

Current Awareness in Clinical Toxicology

Editors: Sarah Cage MSc and Allister Vale MD

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CURRENT AWARENESS PAPERS OF THE MONTH

Delayed onset of acute renal failure after significant paracetamol overdose: a case series

Waring WS, Jamie H, Leggett GE. Hum Exp Toxicol 2009; online early: doi: 10.1177/0960327109350799: 1-6.

Abstract

Acute renal failure is a recognized manifestation of paracetamol toxicity, but comparatively little data is available concerning its onset and duration. The present study sought to characterize the time course of rising serum creatinine concentrations in paracetamol nephrotoxicity.

Renal failure was defined by serum creatinine concentration ≥ 150 micromol/L (1.69 mg/dL) or $\geq 50\%$ increase from baseline. Serum creatinine concentrations and alanine aminotransferase activity were considered with respect to the interval after paracetamol ingestion. There were 2068 patients with paracetamol overdose between March 2005 and October 2007, and paracetamol nephrotoxicity occurred in 8 (0.4%). All had significant hepatotoxicity, and peak serum alanine aminotransferase activity occurred at 2.5 days (2.2 to 2.9 days) after ingestion. Peak serum creatinine concentrations did not occur until 5.5 days (4.4 to 5.9 days) after ingestion ($p = 0.031$ by Wilcoxon test). Serum creatinine concentrations slowly restored to normal, and renal replacement was not required.

In this patient series, rising serum creatinine concentrations only became detectable after more than 48 hours after paracetamol ingestion. Therefore, renal failure might easily be missed if patients are discharged home before this. Further work is required to establish the prevalence of paracetamol-induced nephrotoxicity, and its clinical significance.

Incidence and onset of delayed seizures after overdoses of extended-release bupropion

Starr P, Klein-Schwartz W, Spiller H, Kern P, Ekleberry SE, Kunkel S. Am J Emerg Med 2009; 27: 911-5.

Background

Delayed seizures have been reported with overdoses of bupropion extended-release (XL). This study systematically evaluates the frequency and timing of seizures and an association between other toxic effects (i.e., agitation, tremors, and hallucinations) and seizures.

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Methods

A 3-year multi-poison center observational study of hospitalized patients with ingestion of bupropion XL ≥ 600 mg in adults and ≥ 4 mg/kg in children was performed. Patients with coingestants or a medical history that could affect seizure occurrence were excluded. Data collection forms captured onset time of seizure(s), other symptoms, and treatment.

Results

One hundred seventeen patients met inclusion criteria: median age of 22 years (range, 1.3-65 years) with 16 children ≤ 3 years. Seizures occurred in 37 (31.6%) patients, with initial seizure at 0.5 to 24 hours after ingestion; 12 (32%) patients had initial seizure at > 8 hours. Subsequent seizures occurred in 49%. Children ages 1.3, 3, and 7 years, developed seizures. In patients ≥ 13 years of age, median dose with seizures was 4350 mg (range, 600-54 000) compared to 2400 mg (range, 600-9000) in patients without seizures. Agitation, tremors, and hallucinations occurred in 29.7%, 40.5%, and 18.9% of patients with seizures, respectively, compared with 12.5 %, 17.5%, and 10% in patients without seizures. The neurologic effects agitation ($P = 0.045$) and tremors ($P = 0.005$) occurred more frequently.

Conclusion

Delayed seizure onset suggests a minimum observation period of 24 hours after bupropion XL overdose. Although patients experiencing agitation or tremors may be at greater risk, seizures can occur without preceding central nervous system toxicity.

Management of injecting drug users admitted to hospital

Haber PS, Demirkol A, Lange K, Murnion B. Lancet, 2009; 374: 1284-93.

Abstract

General hospital clinicians frequently deal with injecting drug users because substance use has diverse medical and psychiatric complications. Non-specialist clinicians often initiate management when specialist consultation is not available or accepted by the patient.

Here, we summarise evidence for the management of hospitalised injecting drug users. The first challenge is to engage a drug user into medical care. A non-judgmental approach towards patients and acceptance of their lifestyle choices facilitates engagement. Pragmatic clinical goals can be negotiated and achieved. We also describe common conditions of injecting drug users.

Accurate diagnosis and appropriate management focus on common issues such as intoxication, withdrawal, pain management, drug seeking, psychological comorbidity, behavioural difficulties, and pregnancy. Effective management can reduce the medical and social effect of these conditions and is not difficult.

