

Series of Acute Organophosphate Poisoning Cases in Agricultural Workers

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Abstract

Background: Organophosphate (OP) compounds are commonly used pesticides in agricultural practices, especially in rural India. Due to their easy accessibility and limited awareness about safety protocols, acute OP poisoning remains a major occupational hazard among agricultural workers.

Objective: To present a series of acute OP poisoning cases in agricultural workers, highlighting clinical presentation, management, and the importance of timely intervention and preventive measures.

Methods: Three male agricultural workers from rural Tamil Nadu, aged between 28 and 50 years, presented to the emergency department with features of acute cholinergic crisis following occupational exposure to organophosphate pesticides. Detailed clinical evaluation, biochemical investigations, and supportive management were carried out in all cases. Interventions included administration of atropine, pralidoxime, and ventilatory support as required.

Results: All three cases showed classical symptoms of OP poisoning, including bradycardia, miosis, bronchorrhea, fasciculations, and altered mental status. Two patients required mechanical ventilation due to respiratory distress. One patient developed intermediate syndrome requiring prolonged ICU care. All patients recovered fully following aggressive atropinisation and supportive management. Lack of personal protective equipment and delayed hospital presentation were common risk factors.

Conclusion: This series highlights the preventable nature of OP poisoning and emphasizes the importance of increased awareness, proper use of personal protective equipment, and effective regulatory control over pesticide use. Early diagnosis and prompt treatment are critical to reduce complications and improve prognosis.

Keywords: Organophosphate poisoning, agricultural workers, cholinergic crisis, intermediate syndrome, occupational exposure, rural health, pesticide safety.

Introduction

Organophosphate (OP) compounds are a class of widely used pesticides that irreversibly inhibit the enzyme acetylcholinesterase, leading to the accumulation of acetylcholine at nerve endings and resulting in overstimulation of muscarinic and nicotinic receptors. Acute organophosphate poisoning remains a major public health concern, particularly in low- and middle-income countries (LMICs) like India, where agriculture forms the backbone of the economy and the use of chemical pesticides is widespread and often unregulated [1]. According to the World Health Organization, an estimated 3 million cases of pesticide poisoning occur each year globally, with around 220,000 deaths, the vast majority in developing countries [2]. In India alone, organophosphates account for up to 60% of all pesticide poisoning cases, particularly among agricultural laborers who often lack awareness of pesticide safety, have limited access to protective gear, and face inadequate health infrastructure [3,4]. The toxicity of OP compounds can range from mild symptoms such as nausea and headache to life-threatening respiratory failure, seizures, and coma. Clinical presentation typically follows a cholinergic crisis, characterized by salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis (SLUDGE syndrome), along with bradycardia,

bronchorrhea, muscle fasciculations, and miosis [5]. Delayed complications such as Intermediate Syndrome and Organophosphate-Induced Delayed Neuropathy (OPIDN) can further complicate outcomes [6]. The diagnosis of OP poisoning is primarily clinical, supported by laboratory findings such as decreased serum cholinesterase levels. Early and aggressive treatment with atropine and pralidoxime (PAM), along with respiratory support, is essential for survival. However, due to delays in access to tertiary care, many rural patients present late, often in a critical state [7,8]. This case series presents three illustrative cases of acute organophosphate poisoning in agricultural workers from a rural region in South India, highlighting the clinical spectrum, challenges in management, and the need for preventive interventions and occupational safety awareness.

Case Description

Case 1: Severe Organophosphate Poisoning with Intermediate Syndrome

Patient Details

A 42-year-old male agricultural labourer from rural Tamil Nadu presented with vomiting, profuse sweating, excessive salivation, breathlessness, and altered sensorium. He had been spraying *chlorpyrifos* in his paddy field without protective equipment, with 3–4 hours of exposure due to wind-blown pesticide mist. He arrived at the emergency department approximately 2 hours after symptom onset.

