



Case Report

Fasciculating toxicity

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ABSTRACT

Organophosphorus (OP) compound poisoning with suicidal intent is common. It is one of the frequent cause for admission to hospital Emergencies and intensive care units in our region. We describe here a case of 19-year old girl who presented to the Emergency Department with atypical features of OP poisoning. She had perioral, tongue and lower limb fasciculations along with generalized muscle weakness with no or minimal muscarinic effects. OP poisoning with isolated nicotinic receptor mediated effect is often reported in children but in adult it is extremely rare. Based on history and clinical suspicion of nicotinic receptor mediated effect of OP, she was given intravenous atropine along with other supportive treatment. Patient got completely recovered from fasciculations and her motor weakness improved after 6 h of atropine therapy. Emergency physician should keep a high index of suspicion of isolated nicotinic and ganglionic mediated effect of OP and a trial of atropine should be given to the patient.

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1. Introduction

Organophosphate (OP) is used both as an agricultural pesticide as well as a household insecticide. The most common mode of poisoning is voluntary ingestion with suicidal intent. Organophosphate poisoning is one of the frequent causes for admission to hospitals and intensive care units (ICU) in the South East Asia and specially in India. Usual presentation is with muscarinic features like diarrhea, vomiting, sweating, lacrimation, pin-point pupils and increased respiratory secretions. Here we report a case of organophosphate poisoning who presented with only nicotinic features.

2. Case

A 19-year-old girl presented to our emergency with complaints of nausea, vomiting and dull abdominal pain. Her mother gave a clear history of ingestion of a pesticide with suicidal intent one day before, but she was not able to tell either the ingredients or the brand name of the pesticide. On initial assessment the patient did not have garlic breath odour or increased salivation or sweating. The respiratory rate was 16 per minute, oxygen saturation 98% on room air and breath sounds were normal without any crepitations or rhonchi. Her blood pressure was 134/90 mmHg with a pulse of 105 per minute. Her sensorium was intact with Glasgow Coma Score (GCS) of E4V5M6 but had generalized muscle weakness. Pupils were normal-sized and reacting

to light. Her plantar reflexes were flexor bilaterally. During further examination, it was seen that patient had perioral fasciculations, tongue fasciculations, and thigh muscle fasciculation (Videos 1 & 2). On investigation her blood glucose level was 148 mg/dL on arrival. Chest X-ray and ECG were within normal limits. Blood counts showed hemoglobin of 12.3 g/dL, leucocytes of 18,800/ μ L and platelet count of 194,000/ μ L. Arterial blood gas analysis revealed pH of 7.44, pCO₂ of 37.0 mmHg, pO₂ of 94.2 mmHg. Blood chemistry revealed serum bicarbonate of 25.7 mmol/L sodium 145.9 mmol/L, potassium 4.23 mmol/L, ionized calcium 1.2 mmol/L. Urine toxicology screen was positive for tetrahydrocannabinol and paracetamol. She was treated with intravenous fluids and antibiotics. Considering a clear history of pesticide ingestion and presence of clinical signs like muscle weakness and fasciculations patient was treated with atropine and broad spectrum antibiotics in view of increased total leucocyte count. Our patient received injection atropine 1 mg iv bolus followed by 3 further doses of iv boluses of 2 mg, 4 mg and 8 mg at an interval of 5 min. After this, she was given an infusion of atropine at the rate of 1 mg/h and the dose was titrated as per the clinical response. After 6 h of infusion, her fasciculation improved (Video 3). She was discharged after 6 days without any complications.

3. Discussion

Organophosphate compounds are used for manufacturing chemicals which are used for both domestic and industrial purposes. They are also used as insecticides, nerve gases and ophthalmic agents. Organophosphates are mostly used for suicidal purpose as they are commonly available in the household settings. Poisoning with OP compound is

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prevalent in developing countries that include countries of South East Asia and it causes significant mortality and morbidity [1].

According to the WHO task group, about 1 million serious unintentional poisonings cases occur each year while patients who get hospitalized for suicide attempts with pesticides are about 2 million [2]. Although ingestion with suicidal intent is a common mode, accidental exposure and inhalation while spraying in fields is an important modality of poisoning. The clinical presentations and final outcome of organophosphate poisoning depends not only on the type of organophosphate but also on the dose, the route of administration, time between poisoning and initiation and intensity of the treatment administered [3]. Our patient was one of the suicidal organophosphate poisoning. Organophosphate acts by blocking acetylcholinesterase enzyme which degrades acetylcholine. Excess of neurotransmitter acetylcholine at neuromuscular junction and central nervous system (CNS) can lead to cholinergic crisis with varied clinical presentation based on the receptor on which it acts [4]. Muscarinic effects of organophosphate poisoning are excessive salivation, lacrimation, urination, defecation, increased GI secretion, miosis, bradycardia, bronchorrhea and bronchospasm. Nicotinic effects include fasciculations, muscle weakness, and paralysis via acetylcholine stimulation of nicotinic receptors at the neuromuscular junction while central nervous system effect include central respiratory depression, emotional lability, lethargy, anxiety, tremors, seizure and coma. Intermediate syndrome and delayed polyneuropathy are the complications of organophosphate poisoning [5]. Our case presentation was atypical where nicotinic receptor mediated symptoms like generalized muscle weakness, fasciculations and tachycardia were the main features with little or no muscarinic mediated symptoms. Though cases are reported where fasciculations are predominant features but isolated presentation of generalized fasciculations and weakness are rare [6,7]. Isolated nicotinic mediated symptoms are common with parathion poisoning but can be seen in other OP poisoning as well. Diagnosis of OP poisoning should ideally be confirmed with an assay to measure butyrylcholinesterase activity in plasma or acetylcholinesterase in red blood cell [8]. However, like in our case the results of such assays are rarely available in time to affect clinical decision making. In our region parathion, malathion, fenthion, chlorpyrifos, diazinon and dichlorvos are easily available, hence poisoning with these OP compounds are common [9]. Parathion and dichlorvos are highly toxic while malathion and chlorpyrifos are less toxic. Toxicity of OP, their onset of action and duration of toxicity also depend their lipid solubility. Lipid solubility of OP compound is defined by its octanol/water coefficient. Intermediate to high lipid soluble OP compound like fenthion, malathion will have delayed onset of poisoning and may need prolonged

hospitalization and mechanical ventilatory support because of redistribution of OP compound from lipid store [10]. Assessment and management of airway, breathing and circulation should be done first followed by decontamination in early presentation. Atropine is the mainstay of treatment for muscarinic features. Oximes use in OP is controversial but can be used early and in moderately severe OP poisoning.

4. Conclusion

Onset of symptoms in organophosphate poisoning may be delayed and presentations may be atypical. Even though the symptoms are mild initially, observation for longer period is required. The treating physicians should be vigilant to pick subtle and atypical signs of organophosphate poisoning. Even in the condition of slight suspicion of poisoning, appropriate treatment should be started promptly. Prognosis is excellent if timely treatment is given.

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