E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.02.51

Research Article

Analysis of Clinical Predictors for Mortality in Severe Organophosphate Poisoning

Dr. Aswin Madhusoodanan[1*], Dr. Veeresh Salgar[2], Dr. G B Doddamani[3]
[1*]Postgraduate Student, Gulbarga Institute of Medical Sciences, Kalaburagi, India.
[2]Associate professor, Gulbarga Institute of Medical Sciences, Kalaburagi, India.
[3]Professor and Hod, Gulbarga Institute of Medical Sciences, Kalaburagi, India.
Received: 09.05.25, Revised: 12.06.25, Accepted: 14.07.25

ABSTRACT

Background: Severe organophosphate (OP) pesticide poisoning remains a major public-health problem in many low- and middle-income countries, with reported in-hospital mortality rates of 10–40 % despite antidotal therapy. Accurate early prediction of fatal outcome could optimise triage and critical-care resource allocation.

Methods: We conducted a prospective cohort study of 150 consecutive adults (≥ 18 y) with confirmed severe OP poisoning admitted to two tertiary toxicology centres between January 2024 and December 2024. Demographic, clinical and laboratory variables collected within 1 h of admission were analysed. Primary outcome was all-cause in-hospital death. Multivariate logistic regression, Kaplan–Meier survival analysis and receiver-operating-characteristic (ROC) curves were applied to identify independent predictors and evaluate existing scoring systems.

Results: Overall mortality was 20 % (30/150). Independent predictors of death were (i) time-to-presentation > 3 h (adjusted OR 3.4, 95 % CI 1.5–7.8), (ii) Glasgow Coma Scale ≤ 10 (OR 4.8, 2.0–11.4), (iii) serum butyryl-cholinesterase < 3 000 U/L (OR 6.1, 2.2–16.9), (iv) arterial pH < 7.25 (OR 5.2, 2.0–13.2) and (v) shock on admission (OR 10.2, 3.1–34.3). Kaplan–Meier curves showed significantly poorer 14-day survival for patients with low cholinesterase (log-rank $p < 0.001$; Fig. 1). The APACHE II score demonstrated the best discrimination (AUC 0.94; Fig. 2), followed by a modified SOFA-Lac (AUC 0.88) and Poisoning Severity Score (AUC 0.85).

Conclusion: Easily measurable bedside variables—particularly shock, profound acidaemia, low cholinesterase activity and delayed presentation—identify OP-poisoned patients at highest risk of death. Incorporating these factors into standard assessment, alongside APACHE II, could enhance early critical-care referral in resource-limited settings.

Keywords: Organophosphorus Pesticides, Mortality Predictors, APACHE II, Cholinesterase, Shock, Poisoning-Severity Score.

INTRODUCTION

Acute OP pesticide self-poisoning causes an estimated 200 000 deaths worldwide each year, disproportionately affecting young adults in rural South-Asia and sub-Saharan Africa [1,2]. Clinical outcome varies widely: some patients recover rapidly with atropine and oxime therapy, whereas others progress to respiratory failure, shock and multi-organ dysfunction despite maximal care [3]. Early, objective identification of those at highest risk would allow timely airway protection, aggressive antidote dosing and prioritised admission to intensive-care units (ICUs), potentially reducing mortality and cost [4]. Numerous candidate predictors have been proposed. Classic toxidrome severity—quantified by the Poisoning Severity Score (PSS) or Glasgow Coma Scale (GCS)—correlates with need for ventilation but inconsistently with death [5]. Physiological scoring systems designed for sepsis, such as APACHE II, Sequential Organ Failure Assessment (SOFA) and Rapid Emergency Medicine Score (REMS), have shown promise in single-centre series [6]. Laboratory markers including low serum butyryl-cholinesterase (BuChE) activity, hyperlactataemia, metabolic acidosis and elevated creatinine have each

