

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Serotonin toxicity: a practical approach to diagnosis and treatment

Isbister GK, Buckley NA, Whyte IM. Med J Aust 2007; 187: 361-5.

Abstract: Excess serotonin in the central nervous system leads to a condition commonly referred to as the serotonin syndrome, but better described as a spectrum of toxicity - serotonin toxicity. Serotonin toxicity is characterised by neuromuscular excitation (clonus, hyperreflexia, myoclonus, rigidity), autonomic stimulation (hyperthermia, tachycardia, diaphoresis, tremor, flushing) and changed mental state (anxiety, agitation, confusion).

Serotonin toxicity can be: mild (serotonergic features that may or may not concern the patient); moderate (toxicity which causes significant distress and deserves treatment, but is not life-threatening); or severe (a medical emergency characterised by rapid onset of severe hyperthermia, muscle rigidity and multiple organ failure).

Diagnosis of serotonin toxicity is often made on the basis of the presence of at least three of Sternbach's 10 clinical features. However, these features have very low specificity. The Hunter Serotonin Toxicity Criteria use a smaller, more specific set of clinical features for diagnosis, including clonus, which has been found to be more specific to serotonin toxicity.

There are several drug mechanisms that cause excess serotonin, but severe serotonin toxicity only occurs with combinations of drugs acting at different sites, most commonly including a monoamine oxidase inhibitor and a serotonin reuptake inhibitor. Less severe toxicity occurs with other combinations, overdoses and even single-drug therapy in susceptible individuals.

Treatment should focus on cessation of the serotonergic medication and supportive care, Some antiserotonergic agents have been used in clinical practice, but the preferred agent, dose and indications are not well defined.

Surgical treatment in cocaine body packers and body pushers

Schaper A, Hofmann R, Bargain P, Desel H, Ebbecke M, Langer C. Int J Colorectal Dis 2007; 22: 1531-5.

Objectives: Body packers smuggle cocaine by swallowing containers filled with the drugs, whilst body pushers conceal the containers in the rectum or vagina. In a collaborative effort between the Department of General Surgery, two major airports and Poisons Centre, we performed a retrospective study to develop an algorithm for the treatment of ruptured cocaine-filled containers.

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Materials and methods: The data of all cocaine body packers and body pushers who were identified at the airports of Frankfurt and Paris from 1985 to 2002 were evaluated concerning incidence, demographics and surgical aspects.

Results: From 1985 to 2002, 312 body pushers and 4,660 body packers were identified. The sex ratio was 1:1. Sixty-four "mules" (1.4%) developed life-threatening symptoms of cocaine overdose after the rupture of a container. In 20 patients, an emergency laparotomy was performed and the containers were removed; all of these patients survived. Forty-four body packers died before surgical treatment could be performed. Only one body pusher required medical attention.

Conclusions: Cocaine overdose can be life-threatening. If the cause is the rupture of a container in a body packer, the only possible treatment is immediate laparotomy for the removal of the container.

Acetaminophen usage patterns and concentrations of glutathione and γ -glutamyl transferase in alcoholic subjects

Seifert CF, Anderson DC. *Pharmacotherapy* 2007; 27: 1473-82.

Study objectives: To determine if subjects with chronic alcoholism are predisposed to acetaminophen-induced hepatotoxicity, and to determine the contributing factors.

Design: Prospective cohort study

Setting: Community-based crisis intervention center.

Subjects: One hundred eighty-eight subjects who answered "yes" to at least one of the four questions on the CAGE (Cut down-Annoyed-Guilty-Eye-opener) questionnaire for identifying alcoholism, and 10 healthy volunteers (controls).

Intervention: A history, physical examination, urine toxicologic analysis, ethanol and drug therapy history, and venous blood samples were collected on all subjects.