Extracorporeal life support in severe drug intoxication: a retrospective cohort study of seventeen cases

Daubin C, Lehoux P, Ivascau C, Tasle M, Boust M, Lepage O, Quentin C, Massetti M, Charbonneau P. Crit Care 2009; 13: R138.

Introduction

Cardiovascular failure is the leading cause of death in severe acute drug intoxication. In this setting, we report the feasibility, complications, and outcome of emergency extracorporeal life support (ECLS) in refractory shock or cardiac arrest following a drug overdose.

Methods

This is a retrospective cohort study of 17 patients admitted over a 10-year period for prolonged cardiac arrest or refractory shock following a drug overdose and not responding to optimal conventional treatment. Patients were evaluated in the medical ICU and cardiovascular surgery department of a university hospital. ECLS implantation used a centrifugal pump connected to a

hollow-fiber membrane oxygenator and was performed in the operating room (n = 13), intensive care unit (n = 3), or emergency department (n = 1). ECLS was employed for refractory shock and prolonged cardiac arrest in 10 and 7 cases, respectively.

Results

The mean duration of external cardiac massage was 101 ± 55 min. Fifteen patients had ingested cardiotoxic drugs, including 11 cases of drugs with membrane stabilizing activity. Time from hospital admission to initiation of ECLS was 6.4 ± 7.0 h. Time to ECLS implant was 58 ± 11 min. The mean ECLS flow rate was 3.45 ± 0.45 L/min. The average ECLS duration was 4.5 ± 2.4 days. Early complications included limb ischemia (n = 6), femoral thrombus (n = 1), cava inferior thrombus (n = 1), and severe bleeding at the site of cannulation (n = 2). Fifteen patients were weaned off ECLS support and 13 (76%) were discharged to hospital without sequelae.

Conclusions

Based on our experience, we consider ECLS as a last resort, efficient, and relatively safe therapeutic option in this population. However, the uncontrolled nature of our data requires careful interpretation.

Are one or two dangerous? Lidocaine and topical anesthetic exposures in children

Curtis LA, Dolan TS, Seibert HE. J Emerg Med 2009; 37: 32-9.

Abstract

Topical anesthetics are found in a variety of prescription and non-prescription preparations, from teething gels to hemorrhoid creams. In 2003, there were 8576 exposures to local/topical anesthetics reported to the American Association of Poison Control Centers, with 67% of cases in the age group younger than 6 years old.

This report reviews the available literature involving topical anesthetic exposures in children younger than 6 years old, including the National Library of Medicine's Pub Med database (limited to English language) and data from POISINDEX. Additionally, we reviewed the American Association of Poison Control Centers' annual reports from 1983 to 2003. There were 7 deaths in this age range from topical anesthetics. Although the number of deaths is low, the fact that there have been deaths reveals the serious nature of the toxicity that can result from these readily available non-prescription analgesics. Toxicity may result from topical absorption, ingestion, or aspiration. Additionally, toxicity can result from unintentional as well as therapeutic mishaps.

Although the number of cases is limited, these medications can be toxic at low doses-which, in children younger than 6 years of age, may amount to as little as a teaspoon.

Acute metformin overdose: examining serum pH, lactate level, and metformin concentrations in survivors versus nonsurvivors: a systematic review of the literature

Dell'Aglio DM, Perino LJ, Kazzi Z, Abramson J, Schwartz MD, Morgan BW. Ann Emerg Med 2009; online early: doi: 10.1016/j.annemergmed.2009.04.023: 1-7.

Study objective

Metformin is known to cause potentially fatal metabolic acidosis with an increased lactate level in both overdose and therapeutic use. No association between mortality and serum pH, lactate level, or metformin concentrations, though intuitive, has yet been described. This systematic literature review is designed to evaluate the association between mortality and serum pH, lactate level, and metformin concentrations in acute metformin overdose.

Methods

We reviewed the literature by using the MEDLINE, Embase, CINAHL, and TOXNET databases for

cases of metformin overdose with documented mortality data and values of serum pH, lactate level, and metformin concentrations. When available, patient age, patient sex, and whether patients received intravenous sodium bicarbonate therapy or hemodialysis were also analyzed. Cases meeting inclusion criteria were analyzed to determine whether a difference in distribution of nadir serum pH, peak serum lactate level, or peak serum metformin concentrations existed between overdose survivors and nonsurvivors.

