Organophosphate poisoning: A case report, overview of management and nursing interventions
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Abstract: Organophosphate poisoning is a common cause of acute poisoning in India with high mortality. Prompt recognition and aggressive treatment of acute intoxication is essential to minimize the mortality and morbidity. Nurses play a vital role in the management of poisoning, as it demands close observation, timely administration of antidotes in adequate doses and skillful nursing interventions. This article presents a case report with a literature review of organophosphate poisoning, and its management.

Key words: Organophosphate, poisoning, Anticholinergics, Decontamination.

INTRODUCTION

Organophosphates (OP) are commonly used as insecticides and are among the most common suicidal agents in developing countries like Pakistan, Sri Lanka, and the other Asian and South East Asian countries. Poisoning due to OP is a major health problem as consumption of these insecticides is associated with significant morbidity and mortality. OP poisoning contributes to a large proportion of admissions to hospitals and intensive care units as OPs are comparatively more toxic and easily available than other insecticides like organocarbamates, organochlorides, and pyrethroids.¹

Poisoning is common among the young, especially adolescents and young adults and in farmers, and accounts for 35-40% of all suicidal deaths in India.² According to John G³ from Christian Medical College, Vellore, in the year 2005 alone, 11.7% of total ICU admissions were due to OP poisoning which accounted for 14.6% of Intensive care unit (ICU) deaths. Studies have reported that three million cases of poisoning and forty thousand deaths occur worldwide per year throughout the world, predominantly in the developing countries.⁴,⁵

TYPES OF ORGANOPHOSPHATES: OP compounds may be divided into two types: diethyl (e.g.chlorpyrifos, diazinon, parathion, phorate and dochlofenthion) and dimethyl (e.g. dimethoate, dichlorvos, fenitrothion, malathion and fenthion)⁶,⁹. The route of entry into the body is either through accidental or suicidal ingestion, inhalation or absorption through skin.

PATHOPHYSIOLOGY

Acetylcholine is a neurotransmitter found in neuromuscular junction and peripheral /central nervous systems. Acetylcholinesterase (AChE) is the enzyme responsible for the degradation of acetylcholine. OPs inhibit and inactivate AChE, leading to accumulation of acetylcholine. This results in overstimulation of the muscarinic and nicotinic receptors in the nervous system leading to toxic effects.⁷,⁸,⁹

CLINICAL MANIFESTATIONS

The onset, severity and duration of OP poisoning depend on the route of exposure and amount of agent involved. Clinical Manifestations of OP poisoning may be summarized as shown in Table 1

The most important sequela in patients with acute OP poisoning is neuromuscular weakness. This requires prolonged ventilation. Based on the time of occurrence of weakness, paralysis may be categorized into two types.

Type I: (Acute paralysis) usually develops within 24-48 hrs. This is due to the persistent depolarization at the neuromuscular junction resulting from blockade of acetylcholine esterase. Some of the important clinical
features are muscle fasciculations, cramps, twitching, and weakness. Paralysis of the respiratory muscles may lead to respiratory failure which requires mechanical ventilation.\textsuperscript{1,3}

Table 1 – Symptoms of OP poisoning\textsuperscript{18}

<table>
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<tr>
<th>Muscarinic symptoms</th>
<th>Nicotinic Symptoms</th>
<th>CNS symptoms</th>
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<tr>
<td>Mnemonic “SLUDGE/DUMBLES”</td>
<td>Fasciculations, Paralysis, Pallor, Muscle weakness, Hypertension, Tachycardia, Mydriasis (rare)</td>
<td>Unconsciousness, Confusion, Toxic psychosis, Seizures, Fatigue, Respiratory Depression, Dysarthria, Ataxia, Anxiety</td>
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Type II: (Intermediate syndrome/IMS) develops after the acute cholinergic crisis. It occurs 24-96 hours after the poisoning and the predominant muscle groups involved are respiratory, proximal limb muscle and neck flexors. It persists for about 14 – 20 days. One of the earliest manifestations in these patients is the presence of marked weakness of neck flexion with the inability to lift the head from the pillow.\textsuperscript{1,3}

Type III: OP Induced Delayed Polyneuropathy (OPIDP). It can be pure sensory or motor neuropathy occurring 2-3 weeks after the episode of poisoning. It is predominantly distal. Recovery may take 6-12 months.\textsuperscript{1,3}

DIAGNOSIS

Diagnosis is based on:

1. **History of exposure to a known OP compound.**
2. **Clinical features:** laboured breathing, sweating, miosis (small or pinpoint pupils), bradycardia and typical odour (garlic/petrol). The nicotinic effects like tachycardia and mydriasis may be seen in some patients (rather than the more common miosis and bradycardia). (Table 1).

