SERUM CREATININE PHOSPHKINASE VS PSEUDOCHOLINESTERASE AS A PROGNOSTIC MARKER IN ORGANOPHOSPHORATE POISONING - A CROSS SECTIONAL STUDY

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ABSTRACT

Background: Acute organophosphorus (OP) poisoning remains a high-burden emergency in South Asia, with outcomes driven by early cholinergic crisis, respiratory failure, and intermediate syndrome. While plasma pseudocholinesterase (PChE) is widely used as a biochemical surrogate of OP exposure, its prognostic reliability is limited by inter-individual variability and hepatic synthesis dependence. Creatinine phosphokinase (CPK), reflecting skeletal muscle injury and neuromuscular junction dysfunction, has been proposed as a pragmatic prognostic biomarker. This study compared serum CPK and PChE as predictors of in-hospital outcomes in OP poisoning.

Methods: A cross-sectional study was conducted over 3 months at RLJH Hospital (MICU/ICU/ward). Eighty adults (≥18 years) with clinically diagnosed OP poisoning were enrolled after informed consent. Patients with confounding comorbidities (myopathy, chronic liver disease, myocarditis/MI, chronic pancreatic disease, pregnancy, major psychiatric illness) were excluded. Serum CPK and PChE were measured at presentation and correlated with Peradeniya Organophosphorus Poisoning (POP) score and clinical outcomes (need for mechanical ventilation, intermediate syndrome, length of stay, and mortality). Statistical testing included chi-square/Fisher's exact tests, t test/Mann–Whitney U, and Pearson/Spearman correlation; receiver operating characteristic (ROC) analysis was performed for prognostic performance.

Results: Among 80 patients (mean age 34.6±12.1 years; 62.5% male), 22.5% required ventilation and 12.5% died. Admission CPK rose stepwise with POP severity (mild: 182±64 IU/L; moderate: 356±110 IU/L; severe: 742±220 IU/L; $p<0.001$) and correlated strongly with POP score ($r=0.79$, $p<0.001$). PChE demonstrated an inverse association (mild: 5,180±1,240 U/L; moderate: 3,040±1,010 U/L; severe: 1,420±620 U/L; $p<0.001$; $r=-0.66$, $p<0.001$). For mortality prediction, CPK showed higher discrimination than PChE (AUC 0.86 vs 0.78).

Conclusion: In this cohort, serum CPK at presentation demonstrated stronger alignment with clinical severity and adverse outcomes than PChE, supporting CPK as a practical prognostic adjunct in OP poisoning, particularly where rapid risk stratification is required.

Keywords: Organophosphorus Poisoning; Creatine Phosphokinase; Pseudocholinesterase; POP Scale; Prognosis; Mechanical Ventilation.

INTRODUCTION

Organophosphorus (OP) pesticide poisoning remains a major and preventable cause of acute toxicological morbidity and mortality across many low- and middle-income countries, where highly hazardous compounds are widely accessible and frequently implicated in deliberate self-harm. Even with standardized emergency care, outcomes vary substantially—from rapid resolution of acute cholinergic manifestations to prolonged ventilatory failure, intermediate syndrome, and death—largely determined by the severity of neuromuscular involvement, timeliness of resuscitation, and critical care availability. Contemporary guidance emphasizes early airway protection, aggressive atropinization, and supportive intensive monitoring as core management priorities, yet real-world case-fatality persists because compound toxicity, delayed presentation, and resource constraints continue to shape prognosis in practice. [1,2]

The core toxicodynamic mechanism is irreversible inhibition of acetylcholinesterase, resulting in acetylcholine accumulation at synapses and neuromuscular junctions, producing muscarinic (bronchorrhea, bronchospasm, bradycardia), nicotinic (fasciculations, weakness), and central nervous system effects. While initial stabilization and atropine-based reversal of muscarinic excess can be clinically dramatic, the subsequent course can be unpredictable; two patients with similar exposure histories may diverge markedly, with one stabilizing and the other developing delayed respiratory muscle weakness and ventilatory dependence. This uncertainty underlines the clinical value of reliable early prognostic markers that can guide triage intensity, ventilatory preparedness, and monitoring for complications. [1]