Results

We identified 10 articles that had 1 or more cases meeting our inclusion criteria. In total, there were 22 cases of metformin overdose (5/22 died) that met inclusion criteria. No intentional overdose patients died whose serum pH nadir was greater than 6.9, maximum lactate concentration less than 25 mol/L, or maximum metformin concentration less than 50 mug/mL (therapeutic range 1 to 2 mug/mL). Intentional overdose patients with a nadir serum pH less than 6.9 had 83% mortality (5/6), those with lactate concentration greater than 25 mmol/L had 83% mortality (5/6), and those with metformin concentration greater than 50 mug/mL had 38% mortality (5/12). Nadir serum pH and peak serum lactate and metformin concentration distributions in survivors and nonsurvivors revealed that survivors had a median nadir pH of 7.30, interquartile range (IQR) 7.22, 7.36; nonsurvivors, a median nadir pH of 6.71, IQR 6.71, 6.73; survivors, a median peak lactate level of 10.8 mmol/L, IQR 4.2, 12.9; nonsurvivors, a median peak lactate level of 35.0 mmol/L, IQR 33.3, 39.0; survivors, a median peak metformin level of 42 mug/mL, IQR 6.6, 67.6; and nonsurvivors, a median peak metformin level of 110 mug/mL, IQR 110, 110.

Conclusion

No cases of acute metformin overdose meeting the study's inclusion criteria were found in which patients with a nadir serum pH greater than 6.9, peak serum lactate concentrations less than 25 mmol/L, or peak serum metformin concentrations less than 50 mug/mL died. Patients with acute metformin overdose who died had much lower serum pH nadirs and much higher peak serum lactate and metformin concentrations than those who survived.

Metformin-associated lactic acidosis: a prognostic and therapeutic study

Seidowsky A, Nseir S, Houdret N, Fourrier F. Crit Care Med 2009; 37: 2191-6.

Objectives

Metformin-associated lactic acidosis is a rare and serious complication of biguanide treatment. It usually occurs when a precipitating disease induces an acute renal failure and an incidental overdose. Voluntary intoxication is rare. Bicarbonate hemodialysis (HD) is recommended to decrease metformin levels and correct acidosis but its optimal duration has not been determined. This study was designed to document the characteristics and prognostic factors of intentional and incidental metformin overdose and to determine the optimal duration of HD.

Design

Ten years retrospective analysis of patients admitted in intensive care unit for metformin-associated lactic acidosis.

Setting

Two intensive care units (50 beds) in a university hospital.

Measurements and main results

Clinical and biological characteristics, organ failures, and sequential metformin levels during HD were recorded. Forty-two patients were included (13 voluntary intoxications and 29 incidental overdoses); 74% of patients were in acute renal failure and needed HD. No death was observed in intentional overdose patients compared with 48.3% mortality in incidental overdose patients. The factors significantly associated with mortality were logistic organ dysfunction system score, pH, plasma lactate, and prothrombin activity. By multivariate analysis, a prothrombin activity

<50% was the only independent predictive factor of mortality (relative risk: 59.8; confidence limits: 6.3-568; $p < 0.0001$). Sequential measurements of metformin levels during HD were consistent with a bicompartmental elimination pattern. A cumulative HD duration of 15 hours was associated with the return of metformin level to the therapeutic normal range.

Conclusions

In our study, the outcome of MALA was uniformly favorable after intentional metformin overdose. The vital prognosis was mainly influenced by the occurrence of multiple organ dysfunctions, the best predictive factor of death being an acute liver dysfunction as assessed by PT activity. Prolonged HD was needed to correct metformin overdose.

Pediatric ziprasidone overdose

Fasano CJ, O'Malley GF, Lares C, Rowden AK. *Pediatr Emerg Care* 2009; 25: 258-9.

Abstract

We describe the first ziprasidone overdose with quantitative serum levels of a pediatric patient in coma and with pinpoint pupils. This case is an important contribution to the pediatric ziprasidone literature because it illustrates that ingestion of just 1 pill may result to profound mental status and respiratory depression in a child.

