Current Awareness in Clinical Toxicology

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CONTENTS

CURRENT AWARENESS PAPERS OF THE MONTH

Tramadol overdose causes seizures and respiratory depression but serotonin toxicity appears unlikely

Ryan NM, Isbister GK. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1036279:

Context

Tramadol is a commonly used centrally acting analgesic associated with seizures and suspected to cause serotonin toxicity in overdose.

Objective

This study sought to investigate the effects of tramadol overdose, and included evaluation for serotonin toxicity based on the Hunter Serotonin Toxicity Criteria where the seven clinical features of spontaneous clonus, inducible clonus, ocular clonus, agitation, diaphoresis, tremor and hyperreflexia are examined for in all patients taking serotonergic medications; seizures and central nervous system depression.

Materials and methods

This was an observational cases series based on a retrospective review of tramadol overdoses (> 400 mg) admitted to a tertiary toxicology unit from November 2000 to June 2013. Demographic details, information on ingestion (dose and co-ingestants), clinical effects,

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complications (seizures, serotonin toxicity and cardiovascular effects) and intensive care unit (ICU) admission were extracted from a clinical database.

Results

There were 71 cases of tramadol overdose (median age: 41 years, range: 17–69 years; and median ingested dose: 1000 mg, interquartile range [IQR]: 800–2000 mg). Seizures were dose related and occurred in 8 patients, one of them co-ingested a benzodiazepine compared with 16 patients without seizures. There were no cases of serotonin toxicity meeting the Hunter Serotonin Toxicity Criteria. Tachycardia occurred in 27 and mild hypertension occurred in 32. The Glasgow Coma Score was < 15 in 29 and < 9 in 5 patients; three co-ingested tricyclic antidepressants and two tramadol alone (3000 mg and 900 mg). Respiratory depression occurred in 13, median dose: 2500 (IQR: 1600–3000) mg which was significantly different ($p = 0.003$) to patients without respiratory depression, median dose: 1000 (IQR: 750–1475) mg. Eight patients were admitted to ICU, five due to co-ingestant toxicity and three for respiratory depression.

Discussion

Tramadol overdose was associated with a significant risk of seizures and respiratory depression in more severe cases, both which appear to be related to the ingested dose. There were no cases of serotonin toxicity, while opioid-like effects and adrenergic effects were prominent. Conclusion. Tramadol overdose is associated with seizures and respiratory depression, but is unlikely to cause serotonin toxicity.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1036279

Extracorporeal treatment for valproic acid poisoning: systematic review and recommendations from the EXTRIP workgroup

Ghannoum M, Laliberté M, Nolin TD, MacTier R, Lavergne V, Hoffman RS, Gosselin S. Clin Toxicol 2015; 53: 454-65.

Background

The EXtracorporeal TReatments In Poisoning (EXTRIP) workgroup presents its systematic review and clinical recommendations on the use of extracorporeal treatment (ECTR) in valproic acid (VPA) poisoning.

Methods

The lead authors reviewed all of the articles from a systematic literature search, extracted the data, summarized the key findings, and proposed structured voting statements following a predetermined format. A two-round modified Delphi method was chosen to reach a consensus on voting statements and the RAND/UCLA Appropriateness Method was used to quantify disagreement. Anonymous votes were compiled, returned, and discussed in person. A second vote was conducted to determine the final workgroup recommendations.

Results

The latest literature search conducted in November 2014 retrieved a total of 79 articles for final qualitative analysis, including one observational study, one uncontrolled cohort study with aggregate analysis, 70 case reports and case series, and 7 pharmacokinetic studies, yielding a very low quality of evidence for all recommendations. Clinical data were reported for 82 overdose patients while pharmaco/toxicokinetic grading was performed in 55 patients. The workgroup concluded that VPA is moderately dialyzable (level of evidence = B) and made the following recommendations: ECTR is recommended in severe VPA poisoning (1D); recommendations for ECTR include a VPA concentration > 1300 mg/L (9000 µmol/L)(1D), the presence of cerebral edema (1D) or shock (1D); suggestions for ECTR include a VPA concentration > 900 mg/L (6250 µmol/L)(2D), coma or respiratory depression requiring mechanical ventilation (2D), acute hyperammonemia (2D), or pH = 7.10 (2D).

Cessation of ECTR is indicated when clinical improvement is apparent (1D) or the serum VPA concentration is between 50 and 100 mg/L (350–700 µmol/L)(2D). Intermittent hemodialysis is the preferred ECTR in VPA poisoning (1D). If hemodialysis is not available, then intermittent hemoperfusion (1D) or continuous renal replacement therapy (2D) is an acceptable alternative.

Conclusions

VPA is moderately dialyzable in the setting of overdose. ECTR is indicated for VPA poisoning if at least one of the above criteria is present. Intermittent hemodialysis is the preferred ECTR modality in VPA poisoning.

