Organophosphorus poisoning: A case report with review of literature

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Abstract

Organophosphorus (OP) poisoning is a commonly encountered major clinical problem in many countries of the world, more so in the developing countries. The authors present a case of suicidal OP poisoning with an overview of management of the acute intoxication. The early recognition of symptoms and prompt treatment helps in reducing the morbidity and mortality.

Keywords: Atropine; Insecticide; Intoxication; Intermediate syndrome; Organophosphorus compound; Pesticides; Plasma cholinesterase; Poisoning

Introduction

Organophosphorus (OP) pesticide suicidal poisoning is a major public-health concern across the majority of the rural Asia, including the rural Indian population, owing to its easy and cheap availability in these countries[1-4]. It is also of great importance to the clinicians due the high morbidity and mortality associated with its ingestion[1-4]. Early recognition of the toxic syndrome and its prompt management is the key for saving the patient's life. The authors present a case of alleged suicidal OP poisoning with an overview of management of acute intoxication.

Case Report

A 45-year old Indian male presented to the emergency department with an alleged history of ingestion of a few drops of insecticide (dichlorvos) which was followed by multiple episodes of vomiting and breathing difficulty. The patient was initially taken to a local hospital where gastric lavage was done with normal saline and then was shifted to our hospital for further management. There was no history of seizure, loss of consciousness or chest pain. On examination, he was conscious, but restless, no focal deficit, no nuchal rigidity, with both pupils pinpoint. His blood pressure 150/80mmHg, pulse 60/minute, tachypnea with respiratory rate 30/minute, random blood sugar 200mg%, with excessive oral secretions, chest auscultation-bilateral crepitation, heart sounds normal with no murmurs. The clinical features were suggestive of OP poisoning. He was immediately started on intravenous (IV) fluids, oxygen support and IV atropine. He required 6mg of atropine for complete atropinization which was maintained by infusion at 1.8mg/hour for next two days and then gradually tapered off. He was shifted to the intensive care unit and started on pralidoxime (2gm IV bolus then 2gm every 4th hourly). Patient's initial investigations, including complete haemogram, coagulation profile, liver function and renal function tests were normal. His plasma cholinesterase enzyme was low, thus confirming the diagnosis. He showed improvement in symptoms with no further signs of intoxication over the next five days. He was transferred to room in a stable condition with advice about the long term effect of the OP poisoning and psychiatry follow-up.

Discussion

OP compounds are widely used as pesticides in agriculture, vector control, and domestic purposes. OP compounds act by irreversible inhibition of cholinesterase and pseudo cholinesterase (butyrylcholinesterase) enzymes, which are responsible for hydrolysis of acetylcholine (neurotransmitter) into choline and acetic acid[4,5]. Thus inhibition of cholinesterase enzymes leads to an accumulation of acetylcholine at neural synapses, causing overstimulation of neurotransmission in central nervous system, autonomic nervous system, and the neuromuscular junctions[4,5].

The various modes of intoxication due to exposure to OP compounds include gastrointestinal (suicidal or accidental ingestion), dermal (absorption from broken skin), mucosal (conjunctiva, buccal mucosa), respiratory (inhalation) and intravenous routes[4,5].

Clinical Features

Acute toxicity: The signs and symptoms of OP poisoning usually manifests in thirty minutes to three hours, and are divided into- a) muscarinic effects, b) nicotinic effects, and c) central nervous system (CNS) effects[4-7] (Table 1).

Clinical features and its severity vary according to the poisonous agent, the route of intoxication and the dose.

Intermediate Syndrome: It was initially described in 1974 by Wadia et al. (known as type II paralysis) and later renamed as an intermediate syndrome by Senanayake et al. in 1987[7]. This refers to the muscle paralysis (main involvement of bulbar, respiratory, and proximal muscles) following the acute cholinergic phase, occurring 24–96 hours after exposure. It usually resolves in 1–3 weeks[7]. The patient may require mechanical ventilation due to respiratory muscle paralysis, and complications may occur due to infections or cardiac arrhythmias[4-7].