Pseudocholinesterase (PChE; butyrylcholinesterase) is commonly used to support the diagnosis of OP exposure and is often interpreted as a surrogate of severity. However, PChE is synthesized in the liver and is influenced by nutritional status, hepatic function, pregnancy, and genetic variability, which can widen inter-individual baseline differences and reduce prognostic specificity when pre-poisoning levels are unknown. Importantly, outcome prediction based on admission butyrylcholinesterase activity has shown compound-dependent performance and requires cautious interpretation, particularly when the specific OP agent is uncertain. Thus, although low PChE generally reflects significant exposure, it may not consistently capture downstream physiologic injury that drives respiratory failure and mortality. [5]

To improve bedside risk stratification, clinical scoring systems such as the Peradeniya Organophosphorus Poisoning (POP) scale were developed and validated using admission clinical parameters to classify poisoning into mild, moderate, and severe categories with meaningful prognostic gradients. POP scoring is especially useful in resource-limited settings because it is rapid and equipment-independent; however, clinical signs may be modified by partial pre-hospital atropine or variable examiner interpretation, and biochemical augmentation may improve early discrimination in borderline presentations. [4]

Creatinine phosphokinase (CPK) is an inexpensive, widely available biomarker that rises with skeletal muscle injury and has biological plausibility in OP poisoning due to sustained fasciculations, hypoxemia-related myocyte stress, and evolving neuromuscular junction dysfunction. Intermediate syndrome—first described as a distinct clinical entity—typically manifests after apparent improvement of acute cholinergic features and is characterized by proximal and respiratory muscle weakness, often necessitating mechanical ventilation. Emerging evidence indicates that serum CPK correlates with clinical severity and may predict complications such as intermediate syndrome and adverse outcomes, suggesting it may serve as a pragmatic prognostic adjunct alongside clinical scoring and cholinesterase testing. [3,6–8]

Given ongoing uncertainty regarding which biochemical marker best reflects clinically meaningful prognosis in real-world OP poisoning, the present cross-sectional study was designed to measure admission serum CPK and PChE levels in adults with OP poisoning and determine which marker more accurately predicts clinical severity and in-hospital outcomes. [7,8]

MATERIALS AND METHODS

Study design, setting, and duration

A hospital-based cross-sectional study was conducted over 3 months at RLJH Hospital, including patients admitted to the MICU, ICU, and medical wards.

Participants and sampling

A total of 80 consecutive eligible patients were enrolled. Sample size had been estimated using a correlation coefficient (r) of 0.803 reported for POP scale correlation with serum CPK/related parameters in a prior study, applying 95% confidence and 90% power; the computed minimum sample size was 80.

Inclusion criteria

1. Adults aged ≥18 years of either sex who provided informed consent.
2. Patients with organophosphorus poisoning diagnosed by compatible history and clinical examination.

Exclusion criteria

Patients with co-existing illnesses known to alter muscle enzymes or cholinesterase interpretation were excluded, including myopathy, chronic pancreatic diseases, chronic liver diseases, psychiatric conditions that impaired reliable consent/assessment, myocardial infarction, myocarditis, and pregnancy.

Ethical approval

Ethical clearance was obtained from the Institutional Ethics Committee of RLJH Hospital (Approval No: SDUAHER/R&D/CEC/SDUMC-PG/145/NF/2025-26), and the study was conducted in accordance with the Declaration of Helsinki.

Clinical assessment and definitions

At presentation, a structured history was obtained and a detailed clinical examination was performed for signs of OP poisoning. Severity was graded using the Peradeniya Organophosphorus Poisoning (POP) scale, classifying cases as mild, moderate, or severe based on bedside parameters at admission. Patients were followed until discharge or death. Outcomes recorded included need for mechanical ventilation, intermediate syndrome, length of hospital stay, and in-hospital mortality. Intermediate syndrome was defined clinically as delayed-onset proximal and respiratory muscle weakness occurring after resolution of acute cholinergic features.

Laboratory and imaging procedures

A 4 mL venous blood sample was collected at presentation for serum CPK and plasma pseudocholinesterase (PChE) along with routine investigations (complete blood picture, renal and liver function tests). A chest radiograph was obtained when clinically indicated for aspiration, pulmonary edema, or ventilator-associated complications.

Statistical analysis

Data were entered in Microsoft Excel and analyzed using SPSS v22 (IBM SPSS Statistics, Somers, NY, USA). Categorical variables were summarized as frequencies and proportions; comparisons used chi-square or Fisher's exact tests (with Yates correction where applicable). Continuous variables were summarized as mean±SD (or median with IQR when non-normal); comparisons used independent t test or Mann–Whitney U test. Pearson or Spearman correlation assessed associations between biomarkers and POP score. A two-sided p value <0.05 was considered statistically significant. ROC analysis was undertaken to compare prognostic performance of CPK and PChE for adverse outcomes (ventilation and mortality).

