Original Article

Organophosphate Poisoning Induced Prolonged Intermediate Syndrome Followed By Polyneuropathy And Neuropsychiatric Challenges: A Case Report

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Abstract

Organophosphate (op) poisoning is a common poisoning in India with suicidal and accidental ingestion. It is associated with high morbidity and mortality. We present 23 year old male with history of OP poisoning who developed intermediate syndrome with polyneuropathy. Patient managed conservatively along with multidisciplinary team approach leading to a successful management.

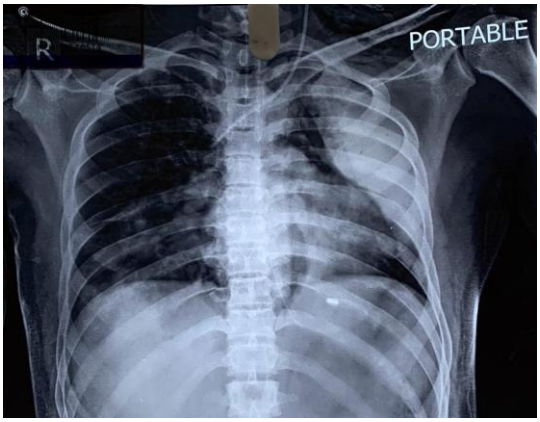
Introduction

Organophosphorus (OP) Pesticides are widely and easily available in market for agricultural use. OP poisoning is most common poisoning seen in India mainly due to suicidal ingestion. The pathophysiological basis for OP poisoning is inactivation of enzyme acetylcholinesterase at the peripheral muscarinic and nicotinic nerve terminal junctions. Three clinical phases of OP poisoning include cholinergic crisis, intermediate syndrome and delayed polyneuropathy [(1,2)].

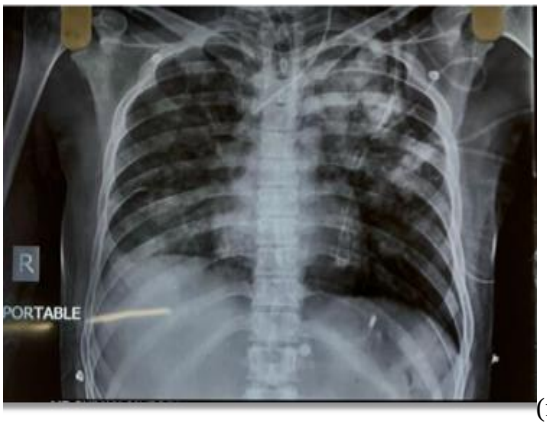
The intermediate syndrome usually occurs 24 - 96 hours following OP poisoning and presents with weakness of muscles of different groups [(3)], with recovery usually occurring in 5 - 18 days. Inhibition of the enzyme Neuropathy target esterase (NTEs) could be responsible for delayed polyneuropathy [(4)]. Various factors that account for variable clinical presentation and stages could be due to the nature of OP compound, severity of poisoning, dose of poisoning, inadequate oxime therapy and delayed treatment.

