

# ***Current Awareness in Clinical Toxicology***

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## ***CURRENT AWARENESS PAPERS OF THE MONTH***

### **Risk factors and mechanisms of anaphylactoid reactions to acetylcysteine in acetaminophen overdose**

**Pakravan N, Waring WS, Sharma S, Ludlam C, Megson I, Bateman DN. Clin Toxicol 2008; 46: 697-702.**

#### ***Background***

Adverse effects to *N*-acetylcysteine (NAC) are well recognized, but their etiology and incidence are unclear.

#### ***Methods***

The nature and severity of adverse effects were prospectively studied in 169 patients and potential reaction mediators studied in 22 patients.

#### ***Results***

Adverse effects were minimal in 101 (59.8%), moderate in 51 (30.2%), and severe in 17 (10.1%). Features were nausea (70.4%), vomiting (60.4%), flushing (24.9%), pruritus (20.1%), dyspnea (13.6%), chest pain (7.1%), dizziness (7.7%), fever (4.7%), wheeze and bronchospasm (7.1%), and rash and urticaria (3.6%). Serum acetaminophen concentration was lower in patients with severe adverse effects: median (IQR) 46 mg/L (0 to 101 mg/L), moderate 108 mg/L (54 to 178 mg/L), and minimal 119 mg/L (77 to 174 mg/L),  $p = 0.002$ . Family history of allergy and female gender were independent risk factors for adverse effects. Severity of adverse effects was associated with histamine release: AUC for change from baseline histamine was -6 ng/mL min (-60 to 11 ng/mL min) in the minimal group, 26 ng/mL min (3-129 ng/mL min) in the moderate group, and 49 ng/mL min (21-68 ng/mL min) in the severe group ( $p = 0.01$ ). There was no increase in tryptase and no differences between groups for NAC concentrations or hemostatic and inflammatory variables (factors II, VII, IX, X, vWF, tPA, IL6, and CRP).

#### ***Conclusion***

Severity of adverse effects correlates with the extent of histamine release. Histamine release appears independent of tryptase suggesting a non-mast cell source. Acetaminophen is protective against adverse effects of NAC, and mechanisms by which acetaminophen might lessen histamine release require further attention.

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## **Effect of delayed activated charcoal on acetaminophen concentration after simulated overdose of oxycodone and acetaminophen**

**Mullins M, Froelke BR, Rivera MRP. Clin Toxicol 2008; online early: doi: 10.1080/15563650802093681: 1-4.**

### ***Objective***

To determine the effect of activated charcoal (AC) on acetaminophen (APAP) absorption kinetics when administered at 1, 2, or 3 h after combined oral overdose with oxycodone.

### ***Method***

IRB-approved, prospective cross-over study of nine healthy human volunteers ingesting 5 g of APAP + 0.5 mg/kg of oxycodone on each of four study days. On the control day, subjects received no AC. On the remaining study days, subjects ingested 50 g of AC at 1, 2, or 3 h after drug ingestion. We measured serum APAP concentration hourly from 0 through 8 h and compared basic non-compartmental pharmacokinetic parameters.

### ***Results***

Compared to the control, AC reduced area under the curve by 43% when given at 1 h ( $p < 0.0001$ ), 22% when given at 2 h ( $p = 0.02$ ), and 15% when given at 3 h ( $p = 0.26$ ). AC at 1 h resulted in a 25% reduction in peak APAP concentration from 48.6 to 36.3  $\mu\text{g/mL}$  ( $p = 0.012$ ) with no significant difference when given at 2 or 3 h. There was no significant difference in elimination half-life among the four study days.

### ***Conclusion***

The effect of AC rapidly declines between 1 and 3 h after combined oral overdose of APAP and oxycodone. AC is unlikely to be beneficial at or beyond 2 h after an overdose of acetaminophen and oxycodone.

## **Evidence and consequences of spectrum bias in studies of criteria for liver transplant in paracetamol hepatotoxicity**

**Ding GKA, Buckley NA. QJM 2008; 101: 723-9.**

### ***Objective***

In severe paracetamol hepatotoxicity, orthotopic liver transplant (OLT) is a standard treatment in patients judged to have a hopeless prognosis. The most commonly used criteria to make this decision are the King's College Criteria (KCC). We aimed to compare the expected survival for patients who meet the KCC and do not receive transplant and those who receive OLT.

### ***Methods***

A systematic review of studies of survival in patients who met the KCC according to whether they were transplanted. Data from these studies was extrapolated to compare long-term survival with and without adjustment for Quality of Life.

