

# *Acute Poisoning with Organophosphate Insecticides: A Report of 26 Cases and Literature Review*

*Nabila Choubane*

*University of Health Sciences, Faculty of Medicine Youcef El Khatib, Department of Emergency Medicine, University Hospital of Algiers, Algeria*

*Corresponding author: Nabila Choubane; University of Health Sciences, Faculty of Medicine Youcef El Khatib, Department of Emergency Medicine, University Hospital of Algiers, Algeria; email: email: nabila.choubane@yahoo.com*

## **Abstract**

**Introduction:** Acute organophosphate (OP) poisoning remains a major public health concern in regions with intensive agricultural activity. This study aimed to describe the clinical presentation, biological findings, therapeutic management, and outcomes of patients admitted with acute OP poisoning to a tertiary emergency department.

**Methods:** We conducted a retrospective descriptive study over 12 months (January–December 2023) at the University Hospital of Algiers emergency department. Patients with a confirmed diagnosis of acute OP poisoning were included. Data collected encompassed demographics, clinical presentation, laboratory findings, therapeutic interventions, and patient outcomes.

**Results:** Twenty-six patients were included, with a mean age of  $29.1 \pm 10$  years. Females accounted for 69% of cases, and 65% of poisonings were intentional. Muscarinic manifestations were present in all patients, while central nervous system signs and nicotinic manifestations were observed in 42% and 19%, respectively. The mean plasma cholinesterase level was 800 IU/L. All patients received atropine; pralidoxime was administered in 19% due to limited availability. Mechanical ventilation was required in 35%, vasopressors in 23%, and anticonvulsants in 8% of cases. The overall mortality rate was 23%, and the mean hospital stay was  $11 \pm 2.3$  days.

**Conclusion:** Acute OP poisoning is common, severe, and potentially fatal. Early recognition, prompt supportive care, and improved access to specific antidotes such as oximes are crucial. Preventive strategies, including pesticide regulation and psychosocial support, are essential to reduce morbidity and mortality.

**Keywords:** Organophosphate; Poisoning; Clinical characteristics; Therapeutic management; Outcomes

## **INTRODUCTION**

Organophosphate (OP) insecticides are widely used in agriculture due to their efficacy, low cost, and availability (1,2). However, their high toxicity makes them a common cause of acute poisoning, particularly in low- and middle-income countries and rural areas (1,2). OPs exert their

toxic effects through irreversible inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synaptic junctions and overstimulation of muscarinic, nicotinic, and central cholinergic receptors (3).

Pesticide poisoning represents a major public health problem worldwide. According to WHO estimates reported by Mew et al.,

approximately 3 million cases occur each year, resulting in over 250,000 deaths, the majority of which are attributable to organophosphates (3). Suicide due to pesticide ingestion constitutes a significant proportion of these fatalities, particularly in South Asia, where nationally representative surveys have documented high suicide mortality rates (4).

Clinically, acute OP poisoning presents with muscarinic signs (hypersalivation, bradycardia, bronchospasm), nicotinic manifestations (fasciculations, muscle weakness), and central nervous system disturbances (confusion, seizures, coma) (5,6). Prognosis depends on the ingested dose, compound type, route of exposure, and timeliness of medical intervention (3,7,8).

In Algeria, epidemiological data on OP poisoning remain limited, despite frequent exposure in rural areas. This study aimed to describe the clinical, biological, therapeutic, and prognostic characteristics of patients admitted for acute OP poisoning to a university emergency department in Algiers, to inform management strategies and preventive measures.

## **METHODS**

This was a retrospective descriptive study conducted in the emergency department of the University Hospital of Algiers over a 12-month period, from January 1<sup>st</sup> to December 31<sup>st</sup>, 2023. All patients admitted for acute organophosphate poisoning were included if

the diagnosis was confirmed both clinically (presence of cholinergic syndrome) and biologically (marked decrease in plasma cholinesterase activity). Patients with mixed or undocumented poisonings were excluded.

Data were extracted from medical records and included: Demographics (age, sex); Circumstances of poisoning: intentional vs. accidental; Clinical presentation (muscarinic, nicotinic, and central nervous system signs); Biological investigations (plasma acetylcholinesterase levels (Ellman's method)); Therapeutic management (atropine, pralidoxime, mechanical ventilation, vasopressors, anticonvulsants, gastric lavage); and Outcomes (length of hospital stay, mortality).

