



Parenteral Administration of Organophosphorus Compound- An Unusual Route of Poisoning Presenting With Severe Toxicity

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Abstract

Acute Organophosphorus Compound poisoning (OPC) usually manifest in 3 different phases of toxic effects like acute cholinergic crisis, intermediate syndrome (IMS) and lastly delayed neuropathy. Poisoning other than by ingestion is rarely seen in clinical practice. Here we report a 22 year male who presented in altered sensorium and erythema with induration in left arm. Treatment was started with high clinical suspicion of OPC poisoning. History of injecting about 15ml of parathion into his left arm was revealed after the patient regained consciousness. He developed features of IMS with necrosis and infection at the site of injection. He required ventilator support. He recovered after prompt administration of Pralidoxime and Atropine along with broad spectrum antibiotics and wound debridement. Parenteral administration of organophosphorus poison can cause severe toxicity especially with delayed manifestations and early recognition and treatment is essential for recovery.

Keywords: Intermediate syndrome, Organophosphorus Poisoning, Parenteral

Case Report

A 22 year male, working at a drug store, was brought to the emergency with a history of altered sensorium and generalized weakness since last 4 hours. The patient's attenders gave a history of finding the patient in an altered sensorium at his house, following which they brought the patient to the hospital. The patient was drowsy, was responding to painful stimuli only, he had

bradycardia, was tachypneic and BP was elevated. His pupils were 2mm in size, bilateral, reacting to light. On local examination, a blackish discolouration and induration was noticed on his left arm (Figure1). He had bilateral crepitations over all lung fields and other systems were unremarkable. Further questioning, revealed that the patient had attempted suicide one year back, by consuming organophosphorus poison for

which further details were not available. Based on the past history and clinical presentation a provisional diagnosis of organophosphorus poisoning was made and the patient was started on IV fluids, Inj.Atropine infusion at 2mg/Hr , along with Inj.Pralidoxime 1gm IV followed by a 500 mg/hr infusion. After around six hours, the patient regained consciousness and on further questioning, mentioned that he had injected about 15ml of Parathion into his left arm. Patient developed respiratory distress and in view of impending respiratory failure was electively intubated and put on ventilator support. Atropine and Pralidoxime infusion was continued as per the clinical signs of recovery. Patient also received broad spectrum antibiotic. His labs revealed leucocytosis (15400) and a normal renal and liver functions. His serum electrolytes, calcium and magnesium were normal. Pseudocholinesterase level was very low at 448

U/L (Normal being 2710 to 11510). On the 3rd day injection site started developing ulcer, hence a surgical opinion was sought. Incision and drainage was done and wound care was given (Pus culture grew Staphylococcus aureus). (Figures 2 and 3) Patient's general condition improved over the next few days, was weaned off the ventilator and atropine was stopped. On the 7th day of his hospital stay, patient developed breathing difficulty and neck weakness. With a clinical diagnosis of intermediate syndrome patient was shifted to ICU and had to be reintubated and placed on ventilator support. Patient was restarted on atropine infusion. On the 17th day patient had good respiratory effort and extrubated on the 18th day of hospital stay. Patient was stabilized and shifted to the ward after 2 days. On the 28th day of admission patient did not have any specific symptoms and hence discharged with psychiatric counselling and antidepressants.

Figure 1- Erythema with necrosis in left arm



Figure 2 – Infected ulcer at the site



Figure 3 – Wound after debridement



Discussion

Acute organophosphorus compound poisoning usually manifests in 3 different phases of toxic effects, like, acute cholinergic crisis, intermediate syndrome (IMS) and delayed neuropathy. Among them, IMS is usually considered as a major factor of organophosphate-related morbidity and mortality. Injected OPC behave differently than OPC taken orally. Injected OPC stays for a long time in the local area and causes intermediary syndrome. The hydrocarbon vehicle used in these insecticides is poorly cleared by the local tissues and is responsible for fever, leukocytosis, and liquefaction necrosis.²

In cases of parenteral injection of organophosphates, attention must be paid to the possibility of late onset of the clinical manifestations of the poisoning. Local complications at the site of the injection, like necrosis and abscesses are also expected findings⁴

The clinical picture in this patient was of late onset which was severe with the manifestation of IMS. This could be explained by the poison being absorbed slowly from the site of injection.

Despite a high incidence, the patho-physiology of IMS remains unclear. Following are some of the proposed mechanisms of IMS. Differences in the susceptibility of various cholinergic receptors, muscle necrosis, prolonged acetylcholinesterase suppression, inadequate oxime dose, down regulation of postsynaptic acetylcholine receptors, failure of postsynaptic acetylcholine release, and oxidative stress-related myopathy have all been postulated. The clinical manifestations of IMS usually occur within 24 to 96 hours, affecting conscious patients without cholinergic signs, and involve the muscles of respiration, proximal limb muscles, neck flexors, and muscles innervated by motor cranial nerves.

With appropriate ventilator therapy, complete recovery develops 4-16 days later. The treatment of IMS is mainly supportive. Nevertheless, because IMS generally concurs with severe organophosphate toxicity and persistent inhibition of acetylcholinesterase, early aggressive decontamination, appropriate antidotal therapy, and prompt institution of ventilatory support should be helpful in ameliorating the magnitude and/or the incidence of IMS. Although IMS is well recognized as a disorder of neuromuscular junction, its exact etiology, incidence, and risk factors are not clearly defined because existing studies are largely small-scale case series and do not employ a consistent and rigorous definition of IMS. Without a clear understanding of the pathophysiology of IMS, specific therapy is not available. The prognosis of IMS, however, is likely to be favorable if respiratory failure can be promptly recognized and treated accordingly.

Conclusion

Patients who are received in the emergency room in an unconscious state should have a good history and thorough clinical examination which can give a clue to the clinical diagnosis. Early and prompt recognition of the IMS can bring down the mortality and the morbidity in OPC poisoning. Various conclusions can be made from this case report. The route of administration of the insecticide also determines the prognosis along with the time delay in presentation and the type of compound. Also local necrosis and infection is a

very common occurrence with parenteral administration and adequate antibiotics and debridement is required.

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