e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 826-831

Original Research Article

Association of Serum Pseudocholinesterase Levels with Severity and Mortality in Acute Organophosphorus Poisoning: A Prospective Observational Study

Manu Kiran B. T.[1], Ramya D.N.[2], Chaithra H.[3]

[1]Senior Resident, Department of General Medicine, Farookh Academy of Medical Education Hospital & Research Institute, Mysuru, Karnataka, India

[2]Assistant Professor, Department of General Medicine, Farookh Academy of Medical Education Hospital & Research Institute, Mysuru, Karnataka, India

[3]Assistant Professor, Department of General Medicine, Farookh Academy of Medical Education Hospital & Research Institute, Mysuru, Karnataka, India

Received: 01-07-2026 / Revised: 25-07-2026 / Accepted: 11-08-2026

Corresponding author: Dr. Manu Kiran B T

Conflict of interest: Nil

Abstract

Background: Acute organophosphorus (OP) poisoning is an important cause of morbidity and mortality, with severe cases frequently requiring mechanical ventilation and intensive care. Serum pseudocholinesterase (PChE), which decreases following OP exposure, may provide a readily available biochemical marker for assessing severity and prognosis.

Objectives: To evaluate serum pseudocholinesterase levels as a predictor of clinical severity and mortality in acute OP poisoning and to determine their association with mechanical ventilation and patient outcome.

Materials and Methods: This prospective observational study included 100 adult patients with acute OP poisoning. Severity at admission was assessed using the Peradeniya Organophosphorus Poisoning (POP) scale. Admission serum PChE levels were categorized as ≤1000, 1001–2000, 2001–3000, and >3000 U/L. Associations of PChE levels with POP severity, mechanical ventilation, and in-hospital mortality were analyzed, with $p<0.05$ considered statistically significant.

Results: Mean serum PChE was 1821.78±1044.91 U/L. All patients with severe poisoning had PChE ≤1000 U/L ($p<0.001$). Among patients with PChE ≤1000 U/L, 79.41% required mechanical ventilation compared with 1.52% of those with levels >1000 U/L ($p<0.001$). Mortality was 67.65% in the ≤1000 U/L group, compared with 3.70% at 1001–2000 U/L, with no deaths above 2000 U/L ($p<0.001$). Mean PChE was significantly lower among non-survivors than survivors (698.42±255.75 vs. 2176.53±943.30 U/L; $p<0.001$).

Conclusion: Low admission serum pseudocholinesterase levels were strongly associated with greater poisoning severity, requirement for mechanical ventilation, and mortality. Serum PChE, particularly a level ≤1000 U/L, may serve as a simple and inexpensive adjunct for early risk stratification in acute OP poisoning, although it should be interpreted together with clinical severity assessment.

Keywords: Organophosphorus poisoning; pseudocholinesterase; Peradeniya Organophosphorus Poisoning scale; mechanical ventilation; mortality; prognosis.

DOI: 10.25258/ijcpr.18.8.128

Introduction

Organophosphorus (OP) compounds are widely used agricultural insecticides and remain an important cause of acute poisoning, particularly in low- and middle-income countries where pesticides are readily accessible. Acute OP poisoning contributes substantially to pesticide-related morbidity, hospitalization, and preventable mortality. Severe poisoning frequently requires intensive care because respiratory failure, cardiovascular instability, altered consciousness, seizures, and intermediate syndrome may complicate the clinical course. [1,2] Recent evidence continues to identify OP insecticides as an important contributor to pesticide-associated deaths and hospital admissions worldwide.[3] The principal toxic effect of OP compounds results from inhibition of acetylcholinesterase, producing accumulation of acetylcholine at muscarinic, nicotinic, and central nervous system synapses. Consequently, patients may develop excessive

salivation, bronchorrhea, bronchospasm, miosis, bradycardia, fasciculations, muscle weakness, altered sensorium, and respiratory paralysis. Respiratory failure is the major life-threatening complication and may result from a combination of excessive airway secretions, central respiratory depression, bronchoconstriction, and weakness or paralysis of respiratory muscles. [2,4]