### ***Results***

The survival of patients meeting KCC and undergoing transplant has not been specifically studied. UK data on transplants for acute liver failure indicate 1 and 10 year survival rates of 65 and 44%, respectively. Survival in those without transplant was documented in 15 studies. The average long-term survival rate was 24.9%. Survival was worse in studies originating in the King's unit (13.8 vs. 30.0%). It was apparent that this may be due to spectrum bias occurring in this much larger unit. There was clear evidence that those with the best prognosis were preferentially transplanted at the Kings liver unit, indicating the criteria may perform significantly worse at predicting death without transplant than previously estimated. Even so, for a 20-year-old meeting KCC, the best estimate of life expectancy with transplant (13.5 years) is no better than without

(13.4 years). Adjustment for quality of life made OLT clearly a worse option.

### **Conclusion**

Criteria for OLT that have a much higher positive predictive value (for death without transplant) are required. Such studies must be conducted only on those who would be considered suitable for transplant. Non-orthotopic liver transplant may be a preferred option in such circumstances, although much more data on survival after this procedure are required.

## **Toxicity from modafinil ingestion**

**Spiller HA, Borys D, Griffith JRK, Klein-Schwartz W, Aleguas A, Sollee D, Anderson DA, Sawyer TS. Clin Toxicol 2008; online early: doi: 10.1080/15563650802175595: 1-4.**

### **Introduction**

Modafinil, a non-amphetamine stimulant, is used for narcolepsy, sleep apnea, and shift work sleep disorder. There is little available information on the toxicity of modafinil overdose.

### **Method**

We performed a retrospective multi-poison center chart review of patients from 11 states who had a single substance ingestion of modafinil with follow up to a known outcome for the years 2000-2007. Data collected included age, gender, dose ingested, clinical effects, length of hospital stay, and medical outcome.

### **Results**

There were 137 patients, of whom 85 (63%) were female. Ages ranged from 1 to 82 years with a mean and median of 22 years ( $\pm 18$ ) and 20 years, respectively, with 43 patients (31%) aged <6 years. Most frequently reported clinical effects were tachycardia ( $n = 38$ ), insomnia ( $n = 33$ ), agitation ( $n = 27$ ), dizziness ( $n = 25$ ), and anxiety ( $n = 24$ ). Forty-five patients were managed at home and 92 in a health-care setting, with only 23 (17%) requiring a medical admission. Therapies included benzodiazepines ( $n = 14$ ), diphenhydramine ( $n = 5$ ),  $\beta$ -blockers ( $n = 3$ ), haloperidol ( $n = 2$ ), IV fluid hydration ( $n = 2$ ), and one each of nitroglycerin, epinephrine, benztropine, and promethazine.

### **Conclusions**

In this case series, clinical effects of modafinil overdoses were generally mild with predominantly tachycardia and CNS toxicity. However, clinically significant effects warranting specific therapy occurred in a minority of patients.

## **Prolonged toxicity after massive olanzapine overdose: two cases with confirmatory laboratory data**

**Tse GH, Warner MH, Waring WS. J Toxicol Sci 2008; 33: 363-5.**

### **Abstract**

Olanzapine is a second-generation atypical antipsychotic that is increasingly used in preference to older antipsychotic agents. Limited data is available concerning the toxic effects of olanzapine after deliberate overdose.

Two patients presented to our institution after massive olanzapine ingestion, and required prolonged ventilatory support due to the development of coma and respiratory depression. Serum olanzapine concentrations were orders of magnitude higher than those associated with therapeutic doses, and remained elevated for several days after ingestion. Both patients made a full recovery with only supportive care, despite having initial serum drug concentrations > 2500  $\mu\text{g/l}$ .

These reports indicate the potential for olanzapine ingestion to cause coma that may persist for several days after overdose.

## **Antenatal use of selective serotonin-reuptake inhibitors and QT interval prolongation in newborns**

**Dubnov-Raz G, Juurlink DN, Fogelman R, Merlob P, Ito S, Koren G, Finkelstein Y. *Pediatrics*, 2008; 122: e710-e715.**

### ***Objectives***

Prolongation of the QT interval is a risk factor for sudden death. Selective serotonin-reuptake inhibitor antidepressants can prolong the QT interval and are widely used by pregnant women. Whether antenatal exposure to selective serotonin-reuptake inhibitor causes QT prolongation in offspring is unknown. The aim of this study was to determine the effect of maternal use of selective serotonin-reuptake inhibitor antidepressants during pregnancy on the QTc interval of the offspring.

