

Research Article

# Correlation of Serum Cholinesterase Levels with Clinical Severity and Outcomes in Paediatric Organophosphorus Poisoning

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## Abstract

**Background:** Organophosphorus (OP) poisoning is a significant cause of morbidity and mortality in children, particularly in agricultural regions. Serum cholinesterase (ChE) activity is widely used for diagnosis, but its prognostic value in paediatric patients remains uncertain.

**Methods:** A retrospective analysis was conducted on paediatric patients with confirmed OP poisoning admitted to a tertiary care centre over one year. Severity was assessed based on presenting symptoms, need for mechanical ventilation, and ICU stay duration. Serum ChE levels were measured at admission and serially thereafter. Correlations between ChE levels and clinical outcomes were analysed.

**Results:** All patients had reduced serum ChE levels at admission (mean:  $XX \pm SD$  U/L). No statistically significant correlation was found between baseline ChE levels and mortality, need for ventilatory support, or ICU stay duration ( $p > 0.05$ ). Patients with extremely low ChE ( $<400$  U/L) tended to have prolonged recovery and higher complication rates. Most survivors showed substantial improvement in ChE activity within 24-36 hours.

**Conclusion:** In paediatric OP poisoning, serum ChE levels confirm the diagnosis but have limited prognostic value. Clinical assessment remains essential for severity grading and management.

**Keywords:** Organophosphorus Poisoning, Serum Cholinesterase, Paediatric, Prognosis, Mechanical Ventilation, Clinical Severity.

## INTRODUCTION

Organophosphorus (OP) compounds are widely used pesticides in agricultural communities, and poisoning by ingestion, inhalation, or dermal absorption is a common medical emergency in children, particularly in rural and developing regions [1,5,12].

OP compounds exert toxicity by irreversibly inhibiting acetylcholinesterase (AChE) and butyrylcholinesterase (pseudocholinesterase), enzymes responsible for the breakdown of acetylcholine at synaptic junctions [10,15]. This inhibition leads to excessive acetylcholine accumulation, producing overstimulation of muscarinic, nicotinic, and central nervous system (CNS) receptors, with clinical manifestations such as miosis, bronchorrhoea, fasciculations, seizures, and respiratory failure [7,9,11,13].

Serum ChE assays are frequently employed in the diagnostic work-up of suspected OP poisoning because significantly reduced levels strongly support exposure [1,7,10,15]. However, their role in predicting prognosis remains controversial. Several adult studies have demonstrated poor correlation between

baseline ChE activity and clinical severity [2,4,6,8].

In the paediatric population, data are scarce. El-Naggar et al. (2009) observed that although ChE levels confirm the diagnosis, they do not reliably predict the need for mechanical ventilation or mortality risk [1]. Similarly, Nouira et al. (1994) found no significant relationship between ChE levels and atropine requirement, ICU stay, or severity grading [2]. Carbamate poisoning, though related, generally has a more benign and self-limiting course due to reversible enzyme inhibition [5]. Early and accurate severity assessment in OP poisoning is critical for guiding aggressive management, triage, and resource allocation [8,14]. While scoring systems such as the Bardin classification [3] and the Simplified Acute Physiology Score (SAPS) [2] are used in adults, their application to paediatric OP poisoning remains poorly validated. This study aims to assess the correlation between baseline serum ChE levels, clinical severity, and outcomes in paediatric OP poisoning.

## METHODS

### Study Design and Setting

This retrospective observational study was conducted in the Paediatric Intensive Care Unit (PICU) of ABVIMS and Dr. Ram Manohar Lohia Hospital, New Delhi, over a period of one year.

### Participants

Children aged 0–18 years admitted with confirmed OP poisoning were included. Diagnosis was established by history of exposure, characteristic clinical features, and low serum ChE levels. Patients with carbamate-only poisoning, co-ingestion of other toxins, or pre-existing neurological disease were excluded.

### Data Collection

Data retrieved from medical records included demographic variables, source and route of exposure, latency between exposure and hospital arrival, presenting symptoms, baseline and serial serum ChE levels, and treatment details (atropine, oximes). Complications such as aspiration pneumonia, seizures, and septic shock were recorded. Outcomes included need for mechanical ventilation, ICU stay duration, and survival status.

### Laboratory Measurement

Serum ChE activity was determined by spectrophotometry (Ellman method), with a laboratory reference range of 1,900–3,800 U/L. Measurements were taken at admission and repeated every 12 hours until recovery or discharge.

### Severity Classification

Severity was graded using a modified Bardin classification:

- **Mild:** Muscarinic symptoms without CNS or respiratory compromise
- **Severe:** Muscarinic plus CNS symptoms but stable respiratory function
- **Life-threatening:** Respiratory failure requiring ventilatory support or severe CNS depression

### Statistical Analysis

Descriptive statistics were calculated. Pearson's correlation assessed relationships between baseline ChE levels and continuous outcomes; Chi-square or Fisher's exact test was used for categorical outcomes. A p-value <0.05 was considered statistically significant.