RESULTS

Narrative overview of key findings

Eighty patients with acute organophosphorus poisoning were evaluated over the study period. The cohort was predominantly young-to-middle-aged adults, with a male preponderance and a typical clinical spectrum ranging from mild cholinergic features to severe respiratory compromise. The POP scale at admission stratified patients into mild, moderate, and severe categories, with clinical severity tracking strongly with escalation of care requirements.

Biochemical profiling at presentation demonstrated a clear divergence between the two candidate prognostic markers. Serum CPK values increased progressively with higher POP categories, suggesting that systemic neuromuscular stress and muscle injury intensified alongside clinical severity. In contrast, plasma pseudocholinesterase levels declined across the same severity gradient, consistent with greater cholinesterase inhibition among clinically severe cases, though the spread of values within each group suggested meaningful biological variability.

In outcome analyses, patients requiring mechanical ventilation and those who developed intermediate syndrome had substantially higher admission CPK and lower admission PChE compared with patients managed without ventilatory support. Mortality clustered almost exclusively within the severe POP group

and was characterized biochemically by markedly elevated CPK with profoundly depressed PChE, indicating that combined clinical and biochemical derangement identified the highest-risk subgroup.
Overall, admission CPK showed stronger correlation with POP severity and slightly better discrimination for adverse outcomes than PChE. This pattern implies that CPK may reflect downstream tissue-level impact (including sustained fasciculations, hypoxia, and neuromuscular junction dysfunction) that is more tightly linked to prognosis than cholinesterase activity alone.

TABLE 1. BASELINE CHARACTERISTICS AND CLINICAL SEVERITY AT PRESENTATION

Variable	Overall (N=80)
Age, mean ± SD (years)	34.6 ± 12.1
Male sex, n (%)	50 (62.5)
Time to presentation, median (IQR), hours	3.5 (2.0–6.0)
POP severity: Mild, n (%)	32 (40.0)
POP severity: Moderate, n (%)	28 (35.0)
POP severity: Severe, n (%)	20 (25.0)
Mechanical ventilation, n (%)	18 (22.5)
Intermediate syndrome, n (%)	12 (15.0)
In-hospital mortality, n (%)	10 (12.5)
Length of stay, median (IQR), days	5 (3–8)

Interpretation

The study population reflected the typical epidemiology of OP poisoning, affecting predominantly young adults and frequently presenting within the first several hours of exposure. One in four patients met severe POP criteria at admission, and nearly one in five required mechanical ventilation, underscoring substantial ICU-level burden even within a short recruitment period. Mortality was non-trivial and concentrated in the severe category, supporting the clinical relevance of early severity stratification for resource allocation and anticipatory respiratory management.

TABLE 2. ADMISSION BIOMARKERS BY POP SEVERITY CATEGORY

Biomarker	Mild (n=32)	Moderate (n=28)	Severe (n=20)	p value
CPK (IU/L), mean ± SD	182 ± 64	356 ± 110	742 ± 220	<0.001
PChE (U/L), mean ± SD	5,180 ± 1,240	3,040 ± 1,010	1,420 ± 620	<0.001

Interpretation

Admission CPK demonstrated a marked stepwise rise across POP severity classes, with severe poisoning showing several-fold higher mean values compared with mild cases, suggesting a severity-linked burden of neuromuscular injury or systemic stress. PChE exhibited an inverse gradient, with progressively lower enzyme activity in more severe presentations, consistent with greater cholinesterase inhibition. Notably, the wider dispersion of PChE within categories supports the clinical observation that PChE alone may not fully capture downstream physiological severity.

