Case Report

Prallethrin poisoning: A diagnostic dilemma

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Abstract

Pyrethroid insecticides are very widely used in agriculture and household due to their high effectiveness and low toxicity in humans. Despite their extensive worldwide use, there are a few reports of human pyrethroid poisoning. The poisoning has a varied presentation and its symptoms overlap with those of other compounds, which can lead to misdiagnosis. We present a case of poisoning with prallethrin, a pyrethroid compound, commonly available as All-Out.

Key words: Prallethrin, pyrethroid insecticides, status epilepticus

Introduction

Pyrethroids are 2250 times more toxic to insects than mammals because insects have increased sodium channel sensitivity, smaller body size, and lower body temperature. Their ingestion gives rise to sore throat, nausea, vomiting, and abdominal pain within minutes. There may be mouth ulceration, increased secretions, and/or dysphagia. Systemic effects are seen 4-48 h after exposure. Dizziness, headache, and fatigue are common; palpitation, chest tightness, and blurred vision are less frequent; and coma and convulsions are the principal life-threatening features.[1]

Case Report

A 20-year-old girl was brought to the emergency department with a history of sudden onset convulsions. There was no history of fever, drug usage, trauma, or any past history of convulsions before the onset of convulsions. The patient was given a single dose of intravenous (IV) diazepam 10 mg for control of generalized tonic-clonic convulsion but since control over the convulsions was achieved, a loading dose of phenytoin 1 g was given. As the convulsions persisted, propofol 50 mg IV was given, which controlled the convulsions. Computed tomography (CT) scan head revealed no abnormality. Electroencephalograpy could not be done due to lack of facilities. Blood sugar, electrolytes, and arterial blood gas analysis showed no deviation from normal values. Noninvasive blood pressure, electrocardiography, and oxygen saturation monitoring was done. The patient was then shifted to the intensive care unit (ICU) and IV infusion of midazolam 0.1 mg/kg/h was initiated and phenytoin 100 mg given 8 hourly. There was no episode of convulsions thereafter. The patient developed hypotension for which inotropic support was started. A central venous cannulation was done to guide the fluid therapy and titrate the dose of inotrope. Breathing was normal and there was no need to mechanically ventilate the patient.

On arrival in the ICU, the relatives accompanying gave some history of domestic dispute and doubt of ingestion of some substance. A Ryle's tube was inserted and gastric lavage given. The patient had complaint of nausea, vomiting, and abdominal discomfort for which symptomatic treatment was given. The patient's sensorium improved the next day and she gave the history of ingestion of contents of two bottles of mosquito repellent available in the house commercially marketed as All-Out (prallethrin 1.6% w/w liquid, 35 mL in each bottle, that is, total dose of 1120 mg). The patient had excessive secretions so atropine 0.6 mg IV was started at 4 hourly intervals, in addition to antiemetics and proton-pump inhibitors. The condition of the patient improved gradually and by 5th day the patient was shifted to the ward from where she was discharged on the 7th day.

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Discussion

Acute human poisoning from exposure to pyrethroids is rare and no clinical case of acute pyrethroid poisoning had been reported in the literature until the outbreak of acute deltamethrin poisoning in spraymen in China in 1982. After that, there have been few reports of pyrethroid poisoning, but most of them are of occupational poisoning.[2] Commonly used synthetic pyrethroid insecticides are Allethrin (Pynamin), Cyfluthrin (Baythroid), Cypermethrin (Ammo), Esfenvalerate (Asana), Fenvalerate (Pydrin), Flucythrinate (Pay-off), Fluvalenate (Mavrik), Permethrin (Ambush), Resmethrin (outdoor insect Fogger), Tetramethrin (Fleakiller II), and Tralomethrin (Scout).[1]

Prallethrin is a structural derivative of naturally occurring pyrethrins. Pyrethrin is an extract from the flower Chrysanthemum cinerarilifolium and is potent against insects. However, its use is limited by its rapid biodegradability. The increase in the potency and toxicity profile is due to the structural modifications.[3,4] The mechanism of pyrethroid toxicity is complex. Their main effects are on sodium and chloride channels. Pyrethroids modify the gating characters of voltage-sensitive sodium channels to delay their closure. A protracted sodium influx (referred to as sodium tail current) results which, if it is sufficiently large or long, lowers the action potential threshold and causes repetitive firing, which may be the mechanism of paresthesia. At relatively high concentrations, pyrethroids can also act on GABA-gated chloride channels, which may be responsible for the seizures.[1] Convulsions generally occur after consumption of doses above 500 mg and their frequency can be 10-30 times a day. Severe cases may be associated with pulmonary edema. Management of pyrethroid poisoning is mainly supportive care and symptomatic management as there is no specific antidote. Gastric lavage should be given. Atropine should be given to decrease secretions in cases with salivation and pulmonary edema. Low doses of atropine (0.5-10 mg) are generally sufficient in these cases.[2]

In this case, the patient presented with convulsions and was diagnosed as status epilepticus. It did not respond to a firstline benzodiazepine (diazepam) or secondline drug (phenytoin) but responded to propofol. Status epilepticus is defined as a continuous seizure lasting for at least 30 min or two or more discrete seizures between which the patient does not recover consciousness. In the 15-30 patients per 100,000 per year, who present in status epilepticus, mortality is as high as 10%.[5] Status epilepticus is a medical and neurologic emergency requiring prompt and aggressive treatment particularly for elderly individuals, in whom comorbid conditions may increase the severity of consequences in status epilepticus. Generalized convulsive status epilepticus (GCSE) is the most common and life-threatening type of status epilepticus, which may lead to systemic complications and neuronal damage. It is often fatal, if untreated or inadequately treated. The treatment of GCSE should begin with basic life support measures and monitoring. Ideally, pharmacologic treatment should be easy to administer and fast acting. The choice of agent to be used as firstline treatment depends on individual patient characteristics.[6] GCSE commonly occurs in patients with no history of seizure or epilepsy. The morbidity associated with status epilepticus is related to the underlying precipitating factors, age of the patient, and duration of the seizure activity.[7] In our case, since there was no history of epilepsy and all other parameters were normal the general treatment of status epilepticus was done. Later when the history revealed ingestion of prallethrin, gastric lavage was done, atropine started, and symptomatic treatment done along with continuous monitoring in the ICU.

Cardiac conduction disturbance due to prallethrin poisoning has been reported, which can probably occur due to its effect on sodium channels in the heart.[8] Pyrethroid poisoning can be easily misdiagnosed as organophosphate or organochlorine poisoning. There are few reports in literature where patients were mistakenly diagnosed as acute organophosphorous poisoning. To differentiate between these two kinds of pesticide poisoning, exposure history is more important.[9]

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