

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

The effects of paracetamol (acetaminophen) on hepatic tests in patients who chronically abuse alcohol - a randomised study

Dart RC, Green JL, Kuffner EK, Heard K, Sproule B, Brands B. *Aliment Pharmacol Ther* 2010; 32: 478-86

Background

Retrospective accounts suggest therapeutic doses of paracetamol can produce severe hepatic injury in patients with putative high risk conditions, including alcoholism and infectious hepatitis. Metabolism of paracetamol to its hepatotoxic metabolite is enhanced in patients who abuse alcohol, who also have compromised liver defences from depressed hepatic glutathione.

Aim

To determine the effect of paracetamol on serum liver tests of newly abstinent subjects that abuse alcohol, including subjects with hepatitis C infection.

Methods

A randomised, double-blind, placebo-controlled study. Adult alcohol abusers with a current drinking episode longer than 7 days received either placebo or paracetamol 4 g/day for five days.

Results

Of 142 subjects enrolled, 74 received paracetamol and 68 received placebo. Mean ALT activity during treatment increased from 48 to 62 IU/L in the paracetamol group and 47 to 49 IU/L in the placebo group. Maximum ALT was 238 and 249 IU/L in the paracetamol and control groups, respectively. The INR remained unchanged and serum bilirubin decreased in both groups. Subgroup analyses for subjects with alcoholic hepatitis, hepatitis C virus antibody, and other subgroups showed no statistical difference between groups.

Conclusion

Administration of paracetamol 4 grams/day appears safe in newly abstinent patients who abuse alcohol.

A multicenter comparison of the safety of oral versus intravenous acetylcysteine for treatment of acetaminophen overdose

Heard K. *Clin Toxicol* 2010; 48: 424-30.

Abstract

Oral and intravenous (IV) N-acetylcysteine (NAC) are used for the treatment of acetaminophen

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poisoning. The objective of this multicenter study was to compare the safety of these two routes of administration.

Methods

We conducted a multicenter chart review of all patients treated with NAC for acetaminophen poisoning. The primary safety outcome was the percentage of patients with NAC-related adverse events.

Results

A total of 503 subjects were included in the safety analysis (306 IV-only, 145 oral-only, and 52 both routes). There were no serious adverse events related to NAC for either route. Nausea and vomiting were the most common related adverse events and were more common with oral treatment (23 vs. 9%). Anaphylactoid reactions were more common with IV administration (6 vs. 2%).

Conclusions

IV and oral NAC are generally mild adverse drug reactions.

Long-term outcome following liver transplantation for paracetamol overdose

Khan LR, Oniscu GC, Powell JJ. *Transpl Int* 2010; 23: 524-9.

Abstract

Paracetamol overdose (POD) is a major cause of acute liver failure (ALF) requiring liver transplantation in the United Kingdom. To characterize the early and late outcome after orthotopic liver transplantation (OLT) for POD in the Scottish Liver Transplant Unit over a 14-year period (1992-2006) data were obtained from a prospective database combined with case-note review.

Of 127 liver transplants performed for ALF, 44 were for POD. The median age was 30 (range 18-51). In 18 patients (63.7%), POD was associated with alcohol/other drugs, nine (20.5%) had a staggered overdose and four patients (9.1%) accidentally overdosed. Nineteen patients (43.2%) had a history of previous overdose/psychiatric illness. Post-transplant mortality during the index admission was 30% (13 patients), whilst five patients died during follow-up. The actuarial 5-year patient survival was 54.5%, whilst graft survival was 49.5%. Some 23% of the patients were re-transplanted: primary nonfunction (1), hepatic artery thrombosis (3) and chronic rejection (2). Three patients had a subsequent transplant; three patients had two further transplants. Nine patients (35%) continue to have social/psychiatric issues.

OLT for POD is associated with significant early and late morbidity and mortality. A multidisciplinary approach is required to identify the suitable candidates, in whom transplantation should be pursued promptly.

