Intermediate syndrome in organophosphorous poisoning-A case report
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Abstract
A case of organophosphorous poisoning in a 29 year old male who developed intermediate syndrome manifested by features of respiratory depression as evidenced by marked weakness of the respiratory muscles, tachypnoea, and drop in oxygen saturation despite reversal of nicotinic and muscarinic effects of organophosphorous poisoning. The case highlights its early recognition and prompt institution of mechanical ventilation with continuation of anticholinergic drugs. The mechanical ventilation had to be continued for 9 days with successful outcome.

Discussion
Intermediate syndrome (IMS)-first termed by Wadia et al2 as type II paralysis (1974), is a syndrome characterized by muscle paralysis following the acute cholinergic phase. The terminology was later changed by Senanayake and Karalliedde3 in 1987 to intermediate syndrome due to the fact that it arises between the period of early cholinergic syndrome and late onset peripheral neuropathy.

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The incidence of IMS in different studies has been reported to be between 20-68%\(^4\). However it was 8.33% in our cases. IMS could be due to a conformational change in acetylcholine receptor altering the depolarization neuromuscular block to a nondepolarisation block, characterized by fade on tetanic stimulation as reported by Senanayake and Karelliede\(^3\).

IMS develops 12-96 hrs after exposure and reflects a prolonged action of acetyl choline on the nicotinic receptors. The clinical features are muscular weakness in ocular, neck, bulbar, proximal limb and respiratory muscle with occasional dystonic posturing requiring mechanical ventilation in an ICU for several days. Cranial nerve palsies are common. The risk of mortality is due to the associated respiratory depression. The sensory functions characteristically remain normal and full recovery is evident in 4-18 days. Our patient had developed respiratory muscle weakness as evidenced by drop in oxygen saturation, decrease in respiratory rate and failure to wean off from ventilator. There was no evidence of weakness of ocular, neck, bulbar, proximal limb muscle or cranial nerve palsies.

It has been commonly associated with organophosphorous compounds like diazinon, dimethoate, methyl parathion, methamidaphes, monocrotophos, fenthion and ethyl parathion\(^5\). Despite its common occurrence, data on risk factors of IMS, early diagnosis and prediction have remained elusive. Commonly used tests such as levels of plasma cholinesterase correlate poorly with the onset of IMS\(^6\).

**Conclusion**

IMS is an important complication of OP poisoning and should be recognized and treated adequately. There should not be delay in intubation and mechanical ventilation. Atropine has to be continued depending upon the clinical response. Mechanical ventilation might have to be continued for long time depending upon clinical response of the patients.

**References**