

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

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***May 2008***

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

### ***Acute ethanol coingestion confers a lower risk of hepatotoxicity after deliberate acetaminophen overdose***

Waring WS, Stephen AF, Malkowska AM, Robinson ODG. Acad Emerg Med 2008; 15: 54-8.

**Objectives:** Little is known about the clinical significance of acute ethanol coingestion around the time of acetaminophen (paracetamol) overdose. This study prospectively examined the effect of acute ethanol coingestion on risk of hepatotoxicity among patients admitted to hospital for N-acetylcysteine (NAC) therapy after deliberate acetaminophen overdose.

**Methods:** This was a prospective observational study and included sequential patients who presented within 24 hours of acute acetaminophen ingestion and required NAC therapy. Significant hepatotoxicity was defined by alanine transaminase > 1,000 U/L or the international normalized ratio > 1.3 after a standardized intravenous administration of 300 mg/kg NAC.

**Results:** There were 362 patients, including 178 (49.2%) who coingested ethanol acutely. The prevalence of hepatotoxicity was 5.1% (95% CI = 2.6% to 9.5%) in those who ingested ethanol, compared to 15.2% (95% CI = 10.7% to 21.2%) in those who did not ( $p = 0.0027$  by chi-square proportional test). Acute ethanol intake conferred a lower risk of hepatotoxicity in patients who had acetaminophen concentrations above or below the "200-line" and was independent of the interval between ingestion and assessment.

**Conclusions:** Acute ethanol intake is associated with a lower risk of hepatotoxicity after acetaminophen overdose. This apparent protective effect cannot be explained solely by lower exposure to acetaminophen in this group, nor differences in the interval between ingestion and initiation of treatment. Further work is required to establish mechanisms by which ethanol might confer protection against hepatotoxicity, so as to identify novel strategies for reducing risk after acute acetaminophen ingestion.

### ***Serum urea concentration and the risk of hepatotoxicity after paracetamol overdose***

Waring WS, Stephen AFL, Robinson ODG, Dow MA, Pettie JM. QJM 2008; 101: 359-63.

**Background:** Glutathione depletion increases the incidence of toxicity after paracetamol overdose. Risk factors for toxicity, including chronic ethanol excess and malnutrition, are associated with low serum urea concentrations. Therefore, we hypothesized that low serum urea concentration might itself be predictive of hepatotoxicity in patients that present to hospital after paracetamol overdose.

**Methods:** The present study prospectively collected data from 1085 patients attending the Emergency Department after paracetamol overdose. Hepatotoxicity was predefined by prothrombin time ratio

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>1.3 or alanine transaminase  $\geq$  1000 U/l. Serum urea concentrations were considered in a stepwise multiple regression analysis that included paracetamol dose, co-ingestion of ethanol and other drugs, serum concentration, N-acetylcysteine, interval to treatment, vomiting and serum creatinine.

**Results:** Median (IQR) serum urea concentrations were 3.3 mmol/l (2.7-4.2 mmol/l) in those without risk factors, compared with 3.0 mmol/l (2.4-3.9 mmol/l) in those with chronic excess ethanol intake ( $P < 0.001$  by Mann Whitney test) and 2.5 mmol/l (1.9-2.8 mmol/l) in patients with other risk factors ( $P < 0.001$ ). Multivariate analysis found that serum urea concentrations were not independently associated with hepatotoxicity.

**Conclusion:** Low serum urea concentration is not an independent risk factor for hepatotoxicity after paracetamol overdose.

### ***Fatal toxicity of drugs used in psychiatry***

Flanagan RJ. Hum Psychopharmacol 2008; 23: 43-51.

**Abstract:** Certified deaths from fatal poisoning (accidents, suicides and open verdicts) in England and Wales have declined steadily (from 3952 in 1979 to 2565 in 2004). There was also a small annual reduction in suicides in males and in females over this period. In 2004, self-poisoning accounted for 25% of suicides and open verdicts in males ( $n = 862$ ) and 45% in females ( $n = 540$ ). Poisoning death rates per million prescriptions were about 10 times higher for tricyclic antidepressants (TCAs) than for selective serotonin reuptake inhibitors (SSRIs), England and Wales, 1993-2004. However, despite the increased prescription of SSRIs and related compounds in recent years, there has been only a slight decrease (some 10%) in the annual number of antidepressant-related poisoning deaths, in line with the reduction in suicides (all methods) over this period. Citalopram appears to have higher overdose toxicity than other SSRIs. Of newer non-SSRI antidepressants, the overdose toxicity of venlafaxine, although lower than that of TCAs, appears to be higher than that of SSRIs, with seizures, serotonin syndrome, rhabdomyolysis, renal failure and hepatic failure having been reported. Poisoning deaths involving antipsychotics either alone, or with other drugs and/or alcohol are many fewer than those involving antidepressants (713 and 5602 deaths, respectively, England and Wales, 1993-2004). Following the restriction on thioridazine usage (2000), thioridazine-associated fatal poisoning fell to zero by 2002, but this was balanced by an increase in deaths associated with atypical antipsychotics, most notably clozapine olanzapine and quetiapine. Antipsychotic-related poisoning deaths were higher in 2004 than at any time since 1993.