Early assessment of poisoning severity is therefore essential for identifying patients who require intensive monitoring, mechanical ventilation, and aggressive antidotal therapy. Several clinical scoring systems have been developed for this purpose, of which the Peradeniya Organophosphorus Poisoning (POP) scale is commonly used. Although clinical scoring provides useful prognostic information, an objective and readily measurable biochemical marker capable of predicting severity and outcome would further improve early risk stratification.[5]

Serum pseudocholinesterase, also known as plasma cholinesterase or butyrylcholinesterase, is inhibited following exposure to OP compounds and its activity typically decreases considerably after significant poisoning. Measurement of serum pseudocholinesterase is relatively simple and widely available in clinical laboratories. Previous studies have demonstrated an inverse relationship between serum pseudocholinesterase activity and the clinical severity of OP poisoning, with markedly reduced levels being observed more frequently among patients with severe manifestations, prolonged intensive care requirements, mechanical ventilation, and adverse outcomes. [5–7]

However, the prognostic performance of pseudocholinesterase has not been uniform across studies because enzyme activity may be influenced by the specific OP compound, quantity and route of exposure, interval between poisoning and blood sampling, hepatic function, nutritional status, and individual biological variation. Consequently, serum pseudocholinesterase should preferably be evaluated in conjunction with clinical findings rather than as an isolated diagnostic parameter. [6,7] Nevertheless, its potential association with severity and mortality makes it an attractive, inexpensive biomarker for early prognostication, particularly in resource-limited settings.

Therefore, the present study was undertaken to evaluate serum pseudocholinesterase levels as a predictor of clinical severity and mortality in patients with acute organophosphorus poisoning and to determine their relationship with clinical severity, requirement for ventilatory support, and patient outcome.

Materials and Methods

Study design and setting: This prospective observational study was carried out in the Department of General Medicine at Farookh academy of Medical Education, Mysuru, Karnataka, over a period of 10 months from September 2025 to June 2026. A total of 100 patients fulfilling the eligibility criteria were included in the study. Adult patients admitted with acute organophosphorus (OP) compound poisoning constituted the study population.

Inclusion Criteria

- Adults aged ≥18 years
- Patients admitted with acute OP poisoning
- Patients providing written informed consent

Exclusion Criteria

- Patients with consumption of other poisons along with an OP compound
- Patients those with chronic neurological illness

Data Collection and Clinical Assessment: After obtaining informed consent, a detailed history was recorded and a comprehensive clinical examination was performed. The severity of poisoning at admission was assessed using the Peradeniya Organophosphorus Poisoning (POP) scale, and patients were classified according to clinical severity. For the present paper, the principal variables of interest were POP severity, serum pseudocholinesterase level, requirement for mechanical ventilation, and in-hospital mortality.

Diagnosis of Organophosphorus Poisoning: Organophosphorus poisoning was diagnosed based on a definite or suspected history of exposure to an organophosphorus compound, characteristic clinical features of the cholinergic and response to atropine therapy. The diagnosis was further supported by a reduced serum pseudocholinesterase (butyrylcholinesterase) level compared with the laboratory reference range. When available, the pesticide container, label, or information provided by relatives was used to confirm the nature of the compound involved.

Laboratory Investigations: Venous blood was collected at the time of admission for estimation of serum pseudocholinesterase (PChE). Serum pseudocholinesterase (PChE; butyrylcholinesterase) activity was measured at admission before or as early as possible after initiation of treatment using a kinetic colorimetric method on an automated biochemical analyser. Serum pseudocholinesterase values were analysed both as a continuous variable and according to clinically relevant categories. The laboratory normal reference range for serum PChE was approximately 4,000–12,000 U/L. For the present analysis, patients were grouped as ≤1000, 1001–

2000, 2001–3000, and >3000 U/L, consistent with the categories used in the manuscript results.

Outcome Assessment: The primary outcomes were the relationship of admission serum pseudocholinesterase levels with clinical severity and in-hospital mortality. Requirement for mechanical ventilation was evaluated as an additional marker of severe poisoning.

Ethical Status: The study was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants.

Statistical Analysis: Data were analysed using SPSS for Windows, version 21.0. Continuous variables were summarized using mean, median, standard deviation, minimum, and maximum values. Categorical variables were expressed as frequencies and percentages. Associations between categorical variables were evaluated using the Chi-square test or Fisher's exact test, as appropriate. The independent-samples t-test was used to compare mean values between two groups. A p-value <0.05 was considered statistically significant.