Colchicine poisoning: the dark side of an ancient drug

Finkelstein Y, Aks SE, Hutson JR, Juurlink DN, Nguyen P, Dubnov-Raz G, Pollak U, Koren G, Bentur Y. *Clin Toxicol* 2010; 48: 407-14.

Introduction

Colchicine is used mainly for the treatment and prevention of gout and for familial Mediterranean fever (FMF). It has a narrow therapeutic index, with no clear-cut distinction between nontoxic, toxic, and lethal doses, causing substantial confusion among clinicians. Although colchicine poisoning is sometimes intentional, unintentional toxicity is common and often associated with a poor outcome.

Methods

We performed a systematic review by searching OVID MEDLINE between 1966 and January 2010. The search strategy included "colchicine" and "poisoning" or "overdose" or "toxicity" or "intoxication."

Toxicokinetics

Colchicine is readily absorbed after oral administration, but undergoes extensive first-pass metabolism. It is widely distributed and binds to intracellular elements. Colchicine is primarily

metabolized by the liver, undergoes significant enterohepatic re-circulation, and is also excreted by the kidneys.

Therapeutic and toxic doses

The usual adult oral doses for FMF is 1.2-2.4 mg/day; in acute gout 1.2 mg/day and for gout prophylaxis 0.5-0.6 mg/day three to four times a week. High fatality rate was reported after acute ingestions exceeding 0.5 mg/kg. The lowest reported lethal doses of oral colchicine are 7-26 mg.

Drug interactions

CYP 3A4 and P-glycoprotein inhibitors, such as clarithromycin, erythromycin, ketoconazole, ciclosporin, and natural grapefruit juice can increase colchicine concentrations. Co-administration with statins may increase the risk of myopathy.

Mechanisms of toxicity

Colchicine's toxicity is an extension of its mechanism of action - binding to tubulin and disrupting the microtubular network. As a result, affected cells experience impaired protein assembly, decreased endocytosis and exocytosis, altered cell morphology, decreased cellular motility, arrest of mitosis, and interrupted cardiac myocyte conduction and contractility. The culmination of these mechanisms leads to multi-organ dysfunction and failure.

Reproductive toxicology and lactation

Colchicine was not shown to adversely affect reproductive potential in males or females. It crosses the placenta but there is no evidence of fetal toxicity. Colchicine is excreted into breast milk and considered compatible with lactation.

Clinical features

Colchicine poisoning presents in three sequential and usually overlapping phases: 1) 10-24 h after ingestion - gastrointestinal phase mimicking gastroenteritis may be absent after intravenous administration; 2) 24 h to 7 days after ingestion - multi-organ dysfunction. Death results from rapidly progressive multi-organ failure and sepsis. Delayed presentation, pre-existing renal or liver impairment are associated with poor prognosis. 3) Recovery typically occurs within a few weeks of ingestion, and is generally a complete recovery barring complications of the acute illness.

Diagnosis

History of ingestion of tablets, parenteral administration, or consumption of colchicine-containing plants suggest the diagnosis. Colchicine poisoning should be suspected in patients with access to the drug and the typical toxidrome (gastroenteritis, hypotension, lactic acidosis, and prerenal azotemia).

Management

Timely gastrointestinal decontamination should be considered with activated charcoal, and very large, recent (<60 min) ingestions may warrant gastric lavage. Supportive treatments including administration of granulocyte colony-stimulating factor are the mainstay of treatment. Although a specific experimental treatment (Fab fragment antibodies) for colchicine poisoning has been used, it is not commercially available.

Conclusion

Although colchicine poisoning is relatively uncommon, it is imperative to recognize its features as it is associated with a high mortality rate when missed.

Comparison of toxicity of acute overdoses with citalopram and escitalopram

Hayes BD, Klein-Schwartz W, Clark RF, Muller AA, Miloradovich JE. J Emerg Med 2010; 39: 44-8.

Background

Seizures and QTc prolongation are associated with citalopram poisoning; however, overdose experience with escitalopram is more limited.

Objectives

The goals of this study were to compare citalopram's vs. escitalopram's clinical effects in overdose,

including the incidence of seizures.