Initial Clinical Findings

- Drowsy; GCS: 10/15
- HR: 52 bpm | BP: 90/60 mmHg | RR: 32/min | SpO₂: 88% (room air)
- Pupils: Pinpoint, reactive
- Fasciculations: Facial and lower limbs
- Chest: Bilateral crepitations
- Classic cholinergic signs: Miosis, bronchorrhea, bradycardia, muscle twitching

Management:

The patient was shifted to ICU and treated with:

- IV atropine (2 mg every 5 min, titrated)
- Pralidoxime (2 g IV stat, followed by 500 mg/hr infusion)
- Oxygen therapy, followed by intubation and mechanical ventilation

Investigations:

- Serum cholinesterase: 220 U/L (↓)
- Chest X-ray: Aspiration pneumonitis
- ABG: Metabolic acidosis
- ECG: Sinus bradycardia

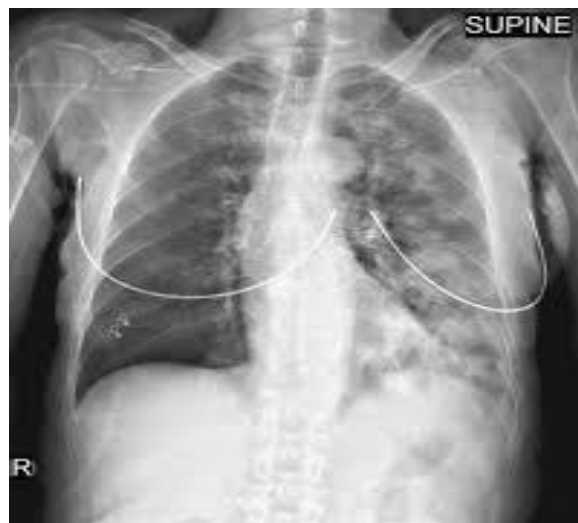


Figure 1: Chest X-ray findings of Aspiration pneumonitis

Progress:

Atropinization was achieved in 48 hours. On Day 4, the patient developed Intermediate Syndrome—with neck weakness, bilateral ptosis, and respiratory muscle weakness—requiring prolonged ventilation. Pralidoxime was continued for 5 days.

Outcome:

Gradual neurological recovery led to successful extubation on Day 8. He was discharged on Day 12 with no residual deficits. Counselling was provided on pesticide safety and protective measures, and he was referred for occupational health follow-up.

Case 2: Moderate Organophosphate Poisoning in a Female Farm Worker

Patient Details

A 35-year-old female farmworker from a semi-rural area in Tamil Nadu was brought to the primary health centre with complaints of nausea, abdominal cramps, excessive sweating, lacrimation, and muscle weakness. She had been involved in hand-weeding a recently sprayed field, unaware that *malathion* had been applied the previous evening. She wore no protective footwear or gloves, and exposure occurred through dermal contact with wet foliage for approximately 2 hours.

Initial Clinical Presentation

- Conscious but anxious and restless
- HR: 64 bpm | BP: 110/70 mmHg | RR: 24/min | SpO₂: 95%
- Pupils: Constricted
- Muscles: Mild tremors and generalized weakness
- Other signs: Lacrimation, salivation, diarrhea

Management

At the PHC, she was immediately stabilized and referred to the district hospital. There, she received:

- IV atropine boluses (1 mg every 10 minutes) until signs of atropinization (dry mouth, increased HR)
- Pralidoxime (1 g IV stat, followed by 500 mg every 6 hours for 48 hours)
- IV fluids and supportive care
- Oxygen via nasal cannula

Investigations

- Serum cholinesterase: 480 U/L (significantly reduced)
- ECG: Normal sinus rhythm
- Chest X-ray and ABG: Normal findings

Progress

She responded well to atropine and pralidoxime therapy within 24–36 hours. Symptoms gradually subsided without any signs of intermediate syndrome or delayed neuropathy. She was monitored for 72 hours and did not require ventilatory support.

Outcome

The patient was discharged on Day 4 with full recovery. She received education on the risks of pesticide exposure and was encouraged to wear protective clothing while working in the fields. Family members and fellow workers were also counselled through a local health outreach program.

Case 3: Fatal Outcome Following Delayed Presentation of Organophosphate Poisoning

Patient Details

A 58-year-old male farmer from a remote tribal village in southern Tamil Nadu was brought to the emergency department in an unresponsive state. According to relatives, he had ingested an unknown quantity of monostrophes (a highly toxic organophosphate) in a suicide attempt approximately 12 hours prior. Due to limited healthcare access and transportation delays, he was only brought to the hospital after worsening symptoms.