3. **Blood levels of serum pseudocholine esterase.**
4. The poison can be identified by some poison centers by analyzing the contents of the stomach.\textsuperscript{1}

INVESTIGATIONS

Specific investigations in the Emergency department include:

1. Blood samples for serum pseudocholine esterase, leucocyte count if infection is suspected and electrolytes.
2. ECG & Chest X-ray

MANAGEMENT

The management of OP may be categorized into emergency, general and specific management.

**Emergency Management:** - OP poisoning is a medical emergency.
1. Initial assessment includes assessment and management of airway, breathing, & circulation. Provide adequate oxygen and ensure a patent airway is maintained (noisy breathing is the best indicator of an obstructed airway).
2. Position the patient in left lateral position to reduce the risk of aspiration.
3. If the patient is drowsy with laboured breathing and has an obstructed airway or poor oxygen saturation, early intubation and ventilation will help overcome the acute crisis.
3. Monitor the vital signs. Arrangements should be made to transfer the patient to ICU if there are signs of low sensorium, laboured breathing, BP less than 90/60 mm Hg or severe muscarinic crisis.

**General and specific management:** The general principles of management of poisoning have to be carried out without delay. The specific management involves neutralization of the poison using specific antidotes. The principles of management are:
1. **Reduce absorption of toxin**
2. **Increase elimination of the toxin**
3. **Neutralization (Using specific antidotes)**
CASE REPORT – Organophosphate poisoning

1. Reduce absorption of toxin
   a) Skin Decontamination:
   Decontamination of the skin is very important and it should be done very thoroughly to prevent further absorption through the skin. The patient’s clothes are removed and the skin is thoroughly washed with soap and water. Gentle cleaning with soap and water is effective and will not abrade the skin or enhance absorption. Skin folds and underside of fingernails and long hairs require special attention. Ocular decontamination is to be carried out by gently washing eyes with water/normal saline. Health-care personnel should wear protective clothing and glasses. Contaminated clothes, shoes and other leather items must be removed from the patients and placed in a separate bag; these should then be incinerated.

   b) Gastrointestinal decontamination:
    Gastric decontamination must be done by induced vomiting only if the patient is fully conscious and oriented. Induced vomiting is not recommended if the individual is drowsy, disoriented or has a poor level of consciousness, as there is a risk of aspiration. Gastric lavage is more effective and is safer than induced vomiting. It is most effective within 60 minutes of ingestion of the poison but can be useful even later in the therapy of poisons which delay gastric emptying. The first aspirate of stomach content is preserved and sent for pharacoanalysis.1,8 Gastric lavage is contraindicated if the GCS score is < 8 as there is risk of aspiration. It may be carried out after intubation & stabilization in patients with low GCS. 3

2. Increase elimination
   a) Activated Charcoal: 17 Activated charcoal (0.5-1g/kg) is useful for gastrointestinal decontamination- it is highly absorbent as it has a large surface area. Sodium sulphate or sorbitol may be used as a cathartic.— its use is however not well established10,1. A single dose of activated charcoal without a cathartic (50gm) is enough because it is ineffective beyond 1-2 hours after consumption of the poison. 19 This may be given through a nasogastric tube in an adult who is either intubated or is fully awake and co-operative. 17
   b) It is a good practice to keep the urine output at the rate of 150-200 ml/hr (2-3ml/kg/hr) with attention to electrolyte balance. 3