Methods

A retrospective review was conducted for single-substance acute overdoses with citalopram and escitalopram, managed in hospitals, that were reported to six U.S. poison centers from 2002-2005.

Results

There were 374 citalopram and 421 escitalopram overdose cases. Gender and ages were similar between the two, with 68-70% females and a median age of 20 years for citalopram and 18 years for escitalopram. Median dose by history was 310 mg for citalopram and 130 mg for escitalopram. More serious outcomes were associated with citalopram overdoses ($p < 0.001$). Most frequently reported clinical effects with citalopram and escitalopram were tachycardia, drowsiness, hypertension, and vomiting. Seizures (30 vs. 1, respectively, $p < 0.001$) and tremor (32 vs. 13, respectively, $p = 0.001$) were more common with citalopram. QTc prolongation occurred in 14 citalopram cases and 7 escitalopram cases ($p = 0.109$). There was an association between increasing dose and severity of outcome for citalopram ($p < 0.001$) and escitalopram ($p = 0.011$). In children < 6 years old, 12 of 66 citalopram and 5 of 57 escitalopram cases experienced toxicity, such as drowsiness, nausea/vomiting, and tachycardia. There were no seizures in this age group.

Conclusions

Escitalopram seems to be less toxic than citalopram after an acute overdose; seizures and tremors were more common with citalopram. Initial management of overdoses should include seizure precautions for citalopram and cardiac monitoring for both drugs.

β -Blockers for chest pain associated with recent cocaine use.

Rangel C, Shu RG, Lazar LD, Vittinghoff E, Hsue PY, Marcus GM. Arch Intern Med 2010; 170: 874-9.

Background

Although β -blockers prevent adverse events after myocardial infarction, they are contraindicated when chest pain is associated with recent cocaine use. Recommendations against this use of β -blockers are based on animal studies, small human experiments, and anecdote. We sought to test the hypothesis that β -blockers are safe in this setting.

Methods

We performed a retrospective cohort study of consecutive patients admitted to the San Francisco General Hospital, San Francisco, California, with chest pain and urine toxicologic test results positive for cocaine, from January 2001 to December 2006. Mortality data were collected from the National Death Index.

Results

Of 331 patients with chest pain in the setting of recent cocaine use, 151 (46%) received a β -blocker in the emergency department. There were no meaningful differences in electrocardiographic changes, troponin levels, length of stay, use of vasopressor agents, intubation, ventricular tachycardia or ventricular fibrillation, or death between those who did and did not receive a β -blocker. After adjusting for potential confounders, systolic blood pressure significantly decreased a mean 8.6 mm Hg (95% confidence interval, 14.7-2.5 mm Hg) in those receiving a β -blocker in the emergency department compared with those who received their first β -blocker in the hospital ward ($P = 0.006$). Over a median follow-up of 972 days (interquartile range, 555-1490 days), after adjusting for potential confounders, patients discharged on a β -blocker regimen exhibited a significant reduction in cardiovascular death (hazard ratio, 0.29; 95% confidence interval, 0.09-0.98) ($P = 0.047$).

Conclusion

β -Blockers do not appear to be associated with adverse events in patients with chest pain with recent cocaine use.

Pharmacological approaches to methamphetamine dependence: a focused review

Karila L, Weinstein A, Aubin H-J, Benyamina A, Reynaud M, Batki SL. Br J Clin Pharmacol 2010; 69: 578-92.

Abstract

Methamphetamine dependence is a serious worldwide public health problem with major medical, psychiatric, socioeconomic and legal consequences. Various neuronal mechanisms implicated in methamphetamine dependence have suggested several pharmacological approaches.

A literature search from a range of electronic databases (PubMed, EMBASE, PsycInfo, the NIDA research monograph index and the reference list of clinicaltrials.gov) was conducted for the period from January 1985 to October 2009. There were no restrictions on the identification or inclusion of studies in terms of publication status, language and design type.