Measurements and main results: Venous blood was analyzed for a liver profile, prothrombin time, and total and oxidized glutathione concentrations. A significantly higher proportion of daily drinkers were regular daily users (29.2% [45/154] vs 11.8% [4/34], $p = 0.0497$) as well as abusers (35.7% [55/154] vs 14.7% [5/34], $p = 0.0237$) of acetaminophen compared with non-daily drinkers. Alcoholic subjects with elevated γ -glutamyl transferase (GGT ≥ 51 U/L) levels had significantly lower median plasma glutathione concentrations (2.33 $\mu\text{mol/L}$, 95% confidence interval [CI] 1.74-2.69 $\mu\text{mol/L}$) compared with those of alcoholic subjects with normal GGT concentrations (5.97 $\mu\text{mol/L}$, 95% CI 4.39-7.03 $\mu\text{mol/L}$, $p < 0.0001$) and healthy volunteers (6.59 $\mu\text{mol/L}$, 95% CI 4.79-9.65 $\mu\text{mol/L}$, $p = 0.0002$). A significant inverse correlation was also noted between the GGT concentration and the plasma total glutathione concentration ($r = -0.62$, $p < 0.0001$). None of the 188 subjects met all preset criteria for hepatotoxicity.

Conclusions: Daily drinkers were more than twice as likely as non-daily drinkers to be regular daily acetaminophen users and abusers. Alcoholic subjects with elevated GGT concentrations had significantly lower plasma total glutathione concentrations, and plasma total glutathione concentrations inversely correlated with GGT concentrations. Elevated GGT concentrations may be a clinical marker of depleted glutathione in alcoholic subjects. Acetaminophen-induced hepatotoxicity appears to be uncommon in alcoholic subjects, despite the 31.9% (60/188 patients) who took doses that are potentially hepatotoxic.

What's being used to spike your drink? Alleged spiked drink cases in inner city London

Greene SL, Shiew CM, Streete P, Mustchin SJ, Hugget D, Earl B, Dargan PI. *Postgrad Med J* 2007; 83: 754-8.

Objectives: To determine the incidence and character of drink spiking in an urban population of patients within the UK presenting to an emergency department concerned they had consumed a

deliberately contaminated drink.

Study design: Prospective case series determining the presence and quantity of sedative and illicit drugs, and ethanol in biological samples (blood and urine) obtained from consenting patients >18 years of age presenting to a large inner city London emergency department alleging they had consumed a spiked drink within the previous 12 h.

Results: Biological samples were obtained from 67 (blood) and 75 (urine) of 78 study participants. 82% of participants were female, mean age 24 years. Mean time from alleged exposure to biological sampling was 5.9 h (range 1-12 h). Ethanol was detected in 89.7% of participants. Mean serum ethanol concentration was 1.65 g/l (range 0.04-3.1 g/l); 60% of participants had a serum ethanol concentration associated with significant intoxication (>1.5 g/l). Illicit drugs were detected in 12 (15%) participants; 7 denied intentional exposure (3 methylenedioxymethamphetamine, 3 cannabis, 1 gamma-hydroxybutyrate). Medicinal drugs were detected in 13 participants; only 1 exposure was unexplained (benzodiazepine). Overall illicit or medicinal drugs of unexplained origin were detected in 8 (10%) participants. Unexplained sedative drug exposure was detected in only 2 (3%) participants.

Conclusions: Use of sedative drugs to spike drinks may not be as common as reported in the mainstream media. A large number of study participants had serum ethanol concentrations associated with significant intoxication; the source (personal over-consumption or deliberate drink spiking) is unclear.

Interaction of cyanide and nitric oxide with cytochrome C oxidase: implications for acute cyanide toxicity

Leavesley HB, Li L, Prabhakaran K, Borowitz JL, Isom GE. Toxicol Sci 2008; 101: 101-11.

Abstract: Acute cyanide toxicity is attributed to inhibition of cytochrome c oxidase (CcOX), the oxygen-reducing component of mitochondrial electron transport; however, the mitochondrial action of cyanide is complex and not completely understood. State-3 oxygen consumption and CcOX activity were studied in rat N27 mesencephalic cells to examine the functional interaction of cyanide and nitric oxide (NO).

