Organophosphate (OP) Poisoning Case Report by the Ingestion of a Potential Lethal Dose; its Management and Appropriate Protocol

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Abstract
Organophosphate (OP) poisoning continues to be a recurrent cause for admission to hospitals and intensive care units (ICU) in under emergent countries. OP poisoning is the most commonly prevailing up to the ratio of (27.64%) and has the highest death ratio (13.88%) of poisoning in Asia. This poisonings causes up to 25% mortality rate worldwide. In this case presentation a young girl age was sent to ICU after ingestion of OP insecticide. Where medical practitioner prescribed some irrational medications which are of veto use and also don’t follow customary protocols for treating this poisoning case, the main reason behind this mishap would probably be the lack of pharmacist intervention in health care team. This is the major drawback of our public sector hospitals in Karachi, Pakistan. Responsiveness and right treatment protocols can trim down both mortality and morbidity rates in the city and prompt appropriate therapeutic dealings can execute better prognosis in these types of urgent situations and may decreased further impediments.

Keywords: Organophosphate poisoning; Case study; Lack of pharmacist; Irrational medications

Introduction
Organophosphorus pesticide self-poisoning is an important concern in rural regions of the developing country, and kills an estimated 200,000 people every year. Accidental poisoning ratio far less but is also a dilemma in places where highly toxic organophosphorus pesticides are available. Organophosphate (OP) compounds have a vast variety of chemicals used in both domestic and industrial settings. Some examples of OP include insecticides, nerve gases, ophthalmic agents and antihelmintics. [1-5].

Signs and symptoms of OP poisoning
These are divided into three different categories.
- Muscarinic effects
- Nicotinic effects
- Central nervous system (CNS) effects

Muscarinic effects of OP are (excess salivation, lacrimation, urination, emesis, GI upset, diaphoresis, diarrhea; urination; miosis; bradycardia, bronchospasm, and salivation). Nicotinic signs and symptoms include muscle fasciculations, cramping and weakness. Autonomic nicotinic effects include hypertension, tachycardia, mydriasis, and pallor. CNS effects include the following: Anxiety, Emotional liability, Restlessness, Confusion, Ataxia, Tremors, Seizures, and Coma.

The key mechanism of action of OP pesticides is inhibition of carboxyl ester hydrolases, mainly acetylcholinesterase (AChE). AChE is an enzyme that is used to degrade the neurotransmitter acetylcholine (ACh) into choline and acetic acid. ACh is found in the central and peripheral nervous system and accumulates throughout the nervous system, resulting in overstimulation of nicotinic and muscarinic receptors [6-8].

Case presentation
A young 20 years old girl was escorted to intensive care units after intake of OP insecticide in a suicidal attempt. She had shortness of breath, decreased altered level of consciousness, diarrhea, miosis, hypersalivation and restlessness and seizures during admission. Vital signs show pulse rate of 62 per min and blood pressure of 120/80 mmHg while respiratory rate of 14 per min [9,10].

<table>
<thead>
<tr>
<th>Brand</th>
<th>Generic</th>
<th>Dose/frequency</th>
<th>Direction</th>
<th>Purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrathione</td>
<td>Pralidoxime methylsulphate</td>
<td>1 g/tid</td>
<td>IV</td>
<td>Cholinesterase reactivators</td>
</tr>
<tr>
<td>Ruling</td>
<td>Omeprazole</td>
<td>40 mg</td>
<td>IV</td>
<td>Acidity/indigestion</td>
</tr>
<tr>
<td>Atropine</td>
<td>Atropine</td>
<td>10 mL/h</td>
<td>IV</td>
<td>Antidote of OPP</td>
</tr>
<tr>
<td>Dayline</td>
<td>Ceftriaxone</td>
<td>750 mg</td>
<td>IV</td>
<td>Treatment of infection</td>
</tr>
</tbody>
</table>
**Patient’s Laboratory and Diagnostic Data**

**Diagnosis**

OP poisoning.

**Major Strategies:** The standard protocol for OP poisoning was not followed. She was not treated with phenytoin or diazepam sodium for seizures. No medication was given for her restlessness. Her restlessness was controlled with diazepam.

Initial antibiotics were ceftriaxone and levofloxacin. It was changed to meropenem and linezolid because of intermediate syndrome and intermittent cholinergic features occurring with fat-soluble organophosphorus.

There was also a foremost drug interaction exited between amikacin and levofloxacin.

**Decontamination**

Take out all the clothing's from the patient and gently cleanse the patients with soap and water because OP are hydrolyzed readily in aqueous solutions. Health care providers should keep themselves away from contamination or utilize proper preventive protocols while handling the patients. Use masks or respiratory protection. Wash eyes of the patients using isotonic sodium chloride solution. Intraosseous administration has been found useful in rapid delivery of atropine into the bloodstream, as shown in the studies of pigs [11,12]. Intravenous glycopyrrolate or diphenhydramine may also provide another centrally acting anticholinergic agent used to treat muscarinic toxicity if atropine is unavailable.

**Discussion and Conclusion**

Medical management of organophosphorus pesticide poisoning is difficult, especially with having few resources in underprivileged localities. Clinical practice is frequently less than ideal, with pitiable antibiotics should be changed to meropenem and linezolid. On maintenance, features of toxicity re-appeared and she again required atropine in bolus dose. She required ventilator support for one week and after 9 days she recovered completely. Her current medication chart was shown in Table 1.

<table>
<thead>
<tr>
<th>Levofloxacin</th>
<th>Levofloxacin</th>
<th>750 mg/OD</th>
<th>IV</th>
<th>Respiratory tract infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>D/W 5%</td>
<td>Fluid</td>
<td>IV 1000 mL</td>
<td>Infusion</td>
<td>Fluid replacement</td>
</tr>
</tbody>
</table>

**Table 1:** Current Medication Chart.

Chest X-ray showed acute respiratory distress. After immediate resuscitation, she was treated with atropine and pralidoxime methylsulphate along with broad spectrum antibiotics. Atropine was given 10 mL/h through IV and the dose was titrated as per her clinical response. The patient also received pralidoxime. Pralidoxime was given at a dose of 1 g infusion, three times per day for initial two days. While on maintenance, features of toxicity re-appeared and she again required atropine in bolus dose. She required ventilator support for one week and after 9 days she recovered completely. Her current medication chart was shown in Table 1.
Conflict of Interest

The authors assert no imminent conflicts of interest with respect to the authorship, research, and/or publication of this article.

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References