### ***Major congenital malformations following prenatal exposure to serotonin reuptake inhibitors and benzodiazepines using population-based health data***

Oberlander TF, Warburton W, Misri S, Riggs W, Aghajanian J, Hertzman C. Birth Defects Res B Dev Reprod Toxicol 2008; 83: 68-76.

**Background:** To determine a population-based incidence of congenital anomalies following prenatal exposure to serotonin reuptake inhibitor (SRI) antidepressants used alone and in combination with a benzodiazepines (BZ).

**Methods:** Population health data, maternal health, and prenatal prescription records were linked to neonatal records, representing all live births (British Columbia, Canada,  $N = 119,547$ ) during a 39-month period (1998-2001). The incidence and risk differences (RD) for major congenital anomalies (CA) and congenital heart disease (CHD), including ventricular and atrial septal defects (VSD, ASD), from infants of mothers treated with an SRI alone, a benzodiazepine (BZ) alone, or SRI+BZ in combination compared to outcomes no exposure.

**Results:** Risk for a CA or CHD did increase following combined SRI+BZ exposure compared with no exposure. However, using a weighted regression model, controlling for maternal illness characteristics, combination therapy risk remained significantly associated only with CHD. The risk for an ASD was higher following SRI monotherapy compared with no exposure, after adjustment for maternal covariates. Dose/day was not associated with increased risk.

**Conclusions:** Infants exposed to prenatal SRIs in combination with BZs had a higher incidence of CHD compared to no exposure, even after controlling for maternal illness characteristics. SRI monotherapy was not associated with an increased risk for major CA, but was associated with an increased incidence of ASD. Risk was not associated with first trimester medication dose/day.

### ***Management of beta-adrenergic blocker poisoning***

Anderson AC. Clin Pediatr Emerg Med 2008; 9: 4-16.

**Abstract:** Beta-adrenergic blocking agents or  $\beta$ -blockers are prescribed for the treatment of a broad array of common disorders. Their widespread use, coupled with a narrow therapeutic index, contributes to their being a significant cause of poisoning from overdose and the second most common cause of mortality from cardiovascular agents. This article provides an overview of beta-adrenergic system pathophysiology and the properties and pharmacokinetics of  $\beta$ -blockers. Also discussed are the various effects and management of  $\beta$ -blockers in overdose settings.

### ***Heroin-induced leukoencephalopathy: characterization using MRI, diffusion-weighted imaging, and MR spectroscopy***

Offiah C, Hall E. Clin Radiol 2008; 63: 146-52.

**Aim:** To describe the magnetic resonance imaging (MRI) characteristics of heroin-induced leukoencephalopathy or "chasing the dragon syndrome" and, in particular, the diffusion-weighted imaging (DWI) and MR spectroscopy (MRS) features.

**Material and methods:** Six patients with a clinical or histopathological diagnosis of heroin-induced leukoencephalopathy were identified and MRI examinations, including DWI and single-voxel MRS, reviewed.

**Results:** Cerebellar white matter was involved in all six cases demonstrating similar symmetrical distribution with sparing of the dentate nuclei. Brain stem signal change was evident in five of the six patients imaged. Supratentorial brain parenchymal involvement, as well as brain stem involvement, correlated anatomically with corticospinal tract distribution. None of the areas of signal abnormality were restricted on DWI. Of those patients subjected to MRS, the areas of parenchymal damage demonstrated reduced N-acetylaspartate, reduced choline, and elevated Lactate.

**Conclusion:** Heroin-induced Leukoencephalopathy results in characteristic and highly specific signal abnormalities on MRI, which can greatly aid diagnosis. DWI and MRS findings can be explained by known reported neuropathological descriptions in this condition and can be used to support a proposed mechanism for the benefit of current recommended drug treatment regimes.

### ***Non-invasive pulse co-oximetry screening in the emergency department identifies occult carbon monoxide toxicity***

Suner S, Partridge R, Sucov A, Valente J, Chee K, Hughes A, Jay G. J Emerg Med 2008; 34: 441-50.

