Intoxications of the new psychoactive substance 5-(2-aminopropyl)indole (5-IT): a case series from the Swedish STRIDA project


Context
5-(2-aminopropyl)indole (5-IT) is a new psychoactive substance (NPS; "legal high" or "research chemical") structurally related to indoleamines and substituted phenethylamines and implicated in several fatalities. We describe the clinical characteristics and results of laboratory investigations of 14 analytically confirmed nonfatal cases of 5-IT intoxication within the Swedish STRIDA project.

Study design
Observational case series of consecutive patients with admitted or suspected intake of NPS presenting to hospitals in Sweden in 2012.

Patients and methods
Blood and/or urine samples were collected from intoxicated patients presenting to emergency departments and intensive care units over the country. Analysis of NPS was performed using an LC–MS/MS multi-component method. Clinical data were collected when caregivers consulted the Poisons Information Centre and also retrieved from medical records. The severity of poisoning was graded retrospectively using the Poisoning Severity Score (PSS).

Results
Eleven male and three female patients (age: 21–53 years, median: 27) tested positive for 5-IT in 2012, all cases appearing in April–July. The 5-IT concentration in serum ranged between 0.015 and 0.59 µg/mL (median: 0.22; n = 8) and in urine between 0.005 and 24.7 µg/mL (median: 5.95; n = 12). Five intoxications were indicated to be caused by 5-IT alone, whereas additional psychoactive substances were detected in the other nine.
cases. Six (43%) of fourteen cases were graded as severe (PSS 3), five (36%) as moderate (PSS 2), and three (21%) as minor (PSS 1) poisonings. In the severe cases, agitation, hallucinations, dilated pupils without light reaction, tachycardia, hypertension, hyperthermia, myoclonus, muscle rigidity, arrhythmias, seizures, rhabdomyolysis, and/or renal failure were noted.

**Conclusions**

The results demonstrated that severe clinical toxicity was commonly present in patients with analytically confirmed 5-IT exposure. The clinical features are consistent with a sympathomimetic toxidrome, and some patients also displayed symptoms associated with serotonin toxicity.

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**Barium toxicity and the role of the potassium inward rectifier current**

**Bhoelan BS, Stevering CH, van der Boog ATJ, van der Heyden MAG. Clin Toxicol 2014; 52: 584-93.**

**Introduction**

Barium is a stable divalent earth metal and highly toxic upon acute and chronic exposure. Barium is present in many products and involved in a number of industrial processes. Barium targets the potassium inward rectifier channels (IRCs) of the KCNJx gene family. Extracellular barium enters and strongly binds the potassium selectivity filter region resulting in blockade of the potassium conducting pore. IRCs are involved in numerous physiological processes of the human body and the most barium sensitive IRCs are highly expressed in all muscle types.

**Objective**

Our purpose was correlate to the clinical outcome of acute barium poisoning in man to current knowledge on IRC function.

**Methodology**

The primary literature search was performed using Medline, Scopus and Google Scholar using search terms "barium AND poisoning"; "barium AND intoxication"; "barium AND case report" and retrieved publications from 1945 through 2012. Additional case reports were retrieved based on the reference lists of the primary hits. Duplicate publications, or publications presenting identical cases were omitted. A total of 39 case reports on acute barium poisoning containing 226 human subjects were identified for review.

**Results**

BaCO$_3$ was the most frequent source and food the most frequent mode of poisoning. Patients suffered from gastrointestinal (vomiting, diarrhea), cardiovascular (arrhythmias, hypertension), neuromuscular (abnormal reflexes, paralysis), respiratory (respiratory arrest/failure) and metabolic (hypokalemia) symptoms. Severe hypokalemia (< 2.5 mM) was observed from barium serum concentrations greater than or equal to 0.0025 mM. Review of the ECG outcomes demonstrated ventricular extrasystoles, ST changes and profound U-waves to be associated strongly with poisoning. Most common treatment modalities were gastric lavage, oral sulfates, potassium i.v. and cardiorespiratory support. 27 patients (12%) died from barium poisoning.

**Conclusions**

Barium is a potent, non-specific inhibitor of the potassium IRC current and affects all types of muscle at micromolar concentrations. Gastrointestinal symptoms frequently occur early in
the course of barium poisoning. Hypokalemia resulting from an intracellular shift of potassium and the direct effect of barium at the potassium channels explain the cardiac arrhythmias and muscle weakness which commonly occur in barium poisoning. Treatment of barium poisoning is mainly supportive. Orally administered sulfate salts to form insoluble barium sulfate in the intestinal tract and potassium supplementation have potential but unproven benefit.

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**Hydrocarbon toxicity: a review**


**Context**

Clinical effects of hydrocarbon exposure have been reported since 1897. These substances are ubiquitous, and their exposures are common. The specific hydrocarbon and route of exposure will determine the clinical effect, and an understanding of this is helpful in the care of the hydrocarbon-exposed patient.

**Objective**

To complete a comprehensive review of the literature on hydrocarbon toxicity and summarize the findings.