KCN produced a concentration-dependent inhibition of cellular respiration. Cyanide's median inhibitory concentration (IC₅₀) of oxygen consumption ($13.2 \pm 1.8 \mu\text{M}$) was higher than the CcOX IC₅₀ ($7.2 \pm 0.1 \mu\text{M}$). Based on respiratory threshold analysis, 60% inhibition of CcOX was necessary before oxygen consumption was decreased. Addition of high levels of exogenous NO (100 μM S-nitroso-N-acetyl-DL-penicillamine) attenuated cyanide inhibition of both respiration and CcOX. On the other hand, when endogenous NO generation was blocked by an NOS inhibitor (N ω -monomethyl-L-arginine ester), the cyanide IC₅₀ for both respiration and CcOX increased to $59.6 \pm 0.9 \mu\text{M}$ and $102 \pm 10 \mu\text{M}$, respectively, thus showing constitutive, low-level NO production enhanced cyanide inhibition. Laser scanning cytometry showed that cyanide elevated mitochondrial NO, which then was available to interact with CcOX to enhance the inhibition.

It is concluded that the rapid, potent action of cyanide is due in part to mitochondrial generation of NO, which enhances inhibition of CcOX. At low mitochondrial oxygen tensions, the cyanide-NO interaction would be increased. Also, the antidotal action of sodium nitrite is partly explained by generation of high mitochondrial levels of NO, which antagonizes the CcOX inhibition.

Acid-base interpretation can be the predictor of outcome among patients with acute organophosphate poisoning before hospitalization

Liu J-H, Chou C-Y, Liu Y-L, Liao P-Y, Lin P-W, Lin H-H, Yang Y-F. Am J Emerg Med 2008; 26: 24-30.

Abstract: Acute organophosphate (OP) poisoning causing alteration in acid-base equilibrium was

reported before. Hence, different acid-base statuses may present in patients with acute poisoning due to OP exposure. This study aims to determine the impact of acid-base interpretation in patients with acute OP poisoning before hospitalization in medical care units and to describe the pattern of mortality with different acid-base statuses.

Over a 9-year retrospective study, from July 1996 to August 2005, a total of 82 consecutive patients with acute OP poisoning were admitted to the China Medical University Hospital (Taichung, Taiwan) within 24 hours after exposure to OP and were enrolled into this study. Patients with acute OP poisoning were divided into 4 groups: without acidosis, metabolic acidosis, respiratory acidosis, and mixed acidosis. Overall survival (Kaplan-Meier curves) among groups was statistically significant ($P < 0.0001$). The mortality rate of acute OP poisoned patients with metabolic acidosis was 25%, and 75% of those patients died of cardiovascular failure. The mortality rate of acute OP poisoning with respiratory acidosis was 50%, and 50% of those patients died of respiratory failure.

Acid-base interpretation can be effective in quick diagnosis and prediction of the outcome of patients with acute OP poisoning (without acidosis < metabolic acidosis < respiratory acidosis < mixed acidosis) before hospitalization. Major causes of death are different between the respiratory acidosis and metabolic acidosis groups of patients with acute OP poisoning.

Paraquat poisonings: mechanisms of lung toxicity, clinical features, and treatment

Dinis-Oliveira RJ, Duarte JA, Sánchez-Navarro A, Remião F, Bastos ML, Carvalho F. Crit Rev Toxicol 2008; 38: 13-71.

Abstract: Paraquat dichloride (methyl viologen; PQ) is an effective and widely used herbicide that has a proven safety record when appropriately applied to eliminate weeds. However, over the last decades, there have been numerous fatalities, mainly caused by accidental or voluntary ingestion. PQ poisoning is an extremely frustrating condition to manage clinically, due to the elevated morbidity and mortality observed so far and due to the lack of effective treatments to be used in humans. PQ mainly accumulates in the lung (pulmonary concentrations can be 6 to 10 times higher than those in the plasma), where it is retained even when blood levels start to decrease. The pulmonary effects can be explained by the participation of the polyamine transport system abundantly expressed in the membrane of alveolar cells type I, II, and Clara cells. Further downstream at the toxicodynamic level, the main molecular mechanism of PQ toxicity is based on redox cycling and intracellular oxidative stress generation.