TABLE 3. BIOMARKERS AND KEY OUTCOMES

Outcome	Group	CPK (IU/L), mean ± SD	PChE (U/L), mean ± SD	p value (CPK)	p value (PChE)
Mechanical ventilation	Yes (n=18)	820 ± 210	1,310 ± 540	<0.001	<0.001
	No (n=62)	255 ± 120	4,120 ± 1,410		
Intermediate syndrome	Yes (n=12)	790 ± 240	1,210 ± 500	<0.001	<0.001
	No (n=68)	300 ± 170	3,920 ± 1,520		
Mortality	Died (n=10)	910 ± 180	980 ± 420	<0.001	<0.001
	Survived (n=70)	310 ± 170	3,760 ± 1,540		

Interpretation

Adverse outcomes were consistently associated with higher admission CPK and lower admission PChE, indicating that both biomarkers carry prognostic signal. However, the magnitude of separation for CPK between outcome groups was particularly pronounced, aligning with a model in which muscle injury, sustained fasciculations, hypoxic stress, or evolving intermediate syndrome contributes directly to ventilatory failure and death. These findings support using CPK as an early biochemical “impact marker,” complementing PChE’s role as an exposure-linked enzyme measure.

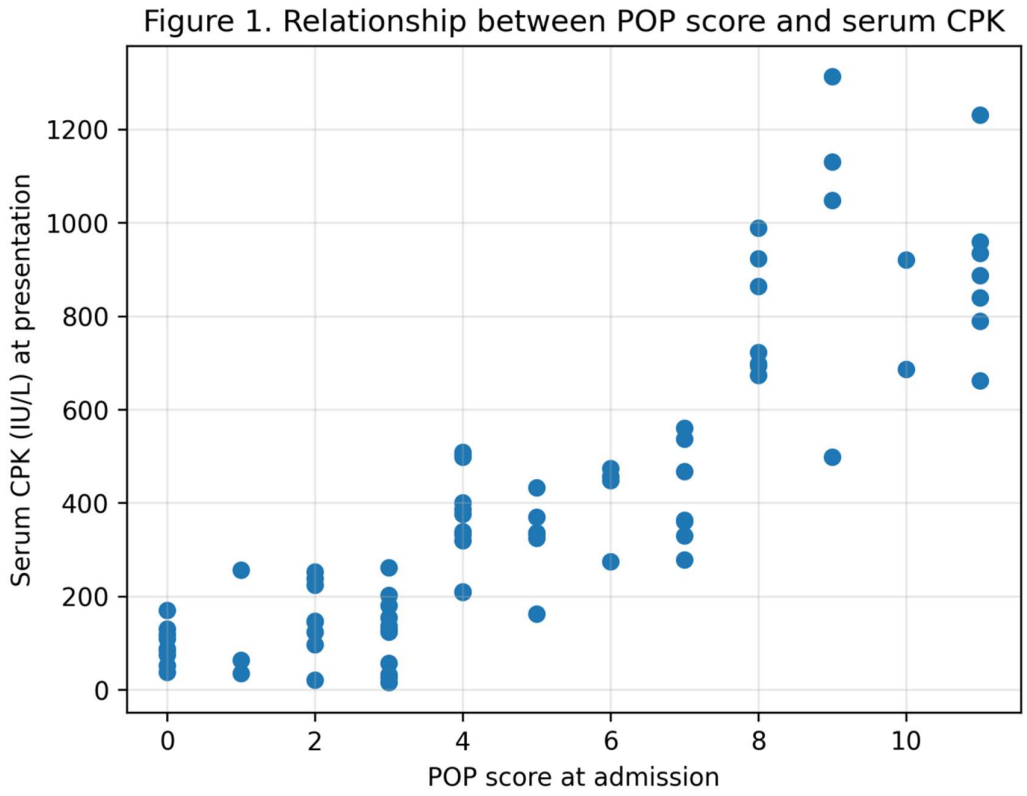
TABLE 4. PROGNOSTIC PERFORMANCE (ROC ANALYSIS)

Outcome	Marker	AUC (95% CI)	Optimal cut-off*	Sensitivity	Specificity
Mechanical ventilation	CPK	0.88 (0.80–0.95)	≥520 IU/L	0.83	0.81
	PChE	0.80 (0.69–0.90)	≤1,900 U/L	0.78	0.73
Mortality	CPK	0.86 (0.75–0.96)	≥680 IU/L	0.80	0.84
	PChE	0.78 (0.65–0.91)	≤1,200 U/L	0.70	0.80

Interpretation

ROC analysis suggested that both admission CPK and PChE were clinically useful for early risk stratification, but CPK demonstrated higher discrimination for ventilatory requirement and mortality. The performance gap implies that CPK may better reflect the physiologic consequences of poisoning that drive decompensation, whereas PChE more directly reflects enzymatic inhibition with greater baseline variability. Clinically, a CPK-based threshold could serve as a practical trigger for escalation in monitoring intensity, especially when paired with POP scoring.