3. Neutralization (Use of specific antidotes)
   a) Inj. Atropine sulphate:
    Inj. Atropine sulphate is a life saving antidote. Complete and early atropinisation is an essential and simple part of early management. It reverses the cholinergic features and improves cardiac and respiratory function15
   Atropinisation Protocol: There is no uniform guideline available for Atropine administration. However, according to a recent guideline which is being followed in Christian Medical College Hospital, Vellore (see Box 1), a loading dose of Atropine (as a bolus) must be initiated followed by monitoring for atropinisation until full atropinisation is achieved. The usual requirement of atropine is about 5-10 mg/hr3,10. The loading dose is followed by an infusion - this produces less fluctuation in plasma atropine concentration and makes weaning easier. The target heart rate is > 100/min on day2, > 90/min on day 3, and > 80/ min on subsequent days.10,16 After initial stabilization, patient should be assessed for the features of adequacy of atropine infusion (Table 2).7

   b) Glycopyrolate: Inj. Glycopyrolate is recommended when there is copious secretion. It has less CNS penetration and may result in less CNS toxicity.11,6

   c) Inj. Pralidoxime (PAM): Current WHO guidelines recommend 30mg/kg loading dose of pralidoxime over 10-20 min, followed by continuous infusion of 8-10 mg /kg per hour until clinical recovery.12,11,9,14 However, several studies failed to show benefit.

d) Antibiotics: Antibiotics are not usually indicated for OP poisoning unless there is strong suspicion of aspiration or evidence of infection.

e) Furosemide: Recommended if pulmonary edema persists, even after full atropinisation.11

f) Sedation: Agitation in OP poisoning may indicate over-atropinisation, hypoxemia or distress due to pain/ discomfort. Intubated patients need a combination of an analgesic and a sedative. Inj. Morphine and lorazepam may be used as an infusion. Haloperidol may decrease seizure threshold and not recommended unless patients are unresponsive to other drugs.
Table: 2 Features used to assess atropine adequacy (Target endpoints)  

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<thead>
<tr>
<th>Features of atropine adequacy</th>
<th>Features of atropine toxicity</th>
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<tr>
<td>Clear chest on auscultation with no wheeze</td>
<td>Confusion</td>
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<tr>
<td>Heart rate between &gt; 80 beats/min</td>
<td>Pyrexia</td>
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<tr>
<td>Pupils no longer pinpoint</td>
<td>Urine retention</td>
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<td>Systolic blood pressure &gt;80mmHg</td>
<td>Bowel ileus</td>
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<tr>
<td>Dry axillae</td>
<td>Hypertension</td>
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<td></td>
<td>Tachycardia</td>
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**Counselling:** Counselling to the poisoned patients will reduce the chances of a repeat attempt at poisoning. It also enables the health care personnel to improve the quality of treatment, minimize the cost of therapy and the period of hospitalization. Family counselling is mandated; this helps the family members to cope with the situation and accept the patient as he is.

**Case report:**
A 27 year old male presented with alleged history of consumption of parathion along with alcohol following a quarrel at home. He had vomiting and three episodes of generalized tonic-clonic seizures lasting for 2-3 min. He was given a gastric lavage and referred for further management. In the Emergency Department his GCS score was 3/15, pulse-70/min, respiratory rate 14/min, BP-110/70 mmHg, SpO₂ 80%. On examination his skin appeared to be flushed, pupils mildly dilated. The rest of the systemic examination was unremarkable. As he developed laboured breathing, emergency endotracheal intubation was done and he was connected to a ventilator. Repeated bolus doses of atropine sulphate were administered till the heart rate reached 110/min. Atropine was continued as an infusion at the rate of 10 ml/hr.

Since he had generalized tonic clonic seizures, he was given a loading dose of intravenous phenytoin (15 mg/kg over 30 min) and continued at 5 mg/kg in three divided doses. He was then shifted to the medical intensive care unit for further management. Blood samples were obtained for complete blood count, electrolytes, and arterial blood gas analysis. Serum pseudo cholinesterase level was 800U/L. (Reference interval 3000 to 8000 U/L & in significant poisoning usually <1000 U/L).

Liver function and renal function tests were normal. Due to persistent neck muscle weakness on day 3, long term ventilation was anticipated and hence an early tracheostomy was done.

To maintain the target heart rate, (Day 1- >110/min, day 2 - 100/min, day 3 - 90/min and thereafter heart rate of at least 80/ min) atropine infusion was initiated. Bolus doses of atropine were intermittently administered when required if the heart rate went below the target rate.

**Nursing interventions**
We approached each of the nursing diagnoses as follows:

1. **Nursing diagnosis:** Ineffective airway clearance related to presence of copious secretions secondary to OP compound effects.

