Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPERS OF THE MONTH

Intoxications involving MDPV in Sweden during 2010-2014: results from the STRIDA project

Beck O, Franzen L, Bäckberg M, Signell P, Helander A. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1089576:

Context

In the recent years, there have been an increasing number of new psychoactive substances (NPS) available through marketing and sale on the Internet. The stimulant 3,4-methylenedioxypyrovalerone (MDPV) is a potent dopamine reuptake inhibitor, which can cause serious intoxications requiring intensive care and even fatality. This report from the STRIDA project presents the prevalence, laboratory results, and clinical features in a series of intoxications involving MDPV over a 5-year period.

Study design

Observational case series of consecutive patients with admitted or suspected intake of NPS presented at hospitals in Sweden from 2010 to 2014.

Patients and methods

Blood and/or urine samples were collected from intoxicated patients with admitted or suspected intake of NPS presenting at hospitals over the country. Analysis of NPS was

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performed by a liquid chromatography-tandem mass spectrometry multicomponent method. Clinical data were collected when caregivers consulted the Swedish Poisons Information Centre and also retrieved from medical records. The severity of poisoning was graded retrospectively using the poisoning severity score.

Results

During the 5-year study period, the number of MDPV-related inquiries to the Poisons Information Centre was 662 out of a total ~4500 suspected NPS-related inquiries (~15%), and 201 analytically confirmed MDPV intoxications were enrolled in the study. The study period covered the period when the use of MDPV in Sweden was at its peak and also the decline to an almost zero level. The age range of patients was 18-68 (mean 36, median 35) years, and 71% were males. The MDPV concentrations in serum ranged between 1.0 ng/mL and 1509 ng/mL (mean 63.6, median 20) and between 1.0 ng/mL and 81 000 ng/mL (mean 3880, median 1160) in urine. The urinary values were also creatinine corrected for variation in urine dilution, and the MDPV/creatinine ratio ranged between 0.10 ng/mmol and 2480 ng/mmol (mean 247, median 92.6). There was a statistically significant association between the serum MDPV concentration and the urinary MDPV/creatinine ratio, for 118 cases where both data were available (r = 0.764; p < 0.0001, Spearman's rank correlation). In 30 (15%) cases, MDPV was the single psychoactive substance identified in the serum or urine specimens. In the other 171 cases, other psychoactive substances were detected together with MDPV. The additional substances (n = 61) comprised of both conventional drugs of abuse, other NPS (n = 39), pharmaceuticals, and ethanol. The cathinone-derivative alpha-pyrrolidinovalerophenone (α-PVP) was the most frequent other NPS, and was detected in 58 (29%) cases, followed by methylone in 14 (7%) cases. The main clinical manifestations reported in patients testing positive for MDPV included agitation, tachycardia (≥100/min), and hypertension (systolic blood pressure ≥ 140 mmHg), which were observed in 130 (67%), 106 (56%), and 65 (34%) cases, respectively. Other symptoms included hallucinations (n = 31, 16%), delirium (n = 29, 15%), hyperthermia (>39°C/102.4°F; n = 18, 10%), and rhabdomyolysis (n = 16, 8%). In MDPV intoxications with serum levels >100 ng/mL, the cases were graded as more severe and hyperthermia was less common.

Conclusions

In a large number of analytically confirmed MDPV intoxications from mostly polydrug users, the urine and serum MDPV concentrations showed a high variability. The clinical features were consistent with a severe sympathomimetic toxidrome. The results also demonstrated that MDPV prevailed as a drug of abuse for a long time, after its classification as a narcotic substance and despite a high incidence of severe poisonings.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1089576

Long-term visual damage after acute methanol poisonings: longitudinal cross-sectional study in 50 patients

Zakharov S, Pelclova D, Diblik P, Urban P, Kuthan P, Nurieva O, Kotikova K, Navratil T, Komarc M, Belacek J, Seidl Z, Vaneckova M, Hubacek JA, Bezdicek O, Klempir J, Yurchenko M, Ruzicka E, Miovsky M, Janikova B, Hovda KE. **Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1086488:**

Context

Visual disturbances due to the toxic effect of formic acid in acute methanol poisonings are generally transient. The subjective symptoms of visual toxicity may resolve within few weeks

and fundoscopic signs of acute optic neuropathy subside within 1–2 months; therefore, the prevalence of long-term visual sequelae in the population of survivors of poisonings may be underestimated.