**Methods**

Relevant literature was identified through searches of Medline (PubMed/OVID) and Cochrane Library databases (inclusive of years 1975–2013), as well as from multiple toxicology textbooks. Bibliographies of the identified articles were also reviewed. Search terms included combinations of the following: hydrocarbons, inhalants, encephalopathy, coma, cognitive deficits, inhalant abuse, huffing, sudden sniffing death, toluene, renal tubular acidosis, metabolic acidosis, arrhythmia, dermatitis, and aspiration pneumonitis. All pertinent clinical trials, observational studies, and case reports relevant to hydrocarbon exposure and published in English were reviewed. Chronic, occupational hydrocarbon toxicity was not included.

**Results**

Exposure to hydrocarbons occurs through one of the following routes: inhalation, ingestion with or without aspiration, or dermal exposure. Inhalational abuse is associated with central nervous system depression, metabolic acidosis, and arrhythmia. The exact mechanism of the CNS depression is unknown, but experimental evidence suggests effects on NMDA, dopamine, and GABA receptors. Chronic toluene inhalation causes a non-anion gap metabolic acidosis associated with hypokalemia. Halogenated hydrocarbon abuse can cause a fatal malignant arrhythmia, termed "sudden sniffing death". Individuals who regularly abuse hydrocarbons are more likely to be polysubstance users, exhibit criminal or violent behavior, and develop memory and other cognitive deficits. Heavy, long-term use results in cerebellar dysfunction, encephalopathy, weakness, and dementia. Neuroimaging may demonstrate leukoencephalopathy in these cases. Acute exposures improve with cessation of exposure. Electrolyte and fluid replacement will improve metabolic acidosis. Arrhythmias are precipitated via catecholamine surge, and beta blockers are presumed protective. Aspiration of hydrocarbons causes a potentially fatal pneumonitis. Symptoms may include cough, wheezing respiratory distress, and hypoxia. Bilateral interstitial infiltrates may be delayed for several hours after the development of pneumonitis. Treatment consists of supportive care, supplemental oxygen, and may require intubation and admission to an intensive care unit in severe cases. Unfortunately, aspiration pneumonitis remains a leading cause of poisoning mortality in children. Dermal exposure can cause dermatitis, chemical burns, and defatting injury. Oral exposure can cause local irritation as well as vomiting,
diarrhea, and abdominal pain.

**Conclusion**

Acute hydrocarbon exposure can result in a wide array of pathology, such as encephalopathy, pneumonitis, arrhythmia, acidosis, and dermatitis. Intentional inhalational and accidental ingestion exposures with aspiration lead to the greatest morbidity and mortality.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.923904](http://dx.doi.org/10.3109/15563650.2014.923904)

**Predicting acute acetaminophen hepatotoxicity with acetaminophen-aminotransferase multiplication product and the Psi parameter**


**Context**

Prediction of potential hepatotoxicity is important for individualizing therapy with N-acetylcysteine (NAC) in patients with acute acetaminophen overdose. Acetaminophen-aminotransferase multiplication product (APAP × AT) and the Psi Parameter (Psi) have been reported to be the predictors of acetaminophen hepatotoxicity.

**Objective**

To determine the validity of APAP × AT and Psi in predicting hepatotoxicity secondary to acute acetaminophen overdose.

**Materials and methods**

We retrospectively reviewed acute acetaminophen overdose cases who were treated with NAC at Siriraj Hospital, Thailand during January 2004–June 2012. The patients' ages were 12 years or more. Initial acetaminophen concentration (mg/L) and aminotransferase (IU/L) were multiplied to obtain APAP × AT. Psi were derived from initial acetaminophen concentrations (mg/L) and lag time (hours) to NAC therapy. The cut-off values for APAP × AT and Psi were 1500 mg·IU/L² and 5 mM·h, respectively. Hepatotoxicity (defined as aspartate or alanine aminotransferase (ALT) greater than 1000 IU/L) was the outcome of interest.

**Results**

A total of 255 patients were included, 32 of whom developed hepatotoxicity. APAP × AT had sensitivity, specificity, and negative likelihood ratio of 90.6%, 62.8%, and 0.2, respectively. The sensitivity of Psi, specificity, and negative likelihood ratio were 96.9%, 91.5%, and 0.0, respectively. The areas under the curve of the receiver operating characteristic (ROC) curve for APAP × AT and Psi were 0.82 and 0.96, respectively, with a statistically significant difference between the two methods (p = 0.002). APAP × AT showed higher specificity (92.5%) in patients who presented 8–24 h after the overdose.

**Discussion and conclusion**

Psi and APAP × AT are valid clinical tools in predicting hepatotoxicity secondary to acute acetaminophen overdose in adults. APAP × AT is useful in predicting a low likelihood of hepatotoxicity after standard NAC therapy among late-presenting patients.

