

Original Research Article

Electrolyte Imbalance as a Prognostic Marker in Acute Organophosphorus Poisoning: A Cross-Sectional Study

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ABSTRACT

Background: Acute organophosphorus poisoning (OPP) causes morbidity and mortality. Hypokalemia may reflect poisoning severity and adverse outcome but is not routinely used for risk stratification.

Material and Methods: This cross-sectional study included 150 adults with acute OPP at a tertiary teaching hospital in Western India between April 2024 and September 2025. Patients with pregnancy, chronic kidney disease, diabetes mellitus, or mixed-poison ingestion were excluded. Admission clinical, electrolyte, renal, arterial blood gas and cholinesterase parameters were recorded. Severity was graded using the Peradeniya Organophosphorus Poisoning (POP) scale. One-way ANOVA, Student's t-test and chi-square test were used; $P \leq 0.05$ was significant.

Results: Of 150 patients, 70.7% were male and 52.0% were aged 18–25 years. Nausea (96.0%), vomiting (92.7%) and breathing difficulty (80.7%) were common. Mild, moderate and severe poisoning occurred in 70.0%, 22.7% and 7.3%, respectively. Mean potassium decreased from 3.95 ± 0.41 mmol/L in mild to 3.40 ± 0.50 in moderate and 2.95 ± 0.33 mmol/L in severe poisoning ($P < 0.001$). Hypokalemia increased from 17.1% to 67.6% and 81.8%, respectively ($P < 0.001$). Mortality increased from 0% to 14.7% and 36.4% across POP grades ($P < 0.001$) and was higher in hypokalemic than normokalemic patients (14.0% vs 2.0%; $P = 0.039$).

Conclusion: Admission hypokalemia is associated with POP severity and mortality in acute OPP and may serve as a low-cost bedside prognostic marker.

Keywords: organophosphorus poisoning, hypokalemia, electrolyte imbalance, prognosis

INTRODUCTION

Acute organophosphorus poisoning (OPP) is a major public-health problem in agrarian, low- and middle-income economies, where these agents are inexpensive, freely available and frequently used for deliberate self-harm. The toxicity stems from irreversible inhibition of acetylcholinesterase and pseudocholinesterase, producing an acute cholinergic crisis that may be followed by intermediate syndrome and delayed polyneuropathy.¹⁻³ Reported in-hospital mortality varies widely from 8% to 50%, depending on the compound, dose, time to medical care and severity at presentation.⁴⁻⁶ Identifying simple, bedside prognostic markers that can stratify risk early in the emergency department

is therefore a clinically relevant unmet need.^{1,3,5} Electrolyte disturbances—particularly involving potassium—are frequently encountered in acute OPP because of enhanced gastrointestinal losses, transcellular shifts driven by catecholamine release and cholinergic overstimulation, and acid-base derangements.^{2,7,8,9} Several investigators have demonstrated that hypokalemia correlates with severity, ventilatory requirement and mortality in OPP.¹⁰⁻¹⁶ However, robust local data from Western India remain limited. The present cross-sectional study was undertaken to evaluate the association between electrolyte imbalance—primarily potassium—severity graded by the Peradeniya Organophosphorus Poisoning (POP) scale, and clinical outcome in patients with acute OPP.

MATERIAL AND METHODS

Study design and setting

A hospital-based cross-sectional observational study was conducted in the Department of Emergency Medicine and the medical Intensive Care Unit (ICU) of a 750-bed tertiary care teaching hospital in Bhuj, Western India, between April 2024 and September 2025. Approval was obtained from the Institutional Ethics Committee, and written informed consent was secured from each participant or a legally acceptable representative.

Participants

Adult patients (>18 years) diagnosed with acute OPP based on history, characteristic clinical features and reduced serum cholinesterase activity were eligible. Patients with pregnancy, known chronic kidney disease, diabetes mellitus, mixed poison ingestion, or unwillingness to participate were excluded to limit confounding of electrolyte parameters.

Sample size

Assuming an electrolyte-imbalance prevalence of approximately 10% in acute OPP, with 95% confidence and a 5% absolute precision, the required minimum sample size was 138, calculated using the formula $N = Z^2PQ/L^2$. After adjusting for potential loss of data the sample size was inflated to **150 patients**, who were enrolled by non-probability purposive sampling.