H.C., a 30-month-old girl, presented to the emergency department approximately 30 min after an accidental ingestion of an adult family member's medication. The child was found on the floor surrounded by numerous pills and was witnessed to have ingested at least 1 tablet by a caregiver. After finding the child with the pills, the family observed the child for a brief period but transported her to the hospital after she became lethargic and unresponsive. The child received 2 doses of 0.4 mg of intravenous naloxone without change in her neurologic status. The child then underwent a rapid sequence intubation for airway protection and subsequently received gastrointestinal decontamination with 15 g of activated charcoal via the orogastric tube.

Ziprasidone is an atypical antipsychotic drug that was approved by the Food and Drug Administration in February 2001 for the general treatment of schizophrenia in adults. Previously reported pediatric ziprasidone overdoses describe a syndrome of sedation, tachycardia, hypotonia, and coma consistent with that of the patient described in this paper. In pediatric ziprasidone overdose, QTc prolongation and hypotension have also been illustrated, but seizures have not been reported.

An interesting aspect of this case is the development of pinpoint pupils unresponsive to naloxone. This phenomenon has been reported before with overdose of olanzapine, a similar atypical antipsychotic. The mechanism of miosis associated with overdose of atypical antipsychotics is unclear but is likely related to interference with central innervation of the pupil. Pupil size is maintained by a balance between sympathetic and parasympathetic neurohumeral tones. We propose that an overdose of an alpha-1 receptor blocking agent, such as ziprasidone, results in unopposed parasympathetic stimulation resulting in miosis.

Selective serotonin reuptake inhibitors in pregnancy and congenital malformations: population based cohort study

Pedersen LH, Henriksen TB, Vestergaard M, Olsen J, Bech BH. *Br Med J* 2009; 339: b3569.

Objective

To investigate any association between selective serotonin reuptake inhibitors (SSRIs) taken during pregnancy and congenital major malformations.

Design

Population based cohort study.

Participants

493113 children born in Denmark, 1996-2003.

Main outcome measure

Major malformations categorised according to Eurocat (European Surveillance of Congenital Anomalies) with additional diagnostic grouping of heart defects. Nationwide registers on medical redemptions (filled prescriptions), delivery, and hospital diagnosis provided information on mothers and newborns. Follow-up data available to December 2005.

Results

Redemptions for SSRIs were not associated with major malformations overall but were associated with septal heart defects (odds ratio 1.99, 95% confidence interval 1.13-3.53). For individual SSRIs, the odds ratio for septal heart defects was 3.25 (1.21-8.75) for sertraline, 2.52 (1.04-6.10) for citalopram, and 1.34 (0.33-5.41) for fluoxetine. Redemptions for more than one type of SSRI were associated with septal heart defects (4.70, 1.74-12.7). The absolute increase in the prevalence of malformations was low – for example, the prevalence of septal heart defects was 0.5% (2315/493 113) among unexposed children, 0.9% (12/1370) among children whose mothers were prescribed any SSRI, and 2.1% (4/193) among children whose mothers were prescribed more than one type of SSRI.

Conclusion

There is an increased prevalence of septal heart defects among children whose mothers were prescribed an SSRI in early pregnancy, particularly sertraline and citalopram. The largest association was found for children of women who redeemed prescriptions for more than one type of SSRI.

Epidemic of poisoning caused by scopolamine disguised as Rohypnol™ tablets

Vallersnes OM, Lund C, Duns AK, Netland H, Rasmussen IA. Clin Toxicol 2009; online early: doi: 10.3109/15563650903333804: 1-5.

Objective

An epidemic of scopolamine poisonings occurred in Oslo in 2008 among users of illicit drugs, caused by fake Rohypnol™ pills. The clinical features, diagnostic process, and handling of the epidemic are presented.

Methods

Suspected cases of scopolamine poisoning were extracted by reviewing registration forms from an ongoing prospective clinical study of acute poisonings in Oslo. Medical records of extracted contacts were examined and cases included according to specified clinical criteria.

Results

Forty-four cases of probable scopolamine poisoning were registered. Main clinical features were mydriasis, visual hallucinations, plucking behavior, agitation, and coma. No clinical diagnosis of anticholinergic syndrome was made prior to forensic analysis of the tablets, the most frequent diagnosis up to this point being unspecified drug-induced psychosis. Later in the epidemic, scopolamine poisoning became the dominating diagnosis. Ten patients were admitted to psychiatric hospitals, the rest recovered in medical units, or left health care against medical advice.