Organophosphate-induced delayed polyneuropathy (OPIDP): This occurs about 2-4 weeks after exposure to large doses of certain OP compounds. It is characterized by distal muscle weakness causing ataxia, foot drop and claw hand, and is due to inhibition of neuropathy target esterase[6]. The recovery may take weeks to months[6].

Diagnosis is based on the clinical history or suspicion, the clinical signs, smell of pesticides or solvents, and reduced levels of butyrylcholinesterase or acetylcholinesterase activity in the blood[4,7]. The decreased levels do not correlate with the severity of the intoxication[7].

Management: Decontamination- Clothes are removed off the patient and the patient is thoroughly cleaned gently with soap and water. Eyes are irrigated with normal saline. Gastric decontamination is done by giving a gastric lavage. Healthcare workers protect themselves by using gloves, gowns and eye/foot wear.

Medication: The mainstay of treatment is atropine, pralidoxime (2-PAM), and benzodiazepines (for anxiety, restlessness and seizure control).

Atropine: 1–3 mg of atropine is given intravenously as a bolus and repeated after every 5 minutes till the end points of atropinization are achieved, they are-- clear chest on auscultation with no wheeze, heart rate> 80 beats/min, pupil no longer pin point, dry axilla/no sweat, and systolic blood pressure > 80 mm of Hg. After achieving atropinization, the effect is maintained by atropine infusion (dose of 20% to 30% of the total amount initially required to atropinize), which is continued for the 2 to 3 days and then tapered off.

Pralidoxime (2-PAM): Oximes, also known as cholinesterase re-activators, are used as antidotes in the cases of OP poisoning. The recommended dose of PAM is 2 g (25 – 50 mg/kg in children) intravenously over 30 minutes, followed by infusion at 8 mg/kg/hour in adults (10 – 20 mg/kg/hour in children) or 2g every 4-6 hours. PAM must not be given without concurrent atropine, as oximes can transiently induce cholinesterase inhibition and worsen the symptoms. Oximes are effective if administered early before the aging (irreversible binding of OP with acetylcholinesterase) occurs (preferably given with 12 hours of intoxication, but can be given up to 48 hours after intoxication).

Table 1: Signs and symptoms of OP poisoning

Muscarinic effects	**SLUDGE:** Salivation, Lacrimation, Urination, Defecation, Gastric upset, Emesis. **Killer Bs:** Bronchorrhea, Bronchospasm, Bradycardia. **DUMBELS:** Defecation, Urination, Miosis, Bronchorrhea/ Bronchospasm/ Bradycardia, Emesis, Lacrimation, Salivation.
Nicotinic effects	Muscle fasciculation, cramping, weakness, diaphragmatic failure. Autonomic effects- hypertension, tachycardia, and pallor
CNS effects	Anxiety, emotional lability, restlessness, confusion, ataxia, tremors, seizures, coma

Supportive measures: These include oxygen support (ventilator support may be required in cases of severe intoxication causing bronchorrhea induced bronchospasm or respiratory muscle paralysis), intravenous fluids, and maintaining electrolyte balance. Psychiatry referral is required in cases of suicidal ingestion and also in cases involving neuro-psychiatric side effects of the poisoning.

Conclusions

This case reminds and emphasizes about the rampant misuse of OP due to its uncontrolled and easy availability in the developing countries, leading to life threatening intoxications (mostly suicidal and occasionally accidental). The treating physicians have to be aware and vigilant of the OP intoxication syndromes for administering early and appropriate treatment to the patient, thus decreasing the morbidity and mortality associated with it. Besides, efforts should be made to control the easy accessibility of the OP compounds and thus the importance of healthcare information to the masses is very important and the agencies involved actively in the dissemination of information in a resource poor country have to work day and night to prevent such accidents in future.[8-17]

Conflicts of interest: None declared

Acknowledgements: None

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