Delayed presentation of intermediate syndrome in a patient with organophosphorus poisoning: A case report

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Abstract

Acute organophosphorus poisoning is the most common cause of deliberate self- poisoning in rural regions of developing countries [1] with a spectrum of presentation depending of the type, degree of exposure and strength of the chemical compound involved. The effects are categorised into acute, delayed and late presentations. Here, we discuss about the delayed presentation of intermediate syndrome in a 42-year-old man who was treated for self- poisoning with organophosphorus pesticide 13 days before his presentation to our ED.

Key-words

Organophosphorus poisoning, Carbon dioxide retention, Management.

Background

Organophosphorus pesticide self-poisoning is an important clinical problem in rural regions of India and other developing countries, with a case fatality rate of >20% (2). The common cause of death in organophosphorus poisoning is respiratory paralysis. Hence, early resuscitation with atropine, oxygen, respiratory support, oximes and fluids is vital in the immediate management, guided by the clinical acumen to recognise the varied and unusual presentations.

Case Presentation

A 42-year-old gentleman presented to our ED with complaints of shortness of breath and altered sensorium for 2 days. He had history of organophosphorus poisoning 13 days earlier for which he was treated in a hospital at Nellore, Andhra Pradesh. The patient was treated with atropine, pralidoxime and other supportive measures initially and was discharged home. The patient started to have difficulty breathing for 2 days that progressed to respiratory distress and drowsiness at the time of presentation.

On Examination

The patient was drowsy, tachycardic but obeyed commands on arrival to ED. He was noted to have weakness of head, neck and proximal limb muscles, with pinpoint pupils.

Management

The initial ABG revealed mild carbon dioxide retention. The patient was initiated on non-invasive ventilatory support, intra venous antibiotics and other supportive measures. Pralidoxime was given as continuous infusion and the patient was nursed in the intensive care unit.

The patient was intubated on day 2 of hospitalisation in view of worsening clinical condition. CT scan of brain was done which showed no abnormality.

CT chest revealed features of bilateral aspiration pneumonia. The levels of serum pseudo choline esterase were monitored serially. Tracheostomy was done in view of prolonged ventilator support.

The patient showed improvement of muscle weakness.

The patient remained stable in the ward, eventually discharged home with complete recovery after 17 days of hospitalisation.

Discussion

The signs and symptoms of organophosphorus poisoning are grouped into acute cholinergic crisis, intermediate syndrome (IMS) and delayed polyneuropathy.

The acute crises are due to the inhibition of acetylcholine esterase which results in the accumulation of acetylcholine and overstimulation of the receptors at the synapses of autonomic nervous system, neuromuscular junction and central nervous system. [3]

The acute presentation occurs within minutes up to 24 hr of ingestion and it includes,

Acute muscarinic symptoms- increased salivation, lacrimation, urination, miosis, bronchospasm, bradycardia and hypotension.

Acute nicotinic symptoms- fasciculation's, cramps, muscle weakness & paralysis.

Acute CNS symptoms include anxiety, restlessness, convulsions & respiratory depression.

The intermediate syndrome (IMS) usually occurs 24–96

hr of ingestion and is characterised by weakness of muscles in head, neck, proximal limbs eventually leading onto weakness of muscles innervated by the cranial nerves and respiratory paralysis [4]. This usually occurs between 2–4 days of organophosphorus pesticide exposure and lasts for 4–18 days with complete recovery [9].

The delayed polyneuropathy symptoms are attributed to the inhibition of neuropathy target esterase (NTE) which occurs 1–4 weeks post exposure.

The delayed presentation of the intermediate syndrome is attributed to the type and amount of exposure to organophosphorus compounds as these are fat soluble, get redistributed and remain in the body for longer duration to produce these delayed symptoms.[8] The initial treatment with inadequate doses of oximes or early discontinuation of oxime therapy is also attributed to the delayed occurrence of intermediate syndrome. The development of IMS has been reported most commonly following ingestion of Diazinon, Dimethoate, Fenthion, Malathion, Methamidophos, Methylparathion, Monocrotophos and parathion.[7]

The treatment with atropine at the dose of 0.5–2 mg every 15 min till secretions have dried, has been recommended.[6] Some studies have found high dose atropine infusion to be better especially in Indian patients. Pralidoxime is administered as bolus dose followed by continuous infusion to maintain a minimum blood concentration of 4 µgm/ml to obtain maximum benefit [5]. It is recommended that pralidoxime should be continued till RBC cholinesterase levels return to 50% of normal value. Decision for mechanical ventilatory support depends on patient's respiratory efforts, sensorium, presence of pinpoint pupils, fasciculations and convulsions. Hence early recognition of the condition and initiation of mechanical ventilatory support is key to reduce mortality.

Conclusion

As most of the presentation is accounted from the rural areas with limited medical and intensive care facility, it is also important to educate the physicians to recognise the clinical syndrome and their deviant presentations. Early recognition of such presentation is crucial and initiation of mechanical ventilatory support is critical in limiting the mortality of such cases. This case report emphasises on the importance to recognise the deviant presentation and timely management.

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