Organophosphate poisoning: the importance of the intermediate syndrome

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Abstract

Organophosphate poisoning is the commonest poisoning in Nepali hospitals, partly due to the easy access of these pesticides from the market. This case report emphasizes the need to closely monitor patients with organophosphate poisoning so that the intermediate syndrome, a well known, life threatening phenomenon related to this poisoning, can be effectively dealt with. The pathophyiology and treatment of the intermediate syndrome is discussed.

Keywords: organophosphate poisoning; intermediate syndrome.

Case Report

A 17 year old girl who just heard of her failure in the SLC exams, presented to Patan Hospital with a history of ingestion of 1 bottle of Metacid (organophosphate pesticide) 30 mins prior to arrival to the hospital. On arrival her blood pressure was 80/50 mm of Hg, heart rate - 120/min, pupils were 5 mm bilaterally, equal and reactive to light. Gastric lavage was done, charcoal and magnesium sulphate was given, intravenous access was secured, intravenous fluids and atropine was started, but she progressively deteriorated; blood pressure dropped further to 60/40 mm of Hg; pupils narrowed to 3 mm, respiration was noisy and laboured. She was intubated, suctioned and was artificially ventilated. She received more atropine and pralidoxime 1 gm was given intravenously over 30 min. Within an hour, she stabilized after 24 mg of atropine. Pupils were 6 mm, blood pressure 90/70 mm of hg, heart rate 132/min and spontaneous respiration was adequate and chest was clear. She was put on regular atropine and more pralidoxime. She continued to improve over the next 4 days and the atropine was tapered. On the 4th day of hospitalization (when she was on atropine 1 mg 8 hourly) she complained of some difficulty in breathing and weakness of her neck and shoulder muscles, then became acutely short of breath and began to gasp. Pupils were 2 mm. She was again intubated and respiration supported artificially. Atropine was again increased to 40 mg/hour and more pralidoxime given. Her condition remained unchanged for the next 4 days. She was then transferred to the intensive care unit and tracheotomy done. Finally after another 6 days she recovered. There was no delayed neuropathy as she continued to recover and went home.

Discussion

Organophosphate group of poisons produce their toxic effect by inhibiting the activity of the enzyme acetylcholinesterase.[1] This results in the build up of acetylcholine in the neuromuscular junction and results in a cholinergic crisis.

Organophosphates are absorbed across the lung, mucous membrane (including gut), skin and symptoms may appear within a few minutes to up to 12 hours; clinical manifestation depends upon inherent toxicity, dosage, rate of absorption and rate of organophosphate metabolism breakdown.

Organophosphates may present with muscarinic effects which include bradycardia, bronchospasm, bronchorrhoea, salivation, lacrimation, diaphoresis, vomiting, diarrhoea, and miosis which is reversed by atropine, the dose of which has individual variation. Some patients may need almost a gram of atropine a day. The dose of atropine is titrated by reversal of the above symptoms, the most important being drying up of the bronchopulmonary secretions. Heart rate and pupil size are not as reliable[1], but very often because pupil size offers a prompt, objective, and quantifiable data, this is used for atropine titration.

Organophosphates' nicotinic effects include tachycardia, fasciculation, mydriasis, muscle cramps, weakness and respiratory paralysis which is helped by pralidoxime, an acetylcholinesterase reactivatior; the earlier this is administered the better. Organophosphates may also demonstrate central effects like CNS depression, agitation, confusion, delirium, coma and seizures.

The intermediate syndrome as demonstrated by this patient when she had to be intubated the second time can be a sinister complication during apparent recovery, but with prompt therapy the outcome is favorable. The

syndrome occurs 1 to 4 days after the acute poisoning.[2] It is called intermediate because it takes place between the acute presentation and the delayed neuropathy, the latter may be linked with fat-solubility of some of the organophosphates.[3]

The most important feature of the intermediate syndrome which may present with facial, extraocular, neck and proximal limb weakness is respiratory paralysis.[4] With ventilatory support as in the patient in question recovery is likely. Without ventilatory support death ensues. Continued treatment with atropine and pralidoxime although said to be ineffective is usually carried out. Improvement may take 5 to 15 days.

What is the cause of the intermediate syndrome ? Experimental and clinical observations seem to suggest that this syndrome is due to the severity of the poisoning relating to prolonged inhibition of the acetylcholinestrase activity.[5] This is not related to the delayed neuropathy.

It may be important to observe the respiratory function (perhaps with a peak flow meter) for up to at least 4 days[4] even as the atropine is being tapered so that ventilatory support if necessary (and where available) can be used. The more severe poisonings may need closer monitoring.[5] Inadequate pralidoxime therapy is proposed to be contributory to the intermediate syndrome[4] but the cost involved for repeated pralidoxime administration may be prohibitive in the Nepali setting.

Organophosphate poisoning is by far the most common poisoning presenting to a hospital in Nepal. Unlike Western countries the local observations is that most people who attempt suicide here do it one time only; repeated attempts are uncommon. Hence if the patient can be saved and psychiatric follow-up planned this may be very beneficial.

References

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