Case Report

# Management of self-inflicted oral organophosphate poisoning in adolescence - a case report

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## ABSTRACT

Organophosphate (OP) poisoning is more common in developing countries such as India. Here, we report a case of self-inflicted oral OP poisoning (monocrotophos) by an adolescent male patient who presented to the emergency department of a tertiary care hospital with tachycardia and frothing without seizure episode (non-linear presentation in OP poisoning). Based on the evidence of consumption of OP compound, the management of the patient went as planned and guarded with i.v. administration of pralidoxime and atropine. Gastric lavage was done soon after the patient came to the hospital and was admitted to the Intensive care unit for 5 days and in the general ward for the next 24 h. The patient was discharged from the hospital in a hemodynamically stable state after 6 days of hospital stay by managing the cardiac, muscarinic, and nervous system events as detailed in this case report.

**Key words:** Acetylcholinesterase, Atropine, Gastric lavage, Organophosphate poisoning, Pralidoxime

Organophosphorus (OP) self-poisoning is an important clinical problem in developing countries. An estimate of 200,000 people per year died due to OP self-poisoning with a fatality rate of >15%. OP inhibits acetylcholinesterase (Ach) enzyme at nerve synapse and butyrylcholinesterase on the red cell membrane, of which inhibition of Ach results in the clinical presentation [1]. Inhibition of Ach results in acetylcholine accumulation and overstimulation of Ach receptors in the synapses of the autonomous nervous system, central nervous system (CNS), and neuromuscular junction. Table 1 provides the clinical presentations of Ach receptors overstimulation at different regions. OP intoxication can be through inhalation, ingestion, or dermal contact. The severity depends on the quantity of OP intoxicated and the route of intoxication. In 10–40% of poisoning cases, characteristic neurological features such as neck flexion weakness, decreased deep tendon reflexes, cranial nerve abnormalities, proximal muscle weakness, and respiratory insufficiency occur which are referred to as "Intermediate Syndrome" (IMS) [2]. OP-induced IMS was firstly reported in Sri Lanka in 1987 [3].

## CASE REPORT

A 16-year-old male with a bodyweight of 60 kg presented to the emergency with an alleged history of consumption of OP compound (Monocrotophos, one of the OP compounds, as indicated on the box presented by the relatives) of an unknown quantity at his residency 4–5 h before the hospital presentation. As soon as, the patient presented to the emergency department in view of the OP compound odor, the patient was undressed and cleaned with normal saline to mask the smell from the OP compound that fell on the dress and adsorbed on the dermal tissue while intoxication, if any.

At the time of arrival, the patient was drowsy and frothing without a history of vomiting and convulsions. Initial vitals were as follows: Blood pressure 160/100 mmHg; pulse rate 135/min; respiratory rate 24/min; $SpO_{2:}$ 92% on 15 liters of $O_2$; and general random blood sugar 200 mg/dl. Physical examination showed bilateral ptosis, pinpoint pupils, neck dropping+, power 0/5 in all the four limbs, OP odor+, and Glasgow Coma Scale 7/15 ($E_2V_2M_3$).

Pathological examination showed serum cholinesterase of 407 U/mL and blood urea of 124 mg/dl. Initial arterial blood gas (ABG) showed severe mixed acidosis with pH: 7.255; $pCO_{2:}$ 44.99 mmHg; $pO_{2:}$ 77.91 mmHg; and $HCO_3^-$: 20.16 mmol/lit. Chest X-ray showed bilateral pneumonia as shown in Fig. 1.

In view of the low saturation and aspiration, the patient was intubated in an emergency, sedated, and paralyzed. Gastric lavage was done with 5 liters of normal saline through Ryle's Tube (Nasogastric tube), given with pralidoxime (PAM) (inj. PAM) 2 g

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**Table 1: Clinical presentations of Ach receptors overstimulation at different regions [1]**

| Overstimulation of Ach receptors in | | | |
|---|---|---|---|
| **Parasympathetic system** | **Sympathetic system** | **Central nervous system** | **Neuromuscular junction** |
| Bronchospasm<br>Bronchorrhea<br>Miosis<br>Lachrymation<br>Urination<br>Diarrhea<br>Hypotension<br>Bradycardia<br>Vomiting<br>Salivation | Tachycardia<br>Mydriasis<br>Hypertension<br>Sweating | Confusion<br>Agitation<br>Coma<br>Respiratory failure | Muscle weakness<br>Paralysis<br>Fasciculation |

