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Acute cypermethrin and other pyrethroid poisoning – An organophosphate-like poisoning: A case report and review

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ABSTRACT

Background: South Africa is one of the largest importers of pesticides in Africa. Pesticides play an integral role in food security and ensuring economic survival. Cypermethrin is a type II pyrethroid and is commonly and widely used as an insecticide. Poisoning arises from exposure through inhalation, skin contact or ingestion. Its toxic effects manifest predominantly as neurological and gastrointestinal symptoms. Severe pyrethroid poisonings are rare but can present as an organophosphate-like toxidrome. This poses a diagnostic conundrum.

Case report: This case concerns a 36-year-old male from a rural town in the Eastern Cape province of South Africa, who was diagnosed with intentional cypermethrin poisoning after a suicide attempt. He was initially diagnosed as a suspected organophosphate poisoning with coma. He made a full recovery after mechanical ventilation and symptomatic treatment with a low dose muscarinic antagonist, atropine sulphate.

Conclusions: With the ease of over-the-counter procurement, cypermethrin and other pyrethroid poisonings pose an increasing diagnostic dilemma to frontline and critical care personnel. This case report intends to raise awareness about the organophosphate-like toxidrome at presentation and the potential complications of high dose atropine treatment, commonly used to treat organophosphate poisoning. The treatment of cypermethrin is largely supportive with dermal decontamination of skin as an essential component. Low dose atropine may be utilized if required.

1. Background

Agriculture is central to the rural economy in South Africa. As such, South Africa is one of the largest importers of pesticides in Africa [1]. Malangu et al. reported on the South African profile of acute poisoning which included 423 patients from four hospitals in KwaZulu Natal Province and four hospitals from Gauteng Province. They found that 9.7% of acute poisonings were as a result of ingestion or exposure to agricultural chemicals and that pyrethroids were the third commonest cause in this group [2]. Cypermethrin is a type II pyrethroid, first synthesized in 1974. It is a synthetic compound derived from pyrethrin, a natural substance found in the flowers of Chrysanthemum spp [3]. Since it is readily available as a household, agricultural and veterinarian insecticide, accidental and intentional exposures are increasing [4]. Cypermethrin exposure manifests in a range of symptoms. The most frequent symptoms are mild local symptoms from dermal contamination [5]. Systemic intoxication from high doses or ingestion manifest as neurological symptoms (tremors, fasciculations, seizures and coma) and gastrointestinal symptoms (nausea, gastrointestinal irritation, and vomiting) [6–8]. We report a case of self-harm after intentional ingestion of cypermethrin that presented as a suspected organophosphate poisoning.

2. Case presentation

A 36-year-old male from a rural part of the Eastern Cape Province in

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South Africa was found unconscious in his home following conflict with his partner. He was found in a pool of vomit, smelling strongly of pesticide. Next to him was an open, unmarked bottle with an oily brown chemical fluid in it. He was breathing, but was unresponsive.

Later it would be revealed that he had ingested approximately 50 mL of a 20% cypermethrin concentrate, equivalent to 10 g. The cypermethrin concentrate was intended as a cattle and sheep dip. Notably, this animal dip changes colour from an oily brown to milky white once mixed with water. A 1:1 000 water ratio is prescribed by the manufacturer.

Intentional pesticide ingestion was suspected and he was immediately taken to his local clinic. The initial assessment noted a Glasgow Coma Score (GCS) of 5/15, constricted pupils bilaterally, blood pressure of 130/70, heart rate of 64/minute, finger prick glucose of 7.5 mmol, respiratory rate of 12 per minute and oxygen saturation of 96%. He was intubated by emergency care practitioners before transfer to Livingstone Tertiary Hospital. Prior to departure, decontamination of skin and clothes was undertaken. A nasogastric tube was placed, draining a creamy, beige liquid (volume: 1 L). A urinary catheter was inserted, and routine monitoring initiated. An initial diagnosis of possible organophosphate poisoning was made, and he was administered 6 mg of atropine sulphate.

At Livingstone Tertiary Hospital, he was still intubated and sedated with a morphine and midazolam infusion. The attending doctor noted a normal cardiovascular examination, with a normal sinus rhythm on the electrocardiogram (ECG). No sign of cardiac toxicity was present. On examination of the respiratory system, the patient had hypersalivation from the mouth, with bilateral crepitations and rhonchi at auscultation. The chest radiograph (CXR) showed minimal non-specific lower lobe alveolar type infiltrates bilaterally. No changes suggestive of pulmonary oedema or acute pneumonitis were seen. These findings were in keeping with a mild case of aspiration pneumonitis and had resolved on the subsequent CXR one day later. The nasogastric tube was still draining a creamy, beige fluid. His Glasgow Coma Score was 2 T, pupils were 4 mm bilaterally with a sluggish response to light. Normal facial symmetry was noted with flaccid extremities and absent reflexes. Selected laboratory investigations are presented in Table 1. The full blood count, electrolytes and liver profile were normal. An infusion containing 40 mg of atropine sulphate in 200 mL of normal saline was prepared and an infusion of 5 mL (or 1 mg) of atropine sulphate per hour was initiated.