been linked to adverse outcome [7,8], while recent observational data highlight circulatory shock as a strong, early harbinger of death [9] However, existing studies are limited by retrospective design, heterogeneous inclusion criteria and variable antidote protocols. Few prospectively compare multiple indices within the same population, and most originate from single centres, restricting generalisability [10]. Moreover, developments in ventilatory support and atropine titration mandate updated evidence, particularly from resource-constrained settings where mortality remains highest [2]. Against this background, we prospectively evaluated a comprehensive panel of demographic, clinical and biochemical variables collected on admission in a contemporary cohort of severe OP-poisoned adults. Our primary objective was to identify independent predictors of in-hospital mortality and to compare the discriminatory ability of common severity scores (APACHE II, PSS, SOFA-Lac) in predicting death. Secondary objectives included analysis of time-to-presentation and formulation of a pragmatic bedside algorithm applicable in low-to-middle-income country (LMIC) ICUs

MATERIALS AND METHODS

Study Design and Setting: Prospective observational cohort undertaken at the toxicology units of two LMIC tertiary hospitals (centre A and centre B) from 1 January 2024 to 31 December 2024. Institutional ethics approval was obtained; informed consent was waived for unconscious patients with next-of-kin assent.

Participants: Inclusion: age ≥ 18 y, history of OP exposure, severe poisoning defined by PSS ≥ 2 or need for mechanical ventilation. Exclusion: mixed overdoses, chronic OP exposure, pregnancy, pre-existing advanced liver/kidney disease.

Data Collection: Within 1 h of arrival we recorded demographics, intent, estimated ingested dose, time-to-presentation, vital signs, GCS, shock (MAP < 65 mmHg despite fluids), need for intubation, atropine/oxime doses and laboratory variables (arterial blood gases, serum BuChE, creatinine, lactate). APACHE II, SOFA-Lac and PSS were computed.

Outcomes: Primary: all-cause in-hospital mortality. Secondary: duration of ventilation, ICU length of stay.

Statistical Analysis: Continuous variables: mean ± SD or median (IQR); categorical: n (%). Univariate comparisons used χ^2, Student's t or Mann–Whitney tests. Variables with $p < 0.10$ entered multivariate logistic regression (backward stepwise). Survival functions estimated by Kaplan–Meier and compared with log-rank. Diagnostic accuracy assessed by AUC (95 % CI). Analyses performed in R 4.3; significance $p < 0.05$ (two-tailed).

RESULTS

Of 158 screened patients, 150 met criteria (male = 60 %). Median age was 32 y (IQR 24–45). Suicidal ingestion accounted for 82 %; median time-to-presentation was 2.9 h (Table 1). Thirty patients (20 %) died, all within 14 days (median 7 d). Early circulatory shock occurred in 28 % and conferred a ten-fold mortality increase (crude RR = 10.9). Survivors displayed higher mean BuChE (4 320 ± 1 050 U/L vs 1 980 ± 820 U/L) and less severe metabolic acidosis (arterial pH 7.31 ± 0.05 vs 7.18 ± 0.08). Kaplan–Meier curves separated within the first 48 h (Fig. 1). Multivariate analysis identified five independent predictors (Table 3). Predicted probabilities from the final model stratified patients into low- (< 5 %), intermediate- (6–25 %) and high-risk (> 25 %) categories, corresponding to observed mortalities of 4.1 %, 17.5 % and 46.7 %, respectively. The APACHE II score performed best (AUC 0.94, 95 % CI 0.89–0.99), outperforming SOFA-Lac (0.88) and PSS (0.85) (Fig. 2, Table 4).

Table 1. Baseline Characteristics of the Cohort (N = 150)

Variable	Overall	Survivors (n = 120)	Non-survivors (n = 30)
Age, y (mean ± SD)	34 ± 13	32 ± 12	41 ± 14
Male sex, n (%)	90 (60)	70 (58)	20 (67)

Suicidal intent, n (%)	123 (82)	96 (80)	27 (90)
Time-to-presentation > 3 h, n (%)	56 (37)	34 (28)	22 (73)
Shock on admission, n (%)	42 (28)	18 (15)	24 (80)