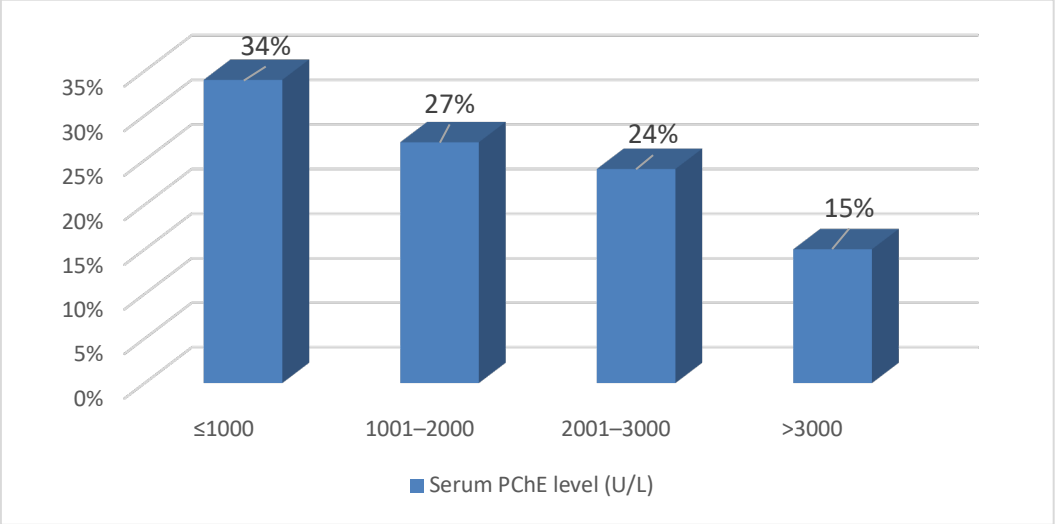
Results

The mean age of the 100 patients was 35.69 ± 14.14 years. The mean POP score was 3.97 ± 2.03, while the mean serum pseudocholinesterase (PChE) level was 1821.78 ± 1044.91 U/L.

Table 1: Baseline clinical and biochemical characteristics of study participants

Variable	Value
Number of patients	100
Age, mean ± SD (years)	35.69 ± 14.135
Age, median (range), years	32 (20–70)
POP score, mean ± SD	3.97 ± 2.027
POP score, median (range)	4 (1–8)
Serum pseudocholinesterase, mean ± SD (U/L)	1821.78 ± 1044.911
Serum pseudocholinesterase, median (range), U/L	1764 (187–4007)

PChE levels ≤1000 U/L were observed in 34% of patients, followed by 1001–2000 U/L in 27%, 2001–3000 U/L in 24%, and >3000 U/L in 15%. Thus, approximately one-third of patients had markedly reduced PChE levels (≤1000 U/L).



Graph 1: Distribution of serum pseudocholinesterase levels among patients with organophosphorus poisoning

Lower PChE levels were significantly associated with greater poisoning severity. All patients with severe poisoning had PChE ≤1000 U/L, compared with 50.0% of moderate and 10.64% of mild cases (p<0.001).

Table 2: Association between serum pseudocholinesterase level and POP severity

PChE level (U/L)	Mild (n=47)	Moderate (n=48)	Severe (n=5)	Total	p-value
≤1000	5 (10.64%)	24 (50.00%)	5 (100%)	34 (34%)	
1001–2000	16 (34.04%)	11 (22.92%)	0	27 (27%)	
2001–3000	15 (31.91%)	9 (18.75%)	0	24 (24%)	
>3000	11 (23.40%)	4 (8.33%)	0	15 (15%)	<0.001
Total	47	48	5	100	

Mechanical ventilation was required in 79.41% of patients with PChE ≤1000 U/L compared with only 1.52% of those with PChE >1000 U/L, demonstrating a significant association between low PChE and ventilatory requirement (p<0.001).