### ***Methods***

Between January 2000 and December 2005, we collected data on all of the newborns born at a single tertiary care hospital. Electrocardiograms of infants exposed to selective serotonin-reuptake inhibitor antidepressants in utero were compared with those of healthy control newborns matched on gestational age. The tracings were interpreted by a pediatric cardiologist who was unaware of the drug exposure.

### ***Results***

We identified 52 newborns exposed to selective serotonin-reuptake inhibitor antidepressants in the immediate antepartum period and 52 matched control subjects. The mean QTc was significantly longer in the group of newborns exposed to antidepressants as compared with control subjects ( $409 \pm 42$  vs  $392 \pm 29$  milliseconds). Five (10%) newborns exposed to selective serotonin-reuptake inhibitor antidepressants had a markedly prolonged QTc interval ( $>460$  milliseconds) compared with none of the unexposed newborns. The longest QTc interval observed among exposed newborns was 543 milliseconds. All of the drug-associated repolarization abnormalities normalized in subsequent electrocardiographic tracings.

### ***Conclusions***

Antepartum use of selective serotonin-reuptake inhibitor antidepressants is associated with QTc interval prolongation in exposed neonates. Additional research using a standardized protocol is needed to determine whether exposure to selective serotonin-reuptake inhibitor antidepressants in late pregnancy is also associated with arrhythmias.

## **Toxicity from the recreational use of 1-benzylpiperazine**

**Gee P, Gilbert M, Richardson S, Moore G, Paterson S, Graham P. *Clin Toxicol* 2008; Online early: doi:10.1080/15563650802307602: 1-6.**

### ***Aim***

This study describes the demographics and symptoms of patients, who presented to the Emergency Department (ED) in Christchurch, New Zealand, with toxicity from 1-benzylpiperazine (BZP)-based "party pills". BZP use has become widespread among the 16- to 30-year age group in New Zealand. This study explores the relationship between plasma BZP level and adverse effects experienced by users. The influence of ethanol co-ingestion was also studied.

### ***Methods***

From 1 April 2005 to 1 July 2007, all BZP-related presentations to the ED were captured on a prospective data sheet. Patients were recruited to obtain plasma BZP levels, and these were correlated with the incidence of seizures and other symptoms. Coexistent ethanol use was also correlated with the frequency of seizures and other common BZP-induced symptoms.

### ***Results***

In total 178 presentations with BZP toxicity were recorded. BZP levels were measured in 96. Sixty-nine percent of patients co-ingested other substances, with the most common substance being

ethanol. In patients who ingested BZP alone, increased plasma BZP levels were associated with increased seizure frequency. Ethanol co-ingestion reduced the incidence of seizures, but significantly increased the likelihood of confusion and agitation.

### **Conclusions**

Adverse effects from BZP commonly include confusion, agitation, vomiting, anxiety, and palpitations. There is strong evidence that higher plasma levels of BZP are associated with an increased incidence of seizures. Co-ingestion of ethanol increases the likelihood of common and distressing BZP-induced symptoms but reduces the incidence of BZP seizures.

## **Management of cocaine-induced cardiac arrhythmias due to cardiac ion channel dysfunction**

**Wood DM, Dargan PI, Hoffman RS. Clin Toxicol 2008; online early: doi: 10.1080/15563650802339373: 1-10.**

### **Abstract**

Cocaine use is common in many areas of the world, particularly the United States and Western Europe. Toxicity following the use of cocaine is associated with a wide range of clinical features.

In this review, we will focus on the cocaine-associated cardiac arrhythmias and, in particular, some of the controversies in their etiology and management. Cocaine can produce arrhythmias either through the production of myocardial ischemia or as a direct result of ion channel alterations. Excessive catecholamines, combined with sodium and potassium channel blockades, give rise to a wide variety of supra-ventricular and ventricular rhythms. The animal and human evidence for ion channel dysfunction is reviewed, and the effects of catecholamines are followed from the cardiac action potential to the development of arrhythmias. Finally, theoretical constructs are combined with existing evidence to develop a rational treatment strategy for patients with cocaine-induced cardiac arrhythmias.

In particular, we review the evidence concerning the controversies relating to the use of lidocaine in comparison with sodium bicarbonate, in terms of QRS prolongation secondary to sodium channel blockade.