A variety of medications have failed to show efficacy in clinical trials, including a dopamine partial agonist (aripiprazole), GABAergic agents (gabapentin) and serotonergic agents (SSRI, ondansetron, mirtazapine). Three double-blind placebo-controlled trials using modafinil, bupropion and naltrexone have shown positive results in reducing amphetamine or methamphetamine use. Two studies employing agonist replacement medications, one with d-amphetamine and the other with methylphenidate, have also shown promise.

Despite the lack of success in most studies to date, increasing efforts are being made to develop medications for the treatment of methamphetamine dependence and several promising agents are targets of further research.

The role of clinical toxicologists and poison control centers in public health

Sutter ME, Bronstein AC, Heard SE, Barthold CL, Lando J, Lewis LS, Schier JG. Am J Prev Med 2010; 38: 658-62.

Background

Poison control centers and clinical toxicologists serve many roles within public health; however, the degree to which these entities collaborate is unknown.

Purpose

The objective of this survey was to identify successful collaborations of public health agencies with clinical toxicologists and poison control centers. Four areas including outbreak identification, syndromic surveillance, terrorism preparedness, and daily public health responsibilities amenable to poison control center resources were assessed.

Methods

An online survey was sent to the directors of poison control centers, state epidemiologists, and the most senior public health official in each state and selected major metropolitan areas. This survey focused on three areas: service, structure within the local or state public health system, and remuneration. Questions regarding remuneration and poison control center location within the public health structure were asked to assess if these were critical factors of successful collaborations. Senior state and local public health officials were excluded because of a low response rate. The survey was completed in October 2007.

Results

A total of 111 respondents, 61 poison control centers and 50 state epidemiologists, were eligible for the survey. Sixty-nine (62%) of the 111 respondents, completed and returned the survey. Thirty-three (54%) of the 61 poison control centers responded, and 36 of the 50 state epidemiologists (72%) responded. The most frequent collaborations were terrorism preparedness and epidemic illness reporting. Additional collaborations also exist. Important collaborations exist outside of remuneration or poison control centers being a formal part of the public health structure.

Conclusions

Poison control centers have expanded their efforts to include outbreak identification, syndromic surveillance, terrorism preparedness, and daily public health responsibilities amenable to poison control center resources. Collaboration in these areas and others should be expanded.

Six years of epinephrine digital injections: absence of significant local or systemic effects

Muck AE, Bebarta VS, Borys DJ, Morgan DL. *Ann Emerg Med* 2010; online early: doi: 10.1016/j.annemergmed.2010.02.019: 1-5.

Study objective

Epinephrine autoinjectors are known to result in accidental digital injections. Treatment recommendations and adverse outcomes are based on case reports. The objective of our study is to determine the frequency of digit ischemia after epinephrine autoinjector digital injections. In addition, we describe the frequency of epinephrine digital injections, treatments used, adverse local effects, and systemic effects.

Methods

We performed a retrospective cohort study on cases reported to 6 poison centers during 6 years, using a search of the Texas Poison Center Network database. Patients who had an epinephrine injection of the hand were reviewed, and digital injections were included. Variables collected included demographics, local and systemic effects, symptom duration, treatments used, comorbidities, and whether admission, surgery, or hand surgery consultation was used. One trained abstractor used a standard electronic data collection form.

Results

There were 365 epinephrine injections to the hand identified for the 6-year period. Of these, 213 were digital injections, and 127 had follow-up. All patients had complete resolution of symptoms. None of the patients were hospitalized or received hand surgery consultation or surgical care. Significant systemic effects were not reported. Pharmacologic vasodilatory treatment was used in 23% (29/127) of patients. Ischemic effects were documented for 4 patients, and 2 of these had symptom resolution within 2 hours. All 4 patients received vasodilatory therapy and were discharged home, with complete resolution of symptoms.

Conclusion

In our series of patients using poison center calls about digital epinephrine autoinjections, there were no cases in which clinically apparent systemic effects were recorded and few patients had ischemia. No patient was admitted or had surgery. Most clinicians did not use vasodilation medications or techniques.

Fomepizole for the treatment of pediatric ethylene and diethylene glycol, butoxyethanol, and methanol poisonings

Brent J. *Clin Toxicol* 2010; 48: 401-6.