**Ach: Acetylcholinesterase**

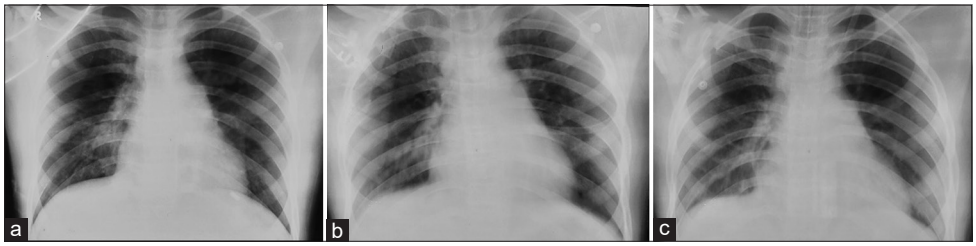


**Figure 1: Chest X-ray showing B/L Pneumonia. (a) Chest X-ray on the day of admission with greater bilateral pneumonia. (b) Chest X-ray on 3rd day of hospital stay showing little decrease in the pneumonia. (c) Chest X-ray on 5th day of admission showing almost recovered lungs with minimal pneumonia**

iv stat dose, and maintained as 500 mg/h infusion. Inj. atropine (inj. Tropine) infusion was started as 1 mg/h when heart rate was <120/min on the day of admission. Despite of heart rate in the higher range, in view of bronchorrhea, miosis, and increased lung secretions, atropine was continued, escalated to the 2 ml/h next day, continued till the 3rd day, and then weaned off in view of increased CNS depression and in plan of extubation. The patient was started with glycopyrrolate (Inj. pyrolate) 1 cc, i.v., QID in place of atropine as this has low CNS penetration. ABG after 1 day showed significant improvement with values as pH: 7.323; $pCO_{2:}$ 52mmHg; $pO_{2:}$ 194mmHg; and $HCO_3^-$: 27 mmol/lit.

The patient presented with fever spikes on the 3rd day after admission which was considered secondary either due to atropine or pneumonia since procalcitonin, blood, and sputum culture sensitivity tested negative. Power in all four limbs was increased from 1/5 on the initial 2 days of admission to 4/5 on the 3rd day and achieved full power 5/5 on the 4th day. Mechanical ventilation was continued for 3 days with positive end-expiratory pressure of six, $FiO_2$ as 50% on the day of admission, 40% next day, 30% on the 3rd day, maintained on T-piece for 24 h with 5 liters, and then extubated on the 4th day. The post-extubation period was uneventful and the patient was provided with 5–6 L of $O_2$ support through face mask for 24 h which was tapered slowly and taken off $O_2$ support after 24 h. Feeds were given to the patient through Ryle's tube for 4 days and extubated on the 5th day after oral liquid compliance and shifted out from the ICU to the general ward. By the 6th day, the patient was brought to a hemodynamically stable state and discharged from the hospital. At the time of discharge, a 6 min-walk test was negative with pupils normal in size and reactive to light and clear lung fields with no bronchorrhea as seen on the chest X-ray. The course of the patient in the hospital is detailed as an algorithm in Fig. 2.

## DISCUSSION

As per the National Crime Bureau of India report in the year 2007, among all the poisoning cases reported, OP poisoning accounted for 19.7%. In developing agricultural countries such as India, the use of OP is more prevalent and so ingestion of OP incidentally in a suicide attempt is more common. However, besides parenteral ingestion, long-term exposure to the OP while spraying in the agricultural lands may also pose a threat to the farmers leading to OP poisoning [4]. An estimate of >3 million people is exposed to OP worldwide every year of which, about 300,000 deaths are being reported. The widely seen reason for deaths in OP toxicity is type 2 respiratory failure secondary to bronchoconstriction, bronchorrhea, central respiratory depression, or paralysis of respiratory muscles [5].

Among various OP compounds, monocrotophos has the highest lethality rate and the need for mechanical ventilation followed by methylparathion [6]. However, methamidophos, dimethoate, and monocrotophos were the most fatal organophosphorus compounds resulting in death on self-intoxication [7]. The chemical formula of monocrotophos is $C_7H_{14}NO_5P$ and the IUPAC name is dimethyl (E)-1-methyl-2-(methyl carbamoyl) vinyl phosphates [8]. The toxic levels of monocrotophos (OP compound) are as oral LD: 50 14 mg/kg body weight; dermal LD 50: 112 mg/kg body weight, and accepted daily intake: 0.0006 mg/kg body weight [9].