Table 3: Association between serum pseudocholinesterase level and requirement for mechanical ventilation

PChE level	Ventilation required	Ventilation not required	Total	p-value†
≤1000 U/L	27 (79.41%)	7 (20.59%)	34	
>1000 U/L	1 (1.52%)	65 (98.48%)	66	<0.001
Total	28 (28.0%)	72 (72.0%)	100	

Mortality was highest among patients with PChE ≤1000 U/L (67.65%) and decreased to 3.70% among those with levels of 1001–2000 U/L. No deaths occurred at PChE levels >2000 U/L (p<0.001).

Table 4: Association between serum pseudocholinesterase level and mortality

PChE level	Death	Recovered	Mortality rate
≤1000 U/L	23	11	67.65%
1001–2000 U/L	1	26	3.70%
2001–3000 U/L	0	24	0%
>3000 U/L	0	15	0%

Non-survivors had significantly lower mean PChE levels than survivors (698.42 ± 255.75 vs. 2176.53 ± 943.30 U/L), with a mean difference of 1478.11 U/L (p<0.001).

Table 5: Comparison of mean serum pseudocholinesterase levels between survivors and non-survivors

Outcome	n	Mean PChE (U/L)	SD	Range (U/L)	Mean difference	p-value
Death	24	698.42	255.749	187–1438		
Recovered	76	2176.53	943.304	567–4007	1478.11	<0.001

Mortality was significantly higher among patients requiring mechanical ventilation (78.57%) than among those not requiring ventilation (2.78%; p<0.001), indicating a strong association between ventilatory requirement and adverse outcome.

Table 6: Relationship between mechanical ventilation and mortality

Ventilator support	Death, n (%)	Recovered, n (%)	Total	p-value
Required	22 (78.57%)	6 (21.43%)	28	
Not required	2 (2.78%)	70 (97.22%)	72	<0.001
Total	24	76	100	

Discussion

The present study demonstrated a strong relationship between serum pseudocholinesterase (PChE) activity and the severity and outcome of acute organophosphorus poisoning.

A clear inverse relationship was observed between serum PChE and POP severity. All five patients classified as having severe poisoning had PChE ≤1000 U/L, whereas progressively higher PChE levels were more frequently encountered among patients with mild poisoning (p<0.001).

This indicates that greater inhibition of circulating cholinesterase activity accompanies more pronounced clinical toxicity. Similar observations have been reported in previous studies in which reduced PChE activity was associated with greater morbidity and severity of OP poisoning [3, 8]. Hiremath et al [6], found admission PChE to be useful for assessing severity and subsequent critical-care requirements. The relationship with respiratory support was particularly striking. Among patients with PChE ≤1000 U/L, (79.41%) required mechanical ventilation, compared with only (1.52%) with levels >1000 U/L (p<0.001). Hiremath et al [6], similarly demonstrated that patients with PChE <1000 IU/L had fewer ventilator-free days and longer ICU stays, suggesting that marked enzyme suppression identifies patients at increased risk of respiratory compromise. This association is clinically relevant because early recognition of patients likely to require ventilatory support can facilitate timely ICU admission and airway management.

Mortality also increased markedly with decreasing PChE activity. Of the 34 patients with PChE ≤1000 U/L, 23 died, corresponding to a mortality rate of 67.65%. In comparison, mortality was only 3.70% in patients with levels of 1001–2000 U/L, with no deaths among those with levels >2000 U/L (p<0.001). Furthermore, mean PChE was substantially lower among non-survivors than survivors (698.42 ± 255.75 vs. 2176.53 ± 943.30 U/L; p<0.001). Previous Indian studies have likewise reported an inverse relationship between

serum cholinesterase activity and adverse outcome [9, 10].

Mechanical ventilation itself identified a particularly high-risk group. Twenty-two of the 28 patients requiring ventilation died, compared with only two deaths among 72 patients who did not require ventilation ($p<0.001$). Previous studies have similarly demonstrated substantial morbidity and mortality among OP-poisoned patients requiring ventilatory support, reflecting the severity of respiratory and neuromuscular involvement [11, 12].

Taken together, these findings suggest that admission serum PChE, particularly a level ≤1000 U/L, may serve as a simple and readily available marker for identifying patients at high risk of severe poisoning, mechanical ventilation, and death. Nevertheless, PChE should not be interpreted in isolation. Eddleston et al [1], demonstrated that the prognostic performance of butyrylcholinesterase varies according to the specific OP compound involved, while another prospective study found no significant association between PChE and clinical outcome. Thus, PChE is best interpreted together with clinical severity scores and the patient's overall clinical condition.