Introduction

The use and clinical efficacy of the alcohol dehydrogenase inhibitor fomepizole is well established for the treatment of ethylene glycol and methanol poisonings in adults.

Methods

A computerized search of the U.S. National Academy of medicine and EMBase databases was undertaken to identify published cases of patients treated with fomepizole. This search strategy identified 14 published cases related to the topic of this review: 10 due to ethylene glycol poisoning, 1 due to diethylene glycol poisoning, 1 due to butoxyethanol ingestion, and 2 due to methanol poisoning. The median age of these cases was 5.5 years old.

Fomepizole in glycol and glycol ether poisoning

For the 10 ethylene glycol poisoned patients, the median recorded values of their arterial pH was 7.27 (range 7.03-7.38), serum bicarbonate concentration was 13 mEq/L (range 2-25), and

ethylene glycol concentration was 2,140 mg/L (range 130-3,840). Eight of these patients were not hemodialyzed. The eight patients who were not hemodialyzed had ethylene glycol concentrations as high as 3,500 mg/L and serum bicarbonate concentrations as low as 4 mEq/L. All 10 patients had resolution of their metabolic acidosis and recovered without sequelae. The half-times of ethylene glycol elimination ranged from 9 to 15 h during fomepizole therapy, which is faster than the 19.7 h reported in adults. The two patients who ingested diethylene glycol or butoxyethanol all recovered without sequelae. The patient who ingested the butoxyethanol had a serum bicarbonate concentration of 13 mEq/L and was not hemodialyzed.

Fomepizole in methanol poisoning

One of the two children who ingested methanol was hemodialyzed. Both cases had a similar degree of severity.

Does fomepizole obviate the need for hemodialysis?

Based on the experience reviewed herein it appears that, as in adults, hemodialysis may not be necessary in most cases of pediatric ethylene glycol poisoning if treated with fomepizole.

Fomepizole pharmacokinetics

Plasma fomepizole concentrations were measured in three cases and were found to be therapeutic with apparent Michaelis-Menton kinetics, having a zero-order elimination rate of 0.6-1 mg/L/h at higher concentrations and a first-order elimination with an apparent elimination half-time of 3.9 h at lower concentrations.

Fomepizole regimen

Most cases used the current U.S.-approved regimen.

Adverse effects of fomepizole

The one adverse effect reported during fomepizole therapy was transient nystagmus in a 6-year-old with a serum ethylene glycol concentration of 130 mg/L and a serum bicarbonate concentration of 2 mEq/L; it is likely that ethylene glycol itself was the cause. Comparison of fomepizole with ethanol therapy. Two cases were originally treated with ethanol but switched to fomepizole because of adverse effects. In both cases, the adverse reactions to ethanol resolved once fomepizole treatment was initiated.

Conclusions

The limited data available suggest that fomepizole, using the same dosage regimen as that used for adults, is efficacious and well tolerated in pediatric patients. In many cases of pediatric ethylene glycol poisoning treated with fomepizole, hemodialysis may not be necessary despite high concentrations and the presence of metabolic acidosis.

Glasgow Coma Scale and laboratory markers are superior to COHb in predicting CO intoxication severity

Grieb G, Simons D, Schmitz L, Piatkowski A, Grottke O, Pallua N. Burns 2010; online early: PM:20434271:

Abstract

Carbon monoxide (CO) intoxications can affect several organ systems and lead to coma or death in severe cases. To date, COHb is routinely used as a marker for detecting CO intoxication.

In this retrospective study, we investigated 173 patients admitted with CO intoxication to our intensive care unit (ICU) over a period of 8 years. Standardised blood tests, chest X-ray and neurological status evaluation were performed on admission and throughout the inpatient treatment. The duration of inpatient treatment was considered to be an indication of the severity of CO-related illness.

Interestingly, the data did not reveal a significant correlation between initial COHb level and the duration of inpatient treatment. Instead, a significant inverse correlation was found between the initial Glasgow Coma Scale and the duration of inpatient treatment. Furthermore, significant correlations were found between the duration of inpatient treatment and the occurrence of

elevated leucocyte numbers, elevated C-reactive protein (CRP) serum concentrations and the presence of lung infiltrates.