Full text available from: [http://dx.doi.org/10.3109/15563650.2014.917180](http://dx.doi.org/10.3109/15563650.2014.917180)
Seeking a role, Psi and APAP×AT as acetaminophen risk assessment tools
Acetaminophen continues to be one of the most commonly ingested pharmaceuticals in overdose. The Rumack-Matthew nomogram has proven to be an invaluable tool for risk assessment and need for antidotal therapy following an acute overdose. There is probably no nomogram as widely or frequently used by toxicologists today. Psi and the acetaminophen aminotransferase product (APAP×AT) are other risk assessment tools introduced by Sivilotti et al. using acute acetaminophen overdoses from the Canadian Acetaminophen Overdose Study (CAOS). Although intriguing, what remains poorly defined is how Psi and APAP×AT can be incorporated into clinical practice.

Full text available from: http://dx.doi.org/10.3109/15563650.2014.917182

Seizures after single-agent overdose with pharmaceutical drugs: analysis of cases reported to a poison center

Context
Seizures during intoxications with pharmaceuticals are a well-known complication. However, only a few studies report on drugs commonly involved and calculate the seizure potential of these drugs.

Objectives
To identify the pharmaceutical drugs most commonly associated with seizures after single-agent overdose, the seizure potential of these pharmaceuticals, the age-distribution of the cases with seizures and the ingested doses.

Methods
A retrospective review of acute single-agent exposures to pharmaceuticals reported to the Swiss Toxicological Information Centre (STIC) between January 1997 and December 2010 was conducted. Exposures which resulted in at least one seizure were identified. The seizure potential of a pharmaceutical was calculated by dividing the number of cases with seizures by the number of all cases recorded with that pharmaceutical. Data were analyzed using descriptive statistics.

Results
We identified 15,441 single-agent exposures. Seizures occurred in 313 cases. The most prevalent pharmaceuticals were mefenamic acid (51 of the 313 cases), citalopram (34), trimipramine (27), venlafaxine (23), tramadol (15), diphenhydramine (14), amitriptyline (12), carbamazepine (11), maprotiline (10), and quetiapine (10). Antidepressants were involved in 136 cases. Drugs with a high seizure potential were bupropion (31.6%, seizures in 6 of 19 cases, 95% CI: 15.4-50.0%), maprotiline (17.5%, 10/57, 95% CI: 9.8-29.4%), venlafaxine (13.7%, 23/168, 95% CI: 9.3-19.7%), citalopram (13.1%, 34/259, 95% CI: 9.5-17.8%), and mefenamic acid (10.9%, 51/470, 95% CI: 8.4-14.0%). In adolescents (15-19y/o) 23.9% (95% CI: 17.6-31.7%) of the cases involving mefenamic acid resulted in seizures, but only 5.7% (95% CI: 3.3-9.7%) in adults (= 20y/o; p < 0.001). For citalopram these numbers were 22.0% (95% CI: 12.8-35.2%) and 10.9% (95% CI: 7.1-16.4%), respectively (p = 0.058). The probability of seizures with mefenamic acid, citalopram, trimipramine, and venlafaxine increased as the ingested dose increased.
Conclusions
Antidepressants were frequently associated with seizures in overdose, but other pharmaceuticals, as mefenamic acid, were also associated with seizures in a considerable number of cases. Bupropion was the pharmaceutical with the highest seizure potential even if overdose with bupropion was uncommon in our sample. Adolescents might be more susceptible to seizures after mefenamic acid overdose than adults.

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Abstract and full text available from: http://dx.doi.org/10.1097/JOM.0000000000000166

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Abstract and full text available from: http://dx.doi.org/10.1542/peds.2013-3331

Efficacy of glucarpidase (carboxypeptidase G2) in patients with acute kidney injury after high-dose methotrexate therapy
Abstract and full text available from: http://dx.doi.org/10.1002/phar.1360

Principles and operational parameters to optimize poison removal with extracorporeal treatments
Abstract and full text available from: http://dx.doi.org/10.1111/sdi.12247

**Review of the use of lipid emulsion in nonlocal anesthetic poisoning**


Abstract and full text available from: http://dx.doi.org/10.1097/PEC.0000000000000155

**Intralipid emulsion treatment as an antidote in lipophilic drug intoxications: a case series**


Abstract and full text available from: http://dx.doi.org/10.1016/j.ajem.2014.05.019

**Intralipid therapy does not improve level of consciousness in overdoses with sedating drugs: a case series**


Abstract and full text available from: http://dx.doi.org/10.1111/1742-6723.12237

**Safety of cotrimoxazole in pregnancy: a systematic review and meta-analysis**


Abstract and full text available from: http://dx.doi.org/10.1097/QAI.0000000000000211

**Atrazine and pregnancy outcomes: a systematic review of epidemiologic evidence**


Abstract and full text available from: http://dx.doi.org/10.1002/bdrb.21101

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Eye drops

Ferrous sulphate

Fluid therapy

Herbal medicines, ethnic remedies and dietary supplements


Heroin (diacetylmorphine)


Iron

Insulin

Ketamine

Levamisole

Lithium

Metformin


Mycophenolate


Immunosuppressants
Fingolimod

Insulin

Iron

Levamisole

Lithium

**Mephedrone**


**Metaxalone**

**Methytryptamine**

**Nicotine**


**NSAIDs**


**Clopidogrel**

**Ibuprofen**

**Opioids**


**Fentanyl**

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