Initial Clinical Presentation

- Unconscious (GCS: 6/15)
- HR: 40 bpm | BP: 80/50 mmHg | RR: 38/min | SpO₂: 84% on room air
- Pupils: Pinpoint, non-reactive
- Profuse secretions, bronchospasm, cyanosis
- Chest auscultation: Crepitations bilaterally
- Fasciculations: Generalized
- Urinary and fecal incontinence present

Management

Immediate resuscitative measures were undertaken:

- Emergency intubation and mechanical ventilation
- IV atropine boluses (starting at 2 mg, rapidly escalated)
- Pralidoxime 2 g IV stat followed by continuous infusion
- Vasopressors for hypotension
- Broad-spectrum antibiotics for suspected aspiration pneumonia

Investigations

- Serum cholinesterase: Critically low at 150 U/L
- Chest X-ray: Diffuse infiltrates suggestive of aspiration and pulmonary edema
- ABG: Severe respiratory and metabolic acidosis
- ECG: Sinus bradycardia with ST depressions

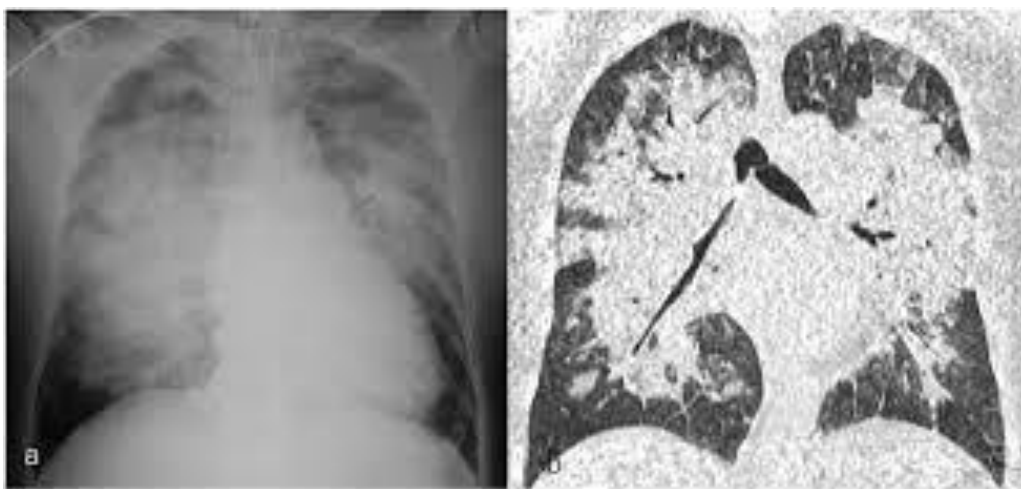


Figure 2: Diffuse infiltrates suggestive of aspiration and pulmonary edema

Progress

Despite aggressive management in the ICU, the patient's condition deteriorated. He developed multi-organ dysfunction, including acute kidney injury, hepatic derangement, and worsening hypoxia. On Day 3 of admission, he suffered a cardiac arrest and could not be revived.

Outcome

The patient succumbed to complications of severe organophosphate poisoning due to delayed treatment initiation. This case highlighted the dangers of late presentation, lack of pre-hospital care, and the need for awareness and accessibility in remote regions.

Discussion

Organophosphate (OP) poisoning continues to be a major public health issue, especially in low- and middle-income countries where agricultural practices are predominant and regulatory measures on pesticide usage are often inadequate. The presented cases reflect the spectrum of acute OP poisoning—from moderate exposure to life-threatening toxicity resulting in death. These cases underscore critical themes such as occupational hazards, lack of protective equipment, delay in accessing care, and complications such

as intermediate syndrome and respiratory failure. The toxic effects of OP compounds are primarily due to inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synapses and neuromuscular junctions, resulting in overstimulation of muscarinic and nicotinic receptors, as described by Eddleston et al. [9]. Clinical manifestations include muscarinic signs (salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis), nicotinic symptoms (muscle fasciculations and weakness), and central nervous system effects such as confusion, seizures, and coma, as reported by Karalliedde and Henry [10].

Case 1 illustrated intermediate syndrome (IMS), a condition that typically manifests 24–96 hours after exposure with characteristic proximal muscle weakness, cranial nerve palsies, and respiratory distress, first described by Senanayake and Karalliedde [11]. The incidence of IMS ranges from 20–68% in OP poisoning depending on the type and amount of OP compound and the adequacy of oxime therapy, as reported by He et al. [12]. Prolonged mechanical ventilation, as observed in our patient, is often required.

Case 2 represents a less severe form of OP toxicity, where early initiation of atropine and pralidoxime led to rapid recovery. Early intervention has been associated with significantly improved outcomes, as emphasized by Johnson et al. [13]. A prospective study conducted in rural Sri Lanka by Dawson et al. demonstrated that patients who received treatment within three hours of exposure had better survival rates compared to those treated later [14].