Data analysis was descriptive, using Microsoft Excel 2023. Results are reported as means, frequencies, and percentages.

The study was conducted in accordance with the Declaration of Helsinki. Due to the retrospective and anonymous nature of the data, formal informed consent was not required. Institutional approval was obtained.

## **RESULTS**

A total of 26 patients were included. The mean age was  $29.1 \pm 10$  years, with females accounting for 69% (n=18) of cases. Most poisonings (65%) were intentional. All patients presented with a muscarinic syndrome. Central nervous system manifestations occurred in 42% of cases, and nicotinic signs in 19%. The mean plasma

cholinesterase activity was 800 IU/L, indicating significant enzymatic inhibition (Table 1).

**Table 1. Demographic and clinical characteristics of patients with acute organophosphate poisoning (n = 26)**

Parameters	Value
Mean age ( $\pm$ SD); years	29.1 $\pm$ 10
Female sex; n(%)	18 (69)
Male; n(%)	8 (31)
Intentional poisoning; n(%)	17 (65)
Accidental poisoning; n(%)	9 (35)
Muscarinic syndrome; n(%)	26 (100)
Central nervous system signs; n(%)	11 (42)
Nicotinic signs; n(%)	5 (19)
Mean cholinesterase level; IU/L	800

All patients received atropine, while pralidoxime was administered in only five cases (19%) due to limited availability. Nine patients (35%) required mechanical ventilation, six (23%) needed vasopressor support, and two (8%) were treated with anticonvulsants. The overall mortality rate was 23%, and the mean hospital stay was 11  $\pm$  2.3 days (Table 2).

**Table 2. Therapeutic management and outcomes of patients with acute organophosphate poisoning**

Therapeutic Intervention/Outcomes	Value
Atropine; n(%)	26 (100)
Pralidoxime; n(%)	05 (19)
Mechanical ventilation; n(%)	09 (35)
Vasopressor support; n(%)	06 (23)
Anticonvulsants (benzodiazepines); n(%)	02 (8)
Mean length of hospital stay; days	11 $\pm$ 2.3
Deaths; n(%)	06 (23)

## DISCUSSION

This retrospective study describes the clinical and epidemiological characteristics of acute OP poisoning in a hospital setting in Algeria. The predominance of intentional exposure in

our cohort aligns with previous hospital-based and epidemiological data, confirming that pesticide ingestion is a major method of self-harm in many low- and middle-income countries [1,2,4]. Systematic reviews and national surveys have highlighted the substantial contribution of pesticide self-poisoning to global suicide mortality [2–4].

In our series, women accounted for a considerable proportion of self-poisoning cases. Similar patterns have been reported in hospital-based cohorts from resource-limited settings, although gender distributions vary across regions and study populations [1,4]. This variability likely reflects sociocultural and demographic differences rather than a uniform epidemiological pattern.

Clinically, the predominance of muscarinic manifestations observed in our patients is consistent with the established mechanism of acetylcholinesterase inhibition, leading to acetylcholine accumulation at synaptic junctions [5]. The frequent occurrence of nicotinic and central nervous system signs further illustrates the broad clinical spectrum described in prospective studies [5–7]. Previous research has shown that clinical severity depends not only on the ingested dose and time to treatment but also on the specific OP compound involved, as toxicity varies markedly among agents [5,12].

Measurement of plasma cholinesterase activity remains a useful supportive diagnostic and severity marker. The markedly

reduced levels observed in our cohort reflect significant enzyme inhibition, consistent with previous clinical and toxicological studies [5,8]. Nevertheless, laboratory results should always be interpreted in conjunction with clinical findings, particularly in settings where timely assays may not be available [5]. The systematic administration of atropine in our cohort is in line with established management principles, where early and adequate atropinization remains the cornerstone of treatment [9,13]. The limited use of pralidoxime reflects issues of availability. While oximes are recommended as specific antidotes, their impact on mortality and clinical outcomes remains debated. Randomized trials and systematic reviews have reported heterogeneous results, with uncertainty regarding their benefit, particularly in severe cases or when treatment is delayed [9–11]. These data support cautious interpretation of oxime efficacy while acknowledging their continued indication when accessible.

The high rates of mechanical ventilation and vasopressor support indicate a severe cholinergic crisis with respiratory and cardiovascular compromise. Comparable needs for advanced supportive care have been reported in intensive care cohorts from regions with high OP exposure [6,12]. Respiratory failure remains a key determinant of prognosis in acute OP poisoning [5,6].