The wide use of monocrotophos in agriculture leads to groundwater and surface water contamination causing neuro and genotoxicity of various organisms [10]. However, fatal dermal toxicity of OP is rarely reported [11]. Mortality associated with OP poisoning was 25% of which, the majority of deaths noted due

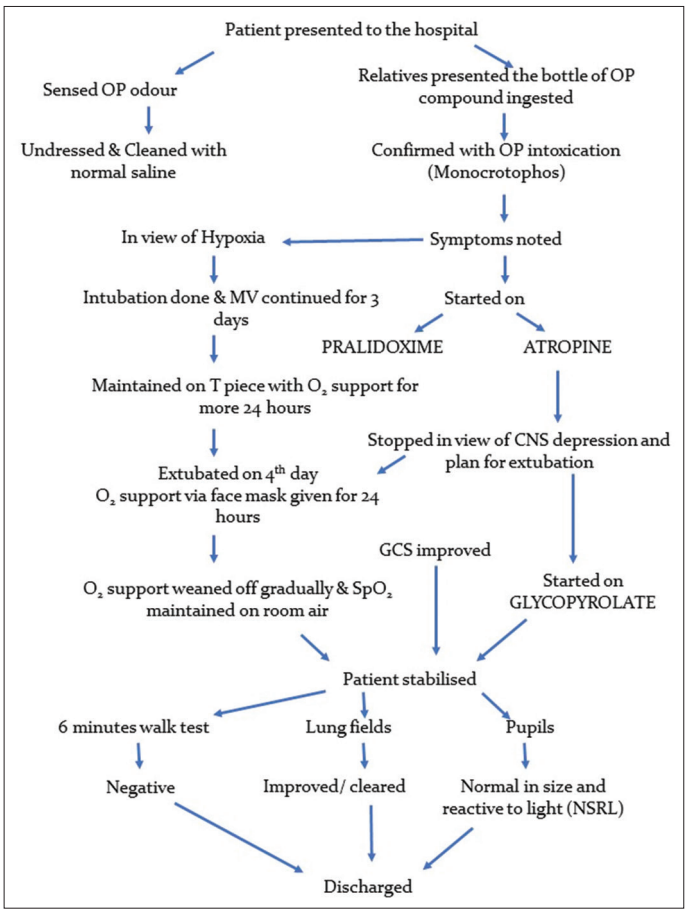


**Figure 2: Algorithm of the case study representing the patient hospital course**

to delay in discovery and transport of the patient to the hospital and due to respiratory failure [12].

However, early diagnosis and proper intervention with the use of atropine and oximes can be life-saving. The use of oximes (PAM) as a life-saving treatment is not significant since the patients treated alone with atropine were also successfully treated. Management of OP poisoning includes the use of antidotes (PAM and atropine) as suggested by the World Health Organization (WHO). PAM regenerates functional Ach and atropine inhibits the build-up of excess Ach [13]. Atropine is to be given before the administration of PAM to avoid the worsening of muscarinic-mediated symptoms. As recommended by the WHO, the dosage of PAM is 30 mg/Kg for adults and 20–50 mg/Kg for children should be given as a bolus over 30 min, followed by the infusion of 8 mg/kg/h for adults and 10–20 mg/kg/h for children should be started and maintained till the neurological improvement is seen [14,15]. Atropine should be given as a loading dose of 2–5 mg intravenously for adults and 0.05 mg/Kg iv for children. Even after 5–10 min of the first dose administration, if pulmonary muscarinic symptoms persist, then double the initial dose administration is advisable [15]. Identification of the type of OP compound intoxicated by the patient plays a key role in the management as few lipid-soluble OP compounds do not present clinically soon after ingestion but may lead to death after a few days, if untreated [16].

## CONCLUSION

As in this case study, the incidental oral ingestion of OP by adolescence was presented with tachycardia and without seizures (non-linear presentation in case of OP poisoning) resulted in hospitalization for 6 days with an initial intubation period of 4 days. However, the management of the patient with atropine, PAM, and mechanical ventilation guarded the prognosis and improved the outcome of the patient.

Informed consent: An informed consent is obtained from the patient's attender for collecting the information and publishing the same by masking the personnel details.

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