Objective

To study the prevalence and character of long-term visual sequelae of acute methanol poisonings based on the data from the Czech mass methanol outbreak in 2012.

Patients and methods

A total of 50 patients with confirmed methanol poisoning were included in this longitudinal cross-sectional study, median age: 48 (range, 23–73) years. The following tests were performed: optical coherence tomography or OCT with evaluation of the retinal nerve fibers layer (RNFL), visual evoked potentials (VEP), magnetic resonance imaging (MRI) of brain, complete ocular examination (visual acuity/field, color vision, contrast sensitivity, and fundus), neurological examinations, and biochemical tests.

Results

Of 50 patients, 7/50 (14%) were discharged with diagnosed visual sequelae and 6/50 (12%) were discharged with both visual and central nervous system sequelae of poisoning. On the follow-up examination, 20/50 (40%) of the patients had long-term visual sequelae, with 8% of blindness. A total of 38% of the patients had abnormal (28% borderline) findings on RNFL, and 40% had abnormal (18% borderline) VEP. Among the patients discharged without detected visual sequelae, 8/37 (22%) had abnormal RNFL and VEP. Patients with visual sequelae had brain lesions more often (70% vs. 27%, $p < 0.01$). MRI identified optic nerve lesions in 2/20 cases with abnormal VEP only. The groups with and without visual sequelae differed in serum methanol, ethanol, HCO_{3-}, formate, pH, anion gap, and base deficit (all $p < 0.01$). Visual disturbances on admission and coma were more prevalent in the patients with visual sequelae ($p < 0.05$). Patients with positive serum ethanol on admission were 93% less likely to have optical axonal damage (OR: 0.07 (95% CI: 0.01–0.8); $p < 0.05$). No association was found between visual sequelae and type of antidote administered, mode of hemodialysis, or folate substitution. Pre-hospital administration of ethanol seemed beneficial: these patients were 90% less likely to have abnormal RNFL findings (OR: 0.10 (95% CI: 0.02–0.52); $p < 0.01$).

Conclusion

The long-term visual sequelae were clearly underestimated on discharge, suggesting a significantly higher amount of patients with long-term sequelae than earlier reported. Thorough examinations before discharge and during follow-up will likely uncover a higher morbidity also after methanol poisonings in general.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1086488

Incidence and causative agents of chemical eye injuries in Switzerland

Tschopp M, Krähenbühl P, Tappeiner C, Kupferschmidt H, Quarroz S, Goldblum D, Frueh BE. Clin Toxicol 2015; online early:

doi: 10.3109/15563650.2015.1094702:

Context

Chemical eye injuries are ophthalmological emergencies with a high risk of secondary complications and severe visual loss. Only limited epidemiological data for such injuries are available for many countries.

Patients and methods

We performed two independent studies. The cause of chemical eye injuries was assessed with a prospective questionnaire study. Questionnaires were sent to all ophthalmologists in Switzerland. A total of 163 patients (205 eyes) were included, between December 2012 and October 2014. Independent of the questionnaire study, the incidence of chemical eye injuries was assessed with a retrospective cohort study design using the database of the mandatory accident insurance.

Results

Ophthalmological questionnaires revealed that plaster/cement (20.5%), alkaline (12.2%) and acid (10.2%) solutions caused the highest number of chemical injuries. Only 2% of all injuries were classified as grade III and none as grade IV (Roper-Hall classification). The official toxicological information phone-hotline was contacted in 4.3% of cases. Using data from the accident insurance, an incidence of chemical eye injuries of about 50/100 000/year was found in the working population.

Conclusion

Here, we present data on the involved agents of chemical eye injuries in Switzerland, and also the incidence of such injuries in the working population. This may also help to assess the need for further education programs and to improve and direct preventive measures.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1094702

Death following intentional ingestion of e-liquid

Chen BC, Bright SB, Trivedi AR, Valento M. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1090579:

Context

Electronic cigarette (e-cigarette) use is growing within the United States, resulting in both intentional and unintentional exposures to concentrated liquid nicotine or "e-liquid." Nicotine has been culpable for severe poisoning and deaths in the past. However, sources of nicotine have traditionally been from cigarettes, cigars, or pesticides. Fatalities due to liquid nicotine are rare, and fatalities following ingestion of e-liquid are even scarcer.