## RESULTS

Table 1. Demographic and Clinical Characteristics

Variable	Findings
Age range (years)	0.2–18
Male:Female ratio	14:33
Time to hospital (hours)	2.4 (range 1–6)
Most common route	Oral ingestion (89.4%)
Most common symptoms	Diarrhoea, vomiting, miosis, salivation, sweating

Table 2. Serum Cholinesterase Levels and Recovery

ChE Level (U/L)	Patients at admission (%)	Normal (>1900 U/L) at 12h (%)	Normal at 24h (%)
<400	34.0	2.0	2.0
400–1000	59.6	27.7	10.6
1000–1900	6.4	21.3	10.6
>1900	0.0	48.9	76.6

Table 3. Treatment Modalities and Outcomes

Treatment/Outcome	Number of patients (%)
Atropine only	19 (40.4%)
Atropine + oxime	28 (59.6%)
Mechanical ventilation	6 (12.8%)
Mortality	4 (8.5%)

Table 4. Complications Observed

Complication	Number of patients (%)
Respiratory failure	8 (17%)
Aspiration pneumonia	5 (10.6%)
Convulsions	3 (6.4%)

Septic shock	1 (2%)
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## DISCUSSION

This study reinforces earlier reports that serum ChE measurement is a valuable diagnostic tool in paediatric OP poisoning but a poor predictor of clinical severity or outcome [1,4,6]. In agreement with Noura et al. (1994), we found no statistically significant correlation between baseline ChE levels and mortality, ventilation requirement, or ICU stay [2]. The prognostic limitations of ChE may stem from variable enzyme inhibition kinetics [7,11,13], differences between plasma and erythrocyte cholinesterase activity [10,15], and non-cholinergic toxic effects [13].

El-Naggar et al. (2009) similarly observed that CNS depression, miosis, and fasciculations were stronger predictors of poor outcomes than enzyme levels [1]. While extremely low ChE (<400 U/L) was associated with a trend toward increased complications, statistical significance was not achieved, possibly due to sample size limitations.

These findings underscore the importance of early aggressive supportive care—including airway protection, atropinisation, and ventilatory support—irrespective of ChE levels [6, 8, 9]. Lifshitz et al. (1997) also highlight

that in carbamate poisoning, which has faster spontaneous enzyme reactivation, oxime therapy is often unnecessary [5].

The role of dermal exposure as a significant but often under-recognised route, especially in children, has been discussed by Phillips (2001) [12], and awareness of this route is important in prevention strategies. Laboratory readiness and the capacity to perform accurate, timely ChE testing are also critical, as emphasised by Flanagan (2004) [14].

Limitations of this study include its retrospective design, single-centre nature, and absence of erythrocyte AChE measurements, which may more closely reflect neuromuscular function [10,15]. Future prospective, multicentre studies combining ChE assays with validated paediatric clinical scoring systems could better define the prognostic role of ChE in OP poisoning [8,14].

## CONCLUSION

Baseline serum ChE levels in paediatric OP poisoning are diagnostic but lack prognostic value. Clinical monitoring and severity-based supportive management remain paramount for improving outcomes.

## REFERENCES

1. El-Naggar AE, Abdalla MS, El-Sebaey AS, Badawy SM. Clinical findings and cholinesterase levels in children of organophosphates and carbamates poisoning. *Eur J Pediatr*. 2009; 168(8):951–956.
2. Noura S, Abroug F, Elatrous S, Boujdaria R, Bouchoucha S. Prognostic value of serum cholinesterase in organophosphate poisoning. *Chest*. 1994;106(6):1811–1814.
3. Bardin PG, Van Eeden SF. Organophosphate poisoning: grading the severity and comparing treatment between atropine and glycopyrrolate. *Crit Care Med*. 1990;18(9):956–960.
4. Tsao ICY, Juang YC, Lan RS, Shieh WB, Lee CH. Respiratory failure of acute organophosphate and carbamate poisoning. *Chest*. 1990;98(3):631–636.
5. Lifshitz M, Shahak E, Bolotin A, Sofer S. Carbamate poisoning in early childhood and in adults. *J Toxicol Clin Toxicol*. 1997;35(1):25–27.
6. Sungur M, Güven M. Intensive care management of organophosphate insecticide poisoning. *Crit Care*. 2001; 5(4):211–215.
7. Kwong TC. Organophosphate pesticides: biochemistry and clinical toxicology. *Ther Drug Monit*. 2002; 24(1):144–149.
8. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet*. 2008; 371(9612):597–607.
9. Aaron CK, Howland MA. Insecticides: organophosphates and carbamates. In: Goldfrank LR, Flomenbaum NE, Lewin NA, et al., eds. *Goldfrank's Toxicologic Emergencies*. 6th ed. Stamford, CT: Appleton & Lange; 1998:1429–1449.
10. Whittaker M. Cholinesterases. In: Bergmeyer HU, ed. *Methods of Enzymatic Analysis*. Vol. 4. Weinheim: Chemie; 1984:52–62.
11. Vale JA. Toxicokinetic and toxicodynamic aspects of organophosphorus insecticide poisoning. *Toxicol Lett*. 1998;102–103:649–652.
12. Phillips A. Assessing dermal exposure to pesticide from nonagricultural use: a UK Health and Safety Executive (HSE)

- perspective. *J Environ Monit.* 2001;3:14N–17N.
13. Hayes WJ. Organophosphate insecticides. In: Hayes WJ, ed. *Pesticides Studied in Man*. Baltimore, MD: Williams & Wilkins; 1982:285–315.
14. Flanagan RJ. Developing an analytical toxicology service. *Toxicol Rev.* 2004;23(4):251–263.
15. Moss DW, Henderson AR. Clinical enzymology. In: Burtis CA, Ashwood ER, eds. *Textbook of Clinical Chemistry*. 3rd ed. Philadelphia: Saunders; 1999:708–711