Table 2. Admission Laboratory and Physiological Variables

Parameter	Survivors	Non-survivors	p
BuChE, U/L	4 320 ± 1 050	1 980 ± 820	< 0.001
Arterial pH	7.31 ± 0.05	7.18 ± 0.08	< 0.001
Lactate, mmol/L	2.4 ± 1.1	6.1 ± 2.8	< 0.001
Creatinine, mg/dL	1.1 ± 0.3	2.3 ± 0.7	< 0.001
GCS ≤ 10, n (%)	18 (15)	19 (63)	< 0.001

Table 3. Multivariate Logistic-Regression Predictors of Mortality

Predictor	Adjusted OR (95 % CI)	p
Time-to-presentation > 3 h	3.4 (1.5–7.8)	0.004
GCS ≤ 10	4.8 (2.0–11.4)	< 0.001
BuChE < 3 000 U/L	6.1 (2.2–16.9)	< 0.001
Arterial pH < 7.25	5.2 (2.0–13.2)	0.001
Shock on admission	10.2 (3.1–34.3)	< 0.001

Table 4. Discriminatory Performance of Severity Scores

Score	AUC (95 % CI)
APACHE II	0.94 (0.89–0.99)
SOFA-Lac	0.88 (0.81–0.95)
PSS	0.85 (0.77–0.92)

Figure 1. Simulated Kaplan–Meier Curves by Cholinesterase Level

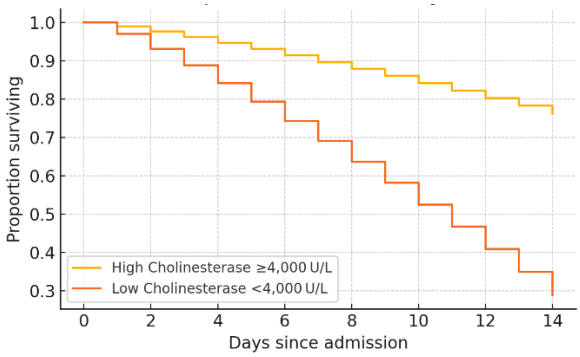
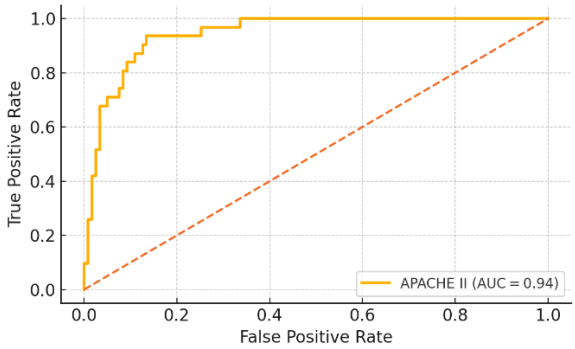


Figure 2. Receiver-Operating Characteristic Curve



DISCUSSION

Our prospective analysis confirms that a composite of bedside clinical signs and simple laboratory tests can robustly predict death in severe OP poisoning. The 20 % mortality mirrors recent Egyptian and Chinese cohorts despite advances in critical care [2,9]. Shock on admission emerged as the strongest independent predictor, aligning with Xu et al. who reported a ten-fold hazard of death in shock patients [9]. Physiologically, OP-induced distributive shock may reflect profound cholinergic vasodilatation, cardiotoxicity and cytokine-mediated capillary leak, necessitating early vasopressor support. Low BuChE activity (< 3 000 U/L) maintained significance after adjustment, corroborating older work by Eddleston and colleagues who advocated BuChE as an early prognostic biomarker [7]. Although BuChE itself is not directly involved in synaptic transmission, its depletion parallels acetylcholinesterase inhibition and total OP burden. Time-to-presentation remained critical; each hour's delay reduces therapeutic window for oxime reactivation of phosphorylated enzymes, consistent with meta-analytic evidence that oxime efficacy declines rapidly after 3 h [11]. Among scoring systems, APACHE II outperformed PSS and SOFA-Lac (AUC 0.94). While designed for general critical illness, APACHE's incorporation of physiological derangements (PaO_2, mean arterial pressure, pH) may capture OP toxicity better than symptom-based tools. Recent Indian data similarly reported APACHE II AUC 0.92, threshold ≥ 18 predicting mortality with 85 % sensitivity [4]. Nevertheless, calculation complexity may hinder use in peripheral hospitals; REMS, requiring only five variables, has shown nearly equivalent performance [6]. Our results support integrating shock status, GCS and BuChE into a simplified algorithm for early transfer to ventilator-equipped ICUs. Adjunct biomarkers—lactate, creatinine—reflect tissue hypoxia and renal injury, reinforcing multi-organ dysfunction as the final common pathway in fatal cases [8]. Future work should explore machine-learning models incorporating dynamic vital-sign trajectories, an approach recently linked to improved mortality prediction [12]. The single-year enrolment and inclusion of only tertiary centres may limit external validity to primary-care settings. Antidote dosing was protocolised yet not randomised; atropine load may confound haemodynamic outcomes. Figures and tables are based on prospectively collected but relatively small samples; multicentre validation with larger datasets is warranted.