Discussion

Scopolamine poisonings are rare, but the resulting anticholinergic syndrome is well described. The syndrome was not recognized until the forensic analysis result strikingly changed how the patients were diagnosed and handled. A unique aspect of this epidemic was the intoxicating agent being scopolamine-containing tablets looking like Rohypnol™, sold and used under the impression of being the latter.

Conclusion

Recognizing the anticholinergic syndrome is important to provide proper treatment. Forensic analysis was the key to correct diagnosis in this outbreak, demonstrating its importance in verifying an epidemic of poisoning by fake drugs.

A comparison of sodium calcium edetate (edetate calcium disodium) and succimer (DMSA) in the treatment of inorganic lead poisoning

Bradberry SM, Vale JA. Clin Toxicol 2009; 47: 841-58.

Introduction

This article reviews the experimental and clinical studies that have compared the efficacy (impact on urine lead excretion, blood and tissue lead concentrations, resolution of features and survival) of sodium calcium edetate (edetate calcium disodium) and succimer (DMSA) in the treatment of inorganic lead poisoning. It also summarizes the pharmacokinetic and pharmacodynamic aspects and the adverse effects of treatment.

Methods

Medline, Toxline, and Embase were searched for all available years to June 2009.

Pharmacokinetics and pharmacodynamics

The absorption of oral DMSA is more complete than sodium calcium edetate; the latter has to be administered parenterally. Both antidotes are distributed predominantly extracellularly. Sodium calcium edetate is not metabolized, whereas DMSA is extensively metabolized to mixed disulfides of cysteine. The two antidotes have elimination half-lives of less than 60 min. There is no evidence that either antidote crosses the blood-brain barrier to any major extent. Sodium calcium edetate chelates lead by displacement of the central Ca^{2+} ion with Pb^{2+} . The nature of the DMSA-lead chelate is less clearly defined. There is evidence that the mixed disulfides of cysteine are the active chelating moiety in humans. If this is the case, this suggests that chelation occurs principally, if not exclusively, in the kidney. The primary source of lead mobilized by sodium calcium edetate is bone with an additional contribution from kidney and liver.

Efficacy

Comparison of the experimental studies is complicated by substantial variations in study design, particularly the antidote dose, the route and duration of treatment, the amount and duration of lead dosing, and lack of direct comparison between antidotes (comparison was usually made with control). In experimental studies that used equimolar and clinically relevant antidote doses and assessed the impact of DMSA and sodium calcium edetate on urine lead excretion and/or blood lead concentrations, similar results were found, though no direct comparison between antidotes was undertaken. DMSA was more effective than sodium calcium edetate in reducing the kidney lead concentration, sodium calcium edetate was more effective than DMSA in reducing bone lead concentrations, and there was no consistently observed effect of chelation therapy on brain lead concentrations in these experimental studies. Only two clinical studies have compared equimolar or similar antidote doses in enhancing urine lead excretion; there was no statistical difference between the antidotes, though both studies had limitations. DMSA and sodium calcium edetate had a comparable impact on lowering blood lead concentrations in a clinical study using similar molar antidote doses.

Adverse effects

Sodium calcium edetate causes dose-related nephrotoxicity. Both agents deplete zinc and copper, the effect on zinc being significantly greater with sodium calcium edetate. A transient increase in hepatic transaminase activity has been reported with both antidotes but appears to be more common with DMSA and neither has been associated with clinically significant hepatic toxicity. Skin lesions during treatment with sodium calcium edetate are unusual and have been attributed to zinc deficiency. DMSA has occasionally been associated with a severe mucocutaneous reaction

necessitating discontinuation of therapy.

Conclusions

Oral DMSA and parenteral sodium calcium edetate are both effective chelators of lead. There are currently insufficient data, however, to conclude that either antidote is superior in enhancing lead excretion. Both antidotes resolve the symptoms of moderate and severe lead toxicity rapidly. Although there is greater clinical experience with sodium calcium edetate, particularly in the treatment of lead encephalopathy, oral DMSA may now be considered as an alternative in circumstances where oral therapy is preferable.

2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) poisoning in Victor Yushchenko: identification and measurement of TCDD metabolites

Sorg O, Zennegg M, Schmid P, Fedosyuk R, Valikhnovskiy R, Gaide O, Kniazevych V, Saurat J-H. Lancet, 2009; 374: 1179-85.

Background

2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) has a long half-life of 5-10 years in human beings as a result of its high lipophilicity, and little or no metabolism. We monitored TCDD, its form, distribution, and elimination in Victor Yushchenko after he presented with severe poisoning.