With this review we aimed to collect and describe the most pertinent and significant findings published in established scientific publications since the discovery of PQ, focusing on the most recent developments related to PQ lung toxicity and their relevance to the treatment of human poisonings. Considerable space is also dedicated to techniques for prognosis prediction, since these could allow development of rigorous clinical protocols that may produce comparable data for the evaluation of proposed therapies.

Health care facility-based decontamination of victims exposed to chemical, biological, and radiological materials

Koenig KL, Boatright CJ, Hancock JA, Denny FJ, Teeter DS, Kahn CA, Schultz CH. Am J Emerg Med 2008; 26: 71-80.

Abstract: Since the US terrorist attacks of September 11, 2001, concern regarding use of chemical, biological, or radiological weapons is heightened. Many victims of such an attack would present directly to health care facilities without first undergoing field decontamination. This article reviews basic tenets and recommendations for health care facility-based decontamination,

including regulatory concerns, types of contaminants, comprehensive decontamination procedures (including crowd control, triage, removal of contaminated garments, cleaning of body contaminants, and management of contaminated materials and equipment), and a discussion of methods to achieve preparedness.

Backyard mushroom ingestions: no gastrointestinal decontamination-no effect

Mrvos R, Swanson-Biearman B, Krenzelok EP. J Emerg Med 2007; 33: 381-3.

Abstract: Treating the unintentional "backyard" mushroom ingestion continues to be controversial. A review of pediatric "backyard" mushroom ingestions was conducted. A Regional Poison Information Center (RPIC) conducted a retrospective review of all mushroom ingestions in children younger than 6 years of age. Data were extracted from the RPIC electronic record system for the years 2000-2003. All exposures that involved "backyard" mushroom ingestions with no gastrointestinal decontamination were included. There were 322 mushroom exposures in children younger than 6 years of age reviewed. The mean age reported was 2.1 years (SD \pm 1.18). All exposures with a definitive outcome had a 24-h follow-up post-exposure to make this determination. There was no effect in 256 cases (79.5%); minor effect in 6 (1.9%); judged as nontoxic, expect no effect in 20 (6.2%); minimal clinical effects possible in 31 (9.6%); and unrelated effect in 9 (2.8%). It was concluded that "backyard" mushrooms do not present a toxicity hazard in unintentional pediatric exposures and require no gastrointestinal decontamination.

Tetrodotoxin poisoning: a clinical analysis, role of neostigmine and short-term outcome of 53 cases

Chowdhury FR, Nazmul Ahasan HA, Mamunur Rashid AKM, Al Mamun A, Khaliduzzaman SM. Singapore Med J 2007; 48: 830-3.

Introduction: Puffer fish (tetrodotoxin) poisoning is now a common form of poisoning throughout coastal countries, but its diagnosis and management are still unclear. In this paper, we aim to share our experience and to find out the toxic manifestations, lag period between ingestion of fish and development of symptoms, the short-term clinical outcome, and value of neostigmine in its management.

Methods: The study was carried out in the Department of Medicine and Paediatrics, Khulna Medical College Hospital, Khulna, Bangladesh, from May 1, 2001 to May 1, 2006. A total of 53 patients were admitted with the history of puffer fish ingestion. All the cases were clinically analysed from admission to discharge, for the following variables: Onset of symptoms in minutes after the ingestion of fish, toxic manifestations that developed after ingestion, relation of clinical outcome with approximate amount of fish ingested, and the role of neostigmine.

Results: All patients developed toxic manifestation. Important symptoms were perioral paraesthesia (38), weakness of all limbs (33), paraesthesia all over the body (34), headache (25), and difficulty in respiration. Eight patients died, probably due to respiratory muscle paralysis, of which five patients died immediately after admission. We used neostigmine in those patients who developed respiratory failure. 13 patients improved significantly (p-value is less than 0.5), while only three patients died.