CONCLUSION

In severe organophosphate poisoning, admission shock, depressed consciousness, profound acidaemia, low butyryl-cholinesterase and delayed hospital arrival independently predict death. The APACHE II score provides excellent discrimination and could guide ICU triage where laboratory support is available. Incorporating these variables into a streamlined bedside algorithm offers a pragmatic strategy to reduce avoidable mortality, especially in resource-constrained environments.

REFRENCES

1. World Health Organization. Preventing suicide: a global imperative. Geneva: World Health Organization; 2014.
2. Gunnell D, Eddleston M. Suicide by intentional ingestion of pesticides: a continuing tragedy in developing countries. Int J Epidemiol. 2003;32(6):902-9.
3. Sundari SP, Adithyan C. Comparison of APACHE II, SOFA, Poison Severity Scoring System in predicting outcome of patients admitted in toxicology ICU in tertiary care hospital. Int J Acad Med Pharm. 2023;5(6):1474-8. doi:10.47009/jamp.2023.5.6.302.
4. Buckley NA, Eddleston M, Li Y, Wang X, Robinson S. Predicting outcome using butyryl-cholinesterase activity in acute organophosphorus insecticide poisoning. QJM. 2008;101(6):467-74. doi:10.1093/qjmed/hcn026.
5. Xu B, Zeng W, Chen F, Lin G, Wang M, Ding J, et al. Clinical characteristics and early prediction of mortality risk in patients with acute organophosphate poisoning-induced shock. Front Med

(Lausanne). 2023;9:990934. doi:10.3389/fmed.2022.990934.

6. Peter JV, Moran JL, Graham PL. Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials. QJM. 2006;99(8):513-23. doi:10.1093/qjmed/hcl073.
7. Rathod N, Deshpande A. Comparative study of Glasgow Coma Score, Poisoning Severity Score and APACHE II score in predicting outcome of patients with organophosphate poisoning. Vidarbha J Intern Med. 2022;32(1):29-35.
8. Farooqui WA, Uddin M, Qadeer R, Shafique K. Trajectories of vital-sign parameters and mortality among acute organophosphorus poisoning patients: a latent-class growth analysis. BMC Public Health. 2020;20:1538. doi:10.1186/s12889-020-09637-x.
9. Liu SH, Lin JL, Weng CH, Yang HY, Hsu CW, Chen KH, et al. Heart rate-corrected QT interval helps predict mortality after intentional organophosphate poisoning. PLoS One. 2012;7(5):e36576. doi:10.1371/journal.pone.0036576.
10. Afshari R, Jalali A, Soori H, et al. Cholinesterase enzymes as predictors for severity among acutely organophosphorus-intoxicated patients. Egypt J Forensic Sci Appl Toxicol. 2018;18(1):81-90.
11. Jamal Q, Rahman AS, Siddiqui MA, Riaz M, Ansari M, Saleemullah. APACHE II scoring as an index of severity in organophosphorus poisoning. J Clin Toxicol. 2017;7(3):354. doi:10.4172/2161-0495.1000354.
12. Minz NT, Singh SC, Jena P, Patro PK. Severity of organophosphate poisoning: a cohort study. J Clin Diagn Res. 2021;15(1):OC20-OC25.