Journal of Clinical Toxicology
ISSN: 2161-0495

Gheshlaghi et al. J Clinic Toxicol 2011, S:1
DOI: 10.4172/2161-0495.S1-001

Case Report Open Access

Status Epilepticus Following Intravenous Injection of Pyrethroid Insecticide for Attempted Suicide: A Not Yet Reported Case

Farzad Gheshlaghi[1,2*], Nastaran Eizadi-Mood[1,2], Mohammad Reza Piri-Ardakani[2,3], Ali Mohammad Sabzghabaee[2] and Alireza Firoozfar[2,3]

[1]*Department of Clinical Toxicology and Forensic Medicine, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran*
[2]*Isfahan Clinical Toxicology Research Center, Isfahan University of Medical Sciences, Isfahan, Iran*
[3]*Medical Students Research Center, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran*

Abstract

Pyrethroid insecticides are very widely used in agriculture and household due to high effectiveness and low toxicity to humans. Intravenous injection with pyrethroids is rarely reported. We describe a 44-year-old male presented with status epilepticus following intravenous injection of a pyrethroid insecticide Cypermethrin. The pathophysiology, clinical features, and management of pyrethroid poisoning are discussed in this article.

Keywords: Cypermethrin; Pyrethroid insecticide; Status epilepticus; Poisoning

Introduction

Despite the wide utilization of all pesticides in the developed world, 99% of all acute pesticide poisoning occurs in developing countries, attempted suicides account for two thirds of all pesticide poisoning fatalities [1]. Pyrethrins are natural extracts derived from flowers of *chrysanthemam cinerarifolium* and *C. cocineum*. Pyrethroids are synthetic analogues of these natural abstracts. Pyrethroids are widely used as insecticides and are also used in the topical treatment of scabies and lice. Because of their increased sodium channel sensitivity, smaller body size, and lower body temperature, insects are 2,250 times more prone to toxicity by pyrethroids than humans [2]. In addition, humans are relatively protected from pyrethroids because of their poor dermal absorption and rapid metabolism to non toxic metabolites. Systemic toxicity of these pesticides is rarely reported despite their widespread use. Local signs of toxicity include paresthesia if skin is contaminated and gastrointestinal irritation if the route of exposure is oral intake. Due to the slow rate of absorption of pyrothroids via skin, systemic toxicity is not expected [3,4]. Fatal pyrethroid poisoning reports are not common in the toxicology literature. To our best of knowledge there are only a few severe, systemic, life-threatening pyrethroid-induced illnesses which have been reported in developing countries [5-7].

Here, we describe a case of Cypermethrin poisoning presenting with status epilepticus, its clinical courses, management and outcome.

Case Report

A 44-year-old male was admitted to a rural hospital with repeated episodes of generalized tonic-clonic seizures following deliberate injection of 3-4 ml Cypertmethrin 1.2% in the right inguinal area.

No history of seizure, head trauma, use of any medication or abuse of any other drug but opium in the recent past was found.

The familial and past medical histories were non-contributing, except for a positive HCV-Ab marker.

On admission to the local hospital, his Glasgow coma scale (GCS)-score was 6-7 and was getting generalized tonic clonic seizures within a few minutes of injecting the poison .His heart rate, blood pressure, and respirations on admission were 85 beats per minute (bpm), 100/60 mm Hg, and 40 per min, respectively. He was having whitish, frothy oronasal discharges which, based on color and odor, seems contained cypermethrin emulsion. No visible intraoral abnormality was found. Physical examination of lungs, heart, and abdomen was normal, as were the deep tendon reflexes.

The patient was treated supportively with crystalloid fluids, endotracheal intubation,gastric lavage and activated charcoal (50 g). He was treated with intra venous Diazepam (10 mg), midazolam (5 mg), phenytoin (500 mg), Phenobarbital (300 mg) to control seizures but there were more episodes of seizure within 3-hours of admission.