The principal limitations include the single-center design, relatively small sample size, and lack of compound-specific analysis. Additionally, the study primarily evaluated PChE at admission rather than serial enzyme measurements. Larger prospective multicenter studies incorporating specific OP compounds and serial PChE measurements would better establish clinically useful prognostic cut-offs.

Conclusion

Serum pseudocholinesterase (PChE) levels showed a significant inverse association with the severity and outcome of acute organophosphorus poisoning. Patients with PChE ≤1000 U/L had substantially greater clinical severity, need for mechanical ventilation, and mortality, while survivors had significantly higher mean PChE levels than non-survivors (2176.53 vs. 698.42 U/L; $p<0.001$).

These findings suggest that admission serum PChE is a simple, inexpensive, and readily available prognostic marker that may assist in early risk stratification and identification of patients requiring intensive monitoring and ventilatory support. However, PChE should be interpreted alongside clinical assessment and severity scores rather than used as an isolated predictor.

References

1. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008;371(9612):597-607. doi:10.1016/S0140-6736(07)61202-1.
2. Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphorus poisoning: a review of different classification systems and approaches. Indian J Crit Care Med. 2014;18(11):735-745. doi:10.4103/0972-5229.144017.
3. Bhat, N., Prabhu H. D., R., & Krishna Bhat, A. G. (2021). A study to assess the utility of poison severity score, pseudocholinesterase levels and Glasgow coma scale in predicting severity and clinical outcome of organophosphorus poisoning. International Journal of Research in Medical Sciences, 9(4), 982–988. https://doi.org/10.18203/2320-6012.ijrms20210999
4. Aroniadou-Anderjaska V, Apland JP, Figueiredo TH, de Araujo Furtado M, Braga MF. Mechanisms of organophosphorus toxicity and the role of acetylcholine. Front Neurosci. 2023; 17:1188612.
5. Chaudhary SC, Singh K, Sawlani KK, Jain N, Vaish AK, Atam V, et al. Prognostic significance of estimation of pseudocholinesterase activity and role of Peradeniya Organophosphorus Poisoning scale in organophosphorus poisoning. Toxicol Int. 2013;20(3):214-217.
6. Hiremath P, Rangappa P, Jacob I, Rao K. Pseudocholinesterase as a predictor of mortality and morbidity in organophosphorus poisoning. Indian J Crit Care Med. 2016;20(10):601-604. doi:10.4103/0972-5229.192047.
7. Arivumani M, Indumathi G, Sasikumar P, Elango P. A study on predictors for mechanical ventilation in organophosphorus poisoning. J Cardiovasc Dis Res. 2023;14(1). DOI: https://doi.org/10.48047/
8. Kumar A, Chaudhary SC, Usman K, Kumar V, Sawlani KK, Patel ML, et al. Correlation of pseudocholinesterase level with clinical, biochemical parameters including cardiac profile and the outcome in organophosphorus poisoning. Ann Afr Med. 2024;23(4):704-709. doi: 10.4103/aam.aam_20_24.
9. Sagah GA, Sobeeh FG, Nassar EH, Selim AF, Sharif AF. PGI Score: A Promising Alternative to Serum Cholinesterase for Predicting Mortality and Adverse Outcomes in Acute Organophosphorus Poisoning: A Cross-Sectional Study. Basic Clin Pharmacol Toxicol. 2025 Dec;137(6): e70145. doi: 10.1111/bcpt.70145. PMID: 41277382.
10. Madhusoodanan A, Salgar V, Doddamani GB. Analysis of clinical predictors for mortality in severe organophosphorus poisoning. Int J Pharm Res Technol. 2025;15(2):356–360.

11. Bhattad PH, Bhattad SP. Organophosphorus poisoning: study of evaluation of clinically relevant indicators. Int J Adv Med. 2022. doi:10.18203/2349-3933.ijam20222401.
12. Goswamy R, Chaudhuri A, Mahashur AA. Pseudocholinesterase as a predictor of mortality and morbidity in organophosphorus poisoning. Indian J Crit Care Med. 2016; 20(10):601-604. doi:10.4103/0972-5229.192052. PMID: 27829717.