During an episode of respiratory instability, the patient was reintubated and it was found that the endotracheal tube was blocked with secretions. The patient was subsequently transferred to the intensive care unit (ICU) for continued care. His ICU stay was uneventful and he was successfully weaned off atropine and the mechanical ventilator within 48 h. He was referred to a social worker and a psychological evaluation was conducted before his discharge home. At 18 months follow up, the patient had no residual symptoms.

Table 1
Selected laboratory investigations:.

Blood results:	Results	References
C-Reactive Protein, mg/L	7	< 10
Cholinesterase, U/L	8100	4620 – 11500
Cardiac Troponin I, ng/L	7	Rule-out ACS: Value < 18
Creatine kinase (CK) U/L	263	20–200
Creatine kinase MB mass (CK-MB), ug/L	13.76	0.60–6.30
INR	1.29	
PTT Ratio	1.0	
SARS-CoV-2 PCR	Negative	
HIV-1/2 Rapid	Negative	
Serum toxicology:	**Results**	**References**
Paracetamol, umol/L	28	66 – 132
Phenobarbital, umol/L	0	65 – 172 L
Theophylline, umol/L	1	Toxic levels: > 111
Ethanol, mmol/L	0.3	< 10.9
Salicylate, mmol/L	< 0.29	
Urine Toxicology Screen:		
Drugs of abuse screen (dipstick):	**Results**	
Amphetamine	Negative	
Barbiturates	Negative	
Benzodiazepines	Negative	
Cocaine	Negative	
Methadone	Negative	
Methamphetamine	Inconclusive	
Opiates	*Positive	
Phencyclidine	Negative	
Tetra-hydro cannabinol	Negative	
Tricyclic antidepressants	Negative	

*Patient was on a morphine infusion for sedation

3. Discussion

Based on their chemical structure and associated toxidromes, pyrethroids are classified into two types. Exposure to type I pyrethroids (allethrin, permethrin) results in reflex hyperexcitability, paresthaesia and fine tremors known as the T-syndrome. Exposure to type II pyrethroids (cypermethrin, deltamethrin) result in salivation, choreoathetosis, coarse tremors, seizures and effects on the skeletal and cardiac muscles, also known as the choreoathetosis–salivation or the CS syndrome [9,10].

The World Health Organization (WHO) categorizes cypermethrin as a moderately hazardous pesticide, having a relatively high potential lethal dose at 50% (LD50) of 250 mg/kg, i.e. around 1–10 g depending on the patient's weight. The toxic oral dose may reportedly manifest at doses from 100 mg/kg body weight upwards. [3,11]. The absorption of pyrethroids is primarily based on excretion studies [12]. From these studies, cypermethrin's dermal absorption in humans is poor, at 1.5% [13]. In contrast to this, oral administration has a 19–57% absorption, with the peak at 3 h and a distributive half-life of 5 h [14,15].

It is more toxic for insects than humans due to its slow dermal absorption in humans and because insects have more sodium channel sensitivity, smaller body size and lower body temperature [16–19]. Pyrethroid metabolism occurs rapidly in the liver through the enzymatic actions of the Cytochrome P450 (CYP) mono-oxygenase family of enzymes. The metabolites are mainly excreted in urine [14,20].

Cypermethrin acts by modifying the voltage gated sodium channels, thereby delaying their closure [21,22]. Moreover, voltage gated calcium channels and gamma amino butyric acid (GABA) mediated chloride channels also have a role in the toxic properties of cypermethrin. Seizures are likely the result of GABA-gated chloride channel dysfunction [23].

3.1. Clinical features

Clinically, the patient may smell of the pesticide. No specific identifiable smell has been attributed to cypermethrin while reported smells range from odourless to organophosphate-like. Exposures often involve commercial formulations with additional hazardous compounds. Notable additives include piperonyl butoxide and organophosphate compounds. Piperonyl butoxide dramatically inhibits Cytochrome P450′s effect by up to 50% within 3 h of exposure [24,25]. The offending agent is often initially unknown, or symptoms are a result of a combination of compounds. Diagnosis may be difficult when confronted with an organophosphate-like toxidrome.

