

Prallethrin And Status- A Rare Association

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Citation

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Abstract

INTRODUCTION Mosquito repellents marketed as vaporizers contain pyrethroids. Most data regarding toxicity of pyrethroids are gathered from dermal or inhalational absorption. Toxicity profile following oral intake is scarcely reported. This is the first case report following oral ingestion of prallethrin. **CASE REPORT** A 19 year old male presented to the ER in status epilepticus. Initial attention to ABC was given. He was managed in the ICU requiring mechanical invasive ventilation. No cause for his status could be determined after all investigations. It was on review of history with the patient later that the cause was determined. He had ingested an entire bottle of liquid mosquito repellent vaporizer after a domestic altercation. Patient has been symptom free on follow up off anti-epileptics. **DISCUSSION** Mosquito repellents contain pyrethroids which act on sodium channels and cause hyperexcitability of neurons. There are no known antidotes. Treatment is symptomatic.

CASE REPORT

A 19 year old healthy Asian boy presented to the ER of our hospital with seizures one hour prior to presentation preceded by unsteady gait. There was no history of trauma, fever or drug usage prior to the onset of seizures. There was no past or family history of seizures. On presentation to our hospital, immediate airway protection was done in view of persistent seizures. Pupils were dilated but reacting to light. There were no signs of bladder or bowel incontinence. Deep tendon reflexes were not elicitable at presentation. Blood sugars checked in the ER were normal. Oxygen saturation was 90%. He was administered intravenous lorazepam (4 mg repeated after 15 mins), phenytoin (1g loading dose), phenobarbitone (1g) and midazolam (2.5 mg/h and loading dose of 1.5 mg) with no control of seizures. The patient required intubation and mechanical ventilation in view of his low Glasgow coma scale. He needed to be paralysed to control the seizures. The MRI brain done was a normal study. Metabolic panel inclusive of electrolytes, renal and liver function tests sent at the time of admission were also normal. His paralytic agents were stopped on the third day and patient was put only on phenytoin. His sensorium improved and was gradually weaned of the ventilator by day 4. He was oriented and fully cooperative by day 4. As prior informant was a friend and parents, on repeated questioning the patient revealed the history of intake of the entire contents of a liquid vaporizer mosquito repellent which contains prallethrin (45 ml, 1.6 % w/w liquid). He was

discharged in a stable condition and is asymptomatic on follow up.

DISCUSSION

This case report describes the clinical manifestation following oral suicidal intake of prallethrin. The patient's ABC was given prime importance. Since no history was available at the time of admission and on all the investigations being normal, other rarer causes of status epilepticus were considered. Status epilepticus refers to a life-threatening condition in which the brain is in a state of persistent seizure. Definitions vary, but traditionally it is defined as one continuous unremitting seizure lasting longer than 30 minutes, or recurrent seizures without regaining consciousness between seizures for greater than 30 minutes¹.

Prallethrin is a structural derivative of naturally occurring pyrethrins. Pyrethrin is an extract from the flower *Chrysanthemum cinerariifolium* and is potent against insects. However its use is limited by its rapid biodegradability. Pyrethroids are the result of research and development efforts in the molecule of pyrethrin so that the potency is retained and is commercially viable^{2,3}. Prallethrin is one such result of these efforts. However with the increase in their potency the toxicity profile has also increased due to the structural modifications.

Pyrethroids exert their neurotoxic effects especially on insects. Their toxicity to humans is at least three orders of

magnitude lower than for insects⁴. Pyrethroids produce reversible impairment of motor function and 'knockdown' in flying insect species that may be followed by death, depending upon the exposure level. The primary action of pyrethroids is on the sodium channel. The interaction with sodium channels leads to a state of hyper excitable cells which is the presumed cause for its neurotoxicity. There is also a hypothesis that other targets like voltage sensitive calcium channel might also be involved. Pyrethroids act most readily on the tetrodotoxin resistant subtype of the sodium channel, expressed in the developing mammalian brain and in the adult dorsal root ganglia. The proportion of sodium channels modified is dose-dependent, but the duration of their hyper excitable state is determined by the structure of the pyrethroid and is independent of dose^{2,5-7}. Hence, the degree of hyperexcitability is dose-related, but the nature of this excitability is structure-dependent. The neurotoxicity of pyrethroids to mammals depends on the stereo chemical configuration at cyclopropane C-1 or the homologous position in compounds lacking the cyclopropanecarboxylate moiety. This structure-dependency is expressed in terms of the variable time constant of prolongation of the sodium current which varies continuously across a range of structures.

Prallethrin is a synthetic pyrethroid with fast knock-down activity against household insect pests. Prallethrin is almost insoluble in water but highly soluble in organic solvents, such as hexane, ethanol, acetone, toluene, etc. IPCS hazard classification of prallethrin is moderately hazardous⁸. An ideal therapeutic agent would antagonize the abnormal,

pyrethroid-evoked, sodium current but leave the normal one unchanged. The approaches have included phenobarbitone for its chloride conductance, ivermectin also for the same and mephensin⁶.

However this is the first case report on acute toxicity following oral ingestion of prallethrin.

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