In conclusion, we postulate that clinical parameters, such as the Glasgow Coma Scale and the laboratory markers CRP and leucocyte count are adequate supportive tools for evaluating the severity of CO-related illness, and further, that the measurement of COHb alone is insufficient for this purpose.

Seven years of cyanide ingestions in the USA: critically ill patients are common, but antidote use is not

Bebarta VS, Pitotti RL, Borys DJ, Morgan DL. Emerg Med J 2010; online early: doi: 10.1136/emj2009.089896:

Background

Cyanide is a common toxin in structural fires and a salt that is ingested for suicide. However, most studies have focused on the effects of inhaled cyanide. The objective of this study was to describe the incidence of cyanide ingestions, symptoms, cardiac arrest and antidotal therapy used as reported to all US poison centres over 7 years.

Methods

A retrospective review of cases over 7 years as reported to 61 poison centres in the USA was performed. Sole ingestions of cyanide were identified. A trained reviewer used a standard data collection sheet within a secured electronic database. Age, intent, clinical effects, treatments, antidotes and outcomes were recorded. One investigator audited a random sample of charts.

Results

Out of 1741 exposures, 435 ingestions were identified. Most were male (68%) and the mean age was 34 years (range 1 month-83 years). 45% of cases were intentional, most commonly as a suicide attempt. 8.3% of cases died and 9% (38/435) of patients had cardiac arrest or hypotension. 13% of all cases and 26% of cases arriving at a healthcare facility received an antidote. In 35% of cases of cardiac arrest or hypotension, and in 74% of intentional ingestions, antidotes were not given.

Conclusions

Suicide attempt was the most common reason for cyanide ingestion. Most of these patients died. Cardiac arrest or hypotension was common, but antidote use was not, particularly in critically ill patients. Research is needed to improve outcomes of cyanide-induced hypotension and cardiac arrest and to reduce barriers to antidote use.

Preventing battery ingestions: an analysis of 8648 cases

Litovitz T, Whitaker N, Clark L. Pediatrics 2010; 125: 1178-83.

Objectives

Outcomes of pediatric button battery ingestions have worsened substantially, predominantly related to the emergence of the 20 mm diameter lithium cell as a common power source for household products. Button batteries lodged in the esophagus can cause severe tissue damage in just 2 hours, with delayed complications such as esophageal perforation, tracheoesophageal fistulas, exsanguination after fistulization into a major blood vessel, esophageal strictures, and vocal cord paralysis. Thirteen deaths have been reported. The objective of this study was to explore button battery ingestion scenarios to formulate prevention strategies.

Methods

A total of 8648 battery ingestions that were reported to the National Battery Ingestion Hotline were analyzed.

Results

Batteries that were ingested by children who were younger than 6 years were most often obtained directly from a product (61.8%), were loose (29.8%), or were obtained from battery packaging

(8.2%). Of young children who ingested the most hazardous battery, the 20 mm lithium cell, 37.3% were intended for remote controls. Adults most often ingested batteries that were sitting out, loose, or discarded (80.8%); obtained directly from a product (4.2%); obtained from battery packaging (3.0%); or swallowed within a hearing aid (12.1%). Batteries that were intended for hearing aids were implicated in 36.3% of ingestions. Batteries were mistaken for pills in 15.5% of ingestions, mostly by older adults.

Conclusions

Parents and child care providers should be taught to prevent battery ingestions. Because 61.8% of batteries that were ingested by children were obtained from products, manufacturers should redesign household products to secure the battery compartment, possibly requiring a tool to open it.

Emerging battery-ingestion hazard: clinical implications

Litovitz T, Whitaker N, Clark L, White NC, Marsolek M. Pediatrics 2010; 125: 1168-77.

Objectives

Recent cases suggest that severe and fatal button battery ingestions are increasing and current treatment may be inadequate. The objective of this study was to identify battery ingestion outcome predictors and trends, define the urgency of intervention, and refine treatment guidelines.