Four hours after the admision of the patient to the clinical poisoning department of a referral teaching hospital, he had persistant seizures with low frequency. Then the patient was tranferred to intensive care unit (ICU). The initial vital signs of the patient were blood pressure 100/50 mmHg, pulse rate 110 beats\min with a respiratory rate of 25 beats\min and the respiratory sounds were normal. For the management of his seizures he was given a 0.3 mg/kg dose of Midazolam as a IV bolus dose which was followed by a 0.1 mg/kg/hr as a maintenance dose and Thiopental-sodium 3 mg/kg as a IV bolus dose followed by 1.5 mg/kg/hr as a maintenance dose. Finally the seizures were controlled and the patient was repiratory-wise supported by mechanical ventilation.

Ten to fifteen hours after the cessation of seizures the first thiopental sodium and then midazolam infusions were tapered and stopped.

An initial arterial blood gas analysis revealed metabolic alkalosis with a pH of 7.44, base excess of 5.8 mmol/L, $Pa0_2$ 48.3 mmHg, and $PaC0_2$ 45.9 mm Hg. Other laboratory data included an increased white blood cell count (WBC) 22.2 000/mm^3 (normal 4.0-10.0 X I d) , red blood cell (RBC) count 5.1 X mil/mm^3 (normal 3.9-5.9106/mm^3), hemoglobin 15.1 g/dL (normal male 14.0-18.0), and hematocrit 45% (normal male 42-52). Renal function tests included creatinine 1.1 mg/

***Corresponding author:** Farzad Gheshlaghi, Department of Clinical Toxicology and Forensic Medicine, Isfahan University of Medical Sciences, Isfahan, Iran, Tel: +98311-2222127; Fax: +98311-2222255; E mail: Gheshlaghi@med.mui.ac.ir

Received October 20, 2011; **Accepted** November 16, 2011; **Published** November 18, 2011

Citation: Gheshlaghi F, Eizadi-Mood N, Piri-Ardakani MR, Sabzghabaee AM, Firoozfar A (2011) Status Epilepticus Following Intravenous Injection of Pyrethroid Insecticide for Attempted Suicide: A Not Yet Reported Case. J Clinic Toxicol S1:001. doi:10.4172/2161-0495.S1-001

dL (normal 0.6-1.6) and blood urea nitrogen 16 mg/dL (normal 8-24). Liver enzyme concentrations in serum were aspartate aminotransferase 24 U/L (up to 40) and alanine aminotransferase 21 U/L (male less than 40) Creatine kinase 650 U/L (male normal 30-190). Serum electrolyte levels were Na 142 mEq/L, K 5 mEq/L. In the macroscopic urine analysis, a 2+ blood was detected and the microscopic test also showed 25-30 RBC counts in a high power filed and a few amorph urate crystals was also seen and no other abnormality was detected.

On next laboratory evaluation about 6 hours after ICU admission the all findings were normal except CPK that was 650 U/I (male normal 30-190).

After 36 hours he was extubated and discharged from ICU. Reassessment of the patient a few hours after ICU discharge showed complete consciousness and there had been no other episode of seizure, hyperactivity, tremor, or motor ataxia. He received a full supportive care and was discharged to have a psychiatrist and followed up by electroencephalogram (EEG) for 3 months to check if any more episode of convulsion happens.

Discussion

Abuse of intramuscular, intravenous and subcutaneous injections of organophosphates for attempted suicide was reported in previous literature [8,9]. Contrarily, the data on the use of pyrethroid type insecticides for the same reasons are quite rare. In our literature review, there was reported only a case of intravenous and subcutaneous injection of a pyrethroid resulted in local toxicity [10] and a single case report of pyrethroid poisoning presenting with status epilepticus [11]. Seizures are a known manifestation of pyrethroid poisoning but there was not any case report of status epilepticus after intravenous injection of pyrethroid.

Marked increase in adrenal activation occurs after poisoning with both types I and II pyrethroids. Type II pyrethroids are generally more toxic than type I. Some authors describe type I pyrethroid poisoning as a “T-syndrome” (coarse tremors) and type II pyrethroid poisoning as a “CS-syndrome” (choreoathetosis or clonic seizures and salivation) [12].

