has caused Haff disease outbreaks worldwide. The bioaccumulation of a new heat-stable, fresh, and/or brackish/salt-water algal toxin in seafood, similar to palytoxin, but primarily myotoxic and not neurotoxic, is suspected for causing Haff disease.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1016165

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MANAGEMENT

General


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Antivenom


Lipid emulsion therapy


Oximes


Beta-blockers


Extracorporeal treatments

Haemodialysis


Plasma exchange

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**Monoclonal antibodies**

**Opioid maintenance therapy**


**Naloxone**


**Naltrexone**


**DRUGS General**


**Acetaminophen (see paracetamol)**

**Amfetamines and MDMA (ecstasy)**


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**Procainamide**


**Antibiotics**

**Aminoglycosides**


**Colistin**


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**Granisetron**

**Antifungal drugs**

**Voriconazole**

**Antimalarial drugs**

**Chloroquine**

**Antineoplastics**


**Bleomycin**

**Methotrexate**

**Nilotinib**

**Ponatinib**

**Sunitinib**

**Vincristine**

**Antipsychotics**


**Aripiprazole**

**Haloperidol**

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**Bufotenine**

**LSD**


Psilocin

Herbal medicines, ethnic remedies and dietary supplements


Heroin (diacetylmorphine)


Hypoglycaemics

**Glyburide**

Immunosuppressants

**Tacrolimus**

**Insulin**


Isotretinoin

Ketamine

**Lithium**


Loparanide


**Memantine**

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**Muscle relaxants**
*Tizanidine*

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**Nicotine**


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Tramadol


**Paracetamol (acetaminophen)**


**Salicylate**


**Salvia divinorum (Diviner's sage)**


**Sodium nitroprusside**


**SSRIs and SNRIs**

**Sertraline**


**Statins**


**Atorvastatin**


**Substance abuse**


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Theophylline

Tobacco

Veterinary products
Closantel

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Pollution and hazardous waste


Water pollution

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General


Acrolein

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Carbon monoxide


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Cement

Ceramics

Chlorine

Clay

Coal dust

Contrast media


**Corrosives**

**Cosmetics**

**Crotonaldehyde**

**Cyanide**

**Detergents**


**Dexmedetomidine**

**Dichloroethane**

**Diethyhexyl phthalate**
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**Dyes**

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**Toluene**


**Tricresyl phosphate**


**Welding fumes**


**METALS**

**General**

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**Aluminium**


**Arsenic**


Cadmium

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Cobalt

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Copper


Gold

Indium

Iron


Lead


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Selenium

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**Zinc**


**PESTICIDES**

**General**


**Pesticides and cancer**


**Aluminium phosphide**


**Carbamate insecticides**

**Carbofuran**


**Mancozeb**


**Fungicides**

**Cyproconazole**


**Prochloraz**


**Herbicides**


**Glyphosate**


**Insecticides**

**Fipronil**


Phenylpyrazole

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Neonicotinoids

Imidacloprid

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Organochlorine pesticides

General

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Pentachlorophenol


Organophosphorus insecticides

General

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Sarin

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Tabun

VX

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