Case Report

Mononeuropathy in Acute Organo-Phosphorous Poisoning

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ABSTRACT

Acute Organo-Phosphorus Compound (OPC) poisoning is one of the most common emergencies encountered in developing countries like India. The occurrence of such poisoning is more in rural India where organo-phosphorus pesticides are used extensively. Poisoning with organo-phosphorus compounds are known to produce early cholinergic symptoms, features of intermediate syndrome and a rare delayed polyneuropathy. But the occurrence of early onset mononeuropathy following OPC ingestion is extremely rare and hence we report such a case of mononeuropathy following parathion ingestion.

Key-words: *Foot Drop, Organo-Phosphorus, Parathion*

INTRODUCTION

Cases of acute pesticide poisoning in particular, organophosphorus compounds (OPC) account for high morbidity and mortality worldwide and more so in developing countries. Commonly the circumstances of poisoning are suicide, homicide, occupational and accidental exposure. Organophosphorus (OP) agents are capable of producing several sub-acute or chronic neurological syndromes. Early neuropathy within 4 days of toxicity is not known to occur and hence we report such a case of early neuropathy caused by parathion.

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CASE HISTORY

A 27 year old housewife was brought to the casualty with history of vomiting followed by sudden unresponsiveness since about 2 hours along with salivation and frothing from mouth. Patient's attendees noted that she came out of the bathroom and developed the above symptoms. Patient did not have any other symptoms and there was no significant past medical history.

On examination, patient was comatose with a pulse rate of 110 bpm, blood pressure of 170/110 mm Hg and respiratory rate of 14 cpm with shallow breathing. She was also noted to have excessive salivation and diaphoresis. Her cardiac examination was normal. Respiratory examination revealed bi-basal crepitations suggestive of possible aspiration. She was found to be comatose, with pinpoint pupils and unresponsive to painful stimulus and mute plantar response.

In view of her depressed sensorium and

poor ventilatory efforts, patient was mechanically ventilated. The history was suggestive of acute cholinergic crisis and physical examination confirmed the same. Hence acute OPC poisoning was suspected and patient was started on atropine and pralidoxime infusions.

Patient was mechanically ventilated for 48 hours following which she was weaned off the ventilator. Atropine infusion was continued for 4 days and then tapered and stopped. Patient was retrospectively asked about the illness and confessed to have consumed about 30 ml of parathion containing pesticide.

On day 5 post admission, she was noted to have a limping gait with numbness over the right lower limb, distally from the lower 1/3rd of the leg. Neurological examination revealed a power of 3/5 at right ankle, weak toe grip, sluggish ankle reflex and intact sensory system. Examination of left lower limb was normal.

Her electrolytes was repeated and found to be in the normal range. Patient was started on vitamin supplements.

Nerve conduction study of bilateral common peroneal, posterior tibial and sural nerves was done which showed absent F waves and mildly reduced compound muscle action potential in right posterior tibial and common peroneal; suggestive of minimal right common peroneal axonal motor neuropathy. She was advised physiotherapy and discharged 2 days later. Patient is currently being followed up on outpatient basis and is found to have mild improvement in right foot drop.

DISCUSSION

This case study evaluates the result of OPC toxicity causing axonal damage. Organophosphorous compounds and carbonates that were first discovered more than 100 years ago, are at present the predominant groups of insecticides employed globally for pest control.

The clinical features of acute OP compound poisoning can be categorized as follows: Acute cholinergic phase, intermediate syndrome and some patients experience a delayed complication of OP induced delayed polyneuropathy.

Acute cholinergic phase: This is the initial phase of acute poisoning resulting in muscaranic and nicotinic effects. The cholinergic phase usually lasts for 24 to 48 hours and constitutes a medical emergency that requires treatment in an ICU.[6]

Table 1: Investigations on admission

Hb – 13.8 mg/dl	LFT – normal
TLC – 33,300 / cu.mm	RBS – 179 mg/dl
Platelet count – 3.44 L/cu.mm	Pseudocholinesterase – 567 U/l
Blood urea – 12 mg/dl	Calcium – 7.9 mg/dl
Serum creatinine – 0.63 mg/dl	Magnesium – 1.9 mEq / l
Sodium – 147 mEq/L	Chest X ray – normal
Potassium – 3.7 mEq/L	ECG – normal sinus rhythm

After recovery from the cholinergic crisis, but before the expected onset of delayed polyneuropathy, some patients develop a muscle paralysis, which is described as Intermediate syndrome. This phenomenon has been reported in between 20-68% of the patients.[7] The cardinal feature of this syndrome is muscle weakness affecting predominantly the proximal limb muscle and neck flexors. Motor cranial nerve palsies (III to VII and X) also occur. Respiratory muscle weakness leading to respiratory failure could lead to a fatal outcome.

Organophosphorus Induced Delayed Polyneuropathy (OPIDP) usually develops following latent periods of 2-4 weeks after the cholinergic crisis. The cardinal symptoms are distal muscle weakness, calf pain preceding the weakness and in some cases paraesthesia in the distal parts of the limbs. Weakness initially appears in the leg muscles causing foot drop, followed by small muscles of the hands. Later it may extend proximally and even involve the truncal muscles. Deep tendon jerks are absent. The prognosis of patients with mild neuropathy is good but those with severe neuropathy are usually left with persistent deficits like claw hand, foot drop, persistent atrophy, spasticity and ataxias. Human and experimental data indicate that recovery is usually complete in the young.[5]

The occurrence of delayed polyneuropathy appears to follow phosphorylation and subsequent aging of an enzyme in axons called as Neuropathy Target Esterase (NTE). The function of this enzyme is not clear yet. It is however present in the brain, spinal cord and the peripheral nervous system.

Delayed polyneuropathy is common following exposure to OPCs, which have weak anticholinesterase activity Eg. Triortho-cresyl-phosphate.

Parathion [O, O-diethyl O-(p-nitro-phenyl) thiophosphate] belongs to the Aryl phosphate class of organophosphorus pesticides. It is available as a Pale-yellow to dark-brown liquid with a garlic-like odour and gets converted to biologically active compound "Paroxon".

Parathion toxicity is known to cause cholinergic symptoms, dizziness, confusion ataxia, convulsions, coma; hypotension; cardiac arrhythmias. The neurologic manifestations on acute parathion intoxication are known to occur as a part of intermediate syndrome or as a part of delayed polyneuropathy. Mononeuropathy, occurring within 4 days of acute Parathion Intoxication is not known to occur.

CONCLUSION

This is a case of residual mononeuropathy following acute OPC toxicity. Majority of cases of OPC poisoning are reported to be lethal or have delayed polyneuropathy. This case study shows acute axonal motor neuropathy is possible and should also be considered.

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