Case

We present a case of a 24-year-old woman who intentionally ingested up to 3000 mg of liquid nicotine intended for e-cigarette use. She was found in pulseless electrical activity and had return of spontaneous circulation (ROSC) after undergoing approximately 10 min of cardiopulmonary resuscitation with a blood pressure of 74/53 mmHg and a pulse rate of 106 beats/min. Despite aggressive supportive care, she ultimately died after she was found to have multiple acute infarcts, consistent with severe anoxic brain injury, on magnetic resonance imaging. The patient's toxicologic testing, obtained shortly after ROSC, was notable for plasma nicotine and cotinine levels each >1000 ng/mL.

Discussion

This fatality highlights the potential toxicity associated with suicidal ingestion of liquid nicotine.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1090579

Urine uranium concentrations and renal function in residents of the United States – 2001 to 2010

Okaneku J, Vearrier D, McKeever R, LaSala G, Greenberg MI. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1094704:

Context

Animal model studies have demonstrated that subchronic oral uranium exposure is associated with renal dysfunction. Little is known about the effects of environmental exposure to uranium in humans.

Objective

To determine whether environmental exposure to uranium is associated with alterations in renal function among residents of the United States.

Methods

We analyzed data from the National Health and Nutrition Examination Survey (NHANES) 2001–2010. Inclusion criteria included the measurement of urine uranium concentration, serum creatinine (sCr), and urine albumin-creatinine ratio. Exclusion criteria included a reported history of diabetes mellitus. Urine uranium concentrations were normalized to urinary creatinine. Respondents with and without detectable urine uranium concentrations were compared using Welch's *t*-test for urine albumin-creatinine ratio and sCr and using Fisher's exact test for a reported history of renal disease. Regression analysis was performed to assess for an association between urine uranium concentration and urine albumin-creatinine ratio, sCr, or a reported history of renal disease.

Results

Uranium was detectable in the urine of 74.1% (n = 9025) of respondents. Urine albumin-creatinine ratio was significantly greater in respondents with detectable urine uranium concentrations (mean 4.84 ± 45.8 mg/g) compared to respondents without detectable urine uranium concentrations (mean 0.77 ± 3.7 mg/g) (p < 0.001). There was no significant difference between the groups with respect to sCr or a reported history of renal disease. Regression analysis did not show a statistically significant association between urine uranium concentration and urine albumin-creatinine ratio (p = 0.45), sCr (p = 0.71), or a reported history of renal disease (p = 0.05).

Conclusions

In this study, a high proportion of the U.S. population had exposure to uranium. We demonstrated an association between detectable urine uranium concentrations and microalbuminuria in residents of the United States but no association with clinical renal disease.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1094704

The pharmacokinetics and extracorporeal removal of *N*-acetylcysteine during renal replacement therapies

Hernandez SH, Howland M, Schiano TD, Hoffman RS. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1100305:

Objective

Acetaminophen-induced fulminant hepatic failure is associated with acute kidney injury, metabolic acidosis, and fluid and electrolyte imbalances, requiring treatment with renal replacement therapies. Although antidote, acetylcysteine, is potentially extracted by renal replacement therapies, pharmacokinetic data are lacking to guide potential dosing alterations. We aimed to determine the extracorporeal removal of acetylcysteine by various renal replacement therapies.

Methods

Simultaneous urine, plasma and effluent specimens were serially collected to measure acetylcysteine concentrations in up to three stages: before, during and upon termination of renal replacement therapy. Alterations in pharmacokinetics were determined by applying standard pharmacokinetic equations.

Results

Over 2 years, 10 critically ill patients in fulminant hepatic failure requiring renal replacement therapy coincident with acetylcysteine were consecutively enrolled. All 10 patients required continuous venovenous hemofiltration (n = 10) and 2 of the 10 also required hemodialysis (n = 2). There was a significant alteration in the pharmacokinetics of acetylcysteine during hemodialysis; the area under the curve (AUC) decreased 41%, the mean extraction ratio was 51%, the mean hemodialytic clearance was 114.01 ml/kg/h, and a mean 166.75 mg/h was recovered in the effluent or 41% of the hourly dose. Alteration in the pharmacokinetics of acetylcysteine during continuous venovenous hemofiltration did not appear to be significant: the AUC decreased 13%, the mean clearance was 31.77 ml/kg/h and a mean 62.12 mg/h was recovered in the effluent or 14% of the hourly dose.

