Management of Unintentional Acute Organophosphate Poisoning After Dermal Exposure in Primary Health Care Setting: A Rare Case Report

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November 09, 2024

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ABSTRACT

Key Clinical Message

Organophosphate (OP) poisoning is the common form of poisoning accounting for approximately 0.9-1.0% of the patient admissions to hospital emergency departments in Nepal. Most cases of unintentional organophosphate poisoning occur in farmers due to unsafe handling and storage of insecticides. Although OP compounds are widely used as insecticide, a few cases of hospital admission after unintentional dermal exposure have been reported so far. The case fatality rate of treated OP poisoning is appropriately 7.4% in Nepal and 5-20% in developing countries of Asia. The severity and onset of the symptoms depends on the amount of

poison ingested, route of absorption, and rate of metabolic breakdown of the insecticide. The most common symptoms of OP poisoning are altered level of consciousness, vomiting, sweating, hypersalivation and lacrimation. Diagnosis of OP poisoning is primarily based on the history, the clinical signs and symptoms, smell of insecticides or solvents involved, and reduced levels of red blood cell or plasma acetylcholine esterase (AchE). We reported a case of a 23-year-old male presented with a history of unintentional chlorpyrifos (CPS) poisoning after dermal exposure with bouts of vomiting and severe epigastric pain. Prompt recognition of signs and symptoms, and immediate decontamination followed by intravenous (IV) atropine and IV fluids with careful monitoring of vitals led to patient's survival without any neurologic sequelae.

Keywords: Organophosphate, Insecticide, Acetylcholine esterase, Chlorpyrifos, Decontamination

1. INTRODUCTION

Organophosphate poisoning is a common prevalent emergency care problem found in Nepal due to its easy access and widespread availability of insecticides and pesticides among farmers.[1,2]Approximately, case fatality rate of treated OP poisoning is 5-20% in developing countries in Asia and 7.4% in Nepal.[3,4]Exposure may occur through inhalation, ingestion, or dermal contact. The severity of the symptoms depends on the amount ingested, route of absorption, and rate of metabolic breakdown of the insecticide. The pathophysiology of OP insecticide is the irreversible inhibition of enzyme acetylcholinesterase (AchE) which breaks down neurotransmitter acetylcholine found in both peripheral and central nervous system, followed by overstimulation of muscarinic and nicotinic receptors resulting in cholinergic toxidrome.[5,6] *Figure 1. Pathophysiology of OP poisoning.*[6]

In Nepal, the grading of OP poisoning is based on the Peradeniya Organophosphorus Poisoning (POP) scale. Parameters such as pupil size, respiratory rate, heart rate, fasciculations, level of consciousness (LOC), and seizures are used to rate the poisoning as mild, moderate, or severe.[3,7] Treatment begins with decontamination, airway control and oxygenation. The mainstays of pharmacological therapy include atropine, pralidoxime (2-PAM), and diazepam. Initial management must focus on adequate use of atropine and optimising oxygenation prior to the use of atropine is recommended to minimise the potential for dysrhythmias.[8]

2. CASE REPORT

2.2 Investigation and Management

First of all, he was decontaminated by washing his hands with soap and water and his clothing was removed. Since he had 2 episodes of vomiting on the way to PHC, gastric lavage was not attempted which may increase the chances of aspiration. Oxygen was attached at 2L/min and IV access with 1L ringer lactate (R/L) was initiated. IV atropine 1 mg loading dose was administered and was repeated every 5 mins until the signs of atropinization appeared. Due to unavailability of 2- PAM, it could not be given. In the meantime, routine blood investigations within the domain of the facility were sent. Electrocardiogram (ECG) showed sinus bradycardia with no other specific changes. Chest X-ray (CXR) was unremarkable. Patient's vitals were monitored with the help of a monitor. His routine investigations during ED presentation were as follows:

Table 1. Laboratory investigations details of patient

Variables	**Value**	**Range**
Total cell count ($\times 10^3/\mu L$)	9480	4000-1100
Haemoglobin (Hb) (mg/dL)	12.6	12-17.5
Platelets ($\times 10^3/\ \mu L$)	145000	150000-450000
Bilirubin (mg/dL)	1.1	0.1-1.2
Random blood glucose (mg/dL)	97	70-100
AST (U/L)	35	8-50
ALT (U/L)	42	7-55
Urea (mg/dL)	27	5-20
Creatinine (mg/dL)	0.9	0.7-1.3