Case Report

We report a case of a 23 years old young boy presenting to the Emergency Room two hours after ingestion of unknown substance. Patient was a known case of depression On arrival, patient was unconscious, vomiting, tachycardiac, pin point pupils, defecation along with profuse sweating and gasping state with desaturation of 68 %. Patient was immediately intubated and mechanical ventilation was started with high suspicion of OP poisoning based on clinical features. Gastric Lavage was done and after initial stabilization and resuscitation. Medical management in the form of inj .Atropine 1.8 mg loading dose was given followed by a maintainence at 4mg / hour and inj . Pralidoxime 1.8 gm loading dose followed by 600 mg / hour was initiated along with other supportive care. Patient was shifted to Intensive Care Unit (ICU) for further management. In ICU, despite modifications in mechanical ventilation , desaturation persisted. Chest radiograph revealed left lower lobe collapse for which emergency bronchoscopy and BAL (Broncho Alveolar Lavage) was done. Sedation and paralysis was continued along with high PEEP and high FiO2. After 3 days, sedation was stopped and weaning started, considering decrease in oxygen demand and clinical improvement. Patient was not able to lift upper limb and lower limb bilaterally . Weakness was more in lower limb as compared to upper limb. Patient also had an episode of seizure which was associated with frothing and upward rolling of eyes with facial twitching. Antiepileptics were started, all metabolic workup was done which was within normal limits. NCCT Head revealed no significant pathology. EEG showed no epileptogenic activity. Power significantly decreased in both limbs, however, the decrease was more in the lower limbs(LL) as compared to upper limbs (UL) and deep tendon reflexes were depressed in both of the limbs. Cranial nerves and sensory nerves examination revealed normal findings. Nerve conduction velocity (LL > UL). During the course of ICU stay patient had higher TLC count, mild renal derangement and spikes of fever. BAL - culture and sensitivity revealed Acinetobacter for which antibiotics modified as per sensitivity. Percutaneous tracheostomy(PCT) was done in view of prolonged ventilatory support and difficult weaning. Multidisciplinary approach with involvement of physiotherapy, occupational physiotherapy, psychiatry consultation along with clinical support , significant clinical improvement was observed . Patient's trachea was therefore decannulated on day 24 and patient tolerated the decannulation very well. There was significant improvement in muscle strength and power of both upper limb and lower limb. The sepsis began to subside, progressive chest X-ray were done which revealed significant improvement in lung fields. For DVT Prophylaxis, VT pump and LMWH was given during the course of treatment. Antibiotics were deescalated and patient was successfully discharged on day 28 and was advised to follow up in OPD. During subsequent follow up patient was perfectly fine with no residual muscle
weakness.



(fig1-chest x-ray shows LL collapse)



(fig2- chest x-ray post bronchoscopy)

Discussion

OP poisoning is one of the most common poisoning seen in Emergency Medicine departments requiring intensive monitoring and urgent interventions. The diagnosis of poisoning is done clinically based on nicotinic and muscarinic signs and symptoms. Intermediate syndrome usually develops in cases of severe OP poisoning within 1 - 4 days after Ingestion and may last up to 7 - 21 days [(5)]. In our case, Intermediate syndrome lasted for a long period of about 24 - 28 days necessitating the prolonged ventilatory respiratory support and rehabilitation for muscle strength and recovery. Intermediate syndrome clinical spectrum is categorized by weakness of proximal muscles, neck muscles, cranial nerve palsies, accessory respiratory, muscles and diaphragm [(6)]. The term Intermediate syndrome (IMS) is coined by Senanayake and Karalliede [(7)]. The exact mechanism is yet not clear, but many electrophysiological studies by EMG and repetitive nerve stimulation suggest it might be due to the combination of pre and post synaptic impairment of neuromuscular junction [(8,9,10)]. Consciousness Is usually preserved unless complicated by any neurological issue like seizure / brain edema, hypoxia related to respiratory failure. In our case, NCCT done revealed no abnormality along with electroencephalogram (EEG) to rule out any epileptogenic forms. Nerve Conduction velocity test revealed bilaterally decreased nerve conduction velocity[(11,12)]. All metabolic reasons to cause seizures were ruled out. In our case, patient required longer duration of

respiratory / ventilatory support due to the ongoing infection in lungs and sepsis and due to the muscle weakness/respiratory weakness. The multidisciplinary approach involving physiotherapy, occupational physiotherapy, dietician, psychiatrist and family support helped in overall improvement in muscle strength and complete functional recovery. Appropriate clinical decisions, supportive care, evaluation and management plays a vital role along with multidisciplinary approach.

Conclusion

There is a high incidence of morbidity and mortality associated with intermediate syndrome in OP poisoning. Early recognition and suspicion in the clinical presentation of OP poisoning is must and timely intervention is crucial. Multidisciplinary team approach along with rehabilitation program holds the key for successful outcome.

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