Ocular exposure causes mild irritation and cases of miosis have been reported [26,27]. *Skin exposure* leads to paresthesia, the most common adverse event [28]. Other symptoms include pruritus, erythema, burning and blisters [29–31]. Symptoms generally start 30 min to 2 h after exposure, peaks at about 6 h and recovers in about 24 h [6, 32–35].

Inhalational exposures are less common. Pyrethroids are not volatile and occurs as small droplets, rather than vapor [36]. Occupational exposure is known to produce irritation of the respiratory tract [37]. Most notable signs and symptoms are sneezing, coughing, rhinorrhea, shortness of breath and wheezing [27,38].

Ingestion is the most likely cause of systemic toxicity. After ingestion, symptoms arise within minutes and include nausea, vomiting, sore throat, abdominal pain and instances of mucosal irritation and ulceration [6,39]. Other symptoms include headache, dizziness and fatigue. Less frequently palpitations, blurred vision, and chest tightness may occur [6]. In severe cases, muscle tremors and torticollis can occur [40]. In cases where upper gastrointestinal endoscopy was performed, oesophagitis, oesophageal ulceration, gastritis, gastric ulcers, and duodenal ulcers have been reported [41,42]. Coma and convulsions are the principal life-threatening features of poisoning [6, 8, 43].

In humans, pyrethroid pulmonary toxicity has not been studied exclusively. Some extrapolations can be drawn from animal studies and individual case reports. Reported pulmonary complications may be attributed to the organic solvents in the formulations. In addition, aspiration pneumonitis and pulmonary oedema have been documented [6, 39, 44].

Cardiotoxicity due to cypermethrin is rare [6,39]. Most of our understanding of the cardiac effects of cypermethrin are from animal studies and isolated case reports. Spencer et al. reported a possible cause for the arrhythmogenic effects as the result of prolonged action potentials [45]. In their case report on prolonged bradycardias after cypermethrin poisoning, Shilpakar and Karki hypothesized sodium channel blockage in the sinoatrial or atrioventricular node as mechanism [4]. Other notable ECG changes may include ST and T wave changes, sinus tachycardia, ventricular ectopic and rarely sinus bradycardias [4, 6, 39]. Biochemical findings reported include a metabolic acidosis, normal serum cholinesterase level, leukocytosis, raised AST and increased serum creatinine [6,39].

4. Management

The diagnosis of severe pyrethroid poisoning can be challenging, owing to the clinical presentation which is similar to that of organophosphate poisoning. The measurement of plasma cholinesterase levels allow for the differentiation of the two conditions. Cholinesterase levels are normal in pyrethroid poisoning[9]. Currently, no antidote is available for poisoning and most exposures result in only minor symptoms. Dermal decontamination of skin with soap and water is an essential component of management.

In the intoxicated individual with systemic symptoms, the focus should be on stabilizing the patient through optimization of the airway, breathing and circulation. This may include hemodynamic stabilization with intravenous fluid resuscitation where appropriate, oxygen supplementation and reduction of absorption by decontamination and enhanced elimination[9]. Following ingestion, activated charcoal (50–100 g for an adult) should be considered if within one hour of ingestion. Gastric lavage is contraindicated, as most formulations contain solvents that can cause chemical pneumonitis[9].

In the event of hypersalivation and pulmonary oedema, low doses of intravenous (IV) atropine sulphate (0.6–1.2 mg) have been used effectively. However, high doses of atropine sulphate, as is used in organophosphate poisoning, have resulted in atropine poisoning and even death[6]. Isolated brief seizures may not require treatment, but the treatment of prolonged convulsions is intravenous (IV) diazepam (5–10 mg)[46]. A Cochrane review demonstrated that IV lorazepam is superior to IV diazepam for aborting seizures[47] and is therefore recommended for the treatment of prolonged seizures[44]. For patients who developed status epilepticus, IV phenobarbital was superior to IV phenytoin in a recent animal study[46,48].

5. Conclusion

With the widespread use and ease of over-the-counter procurement, cypermethrin and other pyrethroid poisonings are increasing and in the absence of a diagnostic assay, may pose a diagnostic dilemma. Though severe systemic poisonings are rare, their organophosphate-like presentation can delay the diagnosis if not suspected and result in inappropriate and potentially harmful treatment. Clinician awareness, decontamination, early administration of activated charcoal, and systemic supportive measures improve overall outcomes.

Ethics approval and consent to participate

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CRediT authorship contribution statement

Leon Daniel Scheepers - the corresponding author did the research/literature review and wrote the manuscript. **Robert Freercks** - assisted with final editing. **Elizabeth van der Merwe** - assisted with final editing. All authors read and approved the final manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

No data was used for the research described in the article.

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Consent for publication

Informed consent obtained in writing from the patient regarding the publication of this case report.

Competing interests

None.

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