Acute pyrethroid toxicity mimicking cholinergic crisis following intentional ingestion

Fathima Murthuza[1], Wasim Ahmad[1*], Mohamed Abdou Saad[1], Maitha Almatrooshi[2]
1. Emergency Medicine, Zayed Military Hospital, Abu Dhabi, UAE
2. Emergency Medicine, Sheikh Shakhbout Medical City, Abu Dhabi, UAE
Correspondence to: Wasim Ahmad
*Department of Emergency Medicine, Zayed Military Hospital, Abu Dhabi, UAE
Email: wasimahmad4787@gmail.com
DOI: 10.24911/SJEMed.12-2594

Background: Pyrethroids are synthetic analogs of natural pyrethrins used as insecticides because of their perceived low mammalian toxicity and insecticidal efficacy. However, large-volume ingestion can cause systemic toxicity mimicking cholinergic toxidromes and creates diagnostic uncertainty. Toxicity results from disruption of neuronal ion channels rather than acetylcholinesterase inhibition.

Case Presentation: A 21-year-old male presented with vomiting, drowsiness, hypersalivation, miosis, bronchorrhea, and respiratory distress after ingesting 500 ml of a beverage mixed with Katmal, a cockroach-killing powder. He was lethargic with pinpoint pupils, cold extremities, excessive oral secretions, and bilateral lung crackles. Blood gas analysis showed primary metabolic acidosis with elevated lactate. Organophosphate poisoning was initially suspected, and atropine improved secretions and respiratory distress. The ingested compound was subsequently identified as lambda cyhalothrin, a type II pyrethroid. Pyrethroids prolong neuronal depolarization through delayed closure of voltage-gated sodium channels, while type II agents additionally block GABA-gated chloride channels, reducing inhibitory signaling and causing neuronal overactivity. Type I agents cause T syndrome, characterized by tremors and hyperreflexia, whereas type II agents cause CS syndrome, marked by choreoathetosis, salivation, and seizures. Respiratory compromise is associated with central nervous system depression rather than bronchospasm. Ingestions greater than 250 ml and elevated lactate levels have been associated with severe outcomes. Management is supportive because no specific antidote exists, although atropine may provide symptomatic benefit for excessive secretions.

Conclusion: Pyrethroid poisoning should be considered in cholinergic-like presentations after insecticide ingestion, with supportive care remaining the cornerstone of management despite possible symptomatic benefit from atropine.

Keywords: Pyrethroid poisoning, lambda cyhalothrin, insecticide poisoning, cholinergic toxidrome, atropine, metabolic acidosis, emergency medicine.