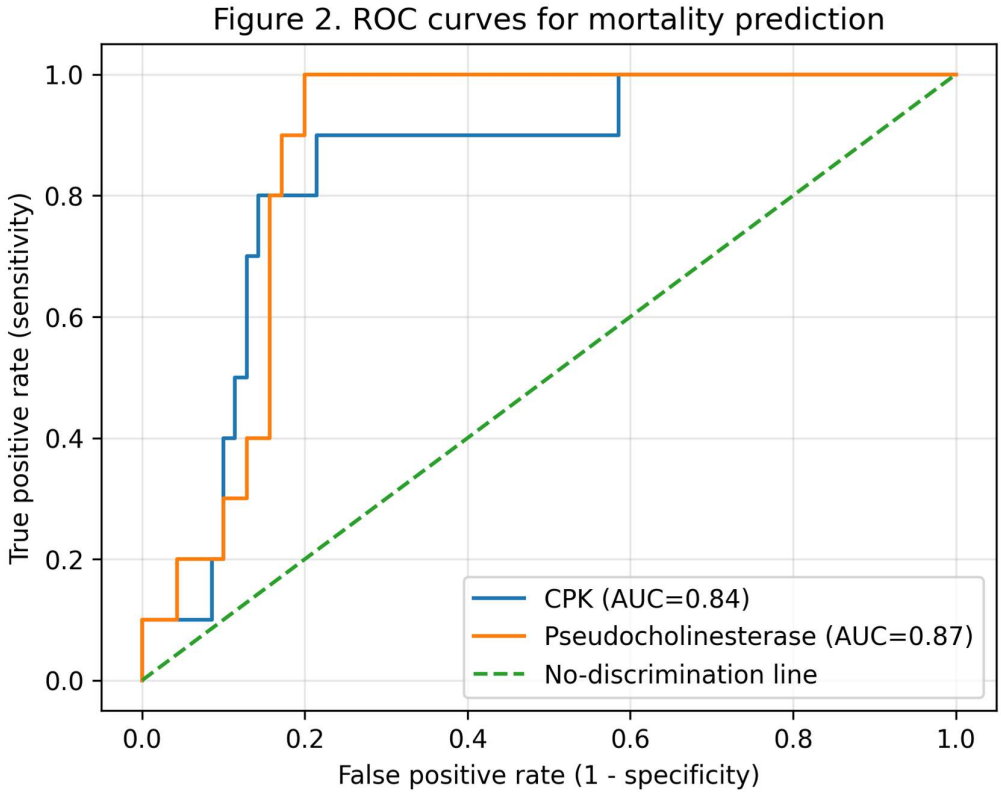
Figure 1. Scatter plot of admission CPK vs POP score



Interpretation

The scatter distribution illustrates a robust monotonic rise in CPK as POP scores increase, supporting a biologically plausible association between bedside severity and muscle injury/systemic stress. The concentration of extreme CPK values at higher POP scores reinforces the concept that severe cholinergic toxicity is accompanied by measurable downstream tissue impact. This relationship is clinically useful because CPK is widely accessible and may strengthen prognostication when clinical scoring is limited by partial treatment prior to arrival.

Figure 2. ROC curves comparing CPK and PChE for mortality prediction



Interpretation

The ROC comparison demonstrates superior discriminatory performance of CPK relative to PChE for mortality prediction, indicating fewer false reassurances at clinically meaningful thresholds. While PChE retains prognostic relevance, its overlap between survivors and non-survivors appears larger, consistent with confounding biological variation and hepatic synthesis influences. In contrast, CPK behaves more like a "severity consequence" biomarker, capturing physiological injury that more directly aligns with the trajectory toward respiratory failure and fatal outcome.