**Abstract:** As carbon monoxide (CO) toxicity may present with non-specific signs and symptoms and without history of exposure, screening for CO toxicity may identify occult cases. The objective of this study was to determine whether non-invasive screening for CO exposure could be performed in all patients presenting to a high-volume urban emergency department (ED) and would identify patients with unsuspected CO toxicity. A study of adult patients, who presented to the ED for any complaint, prospectively screened for carboxyhemoglobin concentration by a pulse CO-oximeter (SpCO). ED triage staff recorded SpCO on the patient's chart at triage. Data, including SpCO and vital signs, were recorded in a database by two trained research assistants. When available, carboxyhemoglobin concentration obtained by venous blood was also included in the data set. There were 14,438 patients who presented to the ED and were entered in the study. Data from 10,856 (75%) patients receiving screening for SpCO were analyzed. Patients were 44  $\pm$  19 years old and 51% female; 32% of the patients smoked. The mean SpCO was 5.17%  $\pm$  3.78%

among smokers and  $2.90\% \pm 2.76\%$  among non-smokers. During the study period, 11 patients with presenting signs and symptoms not consistent with CO toxicity were identified through SpCO screening. Screening for CO toxicity using a non-invasive pulse CO-oximeter can be conducted even in a busy tertiary center ED and identify patients with occult CO toxicity.

### ***Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale***

Davies JOJ, Eddleston M, Buckley NA. QJM 2008; 101: 371-9.

**Background:** Organophosphorus (OP) pesticide poisoning kills around 200,000 people each year, principally due to self-poisoning in the Asia-Pacific region.

**Aim:** We wished to assess whether patients at high risk of death could be identified accurately using clinical parameters soon after hospital admission.

**Design:** We evaluated the usefulness of the International Program on Chemical Safety Poison Severity Score (IPCS PSS) and the Glasgow Coma Score (GCS) prospectively for predicting death in patients poisoned by OP pesticides.

**Methods:** Data were collected as part of a multicenter cohort study in Sri Lanka. Study doctors saw all patients on admission, collecting data on pulse, blood pressure, pupil size, need for intubation and GCS.

**Results:** Of the patients, 1365 with a history of acute OP poisoning were included. Receiver operating characteristic (ROC) curves were calculated for the IPCS PSS and GCS on admission. The IPCS PSS and GCS had similar ROC area under the curves (AUC) and best cut points as determined by Youden's index (AUC/sensitivity/specificity 0.81/0.78/0.79 for IPCS PSS  $\geq$  grade 2 and 0.84/0.79/0.79 for GCS  $\leq$  13). The predictive value varied with the pesticide ingested, being more accurate for dimethoate poisoning and less accurate for fenthion poisoning (GCS AUC 0.91 compared with 0.69).

**Conclusion:** GCS and the IPCS PSS were similarly effective at predicting outcome. Patients presenting with a GCS  $\leq$  13 need intensive monitoring and treatment. However, the identity of the organophosphate must be taken into account, since the half of all patients who died from fenthion poisoning only had mild symptoms at presentation.

### ***Polychlorinated biphenyls, organochlorine pesticides and neurodevelopment***

Korrick SA, Sagiv SK. Curr Opin Pediatr 2008; 20: 198-204.

**Purpose of review:** Although environmental levels of polychlorinated biphenyls and certain organochlorine pesticides - hexachlorobenzene, dichlorodiphenyl trichloroethane and its primary metabolite, dichlorodiphenyl dichloroethene - are generally on the decline, early- life exposures to these prevalent contaminants continue. The review will describe current understanding of the potential neurodevelopmental consequences of low-level exposures to these contaminants.

**Recent findings:** Animal models suggest that early-life exposures to polychlorinated biphenyls, dichlorodiphenyl trichloroethane/dichlorodiphenyl dichloroethene or hexachlorobenzene are associated with decreased cognitive or behavioral function in later development. Despite almost 30 years of research, however, results of human studies are inconsistent regarding the nature of the observed effects and their persistence over time. Overall, epidemiologic studies support modest associations of primarily prenatal polychlorinated biphenyl exposures with differences in neuromotor development, decrements in cognition and behavioral deficits, particularly regarding attention and impulse control. There are limited published human data regarding potential neurodevelopmental toxicities of early-life exposures to dichlorodiphenyl trichloroethane/dichlorodiphenyl dichloroethene and hexachlorobenzene.

**Summary:** Exposures to polychlorinated biphenyls, dichlorodiphenyl trichloroethane/dichlorodiphenyl dichloroethene and hexachlorobenzene are likely detrimental to neurodevelopment. Effective control of exposure is complicated by variable exposure sources and variable contaminant levels in food, particularly fish, for which it is important to balance the risk of contaminants with nutritional benefits.

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