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Malathion Poisoning Presented as Intermediate Syndrome and Organophosphate Induced Delayed Polyneuropathy in Succession: A Case Report

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Abstract

Organophosphate compounds after exposure cause acute and sub acute manifestations depending on the type and severity of the agents like Acute Cholinergic Manifestations, Intermediate Syndrome and Delayed Central Nervous System Complications. It occurs in association with the ingestion of large amounts of organophosphate after the stimulation of cholinergic receptor. We reported a 36 years-old female patient who developed severe Organophosphates Poisoning complicated by Intermediate Syndrome and Organophosphate Induced Delayed Polyneuropathy in India. It is reported to increase awareness of health care workers to this rare complication.

Keywords: Organophosphate (OP); Intermediate; Polyneuropathy; OPIDPN

Introduction

Organ phosphorus compounds are the organic derivatives of Phosphorous containing acids that is widely used in agriculture, residential landscaping, public recreation areas, and in public health pest control programs. In Organophosphate poisoning the Cholinesterase are phosphorylated by the Phosphate end of Organophosphates and cause accumulation of excessive Acetyl Chlorine at synapses with resultant over stimulation of neurotransmission. The clinical features are due to excess acetylcholine is Muscarinic, Nicotinic and central nervous system three well defined clinical phases are seen: Initial Acute Cholinergic Crisis, the Intermediate Syndrome and Delayed Polyneuropathy (OPIDPN). Here severe and prolonged cholinergic crisis with unusual complications, notably Intermediate Syndrome and OPIDPN are described in the same patient in different course of her illness.

Case Report

A female patient, 36 years old to commit suicide, took a large amount of an organophosphate compound, Malathion on December 2, 2012. She was found fallen in her room and complain of severe abdominal pain, salivation, lacrimation and diarrhea a few hours after the ingestion of the insecticide. At the time on examination in emergency room the patient was showing miotic pupils and fasciculation. Atropine 15 mg IV every 15 minutes, Pralidoxime 500 mg/hr continuous IV infusion ,unquantified amount of Charcoal and 1gm Ceftraxone IV every 12 hours were administered. 72 hours after the appearance of the initial symptoms the patient complain difficulty in respiration followed by respiratory insufficiency, and she was admitted to the Medical Intensive Care Unit (MICU) on December 5,2012.On physical examination at MICU his blood pressure was 90/60 mmHG, pulse rate 78/min, respiratory rate 19/min and temperature was 37.8°C. He had Rhonchi all over the chest, pupils were pinpointed and Glasgow Coma Scale (GCS) was 3/15. He also had paradoxical abdominal muscle movement. On investigations white blood cell count was 11,800/dl, Hematocrit 44.2%, Platelet count 139,000/dl, Erythrocyte Sedimentation rate 26/hr and Random Blood Sugar 110 mg/dl. However, Serum Transaminases, Alkaline Phosphates and liver and renal function tests and Serum Electrolytes were in normal range. Futhermore, chest X-ray and ECG were normal. Oxygen saturation was 90% with 6 L/min flow of oxygen through face mask. Her GCS was 3/15 in the first 24 hrs in the MICU after that she started to respond verbal command but subsequently the level of consciousness was waxing and waning. Therefore, she was continued with large dose of atropine with administration of 2 mg every 15 minutes, Pralidoxime 500 mg/hr continuous IV infusion and on the third day of MICU admission she was intubated and put on ventilator support (Figure 1). She stayed for 72 hours on ventilator support without respiratory drive. After that, she was discontinuation of the mechanical ventilation. She shifted in

Figure 1: IV infusion and on the third day of MICU admission she was intubated and put on ventilator support.

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female medical ward on day 9 from admission, without showing any symptoms she discharged on day 12.