Methods

Data were analyzed from 3 sources: (1) National Poison Data System (56535 cases, 1985-2009); (2) National Battery Ingestion Hotline (8648 cases, July 1990-September 2008); and (3) medical literature and National Battery Ingestion Hotline cases (13 deaths and 73 major outcomes) involving esophageal or airway button battery lodgment.

Results

All 3 data sets signal worsening outcomes, with a 6.7-fold increase in the percentage of button battery ingestions with major or fatal outcomes from 1985 to 2009 (National Poison Data System). Ingestions of 20-25 mm diameter cells increased from 1% to 18% of ingested button batteries (1990-2008), paralleling the rise in lithium-cell ingestions (1.3% to 24%). Outcomes were significantly worse for large-diameter lithium cells (≥ 20 mm) and children who were younger than 4 years. The 20 mm lithium cell was implicated in most severe outcomes. Severe burns with sequelae occurred in just 2-2.5 h. Most fatal (92%) or major outcome (56%) ingestions were not witnessed. At least 27% of major outcome and 54% of fatal cases were misdiagnosed, usually because of nonspecific presentations. Injuries extended after removal, with unanticipated and delayed esophageal perforations, tracheoesophageal fistulas, fistulization into major vessels, and massive hemorrhage.

Conclusions

Revised treatment guidelines promote expedited removal from the esophagus, increase vigilance for delayed complications, and identify patients who require urgent radiographs.

Assessing public health risk in the London polonium-210 incident, 2006

Maguire H, Fraser G, Croft J, Bailey M, Tattersall P, Morrey M, Turbitt D, Ruggles R, Bishop L, Giraudon I, Walsh B, Evans B, Morgan O, Clark M, Lightfoot N, Gilmour R, Gross R, Cox R, Troop P. Public Health 2010; 124:313-8.

Objectives

Mr Alexander Litvinenko died in a London hospital on 23 November 2006, allegedly from poisoning with the radionuclide polonium-210 (^{210}Po). Associated circumstances required an integrated response to investigate the potential risk of internal contamination for individuals exposed to contaminated environments.

Study design

Descriptive study.

Methods

Contaminated locations presenting a potential risk to health were identified through environmental assessment by radiation protection specialists. Individuals connected with these locations were identified and assessed for internal contamination with ^{210}Po .

Results

In total, 1029 UK residents were identified, associated with the 11 most contaminated locations. Of these, 974 were personally interviewed and 787 were offered urine tests for ^{210}Po excretion. Overall, 139 individuals (18%) showed evidence of probable internal contamination with ^{210}Po arising from the incident, but only 53 (7%) had assessed radiation doses of 1 mSv or more. The highest assessed radiation dose was approximately 100 mSv.

Conclusions

Although internal contamination with ^{210}Po was relatively frequent and was most extensive among individuals associated with locations judged a priori to pose the greatest risk, a high degree of assurance could be given to UK and international communities that the level of health risk from exposure to the radionuclide in this incident was low.

Pharmacokinetics of obidoxime in patients poisoned with organophosphorus compounds

Thiermann H, Eyer F, Felgenhauer N, Pfab R, Zilker T, Eyer P, Worek F. Toxicol Lett 2010; online early: doi:10.1016/j.toxlet.2010.06.005:

Objectives

Reactivation of inhibited acetylcholinesterase (AChE) with oximes is a causal therapy of intoxication with organophosphorus compounds (OPs). Maximal oxime effects are expected when effective doses are administered as soon as possible and as long as reactivation can be anticipated. An obidoxime plasma level in the range of 10-20 μM was estimated as appropriate. The achievement of this target was assessed in 34 severely OP-poisoned patients.

Methods

After admission to the ICU the obidoxime regimen (250 mg i.v. as bolus, followed by 750 mg/24 h) was started and maintained as long as reactivation was possible. Plasma concentrations of obidoxime were determined by HPLC.