The observed mortality rate in our series was 23.1%, highlighting the severity of acute OP poisoning. This rate is consistent with other hospital-based studies from regions with a high incidence of OP exposure, where reported mortality generally ranges between 15% and 25% [6,12,13]. Differences between studies may reflect variations in clinical severity, access to intensive care, and overall management resources.

From a public health perspective, these findings reinforce the importance of preventive strategies targeting access to highly hazardous pesticides. Restricting availability has been proposed as a key intervention to reduce fatal self-poisoning in developing countries [14].

This study has several limitations. The sample size was relatively small, which limits statistical power, and the retrospective design without long-term follow-up prevented the assessment of functional or psychological sequelae after poisoning. In addition, precise identification of the ingested organophosphate compounds was sometimes not possible, restricting analysis according to compound type. Despite these limitations, this series provides essential data for Algeria and serves as a foundation for prospective multicenter studies aimed at refining therapeutic and preventive strategies.

## **CONCLUSION**

Acute organophosphate poisoning remains a frequent and life-threatening medical

emergency. Our findings confirm the predominance of intentional exposure, the characteristic cholinergic clinical pattern, and the substantial need for intensive supportive care. While atropine remains the cornerstone of therapy, limited access to oximes and variability in compound toxicity may influence outcomes. Strengthening early management and implementing preventive strategies, including pesticide regulation and psychosocial interventions, are essential to reduce mortality. Prospective multicenter studies are warranted to clarify optimal therapeutic approaches.

**Conflict of interest:** The authors declare no conflict of interest.

## REFERENCES

1. Peter JV, Jerobin J, Nair A, et al. Clinical profile and outcome of patients with organophosphate poisoning needing intensive care. *Indian J Crit Care Med.* 2014;18(9):576–80.
2. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. *BMC Public Health.* 2007;7:357.
3. Mew EJ, Padmanathan P, Konradsen F, Eddleston M, Chang SS, Phillips MR, Gunnell D. The global burden of fatal self-poisoning with pesticides 2006–15: Systematic review. *J Affect Disord.* 2017;219:93–105.
4. Patel V, Ramasundarahettige C, Vijayakumar L, et al. Suicide mortality in India: a nationally representative survey. *Lancet.* 2012;379(9834):2343–51.
5. Eddleston M, Eyer P, Worek F, et al. Differences between organophosphorus insecticides in human self-poisoning: a prospective cohort study. *Lancet.* 2005;366(9495):1452–9.
6. Karunarathne A, Jayarathne S, Kularatne SA, et al. Outcome of organophosphate poisoning in rural Sri Lanka: A tertiary care hospital-based study. *Indian J Crit Care Med.* 2021;25(Suppl 2):S139–47.
7. Banerjee I, Tripathi SK, Roy AS. Organophosphorus poisoning: A clinicopathological study in rural Bengal. *N Am J Med Sci.* 2012;4(3):147–50.
8. Shadnia S, Azizi E, Hosseini A, et al. Evaluation of oxidative stress and genotoxicity in organophosphate pesticide manufacturing workers. *Hum Exp Toxicol.* 2005;24(6):297–300.
9. Vale JA, Meredith TJ, Buckley NA. Oximes in organophosphate poisoning: a systematic review of clinical trials. *Clin Toxicol (Phila).* 2016;54(8):627–34.
10. Buckley NA, Eddleston M, Li Y, et al. Oximes for acute organophosphate pesticide poisoning. *Cochrane Database Syst Rev.* 2011;(2):CD005085.
11. Roberts DM, Eddleston M, Wilks MF, et al. Clinical outcomes and kinetics of pralidoxime in acute organophosphate poisoning: a multicenter randomized controlled trial. *Lancet.* 2007;369(9564):1035–41.
12. Dawson AH, Eddleston M, Senarathna L, et al. Acute human lethal toxicity of pesticides: a prospective cohort study. *Crit Care Med.* 2010;38(3):832–8.
13. Pawar KS, Bhoite RR, Pillay CP, et al. Management of organophosphorus poisoning with a single bolus of atropine. *Lancet.* 2006;368(9553):2136–41.
14. Konradsen F, van der Hoek W, Cole DC, et al. Reducing acute poisoning in developing countries, options for restricting the availability of pesticides. *Toxicol Lett.* 2003;137(3):167–74.