However, five day after the discharge she started having hyperesthesia in the plantar area and cramping calf pain followed by distal weakness in the lower limbs and, two days after, in the upper limbs and urinary and fecal incontinence. On physical examination she was conscious and oriented. Power on both lower limbs was 0/5, while on upper extremity it was 3/5. The patellar reflexes were present and symmetric while ankle reflex absent .while tone was reduced on the lower extremity and all modalities of sensory examinations were normal. On investigations after discharge showed normal Hematological and blood chemistry tests; negative for HIV; normal spine x-rays and normal Magnetic Resonance Imaging (MRI) of spine. To rule out AIDP patient lumbar puncture done on at 2nd day of admission which showed 2 cell lymphocyte and protein and sugar within normal limit In addition, Electrophysiological examination showed no motor response from Tibial and Peroneal nerves and there was severe reduction in amplitude of left Median motor nerve. The sural and plantar sensory nerves were normal. CMAPs were not elicited in the lower limbs. These findings are suggestive of predominant motor axonal polyneuropathy involving mostly the lower limbs.

Discussion

Organophosphate poisoning is one of the commonest types of poisoning in India for suicide attempt [1]. We reported here a case report which was presented with severe poisoning and majority of complications described in literatures were observed in succession in this patient [2-6]. Our patient developed respiratory failure at the 3rd day of poisoning which is typical duration for Intermediate Syndrome. Intermediate Syndrome occurs between the initial acute cholinergic manifestations and the late Organophosphate Induced Delayed Neuropathy and was first described by Wadia et al. [5] but name of the syndrome was given by Karalliedde and Senanayake [3]. The cardinal features of intermediate syndrome comprise muscular weakness, predominantly affecting the proximal limbs muscles and neck flexors. Cranial-nerve palsies are common. In our patient respiratory muscle was affected causing respiratory failure but other muscle groups have not been affected. Unlike the delayed polyneuropathy, this syndrome causes death due to associated respiratory tract infections, air way obstruction by secretions, Pulmonary Edema, and respiratory depression.

The late manifestation which occurred in our patient was sub acutely developed weakness of lower and upper extremity which was diagnosed to be Organophosphate Induced Delayed Neuropathy. OPIDN associated signs include foot drop, weakness of the intrinsic hand muscles, absent ankle jerks and weakness of hip and knee flexors. The course is usually sub acute and occurs within two weeks after the initial symptoms. Commonly the Clincopathology is sensory motor involvement of the peripheral nerves but pure or predominant motor axononal involvement similar to our patient has also been reported [7]. Chia-Chang described in a case report of a 28 years Taiwanese woman a prolonged Cholinergic features for many days and needed Atropine up to 80 mg in an hour with a total dose of 11,665 mg in 17 days who also took Pralidoxime [6]. Such delayed manifestations are reported to occur because of Acetylcholiesterase Enzyme (AChE) aging, poor rephosphorylation and decreased synthesis of new enzymes [2]. After ingesting Malathion, our patient presented all signals and symptoms of the three phases of intoxication by organophosphate, a fact that shows that the quantity ingested was substantial. Rarely, some OPs produce delayed neurotoxicity with the onset of clinical symptoms occurring one or two weeks after the exposure. The occurrence of OPIDN is said to follow the phosphorylation and subsequent ageing of an enzyme in axons called as Neuropathy Target Esterase [4]. The management of severe Organophosphates poisoning is supportive care of airway, oxygenation and administration of high dose of Atropine and rephosphorylation attempts by Oximes. Intubation may be necessary in cases of respiratory distress. Treatment of intermediate syndrome and OPIDPN is by early detection and supportive care like respiratory failure management. There is no specific therapy for the Organophosphate Induced Delayed Neuropathy.

Conclusion

Patients with severe Organophosphate Poisoning can have prolonged cholinergic manifestations which would require large dosage of Atropine administration and thorough clinical evaluation is needed to titrate the dose down and discontinue it. Moreover, careful follow-up is needed for the rare complications of Organophosphate Poisoning like Intermediate Syndrome and OPIDIN. Therefore, I recommend health care professional should be aware of these rare complications, their presentation and how to manage them.

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