Deliberate self-harm with parenteral chlorpyrifos: a case report

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ABSTRACT
Organophosphate (OP) pesticide poisoning is one of the most common poisonings in India. Most self-poisoning involves pesticide ingestion. Parenteral pesticide poisoning is unusual. Here we report a case of a middle-aged man who presented with history of deliberate self-harm by injecting chlorpyrifos. The patient presented to the Emergency Department with cholinergic symptoms and blisters and ulcers on the right arm. This case represents an uncommon mode of OP pesticide toxicity and distinctive set of local complications secondary to injection.

Introduction
We report case of a middle-aged man who presented with history of deliberate self-harm by injecting chlorpyrifos. Parenteral organophosphate (OP) poisoning is unusual with few previously reported cases [1–7]. The patient presented to the Emergency Department (ED) with cholinergic symptoms and blisters and ulcers on the right arm. This case, presented in accord with the CARE guidelines, illustrates the clinical feature and possible complication of self-injection of chlorpyrifos.

Case presentation
A 55-year-old male arrived to the ED with alleged history of injection of some substance in his right forearm three days before presenting to our ED. At the time of presentation, the substance injected and the route of injection were unknown. As intravenous route requires a particular expertise that would be unusual for a common person and has a rapid onset of action, we believed the route to be either intradermal or subcutaneous in this case.

The exposure reportedly occurred when patient injected himself with the unknown substance. After three to four hours of injection, the patient developed swelling in his right forearm with blisters formation that later turned into ulcers. On second day, he developed respiratory distress and agitation for which he was taken to a local hospital where supportive managements included oxygen and intravenous fluids. On the third day, patient was referred to our tertiary treatment center for better intensive care management.

On presentation to our ED, patient was in severe respiratory distress with respiratory rate of 30 breaths/min and his oxygen saturation was not recordable. His pulse rate was 48 beats/min and blood pressure was 96/74 mm Hg. He had altered sensorium with Glasgow coma score of 11 (E3V3M5). Bilateral pupils were constricted with sluggish reactivity to light. Plantar reflex was extensor, and deep tendon reflexes were sluggish. Auscultation of chest revealed reduced air entry bilateral with no rales, rhonchi, or wheezes. There were no fasciculations. In view of patient's decreasing oxygen saturation, he underwent intubation and mechanical ventilation. The relatives also gave history of decrease in urine output for last 2 days.

His laboratory reports including serum electrolytes, complete blood count, blood gas, and troponin-I were normal except for an elevated lactate value of 6.8 mmol/L (normal value <2 mmol/L). Initial serum creatinine value was 1.8 mg/dL (159 µmol/L) which increased to 2.5 mg/dL (248 µmol/L) after 4 h, indicating acute kidney injury. Bedside lung ultrasound showed presence of lung sliding with “A” profile (normal) in all lung zones.

Arterial Doppler of right upper limb showed normal flow in the right brachial and radial artery. Flow in the right ulnar artery could not be assessed because of presence of severe edema at the site. On local examination of right upper limb, two large ulcers and one small blister were present on the medial aspect (Figure 1).
Considering the signs and symptoms (respiratory distress, bradycardia, miosis) consistent with OP poisoning, he received intravenous atropine 16 mg followed by intravenous infusion at the rate of 3 mg/h. After bolus doses of atropine heart rate increased to 100 beats/min from 48 beats/min, and miosis improved. He received intravenous noradrenaline infusion starting at the rate of 1 mg/h for hypotensive shock. He received empiric antibiotics (intravenous clindamycin 600 mg and amoxicillin clavulanate 1.2 g) for presumed sepsis.

Three hours later, a family member provided a photograph (Figure 2), identifying the injected substance as Eldriene TC® 20% chlorpyrifos. This corroborated OP poisoning. Plasma cholinesterase and creatine phosphokinase activities were unavailable at our hospital. Patient was admitted to intensive care unit due to respiratory and cardiovascular failure requiring mechanical ventilation. However, after 12 h of presentation, the patient died because of severe metabolic acidosis.

**Discussion**

OP poisoning is the major cause of poisoning mortality and morbidity in India and neighboring countries. OPs inhibit acetylcholinesterase with resulting cholinergic excess at muscarinic and nicotinic receptors. Bradycardia and miosis are common features, and death usually results from bronchorrhea and respiratory muscle failure. Antidotal treatment includes atropine to block muscarinic receptors and pralidoxime to restore cholinesterase enzyme function. The latter is most effective given early in the course [8].

Chlorpyrifos and other OP products contain hydrocarbon solvents. Injection of these products may cause cellulitis, fat necrosis, compartment syndrome, or sterile abscess formation [5–7, 9]. This likely explains the blistering and ulcer formation on the patient’s arm (Figure 1).

The diagnosis of OP poisoning relies upon the history of ingestion or mucocutaneous exposure, clinical features, and plasma cholinesterase activity levels. The depressed plasma cholinesterase activity confirms the diagnosis of OP poisoning. The estimation of red blood cell cholinesterase is more specific [10].

Treatment includes reversal of acetylcholine excess at muscarinic sites with atropine and reversal of toxin binding at active sites on the cholinesterase molecule using pralidoxime or other oximes [11, 12]. As death is primarily due to airway and respiratory failure, supportive care requires airway management. Early antidotal treatment generally produces better outcomes. His delayed presentation to the hospital resulted in graver clinical condition on arrival.

Finally, hydrocarbons solvents in OP formulations can be highly irritating to local tissues and may lead to soft tissue damage. Local injection site reactions like cellulitis, fat necrosis, compartment syndrome, and abscess formation, which tends to be sterile, can occur [13–16]. They may require treatment with anti-inflammatory agents and antimicrobials if contamination is suspected.

**Conclusion**

We describe a fatal poisoning by uncommon self-injection of chlorpyrifos 20% in hydrocarbon vehicle. Toxic effects included local tissue injury, cholinergic excess, bradycardia, hypotension and respiratory failure with fatal outcome.

**Disclosure statement**

No potential conflict of interest was reported by the author(s).
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