

“The Toxic Depot”: Parenteral Insecticide Injection

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ABSTRACT

Background: Organophosphorus (OP) pesticides are extensively used both in developed and developing countries. Organophosphorus poisoning primarily occurs through occupational, accidental, and suicidal exposures. Toxicity through parenteral injections is seldom reported and there are only very few case reports till date.

Case presentation: We report a case of parenteral injection of 10 mL of OP compound (Dichlorvos 76%) into a swelling over the left leg. The compound was injected by the patient himself as adjuvant therapy for swelling. Initial manifestations included vomiting, abdomen pain, and excessive secretions followed by neuromuscular weakness. The patient was subsequently intubated and treated with atropine and pralidoxime. The patient did not improve with antidotes for OP poisoning, attributed to the depot the OP compound had formed. The swelling was excised and the patient immediately showed response to the treatment. Biopsy of the swelling showed granuloma and fungal hyphae. The patient developed intermediate syndrome during the ICU stay and was discharged after 20 days of hospital stay.

Keywords: Atropine, Depot organophosphorus poisoning, Intermediate syndrome, Organophosphorus poisoning, Parenteral injection.

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HIGHLIGHTS

This case represents a rare case of the injected OP compound as a therapeutic modality for chronic leg swelling. Though expected management was atropine and pralidoxime, the lack of improvement in the patient hinted at the possibility of toxin depot in the leg. Surgical excision of the above-mentioned depot was life-saving.

INTRODUCTION

Organophosphorus compound is the most widely used insecticide in India. The common routes of intoxication are oral (intentional/unintentional), inhalational (mostly unintentional), or transcutaneous absorption. Poisoning can also occur rarely through the parenteral route (intramuscular, intravenous, or subcutaneous).¹ Hardly any case reports of parenteral OP poisoning have been described in the literature.²⁻⁶ Organophosphorus poisoning with parenteral injection may present with cholinergic crises, intermediate syndrome, or delayed toxicity.^{2,3} Here, we present a case in which an extremely rare and unusual way of self-administration of OP compound was used as therapy for swelling.

CASE DESCRIPTION

A 49-year-old male presented to the Emergency Department (ED) with a history of parenteral injection of 10 mL OP compound (Dichlorvos 76%) (Fig. 1) over a swelling on his left leg (Fig. 2). The compound was injected by the patient himself as a therapy to decrease the size of swelling as suggested by his friends. Two hours after self-administration, the patient developed nausea and vomiting and was treated with antiemetics and discharged from the local hospital on the same day. The next morning patient developed abdomen pain associated with multiple episodes of vomiting and giddiness. The patient was then referred to tertiary care for intensive care.

On arrival at ED, the patient was conscious, oriented, tachypneic with an RR-35 CPM, PR-120 bpm, BP-160/90 mm Hg, temperature-36.8°C, SpO₂-88%@RA, pupils-bilateral 3 mm

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Fig. 1: Bottle showing the pesticide, Dichlorvos

sluggishly reactive, bilateral crepitations present on auscultation, and fasciculations were present. Bilateral plantar reflex and deep tendon reflexes were normal. The patient was immediately supported with 6 liters of oxygen on NRBM, stat Inj. Atropine 6 mg was given followed by Inj. Atropine 2 mg/hour infusion, the dose was titrated based on signs of atropinization. Inj. PAM 2 gm



Fig. 2: Swelling on the leg, where the pesticide was injected

IV loading dose was administered over 30 minutes followed by a 500 mg/hour infusion. After 45 minutes of arrival into ED, his sensorium deteriorated, tachypnea worsened with abdomino-thoracic breathing pattern and emerging bilateral ptosis. The patient was intubated and mechanically ventilated in view of respiratory distress and low GCS. Arterial blood gas analysis showed pH:7.365, PO₂:68.1, PCO₂:43.7, and HCO₃:23.5. The patient was not improving with conventional therapy for OP poisoning, raising the doubts of the swelling on the leg being a source/depot for prolonged toxin release. The leg swelling was then excised by the surgical team, biopsy showed granuloma with fungal hyphae, for which the patient was started on antifungal medications. He developed features of intermediate syndrome such as respiratory muscle weakness, proximal muscle weakness during ICU stay, and atropine infusion and ventilatory support were continued. The patient subsequently improved and was discharged.

DISCUSSION

Organophosphorus compounds are cholinesterase inhibitors, which result in excess acetylcholine at sympathetic ganglia, parasympathetic ganglia, and synapses. Organophosphorus compounds bind irreversibly to acetylcholinesterase enzyme through the process of phosphorylation.

The time of onset of clinical manifestations depends on poison load, route of exposure, and chemical nature of the pesticide. The severity of symptoms depends on the percentage and amount of poison consumed.⁴

Diagnosis and treatment of suspected organophosphate poisoning are based on history and the presence of a suggestive toxidrome. Laboratory cholinesterase levels take time and waiting for the results delays the administration of life-saving treatment.

Treatment consists of airway control, decontamination, prevention of absorption, and administration of an antidote.⁶

Literature searches have revealed few case reports of parenteral OP poisoning. Badhe and Sudhakar² described a case of parenteral (IV) monocrotophos poisoning, where the patient presented with cholinergic crises within 30 minutes but later developed intermediate syndrome. Malla et al.³ described a case of parenteral injection of chlorpyrifos compound, which showed features of mild cholinergic toxicity with cellulitis and abscess at the injection site. Ghosh et al.⁴ described a case of subcutaneous injection of Dichlorvos, where the patient presented with excessive salivation and pyramidal tract signs but later developed respiratory failure and delayed extrapyramidal manifestations. Nizami et al.⁶ described a case of intramuscular quinolphos with cypermethrin poisoning, where the patient presented with cholinergic crises with intermediate syndrome.

Amongst published cases, the mode of poisoning is suicidal. The case we described here is rare, using an injected OP compound as a therapy for swelling.

CONCLUSION

Organophosphorus poisoning by the parenteral route is rare. The onset of symptoms may be delayed or prolonged despite regular treatment. The treating physician should be vigilant, and prolonged treatment and depot excision, in this case, were life-saving.

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