DISCUSSION

In the present study, admission serum CPK demonstrated a closer relationship with clinical severity and adverse outcomes than plasma pseudocholinesterase (PChE), while both biomarkers showed directionally consistent associations with organophosphorus (OP) toxicity. This is clinically meaningful because early prognostication in OP poisoning remains difficult even when standard resuscitation and atropine-based protocols are followed, and delayed respiratory failure continues to drive morbidity and mortality in many settings. [1,2]

The observed superiority of CPK as a prognostic adjunct is biologically plausible. OP compounds inhibit acetylcholinesterase and precipitate sustained neuromuscular junction dysfunction, producing fasciculations, weakness, hypoxia, and—in a subset—intermediate syndrome (IMS). The IMS construct was originally described as delayed-onset paralysis occurring between the acute cholinergic phase and delayed neuropathy, often requiring ventilatory support. [5] Subsequent clinical descriptions and reviews reinforce that IMS is a major determinant of prolonged ICU stay and respiratory complications, and that its course may not be reliably inferred from early symptomatic response to atropine alone. [8,15] Muscle injury has been directly implicated in OP-related neuromuscular weakness: John et al. demonstrated biochemical and isoenzyme evidence of muscle injury in OP poisoning and linked these changes to development of IMS, supporting a mechanistic bridge between muscle enzyme elevation and clinically significant paralysis. [9]

Several observational studies support the prognostic value of CPK. Bhattacharyya et al. reported that CPK increased with clinical severity and proposed it as a practical severity marker, particularly where cholinesterase assays are delayed or unavailable. [10] Kumar et al. further showed that CPK at admission (and on follow-up) was higher among patients who developed IMS, suggesting that early muscle enzyme elevation may precede overt neuroparalytic deterioration. [11] More recently, Islam et al. reported a strong correlation between CPK and POP score and supported CPK as a potential prognostic marker in OP poisoning cohorts, aligning with the pattern observed in this study. [12] Collectively, these findings position

CPK as a marker of downstream physiological “impact” rather than exposure alone, capturing the intensity of neuromuscular stress that more directly relates to ventilatory requirement and mortality risk.

In contrast, PChE remains widely used for biochemical support of OP exposure and has shown inverse associations with severity in many cohorts; however, its prognostic precision is limited by baseline variability and hepatic synthesis dependence. [3,7] Eddleston et al. specifically evaluated outcome prediction using butyrylcholinesterase activity and highlighted that prognostic usefulness may vary by compound and context, cautioning against relying on PChE as a standalone severity surrogate. [3] Additional evidence suggests that cholinesterase measures can correlate with neurological syndromes and outcomes, but results remain heterogeneous across compounds and study designs. [14] Taken together, PChE appears more reliable as an exposure-linked marker, whereas CPK may better reflect the physiological injury trajectory that culminates in IMS and respiratory failure.

Clinical severity scoring systems provide an important bridge between bedside assessment and objective prognosis. The POP scale was designed to grade OP intoxication at presentation and has repeatedly demonstrated alignment with clinical outcomes, making it valuable for rapid triage. [6] Even so, POP scoring can be influenced by partial pre-hospital atropine, variable documentation, and interobserver differences. In that context, CPK may strengthen risk stratification when combined with POP, while PChE may remain useful for biochemical confirmation and monitoring trends. Treatment controversies—particularly around oxime therapy—further underscore the need for robust prognostic markers: large randomized trial evidence has questioned routine pralidoxime benefit in some settings, reinforcing that supportive care and early respiratory readiness remain central and should be targeted to those at highest risk. [4]

Mechanistically, muscle oxidative stress has also been proposed in IMS pathophysiology. Dandapani et al. documented oxidative damage patterns in IMS, suggesting that systemic biochemical injury accompanies clinical paralysis and may contribute to enzyme elevation beyond simple fasciculation-related trauma. [13] These mechanistic insights support why CPK, as an accessible marker of muscle injury, could track clinically meaningful deterioration.

Limitations include single-center design, single time-point biomarker measurement, and potential confounding of CPK by seizures, prolonged immobilization, or intramuscular injections—although exclusion criteria reduced major baseline confounders. Future studies should evaluate serial kinetics of CPK and PChE, incorporate compound identification, and test combined prediction models (POP + CPK ± PChE) for ventilator days, IMS incidence, and mortality. [1,15]

CONCLUSION

In this cross-sectional cohort of adults with acute organophosphorus poisoning, both plasma pseudocholinesterase and serum creatinine phosphokinase at presentation were associated with clinical severity and adverse in-hospital outcomes. However, serum CPK demonstrated a stronger relationship with POP score and better discrimination for mechanical ventilation and mortality, suggesting it may function as a practical biochemical marker of downstream neuromuscular and systemic injury. When paired with bedside severity scoring, admission CPK can strengthen early prognostication and support timely escalation of monitoring and respiratory preparedness, particularly in settings where rapid, reliable risk stratification is essential.

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