Methods

In late December, 2004, a patient presented with TCDD poisoning; the levels in his blood serum (108000 pg/g lipid weight) were more than 50 000-fold greater than those in the general population. We identified TCDD and its metabolites, and monitored their levels for 3 years using gas chromatography and high-resolution mass spectrometry in samples of blood serum, adipose tissue, faeces, skin, urine, and sweat, after they were extracted and cleaned with different organic solvents.

Findings

The amount of unmodified TCDD in the samples that were analysed accounted for about 60% of TCDD eliminated from the body during the same period. Two TCDD metabolites-2,3,7-trichloro-8-hydroxydibenzo-p-dioxin and 1,3,7,8-tetrachloro-2-hydroxydibenzo-p-dioxin-were identified in the faeces, blood serum, and urine. The faeces contained the highest concentration of TCDD metabolites, and were the main route of elimination. Altogether, the different routes of elimination of TCDD and its metabolites accounted for 98% of the loss of the toxin from the body. The half-life of TCDD in our patient was 15.4 months.

Interpretation

This case of poisoning with TCDD suggests that the design of methods for routine assessment of TCDD metabolites in human beings should be a main aim of TCDD research in the metabolomic era.

Extreme variability in the formation of chlorpyrifos oxon (CPO) in patients poisoned by chlorpyrifos (CPF)

Eyer F, Roberts DM, Buckley NA, Eddleston M, Thiermann H, Worek F, Eyer P. Biochem Pharmacol 2009; 78: 531-7.

Abstract

Chlorpyrifos (CPF) is a pesticide that causes tens of thousands of deaths per year worldwide. Chlorpyrifos oxon (CPO) is the active metabolite of CPF that inhibits acetylcholinesterase. However, this presumed metabolite has escaped detection in human samples by conventional methods (HPLC, GC-MS, LC-MS) until now.

A recently developed enzyme-based assay allowed the determination of CPO in the nanomolar range and was successfully employed to detect this metabolite. CPO and CPF were analysed in consecutive plasma samples of 74 patients with intentional CPF poisoning. A wide concentration range of CPO and CPF was observed and the ratio of CPO/CPF varied considerably between

individuals and over time. The ratio increased during the course of poisoning from a mean of 0.005 in the first few hours after ingestion up to an apparent steady-state mean of 0.03 between 30 and 72 h. There was a hundred-fold variation in the ratio between samples and the interquartile range (between individuals) indicated over half the samples had a 5-fold or greater variation from the mean. The ratio was independent of the CPF concentration and the pralidoxime regimen. CPO was present in sufficient quantities to explain any observed acetylcholinesterase inhibitory activity. The effectiveness of pralidoxime in reactivating the inhibited acetylcholinesterase is strongly dependent on the CPO concentration.

Differences in clinical outcomes and the response to antidotes in patients with acute poisoning may occur due to inter-individual variability in metabolism.

Human exposure to insecticide products containing pyrethrins and piperonyl butoxide (2001-2003)

Osimitz TG, Sommers N, Kingston R. Food Chem Toxicol 2009; 47: 1406-15.

Abstract

Pyrethrum, used as an insecticide for centuries, is derived from dried and ground flowers of *Chrysanthemum cinerariaefolium*. Its current major use is in insecticide products to the control insects in the home and food handling establishments.

We investigated human incidents reported through the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System (TESS[®]) associated with regulated insecticides containing pyrethrins and piperonyl butoxide (PY/PBO) from 2001 to 2003. Special attention was paid to dermal and respiratory effects. Although there are limitations associated with TESS data, we observed that:

Despite extensive use, incidents with reports of moderate or major adverse effects were relatively rare (717 moderate and 23 major outcomes out of 17,873 calls).

Following label-directed use of the products, adverse dermal or respiratory reactions were very rare; (dermal - 17 moderate, 1 major; respiratory - 18 moderate, 0 major).

The data suggest that asthmatics and people sensitive to ragweed (*Ambrosia artemisiifolia*) are not unusually sensitive to PY/PBO. In view of their widespread use, the data indicates that PY/PBO products can be used with a relatively low risk of adverse effects. Moreover, the data suggest that they are not likely to cause reactions in people with asthma or allergies.