Results

A total amount of 2269 ± 1726 mg obidoxime was infused over $65 \text{ h} \pm 55 \text{ h}$ resulting in a steady state plasma concentration of $14.5 \pm 7.3 \mu\text{M}$. Obidoxime was eliminated with $t_{1/2}(1)$ 2.2 and $t_{1/2}(2)$ 14 h. The volumes of distribution amounted to $0.32 \pm 0.1 \text{ L/kg}$ ($V(1)$) and 0.28 ± 0.12 ($V(2)$) L/kg. Post mortem examination of tissue in one patient showed obidoxime accumulation in cartilage, kidney and liver and pointed to brain concentrations similar to plasma concentration.

Conclusions

Using the suggested obidoxime regimen, the targeted plasma concentration could be achieved. Obidoxime was eliminated biphasically and was well tolerated. This result allows the recommendation of using this definite regimen for adults also in case of mass casualties.

Childhood leukaemia and parental occupational exposure to pesticides: a systematic review and meta-analysis

Van Maele-Fabry G, Lantin A-C, Hoet P, Lison D. Cancer Causes Control 2010; 21: 787-809.

Objective

To conduct a systematic review and meta-analysis of published studies on the association between parental occupational exposure to pesticides and childhood leukaemia.

Methods

Studies were identified from a MEDLINE search through 31 July 2009 and from the reference lists of identified publications. Relative risk (RR) estimates were extracted from 25 studies published

between 1985 and 2008. Meta-rate ratio estimates (mRR) were calculated according to fixed and random-effect meta-analysis models. Separate analyses were conducted after stratification for study design, definition of exposure (employment in a farm/agriculture assuming exposure to pesticides versus exposure to pesticides stipulated), exposed parent, window of exposure, type of leukaemia and biocide category.

Results

No statistically significant association between childhood leukaemia and parental occupation as farmers/agricultural workers was observed. When exposure to pesticides was stipulated, positive associations were reported for maternal exposure for all studies combined (mRR: 1.62; 95% CI: 1.22-2.16), in all exposure windows considered and for acute non-lymphocytic leukaemia (ANLL). There was no association with paternal exposure when combining all studies (mRR: 1.14; 95% CI: 0.76-1.69). However, significant increased risks were seen for paternal exposure, in some exposure windows as well as for the biocide category.

Conclusions

The strongest evidence of an increased risk of childhood leukaemia comes from studies with maternal occupational exposure to pesticides. The associations with paternal exposure were weaker and less consistent. These results add to the evidence leading to recommend minimizing parental occupational exposure to pesticides. Our findings also support the need to rely more on studies that clearly stipulate exposure to pesticides rather than those that assume pesticide exposure because of farm/agriculture employment.

Acute intentional toxicity: endosulfan and other organochlorines

Moses V, Peter JV. Clin Toxicol 2010; online early: doi: 10.3109/15563650.2010.494610:

Introduction

Organochlorine pesticides continue to be used in several developing countries despite concerns regarding their toxicity profile. Endosulfan is an organochlorine recognized as an important agent of acute toxicity.

Methods

In this retrospective study, the clinical features, course, and outcomes among patients with acute endosulfan poisoning requiring admission to the hospital during an 8-year period (1999-2007) were reviewed.

Results

Among 34 patients hospitalized during this study period for alleged organochlorine poisoning, 16 patients with endosulfan poisoning were identified. The majority (75%) received initial treatment at a primary or secondary center. Neurological toxicity predominated, particularly low sensorium (81%) and generalized seizures (75%), including status epilepticus (33%). Other features observed included hepatic transaminase elevation, azotemia, metabolic acidosis, and leukocytosis. Mechanical ventilation was required in 69% and vasoactive agents in 19%. In-hospital mortality was 19%. There were no gross neurological sequelae at discharge. In three other patients who presented with organochlorine poisoning, the compounds ingested were lindane, endrin, and dicofol (n = 1 each). The course and outcomes in these patients were unremarkable and all three patients survived.

Conclusions

Endosulfan is capable of high lethality and significant morbidity. The commonest manifestations are neurological although other organ dysfunction also occurs. In the absence of effective antidotes, restriction of its availability, along with prompt treatment of toxicity, including preemptive anticonvulsant therapy are suggested.

TOXICOLOGY

General

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