## **Drug withdrawal, cocaine and sedative use disorders increase the need for mechanical ventilation in medical patients**

**De Wit M, Gennings C, Zilberberg M, Burnham EL, Moss M, Balster RL. Addiction 2008; 103: 1500-8.**

**Aims** Alcohol use disorders increase the need for mechanical ventilation (MV) in critically ill medical, surgical and trauma patients. Studies examining other drug use disorders (DUD) in trauma patients have not demonstrated heightened rates of intensive care unit (ICU) complications. Patients with asthma and concurrent cocaine or heroin use disorders have an increased need for MV. The objective of this study is to determine if the presence of DUD and drug withdrawal syndromes are associated with increased need for MV in medical patients.

**Design** Analysis of a national database.

**Setting** The Nationwide Inpatient Sample, the largest all-payer in-patient database was utilized for the years 2002-2004.

**Participants** Adult patients with one of the six common diagnoses associated with medical ICU admission were included.

**Intervention** None.

**Measurements** Univariate analysis and multivariate logistic regression were performed to determine if DUD and drug withdrawal were associated independently with the use of MV.

**Findings** A total 1 218 875 patients fulfilled one of the six diagnoses; 22 827 (1.9%) had DUD, and 102 841 (8.4%) underwent MV. Independent of the medical diagnosis, DUD was associated

with an increased risk for requiring MV by univariate analysis (relative risk = 1.50,  $P < 0.0001$ ). By multivariate analyses, sedative and cocaine use disorders remained associated with increased need for MV. Independent of medical diagnosis and substance, drug withdrawal was associated with increased odds of MV by both univariate and multivariate analysis (odds ratio = 2.94,  $P < 0.0001$ ).

**Conclusions** DUD are associated with increased need for MV in medical patients. This study demonstrates the importance of screening all medical patients for DUD.

## **Lithium poisoning: is determination of the red blood cell lithium concentration useful?**

**El Balkhi S, Mégarbane B, Poupon J, Baud FJ, Galliot-Guilley M. Clin Toxicol 2008; Online early: doi:10.1080/15563650802392398: 1-6.**

### **Introduction**

Despite a narrow therapeutic index, lithium remains a cornerstone for the treatment of bipolar disease. As lithium poisoning may result in life-threatening neurotoxicity, measurement of the lithium concentration is mandatory in drug monitoring as well as for the diagnosis and prognostic evaluation of lithium poisoning. However, toxic symptoms do not always correlate with plasma concentrations. Therefore, more reliable indicators have been proposed, including measurement of the red blood cell (RBC) lithium concentration and the RBC-to-plasma lithium ratio.

### **Plasma and RBC lithium concentrations**

Few studies have reviewed the relative utility of these measurements both in monitoring therapy and in poisoning, and they have involved only small numbers of subjects. Moreover, factors influencing plasma and RBC lithium concentrations are numerous, including gender, age, dosage, treatment duration, co-medications, and underlying diseases. In treated patients, investigated using in vivo magnetic resonance spectroscopy, there is a significant correlation between plasma and brain lithium concentrations in steady-state conditions. In contrast, lithium transport across erythrocytes markedly differs from its transport into the central nervous system, questioning the relevance of measuring the RBC lithium concentration. In poisoned patients, plasma and RBC lithium concentrations follow a parallel decline irrespective of the type of poisoning.

### **Conclusions**

Based on present evidence, measurement of the RBC lithium concentration and the calculation of the RBC-to-plasma lithium ratio offer no important clinical advantage over the measurement of the plasma lithium concentration, which remains the most important variable to monitor in lithium-treated or lithium-poisoned patients.

## **A meta-analysis of studies investigating the effects of lead exposure on nerve conduction**

**Krieg EF, Jr., Chrislip DW, Brightwell WS. Arch Toxicol 2008; 82: 531-42.**

### **Abstract**

Group means from nerve conduction studies of persons exposed to lead were used in a meta-analysis. Differences between the control and exposed groups, and the slopes between nerve conduction measurements and log<sub>10</sub> blood lead concentrations were estimated using mixed models. Conduction velocity was reduced in the median, ulnar, and radial nerves in the arm, and in the deep peroneal nerve in the leg. Distal latencies of the median, ulnar, and deep peroneal nerves were longer. No changes in the amplitudes of compound muscle or nerve action potentials were detected. The lowest concentration at which a relationship with blood lead could be detected was 33.0 µg/dl for the nerve conduction velocity of the median sensory nerve.

Lead may reduce nerve conduction velocity by acting directly on peripheral nerves or by acting indirectly, for example, on the kidney or liver.

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