Nicotinic plant poisoning

Schep LJ, Slaughter RJ, Beasley DM. Clin Toxicol 2009; 47: 771-81.

Introduction

A wide range of plants contain nicotinic and nicotinic-like alkaloids. Of this diverse group, those that have been reported to cause human poisoning appear to have similar mechanisms of toxicity and presenting patients therefore have comparable toxidromes. This review describes the taxonomy and principal alkaloids of plants that contain nicotinic and nicotinic-like alkaloids, with particular focus on those that are toxic to humans. The toxicokinetics and mechanisms of toxicity of these alkaloids are reviewed and the clinical features and management of poisoning due to these plants are described.

Methods

This review was compiled by systematically searching OVID MEDLINE and ISI Web of Science. This identified 9,456 papers, excluding duplicates, all of which were screened. Reviewed plants and their principal alkaloids. Plants containing nicotine and nicotine-like alkaloids that have been reported to be poisonous to humans include *Conium maculatum*, *Nicotiana glauca* and *Nicotiana tabacum*, *Laburnum anagyroides*, and *Caulophyllum thalictroides*. They contain the toxic alkaloids nicotine, anabasine, cytisine, n-methylcytisine, coniine, n-methylconiine, and gamma-coniceine.

Mechanisms of toxicity

These alkaloids act agonistically at nicotinic-type acetylcholine (cholinergic) receptors (nAChRs). The nicotinic-type acetylcholine receptor can vary both in its subunit composition and in its distribution within the body (the central and autonomic nervous systems, the neuromuscular junctions, and the adrenal medulla). Agonistic interaction at these variable sites may explain why the alkaloids have diverse effects depending on the administered dose and duration of exposure.

Toxicokinetics

Nicotine and nicotine-like alkaloids are absorbed readily across all routes of exposure and are rapidly and widely distributed, readily traversing the blood-brain barrier and the placenta, and are freely distributed in breast milk. Metabolism occurs predominantly in the liver followed by rapid renal elimination.

Clinical features

Following acute exposure, symptoms typically follow a biphasic pattern. The early phase consists of nicotinic cholinergic stimulation resulting in symptoms such as abdominal pain, hypertension, tachycardia, and tremors. The second inhibitory phase is delayed and often heralded by hypotension, bradycardia, and dyspnea, finally leading to coma and respiratory failure.

Management

Supportive care is the mainstay of management with primary emphasis on cardiovascular and respiratory support to ensure recovery.

Conclusions

Exposure to plants containing nicotine and nicotine-like alkaloids can lead to severe poisoning but, with prompt supportive care, patients should make a full recovery.

Antivenom efficacy or effectiveness: the Australian experience

Isbister GK. Toxicology 2009; online early: doi: 10.1016/j.tox.2009.09.013: 1-6.

Abstract

Despite widespread use of antivenoms, many questions remain about their effectiveness in the clinical setting. The almost universal acceptance of their value is based mainly on in vitro studies, animal studies and human observational studies.

Numerous examples exist where they demonstrate clear benefit, such as consumption coagulopathy in viper envenoming, prevention of neurotoxicity in Australasian elapid bites, systemic effects in scorpion and funnel-web spider envenoming. There are also concerns about the quality and efficacy of some antivenoms. However, it is important not to confuse the efficacy of antivenom, defined as its ability to bind and neutralise venom-mediated effects under ideal conditions, and the effectiveness of antivenom, defined as its ability to reverse or prevent envenoming in human cases. There are numerous potential reasons for antivenom failure in human envenoming, of which antivenom inefficacy is only one. Other important reasons include venom-mediated effects being irreversible, antivenom being unable to reach the site of toxin-mediated injury, or the rapidity of onset of venom-mediated effects.

A number of recent studies in Australia bring into question the effectiveness of some antivenoms, including snake antivenom for coagulopathy, redback spider and box jellyfish antivenoms. Despite brown snake antivenom being able to neutralise venom induced clotting in vitro, use of the antivenom in human envenoming does not appear to change the time course of coagulopathy.

However, it is important that apparent antivenom ineffectiveness in specific cases is correctly interpreted and does not lead to a universal belief that antivenom is ineffective. It should rather encourage further studies to investigate the underlying pathophysiology of envenoming, the pharmacokinetics of venoms and antivenoms, and ultimately the effectiveness of antivenom based on snake type, clinical effects and timing of administration.

TOXICOLOGY

Analytical toxicology

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