

Self subcutaneous injection of profenophos with delayed systemic toxicity: a case report

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Abstract

Suicidal poisoning by ingestion of organophosphate (OP) insecticide is a common mode of poisoning in our region. However, attempted suicide via the parenteral route has rarely been reported. We report a case of self subcutaneous injection of 50% profenophos in both hands, a rare and unusual way of intoxication. It causes local toxicity such as toxic cellulitis, abscess and necrosis with delayed systemic toxicity. He responded to parental antibiotics, surgical debridement, atropine and pralidoxime.

Key-words: subcutaneous injection, organophosphate, surgical debridement.

Introduction

Organophosphorous compound among the most popular and most widely used insecticide in India. Because of its easy availability and assess organophosphorous poisoning is very common. Poisoning occur mostly by voluntary ingestion, inhalation or by absorption by skin. Only few case report of parental organophosphorous poisoning have been described [1,2,3,4]. We are

reporting a case of self subcutaneous injection of organophosphorous with atypical systemic manifestation and severe local toxicity, which is an uncommon mode of organophosphorus poisoning, managed in our hospital. He injected 50% profenophos in both hands.

Case Report

A 50 year old male, insecticide shopkeeper by occupation presented to emergency room with history of self injection of 50% profenofos, 2ml each in dorsal aspect of both hands. The exposure occurred 20 hours before presentation. On presentation he had a complaint of giddiness and swelling with pain in both hands.

The pain had made him to seek medical attention. He had no premorbid illness. He consumes alcohol. On examination: He is conscious, oriented and comfortable. Pulse: 88/min regular, BP: 110/80 mmHg, RR: 18/min regular, SpO₂: 99% room air, No pallor, No cyanosis, No clubbing, and No icterus. Cardio vascular system: S1S2 heard, no murmur, JVP not raised.

Respiratory system: Bilateral equal air entry, no added sound. Central nervous system: Pupil 3 mm equally reacting to light on both sides, no excessive salivation, no excessive lacrimation, no loose stools, no fasciculation, power 5/5, tone normal, deep tendon reflexes 2+, plantar bilaterally flexor. Abdomen: soft, non tender, organs not palpable, bowel sound were heard. Local examination of both hand showed swelling with erythema extending up to mid one third of forearm on both upper limb. There was cicatrix of around 3cm in diameter on dorsal aspect of right hand and around 1cm in diameter on dorsal aspect of left hand.

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Fig 1: Day 1 – Right hand with swelling and erythema with cicatrix at the injection site.



Fig 2: Day 1 – Left hand with swelling and erythema with cicatrix at the injection site.



Fig 3: Right hand two days after surgical debridement.



Fig 4: Right hand two days after surgical debridement.



Fig 5: On day of discharge – right hand.



Fig 6: On day of discharge – left hand.

His investigations on admission showed Hb: 10.7 gm%, WBC 10800cells/mm³, lymphocytes 15%, neutrophils 82%, RBS 3980cells/mm³, urea 20mg/dl, creatinine 0.9mg/dl, Na 140mmol/l, K 4.7mmol/L, Serum cholinesterase 4800 U/L (normal 5100 – 11700 U/L). Chest X-ray: Normal cardiac and lung shadow, ECG: within normal limit.

He was admitted in ICU and was started on Inj.Ceftriaxone+sulbactam, inj. Metronidazole, inj. Thiamine, inj pralidoxime and IV fluids. Though the patient didn't have cholinergic signs and symptoms on presentation, on reviewing literature parenteral op poisoning are prone for delayed manifestations [4], he was added on with inj atropine 1mg iv every 15min. He developed atropine delirium after 12 hours of starting atropine and atropine dose was tapered and stopped on 3rd day. The swelling was progressive till the second day of admission. On 3rd day of admission surgical debridement of necrosed tissue were done and pus was drained. Following the swelling was regressive. The antibiotics were continued. Swelling resolved completely on 5th day and debrided wound was healing well. On 6th day of admission he developed wheeze and crackles, pupil were 1mm on both sides with minimal reaction to light, there was no muscle weakness, his serum cholinesterase was found to be 643 U/L and there were no other muscarinic and nicotinic signs or symptoms. Inj. atropine 2mg IV bolus was given and atropine infusion was started at a rate of 4mg/hr. The wheeze and crackles subsided with atropine. Serum cholinesterase was rising on subsequent days. Serum cholinesterase was found to be 2584 U/L on 11th day. Patient was discharged on 13th day of hospital stay and the wound was healing well.

Discussion

Organophosphates are powerful inhibitors of acetylcholinesterase which is responsible for hydrolyzing acetylcholine to choline and acetic acid after its release and completion of function (i.e. propagation of action potential). As a result, there is accumulation of acetylcholine with continued stimulation of cholinergic receptors and eventual paralysis of nerve or muscle [5]. Cholinergic crisis usually occurs within the first 8 hours and nearly all within 24 hours [6].

The initial treatment of poisoning focuses on ensuring adequate oxygenation, followed by the administration of atropine to antagonize the muscarinic and central nervous system effects of the OP. Pralidoxime is usually used in the case of respiratory depression, muscle fasciculations or muscular weakness to antagonize the toxicity of OPs on nicotinic synapses. The dose of atropine and pralidoxime should be controlled flexibly. In reviewing literatures parenteral organophosphorous poisoning is highly toxic especially in intravenous and intramuscular poisoning and they are prone to develop delayed systemic toxicity. Even a case of intermediate syndrome has been reported by Ashok Badhe et al following intravenous organophosphorous poisoning [1].

In the present case, signs and symptoms of systemic toxicity (cholinergic crisis) resulting from the subcutaneous injection of organophosphorous manifested late, 6th day of hospital stay. And the patient had fluctuating serum cholinesterase level, where maximal fall in serum cholinesterase observed during

regressing of swelling. Which may due to transient release of organophosphorous compound from injection site.

Conclusion

This case represents a rare mode of organophosphorous administration for suicide. The patient had immediate local toxic effects which may be due to the contaminated needle or solvent of organophosphorous and delayed systemic toxicity which may be contributed by transient release of organophosphorous compound from subcutaneous tissue. Antidote may be considered while debriding the wound to counteract the release of OP from tissues. As intermediate syndrome is common among parenteral OP poisoning pralidoxime may play a very significant role in parenteral OP poisoning [4]. Parenteral organophosphorous poisoning are prone for delayed systemic effects and patients need prolong vigilant observation.

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