Case 3 demonstrated the severe consequences of delayed presentation. Mortality in OP poisoning increases significantly when treatment is initiated beyond 6–8 hours post-exposure, as noted by Peter et al. [15]. Furthermore, ingestion of highly toxic agents such as monocrotophos, combined with the absence of immediate resuscitative care, carries a poor prognosis, as reported by Singh and Sharma [16].

The socio-demographic profile of our patients—rural male farmers aged 40–60 years—mirrors findings reported by Ramesh and Dutta [17]. Lack of personal protective equipment, poor education on safe pesticide handling, and barriers to emergency medical access are recurrent themes in OP poisoning cases in India and other developing nations, as highlighted by Kishi et al. [18]. A study from Maharashtra conducted by Salvi and Adsul observed that nearly 80% of farm workers used no protective gear while spraying pesticides, and more than 50% had inadequate knowledge regarding pesticide toxicity [19]. Interventions such as community education, stricter regulation of pesticide sales, and distribution of protective equipment are crucial public health measures to reduce the burden of OP poisoning.

Conclusion

This case series highlights the ongoing burden of organophosphate poisoning among agricultural workers in rural settings. Delayed presentation, lack of awareness, and absence of personal protective equipment contribute significantly to morbidity and mortality. Early recognition and prompt treatment with atropine, oximes, and supportive care are crucial in improving outcomes. Preventive strategies such as farmer education, regulation of pesticide availability, and promotion of safe handling practices are essential to reduce the incidence and severity of such poisonings.

Conflict of Interest: Nil

Reference

1. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008;371(9612):597–607.
2. World Health Organization. Public health impact of pesticides used in agriculture. WHO; 1990.

3. Singh S, Sharma N. Neurological syndromes following organophosphate poisoning. *Neurol India*. 2000;48(4):308–313.
4. Balali-Mood M, Abdollahi M. Basic and clinical toxicology of organophosphorus compounds. *Springer Science & Business Media*, 2014.
5. Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. *Indian J Crit Care Med*. 2014;18(11):735–745.
6. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticides: An intermediate syndrome. *N Engl J Med*. 1987;316(13):761–763.
7. Batra AK, Keoliya AN, Jadhav GU. Poisoning: an unnatural cause of morbidity and mortality in rural India. *J Assoc Physicians India*. 2003;51:955–959.
8. Jeyaratnam J. Acute pesticide poisoning: a major global health problem. *World Health Stat Q*. 1990;43(3):139–144.
9. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008;371(9612):597–607.
10. Karalliedde L, Henry J. Effects of organophosphates on the nervous system. *J Neurol Neurosurg Psychiatry*. 1993;56(3):290–293.
11. Senanayake N, Karalliedde L. Neurotoxic effects of organophosphorus insecticides. *N Engl J Med*. 1987;316(13):761–763.
12. He F, Xu H, Qin F, Xu L, Huang J. Intermediate syndrome following acute organophosphate poisoning—an analysis of 21 cases. *Hum Exp Toxicol*. 1998;17(1):40–45.
13. Johnson MK, Jacobsen D, Meredith TJ, et al. Evaluation of antidotes for poisoning by organophosphorus pesticides. *Emerg Med Clin North Am*. 1994;12(2):365–381.
14. Dawson AH, Buckley NA, Whyte IM, et al. Early management and outcome in acute organophosphorus poisoning: a prospective study. *QJM*. 2004;97(11):697–709.
15. Peter JV, Moran JL, Graham PL. Advances in the management of organophosphate poisoning. *Expert Opin Pharmacother*. 2007;8(10):1451–1464.
16. Singh S, Sharma N. Neurological syndromes following organophosphate poisoning. *Neurol India*. 2000;48(4):308–313.
17. Ramesh T, Dutta TK. Clinical profile of organophosphorus poisoning in a tertiary care hospital in South India. *J Assoc Physicians India*. 2010;58:403–407.
18. Kishi M, Hirschhorn N, Djajadisastra M, Satterlee LN, Strowman S, Dilts R. Relationship of pesticide spraying to signs and symptoms in Indonesian farmers. *Scand J Work Environ Health*. 1995;21(2):124–133.
19. Salvi RM, Adsul BB. Knowledge, attitude and practice about pesticide exposure among farm workers in Western Maharashtra. *Indian J Occup Environ Med*. 2019;23(2):70–75.