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The effects of intravenous lipid emulsion on prolongation of survival in a rat model of calcium channel blocker toxicity

Kang C, Kim DH, Kim SC, Lee SH, Jeong JH, Kang T-S, Shin I-W, Kim RB, Lee DH. Clin Toxicol 2015; online early: doi: 10.3109/15563650.2015.1045979:

Context

Intravenous lipid emulsion (ILE) has been shown to ameliorate the toxicity of lipid-soluble agents in animal studies and clinical cases.

Objectives

To investigate the therapeutic effects of ILE in a rat model of toxicity from calcium channel blockers (CCBs), including diltiazem and nicardipine.

Methods

Two sets of experiments of CCB poisoning were conducted. In the first set, 14 male Sprague-Dawley rats were sedated and treated with ILE or normal saline (NS), followed by continuous intravenous infusion of diltiazem (20 mg/kg/h). In the second experiment, the study protocol was the same except the infusion of nicardipine (20 mg/kg/h). The total dose of infused drug and the duration of survival were measured. In addition, mean arterial pressure and heart rate were monitored.

Results

Survival was prolonged in the ILE group (48.4 ± 11.3 vs. 25.0 ± 3.7 min; p = 0.002). Furthermore, the cumulative mean lethal dose of diltiazem was higher in the ILE group (16.1 ± 3.8 mg/kg) than in the NS group (8.3 ± 1.1 mg/kg) (p = 0.002). With nicardipine poisoning, survival was also prolonged in the ILE group (71.0 ± 8.3 min vs. 30.6 ± 6.1 min; p = 0.002). The cumulative mean lethal dose was higher in the ILE group than in the NS group (23.7 ± 2.8 mg/kg vs. 10.2 ± 2.0 mg/kg; p = 0.002).

Conclusions

ILE pretreatment prolonged survival and increased the lethal dose in a rat model of CCB poisoning using diltiazem and nicardipine.

Full text available from: http://dx.doi.org/10.3109/15563650.2015.1045979

Intoxications by the dissociative new psychoactive substances diphenidine and methoxphenidine

Helander A, Beck O, Bäckberg M. Clin Toxicol 2015; 53: 446-53.

Background

Diphenidine (1-(1,2-diphenylethyl)piperidine) and its 2-methoxylated derivative methoxphenidine (MXP, 2-MeO-diphenidine) are substances with dissociative effects that

were recently introduced for "recreational" purpose through the online-based sale of new psychoactive substances (NPS). A number of analytically confirmed non-fatal intoxications associated with diphenidine or MXP have occurred in Sweden and were included in the STRIDA project.

Study design

Observational case series of consecutive patients with admitted or suspected intake of NPS and requiring intensive treatment in an emergency room and hospitalization in Sweden.

Patients and methods

Blood and urine samples were collected from intoxicated patients presenting at emergency departments all over the country. NPS analysis was performed by multi-component liquid chromatography-mass spectrometry methods. Data on clinical features were collected during telephone consultations with the Poisons Information Centre and retrieved from medical records. Information was also obtained from online drug discussion forums.

Case series

Over a 12-month period from January to December 2014, 750 cases of suspected NPS intoxication originating from emergency departments were enrolled in the STRIDA project of which 14 (1.9%) tested positive for diphenidine and 3 (0.4%) tested positive for MXP. Co-exposure to several other NPS (e.g., 5-/6-(2-aminopropyl)benzofuran, 2-4-bromo-methcathinone, butylone, 3,4-dichloromethylphenidate, 5-methoxy-N-isopropyltryptamine, methiopropamine, and alpha-pyrrolidinopentiothiophenone), also including other dissociative substances (3-/4-methoxyphencyclidine), and classical drugs of abuse (e.g., cannabis and ethanol) was documented in 87% of these cases. The 17 patients were aged 20-48 (median: 32) years, and 13 (76%) were men. They commonly presented with hypertension (76%), tachycardia (47%), anxiety (65%), and altered mental status (65%) including confusion, disorientation, dissociation, and/or hallucinations. Eight patients (47%) displayed severe intoxication (Poisoning Severity Score 3). The diphenidine- or MXP-positive patients required hospitalization for 1-3 (median: 2) days. In addition to standard supportive therapy, half of the cases were treated with benzodiazepines and/or propofol.

Conclusion

The adverse effects noted in analytically confirmed cases of NPS intoxication involving diphenidine or MXP were similar to those reported for other dissociative substances such as ketamine and methoxetamine. However, the high proportion of polysubstance use might have played a role in the intoxication and clinical features in some cases.

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Circulating kidney injury molecule-1 predicts prognosis and poor outcome in patients with acetaminophen-induced liver injury

Antoine DJ, Sabbisetti VS, Francis B, Jorgensen AL, Craig DGN, Simpson KJ, Bonventre JV, Park BK, Dear JW. Hepatology 2015; online early: doi: 10.1002/hep.27857:

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CHEMICAL WARFARE, BIOLOGICAL WARFARE AND RIOT CONTROL AGENTS

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INDEX

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