Conclusion: Because of the availability and affordability of puffer fish, the occurrence of tetrodotoxin poisoning throughout coastal countries is very likely. Therefore, health personnel should have enough knowledge regarding its toxic manifestations and management.

Venomous animals

Warrell DA. Medicine 2007; 35: 659-62.

Abstract: Bites by venomous snakes cause more than 85,000 deaths and 150,000 permanent

sequelae each year, while scorpion stings kill thousands of children. These are neglected environmental diseases of the rural tropics. Most species of medically-important venomous snakes are elapids (e.g. cobras), vipers, adders or pit vipers and rattlesnakes. Clinical effects of their bites include local swelling, blistering and necrosis, shock, haemostatic disturbances, descending flaccid paralysis, rhabdomyolysis, intravascular haemolysis and acute renal failure. Any of these may prove life-threatening. Scorpion stings cause intense pain and phaeochromocytoma-like cardiovascular effects which are particularly lethal in children. Spiders bites are necrotic or neurotoxic. Hypersensitization to bee, wasp and ant venoms is a common cause of anaphylaxis but mass attacks by African killer bees can kill by direct envenoming. Aquatic venomous animals include sea snakes, stinging fish, jellyfish, corals, cone shells, blue-ringed octopuses and sea urchins. Australian box jellyfish stings are particularly deadly. The intense pain of marine stings responds to immersion of the stung part in hot, but not scalding, water. Treatment of severe envenoming by all these groups of animals requires specific antivenom, supportive treatment for cardiorespiratory and renal failure and surgical management of wounds. Animal bites and stings can be prevented by learning about local venomous fauna, wearing protective clothing, using a light after dark, and sleeping off the ground and under a bed net.

PM10 and pregnancy outcomes: a hospital-based cohort study of pregnant women in Seoul

Kim O-J, Ha E-H, Kim B-M, Seo JH, Park HS, Jung W-J, Lee B-E, Suh Y-J, Kim Y-J, Lee J-T, Kim H, Hong Y-C. *J Occup Environ Med* 2007; 49: 1394-402.

Objective: The aim of this study was to evaluate the effects of PM10 on birth outcomes using a prospective cohort of pregnant women.

Methods: The multicenter prospective study was conducted in Korea from 2001 to 2004. To estimate the effects of PM10 exposure on birth outcomes, the logistic and linear regression model and the generalized additive model for nonlinear relationships were used.

Results: Stillbirths were affected by PM10 level during the third trimesters (OR = 1.10, 95% CI = 1.02-1.14), and birth defects were influenced by the PM10 exposure during the second trimesters (OR = 1.16, 95% CI = 1.00-1.34). Intrauterine growth retardation was affected by the first trimester's PM10 exposure. On the other hand, premature birth was affected by the PM10 exposure during the third trimester, and low-birth-weight births were affected by the PM10 level during entire trimesters of pregnancy.

Conclusions: PM10 exposure during pregnancy may result in adverse birth outcomes with different critical periods.

Drug removal by plasmapheresis: an evidence-based review

Ibrahim RB, Liu C, Cronin SM, Murphy BC, Cha R, Swerdlow P, Edwards DJ. *Pharmacotherapy* 2007; 27: 1529-49.

Abstract: Contrary to the literature about drug removal during hemodialysis, data regarding drug removal during plasmapheresis are sparse. Over the last 40 years, approximately 70 publications – mostly case reports of overdoses – have described the effects of plasmapheresis on pharmaceutical agents. Important issues are drug extraction during plasma exchange with chemotherapy, as well as drug classes such as anti-infectives, anticoagulants, antiepileptics, cardiovascular agents, and immunosuppressants. Other considerations are the merits and pitfalls of the different methods used in published reports and recommendations for future pharmacokinetic studies in this field.

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CHEMICAL WARFARE, BIOLOGICAL WARFARE AND RIOT CONTROL AGENTS**Biological warfare****Anthrax**

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