Plasma AchE, serum electrolytes, arterial blood gas (ABGs), serum troponin level and coagulation studies were not done due to unavailability of diagnostic kits and rural location of health facility. Patient was from a poor economic background and did not have any insurance plan, his attendants could not afford tertiary care for him and requested us to treat in our setting despite the consequences. His vitals were stable over time with his PR of 83/min, BP of 122/72 mmHg, Temp. of 99°F and SaO_2 of 98% with 2L O_2/min after 6 hours. He showed improvement in symptoms with no further signs of intoxication over 12 hours. Atropine dose was titrated as per his clinical response and signs of atropinization. His chest cleared of crepitations, and ECG showed normal sinus rhythm. Boluses of 1L of RL and 1L of 5% dextrose water (D/W) were attached over a period of 12 hours. His complete blood count (CBC), liver function tests (LFTs), renal function tests (RFTs), all were within normal limits.

2.3 Follow up

Before discharge he was counselled to seek immediate medical attention in case of shortness of breath and weakness of extremities that may follow 3-4 days later. We followed him for next 6 weeks for any neurologic sequelae but none of the aforementioned symptoms were present.

3. DISCUSSION

OP is the common form of poisoning accounting for 0.9-1.0% of the patient admissions to hospital EDs in Nepal.[2,4] The most prevalent symptoms of OP poisoning are SLUDGE (salivation, lacrimation, urination, defecation, gastric cramps, and emesis).[9] Signs and symptoms of OP poisoning can be divided into acute cholinergic crisis, intermediate syndrome (IMS) and type 3 paralysis or organophosphate induced delayed polyneuropathy (OPIDP).[10] IMS is muscle paralysis involving mainly bulbar, respiratory, and proximal muscles of limbs following the acute cholinergic phase, occurring 3-4 days after exposure. It usually resolves in 1–3 weeks but may require mechanical ventilation if indicated.[9] Respiratory failure is the most common cause of death in IMS.[8,11] OPIDP occurs about 2-4 weeks after exposure to large doses of OP insecticide. It is characterised by distal muscle weakness causing ataxia, foot drop and claw hand, and is due to inhibition of neuropathy target esterase. It may take weeks to months to recover. Rare complications of OP poisoning are cardiac arrhythmias, pancreatitis, and hepatic dysfunction.[12] There have been cases of delayed myelopathy after insecticide CPS ingestion.[10]Diagnosis of OP poisoning is primarily based on the clinical history, the clinical signs and symptoms, smell of insecticides or solvents involved, and reduced levels of RBC or plasma AchE. The management of OP poisoning requires prompt identification of toxins and rapid administration of atropine to counteract the cholinergic crisis. Although psychotic symptoms such as restlessness, excitement, hallucinations, and delirium have been reported following atropine administration, atropine is an antidote for OP poisoning.[3] Exposure with dermal contact needs immediate decontamination to reduce systemic absorption.[13] Further management requires adequate supportive care including IV fluids, airway management, seizure control and 2-PAM to reactivate AchE.[2] One of the studies shows atropine seemed to be as effective as atropine plus 2-PAM in the treatment of acute OP poisoning. 2-PAM if used should be given within 48 hrs of poisoning.[8] Immediate decontamination, oxygen, ventilatory support and benzodiazepines for seizure control if required, IV atropine, IV 2-PAM and IV fluids are of utmost importance for saving a patient's life.[4] Also while dealing with cases of suspected poisoning, stomach wash, blood, stool, urine and if possible empty bottles of poison should be collected for medicolegal registry.[14]

CONCLUSION

OP poisoning poses a serious threat especially in developing countries like Nepal where they are easily accessible and widely used in agriculture sectors. Lack of proper government legislature to limit the unauthorised use of these harmful substances led many people to die even of unintentional poisoning. Improvement in emergency and critical care services, and equipment in rural health facilities public awareness about pesticides and insecticides along with proper use, personal protection and cleanliness may lower morbidity and mortality associated with OP poisoning.

AUTHOR CONTRIBUTIONS

Bishal Koirala : Conceptualization; supervision; data curation; writing – original draft; writing – review and editing.

Priyanka Devkota : Conceptualization; data curation; writing – original draft; writing – review and editing.

Raghu Devkota : Supervision; data curation; writing – original draft; writing – review and editing.

FUNDING INFORMATION

No fund was available for this study.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest in this study.

ETHICAL STATEMENT

The patient has provided written informed consent for the publication of this case report.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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