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Case Note

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.

According to WHO one million serious accidental and two million suicidal poisonings due to insecticides occur worldwide every year of which 200,000 patients die with most deaths occurring in developing countries (1).A WHO study on pesticide poisoning in SEARO countries revealed that among total of 258 cases of acute pesticide poisoning in Nepal 91.5% were intentional cases followed by 8.2% occupational exposure and 1.16% accidental cases.

Organophosphorous compounds are irreversible inhibitors of the enzyme acetylcholinesterase, causing accumulation of acetylcholine at synapses with resultant over stimulation of neurotransmission. The clinical features are due to excess acetylcholine at the muscarinic and nicotinic receptors which leads to initial stimulation and eventual exhaustion of cholinergic synapses. The cholinergic effects depend on the balance between muscarinic and nicotinic receptors and is best described by mnemonic *DUMBELS* – *d*iarrhoea, *u*rination, *m*iosis, *b*ronchospasm, *e*mesis, *l*acrimation and *s*alivation. 12 patients were treated for organophosphorous poisoning at Manipal Teaching Hospital during 2004-05, of which one patient developed intermediate syndrome which is being reported here.

Case report
29 years old male, ex-IV drug abuser was admitted in ICU with alleged history of suicidal attempt by consumption of dichlorovos 76% of 7hours duration. On examination, patient was drowsy; responding to commands.Pulse-80/min,BP-140/90mmHg,RR-16/min.Pupils-2mm size bilateral and reacting. There was diffuse crepitations and rhonci on examination of chest. He was treated with atropine infusion 8mg/hr, PAM 500mg iv 8th hourly. On 2nd day despite full atropinization patient developed respiratory arrest with no spontaneous breathing and was intubated and ventilated. Atropine infusion was continued. PAM was stopped on 3rd day. On 4th day, clinical condition deteriorated. Oxygen saturation dropped down to 70% with evidence of collapse of left lung; tracheostomy was done and bronchoscope suction helped the lungs to expand. Patient improved .Oxygen saturation was maintained above 90%.Since patient was fully atropinized with better sensorium; he was weaned off from ventilator and put on T-piece trial. However, on 6th day, the patient could not maintain oxygen saturation and there was persistent respiratory depression with RR-30/min and drop in oxygen saturation. A diagnosis of intermediate syndrome was made clinically and patient was placed again on ventilator after which the saturation was maintained above 90%.Atropine infusion was continued .Patient was weaned off successfully from ventilator on day 9.Atropine infusion tapered and stopped on day 12.

Discussion
Intermediate syndrome (IMS)-first termed by Wadia et al[2] 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 Karalliedde[3] 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, methamidaphe, 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.

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