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

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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 chrysanthemum cinerarifolium and C. coccineum. Pyrethroids are synthetic analogues of these natural extracts. 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 pyrethroids 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 Cypermethrin 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 admission of the patient to the clinical poisoning department of a referral teaching hospital, he had persistent seizures with low frequency. Then the patient was transferred 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 respiratory-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, PaO_2 48.3 mmHg, and PaCO_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 10^9/L) , red blood cell (RBC) count 5.1 X mil/mm^3 (normal 3.9-5.910^6/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/

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poisoning. Only low pyrethroid concentrations are necessary to modify and synaptic disturbances leading to hyper excitatory symptoms of channels resulting in membrane depolarization, repetitive discharges, [12]. as a “T-syndrome” (coarse tremors) and type II pyrethroid poisoning toxic than type I. Some authors describe type I pyrethroid poisoning both types I and II pyrethroids. Type II pyrethroids are generally more of pyrethroid. 

Seizures are a known manifestation of pyrethroid poisoning but there report of pyrethroid poisoning presenting with status epilepticus [11]. 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