Conclusions

There was no significant extraction of acetylcysteine from continuous venovenous hemofiltration. In contrast, there was significant extracorporeal removal of acetylcysteine during hemodialysis. A reasonable dose adjustment may be to double the IV infusion rate or possibly supplement with oral acetylcysteine during hemodialysis.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1100305

Evidence for the changing regimens of acetylcysteine

Chiew AL, Isbister GK, Duffull SB, Buckley NA. Br J Clin Pharmacol 2015; online early: doi: 10.1111/bcp.12789:

Abstract and full text available from: http://dx.doi.org/10.1111/bcp.12789

Summary statement: New guidelines for the management of paracetamol poisoning in Australia and New Zealand

Chiew AL, Fountain JS, Graudins A, Isbister GK, Reith D, Buckley NA. Med J Aust 2015; 203: 215-8.

Abstract and full text available from: http://dx.doi.org/10.5694/mja15.00614

Activated charcoal for acute overdose: a reappraisal

Juurlink DN. Br J Clin Pharmacol 2015; online early: doi: 10.1111/bcp.12793:

Abstract and full text available from: http://dx.doi.org/10.1111/bcp.12793

Carbon monoxide poisoning deaths in the United States, 1999 to 2012

Sircar K, Clower J, Shin MK, Bailey C, King M, Yip F. Am J Emerg Med 2015; 33: 1140-5.

Abstract and full text available from: http://dx.doi.org/10.1016/j.ajem.2015.05.002

Incidence of serum sickness after the administration of Australian snake antivenom (ASP-22)

Ryan NM, Kearney RT, Brown SGA, Isbister GK. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1101771:

Context

Serum sickness is a delayed immune reaction resulting from the injection of foreign protein or serum. Antivenom is known to cause serum sickness but the incidence and characteristics are poorly defined.

Objective

To investigate the incidence and clinical features of serum sickness following the administration of Australian snake antivenoms.

Materials and methods

This was a prospective cohort study of patients recruited to the Australian Snakebite Project who received snake antivenom from November 2012 to March 2014. Demographics, clinical information, laboratory tests and antivenom treatment were recorded prospectively. Patients administered antivenom were followed up at 7–10 days and 6 weeks' post-antivenom. The primary outcome was the proportion with serum sickness, pre-defined as three or more of: fever, erythematous rash/urticaria, myalgia/arthralgia, headache, malaise, nausea/vomiting 5–20 days post-antivenom.

Results

During the 16-month period, 138 patients received antivenom. 23 were not followed up (unable to contact, tourist, child, bee sting) and 6 died in hospital. Of 109 patients followed up, the commonest reason for antivenom was venom induced consumption coagulopathy in 77 patients. An acute systemic hypersensitivity reaction occurred post-antivenom in 25 (23%) and 8 (7%) were severe with hypotension. Serum sickness occurred in 32/109 (29%) patients, including 15/37 (41%) given tiger snake, 6/15 (40%) given polyvalent and 4/23 (17%) given brown snake antivenom. There was no association between the volume of antivenom and serum sickness, $p = 0.18$. The commonest effects were lethargy, headache, muscle/joint aches and fever.

Discussion

The incidence of serum sickness after snake antivenom in Australia was higher than earlier investigations which failed to define symptoms or follow-up patients, but similar to more recent studies of antivenoms in the United States.

Conclusion

Serum sickness is common with Australian snake antivenom but does not appear to be predictable based on the volume of antivenom administered.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1101771

Application of a dynamic in vitro model with real-time determination of acetylcholinesterase activity for the investigation of tabun analogues and oximes

Worek F, Herkert NM, Koller M, Thiermann H, Wille T. Toxicol Vitro 2015; online early: doi: 10.1016/j.tiv.2015.09.010:

Abstract and full text available from: http://dx.doi.org/10.1016/j.tiv.2015.09.010

TOXICOLOGY

General

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