   **Expected outcome:** - Airway clearance as evidenced by maintenance of SpO₂ at 90-100% and prevention of aspiration.
Nursing interventions: Endotracheal tube was secured, frequent suctioning was done; humidified oxygen, and salbutamol alternating with nebulization with ipratropium were administered. Atropine infusion was initiated at 10 mg /hr and tapered to 2mg on the fourth day and then discontinued. He was maintained at 45° head end elevation and was positioned laterally. Chest physiotherapy was given to mobilize the secretions.

Expected outcome: Prevention of seizures and the related injuries.
Nursing implementations: Periodic and regular assessment of GCS score administration of antiepileptic drugs were done. –Additional precautions were initiated with provision of side railed cot, and positioning of patient (left lateral with head elevation at 45 degree). Patient was closely observed.

3. Nursing diagnosis: Decreased cardiac output related to cholinergic effects of OP poisoning.
Expected outcome: Maintenance of cardiac output as evidenced by mean arterial pressure (MAP)>70mm of Hg & heart rate>110/min..
Nursing interventions: Close monitoring of hemodynamic status (blood pressure, MAP and heart rate) . MAP was maintained between 70-80 mmHg. Atropine was administered to maintain the target heart rate [ Day 1: 110/min; Day 2: 100/min; Day 3: 90/min]. Adequate intravenous fluids were administered to prevent dehydration due to salivation & diarrhea.

Expected outcome: Normal hydration status.
Nursing interventions: Intravenous fluids were administered as per plan and urine output was monitored. In addition to intravenous fluids, nasogastric feeds were initiated . A cumulative fluid balance sheet was maintained.

5. Nursing diagnosis: Nutritional imbalance related to ‘Nil per oral’ (NPO) status secondary to risk of aspiration.
Expected outcome: Achieve nutrition balance as evidenced by serum Albumin of3.5-5g/dl%, Hb>10g%.
Nursing interventions: Nasogastric aspirations(q4h) were performed for two days to check the gastrointestinal function. Clear fluids were started on day 2 followed by formula feeds (35- 45 kcal/kg/day) with probiotics (q6h).On day 20, tracheostomy was closed and oral feeds were initiated with soft solid followed by normal diet.

Expected outcome: Enhance the coping ability of the family.
Nursing interventions: Open communication was encouraged among the family members and family counselling was organized. The family members were counselled so they could understand the prognosis by the physician. Arrangements were made for their spiritual comfort.

7. Nursing diagnosis: Risk of complications such as pressure sores, IMS, OPIDP, and ventilator associated pneumonia (VAP) related to poisoning effects and prolonged mechanical ventilation.
Expected outcome: Prevention of complications.
Nursing interventions:
a) Aspiration: The head end of the bed was elevated to 30-45 degree, change of position was carried out before feeds and continuous feeds were given using feed pump which prevented further aspiration.
b) IMS: Assessment was done for breathing pattern & neck muscle weakness for 96 h. Muscle power & reflexes were monitored.
c) OPIDP: The patient was monitored for persistent & delayed onset muscle weakness and seizures. Assessment was done for re-emergence of SLUDGE symptoms.
d) VAP: Standard precautions were followed: The patient was assessed for signs of infection, breathing pattern & characteristics of secretions were monitored; tubing of the ventilator was changed as frequently as possible if sedimented with secretions and suctioning was done as required under aseptic techniques. Adequate chest physiotherapy and nebulisations were
given to mobilize the secretions. Progress was monitored using chest X-rays.

e) Pressure sores: Skin integrity was maintained by back care and 2-hourly change of position.

Evaluation: Patency of the airway was maintained with regular suctioning and optimal positioning. The targeted heart rate was achieved with administration of atropine. Close monitoring, observation, and timely interventions enabled recovery. Meticulous oral care, nebulization, chest physiotherapy and aseptic techniques were strictly adhered to. He did not develop VAP despite being hospitalized for 24 days. He recovered, was extubated and was referred to a psychiatrist for further counselling.

CONCLUSION: Poisoning is a common cause of hospital admissions. Caring of patients with OP poisoning is a challenge for nurses. Assessment and prevention of complications is one of the vital roles of the nurse. Appropriate, evidenced based practice will enhance quick recovery, reduce morbidity and mortality. Nurses must ensure that both patients and family members receive counselling, to cope and live in the community.

References: