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A Suicide Attempt: Deltamethrin Intoxication

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ABSTRACT

Poisoning is a frequent cause of emergency department visits, with attempted suicide accounting for up to two-thirds of all fatalities from pesticide poisoning. Despite extensive pyrethroid use worldwide, relatively few cases of human poisoning have been reported in the literature. Although most pesticide use occurs in the developed world, 99% of all acute pesticide poisoning occurs in developing countries. Deltamethrin poisoning is uncommon, with fewer than 10 deaths reported following ingestion or occupational exposure.

The present case report describes deltamethrin intoxication due to intentional oral ingestion in a suicide attempt, resulting in a life-threatening combination of seizures and coma due to non-convulsive status epilepticus.

LEARNING POINTS

- Up to two-thirds of all fatalities from pesticide poisoning are due to suicide.
- Organophosphate or organochloride poisoning can mimic pyrethroid poisoning.
- Poisoning with deltamethrin, which belongs to the pyrethroid family, is uncommon; there is no antidote, with supportive and symptom-directed treatment being the gold standard.

KEYWORDS

Deltamethrin, pyrethroid, poisoning, coma, non-convulsive status epilepticus

CASE DESCRIPTION

A 76-year-old man with depression and radiation-induced cystitis presented to the emergency department (ED) in a coma and with myoclonic seizures. He had ingested around 30 ml of an unknown chemical 2 hours before admission according to family members. The chemical was eventually identified as deltamethrin, an insecticide belonging to the pyrethroid family. The ambulance service was initially called due to vomiting and obtundation, associated with a strong chemical smell. The patient presented to the ED with a regular pulse, tachypnoea (respiratory rate 40 bpm) and SpO2 99% on room air. On examination he had mid-dilated pupils, sluggish reaction to light, horizontal roving eye movements, bilateral myoclonus, and a Glasgow Coma Scale of 6. Computed tomography (CT) of the head excluded obvious structural aetiologies for the clinical picture described. Arterial blood gas (ABG) analysis revealed mixed respiratory and metabolic acidosis, with hyperlactataemia, pH 7.29, PaO2 69 mmHg, PaCO2 51 mmHg, HCO3 22 mmol/l and lactate 22 mg/dl. A full blood count revealed a neutrophilic leucocytosis with a normal biochemistry panel except for stage 1 acute kidney injury.

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Further toxicology screening was normal apart from a falsely positive amphetamine finding due to cross-reactivity. Normal serum cholinesterase levels were observed (8067 U/l).

The patient was intubated for airway protection and admitted to ITU. Nasogastric lavage was performed and levetiracetam initiated. Within the first 12 h of ITU admission, the patient presented with incomplete cholinergic syndrome with myosis, bradycardia (35/min) requiring multiple doses of atropine, hypotension requiring vasopressors, prolonged QTc (490 ms) and fasciculations of both arms and the left leg. The patient was kept sedated as initial electroencephalographic (EEG) evaluation in the first 24 h of admission showed periodic rhythmic discharges suggesting non-convulsive status epilepticus (NCSE). Serial EEGs documented refractory status epilepticus requiring the addition of multiple antiepileptic drugs (AED) (levetiracetam, sodium valproate, phenobarbital, clonazepam and perampanel) until EEG documented burst-suppression allowing for gradual AED tapering. Although the patient recovered from super-refractory NCSE, he demonstrated severe neurocognitive and motor impairment at ITU discharge. However, following intensive neuropsychological and motor rehabilitation, he made a full recovery back to baseline.

DISCUSSION

Insecticides containing pyrethroids such as deltamethrin are considered to have a lower toxicity potential than other insecticides like those containing organophosphates (OP) [1]. There are several similarities between pyrethroid, OP and organochloride (OC) poisoning but management differs so physicians working in emergency departments and ITU should be aware of this toxidrome, which can clinically mimic and be misdiagnosed as OP or OC poisoning [1].

Moreover, there is no inhibition of serum cholinesterases in pyrethroid poisoning, requiring a lower atropine dosage (usually less than 10 mg), with a better prognosis reported. To differentiate between these two kinds of pesticide intoxication, exposure or collateral history is of the utmost importance [1].

The central nervous system (CNS) is the target for pyrethroid intoxication, which directly binds and modifies the gating characteristics of voltage-sensitive sodium channels delaying their closure [2]. At higher concentrations, they act on GABA-gated chloride channels resulting in seizure activity. CNS symptoms are thought to be dose-dependent but further studies are required to ascertain the exact mechanism of action related to CNS toxicity [3]. Cardiac symptoms have also been described. It is thought that neuroexcitation can lead to cardiac involvement due to a sympathetic surge or to direct blockade of myocardial sodium channels, leading to delayed closure and hyperexcitability [4].

Poisoning with insecticides is common in areas where agriculture is one of the main sources of economic income. Although case series describing intoxication due to occupational exposure have been reported, only a few cases of deltamethrin intoxication after oral ingestion are described in the literature [5].

Generalized signs and symptoms of poisoning include headache, nausea and vomiting, hypersalivation, irritability, ataxia, dizziness, fasciculations, seizures, pulmonary oedema and death [4]. It has been demonstrated, in rats, that deltamethrin is excreted in urine and faeces within 2–4 days. However, the cyano group, which is converted to thiocyanate, is excreted more slowly. A study carried out on three volunteers given a single oral dose of 3 mg of deltamethrin reported the maximum plasma concentration in 1–2 h and a half-life of 10–11.5 h, with 10–26% of the dose excreted in faeces and 51–59% in urine over 5 days [3]. There is no antidote for deltamethrin toxicity, with supportive and symptom-directed treatment being the gold standard.

The inclusion of our case report in the literature provides new information regarding poisoning due to deltamethrin, which will further aid intensivists in managing these patients.

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