Pyrethroids produce prolonged opening of membrane sodium channels resulting in membrane depolarization, repetitive discharges, and synaptic disturbances leading to hyper excitatory symptoms of poisoning. Only low pyrethroid concentrations are necessary to modify sensory neuron function. Type II pyrethroids also decrease chloride currents through voltage-dependent chloride channels and this action probably contributes the most to the features of poisoning with Type II pyrethroids . At relatively high concentrations, pyrethroids can also act on gamma-amino butyric acid-gated chloride channels, which may be responsible for the seizures seen with severe Type II poisoning [11]. There are suggestions that voltage-sensitive calcium ($Ca^{(2+)}$) channels (VSCC) may also be important targets of pyrethroid action. However, currently the data available neither supports nor refutes conclusively the hypothesis that effects on VSCC are important to the acute neurotoxicity of pyrethroids [11].These are the mechanisms that suggested as a causes of seizure in pyrethroid poisoning but because Cypermethrin is a commercial product containing 20% permethrin, 70% xylene, and 10% anionic and nonionic surfactants, xylene might be an important cause of the CNS signs and symptoms (decrease level of consciousness and seiziures).

This article was originally published in a special issue, **Epidemiology of Poisoning** handled by Editor(s). Dr. John F Gamble, Consultant, Somerset, New Jersey, USA; Dr. Monath Sanjaya Kuruppu, Monash University, Australia

Regarding the fact that still there is no antidote for pyrethrin and pyrethroid poisoning, the main measure of treatment for this toxicity remains symptomatic and supportive. Pyrethroid paresthesias are treated by decontamination of the skin. Seizures due to systemic poisoning are sometimes difficult to control with anticonvulsants [13]. Pentobarbitone, is reported effective as a useful therapy against systemic Type II pyrethroid poisoning in rats, probably due to its dual action as a chloride channel agonist and a membrane stabilizer [13]. According to our experience in controlling refractory seizure we suggested use of high dose of midazolam and thiopental sodium in the same cases. In conclusion, the patients presenting with Status epilepticus following intravenous injection of pyrethroid in suicidal attempts should be evaluated carefully so that early diagnosis, immediate planning for ICU admission, intubation, intravenous infusions of high dose of Midazolam and Thiopental sodium for control of seizures is recommended.

References

1. Koh D, Jeyaratnam J (1996) Pesticide’s hazards in developing countries. Sci Total Environ 188: 78–85.
2. Bradberry SM, Cage SA, Proudfoot AT, Vale JA (2005) Poisoning due to pyrethroids. Toxicol Rev 24: 93-106.
3. Soderlund DM, Clark JM, Sheets LP, Mullin LS, Piccirillo VJ et al (2002) Mechanisms of pyrethroid neurotoxicity:implications for cumulative risk assessment. Toxicology 171: 3-59.
4. Ray DE, Forshaw PJ (2000) Pyrethroid insecticides: poisoning syndromes, synergies and therapy. J Toxicol Clin Toxicol 38: 95-101.
5. Goldberg LH, Shupp D, Weitz HH, Zeccardi JA (1982) Injection of household spray insecticide. Ann Emerg Med 11: 626-629.
6. Buchman MT (2000) Upper extremity injection of household insecticide: a report of five cases. J Hand Surg 25: 764-767.
7. Wedin GP, Jones RR (1984) Parenteral administration of hydrocarbons. J Toxicol Clin Toxicol 22: 485-492.
8. Guven M, Unluhizarci K, Goktas Z, Kurtoglu S (1997) Intravenous organophosphate injection: an unusual way of intoxication. Hum Exp Toxicol 16: 279-280.
9. Hadimioglu N, Dosemeci L, Arici G, Ramazanoglu A (2002) Systemic organophosphate poisoning following the percutaneous injection of insecticide: Case report. Skin Pharmacol Appl Skin Physiol 15: 195-199.
10. Frank LoVecchio, Jason Knight (2005) Injection of pyrethroids without significant sequelae. American Journal of Emergency Medicine 23: 406.
11. Ghosh S, Ahlawat A, Rai KK, Arora A (2009) An unusual cause of status epilepticus. Indian J Crit Care Med 13: 106-107.
12. Poulos L, Athanaselis S, Coutselinis A (1982) Acute intoxication with cypermethrin (NRDC 149). Toxicol Clin Toxicol 19: 519–520.
13. Ray DE, Forshaw PJ (2000) Pyrethroid insecticides: Poisoning syndromes, synergies, and therapy. J Toxicol Clin Toxicol 38: 95-101.