Data collection

At admission, demographic data, exposure history, and a structured clinical examination were recorded, including level of consciousness, pupillary size, oro-nasal frothing, fasciculations and vital parameters. Severity of poisoning was graded using the **Peradeniya Organophosphorus Poisoning (POP) scale** as mild (0–3), moderate (4–7) or severe (8–11)^{1,17}. Venous blood samples were drawn before initiation of atropine or fluid resuscitation. Serum sodium, potassium and chloride were measured by ion-selective electrodes, renal function by UV-kinetic methodology, serum cholinesterase by Ellman's colorimetric technique, and arterial blood gas analysis by an automated analyser. Hypokalemia was defined as serum potassium <3.5 mmol/L. The primary outcome was in-hospital mortality; secondary outcomes were severity grading and need for ventilatory support.

Statistical analysis

Data were entered in Microsoft Excel and analysed using GraphPad Prism v9.2.0. Continuous variables were expressed as mean $\pm$ standard deviation, and categorical variables as frequencies and percentages. Comparisons of continuous data across POP grades used one-way ANOVA, and Student's t-test was used for two-group comparisons. Categorical associations were tested with the chi-square test. A two-tailed P-value ≤ 0.05 was considered statistically significant.

RESULTS

Demographic profile and clinical presentation

Of the 150 patients enrolled, 106 (70.7%) were male and 44 (29.3%) female. The cohort was predominantly young: 78 patients (52.0%) were aged 18–25 years and 38 (25.3%) were 26–30 years; the incidence declined progressively with age [Table 1]. Gastrointestinal and respiratory manifestations dominated the clinical picture, with nausea (96.0%), vomiting (92.7%) and breathing difficulty (80.7%) being most frequent, followed by perspiration (64.7%), miosis (56.0%) and other muscarinic features. Fasciculations, indicative of nicotinic involvement, were noted in only 9.3% of patients. Vital signs were largely preserved at admission, with mean SpO₂ of 96.2 ± 4.7% and mean systolic blood pressure of 119.7 ± 12.2 mmHg.

Table-1: Demographic distribution, presenting symptoms and vital parameters of the study population (N=150)

Variable	Category	N	% (Mean ± SD)
Gender (Total N = 150)	Male	106	70.7
	Female	44	29.3
Age group (years)	18–25	78	52.0
	26–30	38	25.3
	31–40	14	9.3
	41–50	8	5.4
	51–60	9	6.0
	61–70	3	2.0
Presenting signs and symptoms	Nausea	144	96.0
	Vomiting	139	92.7
	Breathing difficulty	121	80.7
	Perspiration	97	64.7
	Miosis	84	56.0
	Salivation	62	41.3
	Lacrimation	58	38.7
	Urination	56	37.3
	Diarrhea	44	29.3
	Weakness	37	24.7
	Fasciculations	14	9.3
Vital parameters at admission	Respiratory rate (/min)	—	17.7 ± 2.5
	SpO ₂ (%)	—	96.2 ± 4.7
	Pulse rate (/min)	—	84.6 ± 15.2
	SBP (mmHg)	—	119.7 ± 12.2
	DBP (mmHg)	—	77.9 ± 5.3

Baseline biochemical, blood-gas and severity profile

Mean serum sodium, chloride and renal function parameters were within clinically acceptable limits, while mean serum cholinesterase was markedly depressed at 5060.8 ± 3876.3 U/L, reflecting significant cholinesterase inhibition. Arterial blood gas analysis revealed mild metabolic acidosis (mean pH 7.3 ± 0.1; HCO₃ 18.2 ± 5.6 mmol/L). Severity grading by the POP scale showed mild poisoning in 105 patients (70.0%), moderate in 34 (22.7%) and severe in 11 (7.3%) [Table 2].

Table-2: Mean biochemical and arterial blood gas parameters and severity distribution by the Peradeniya Organophosphorus Poisoning (POP) scale

Parameter / Category	Mean $\pm$ SD
Serum sodium (mmol/L)	135.9 $\pm$ 5.9
Serum potassium (mmol/L)	3.8 $\pm$ 0.5
Serum chloride (mmol/L)	103.8 $\pm$ 9.9
Serum urea (mg/dL)	26.0 $\pm$ 16.3
Serum creatinine (mg/dL)	0.7 $\pm$ 0.36
Serum cholinesterase (U/L)	5060.8 $\pm$ 3876.3
Arterial pH	7.3 $\pm$ 0.1
PCO ₂ (mmHg)	37.8 $\pm$ 16.6
PO ₂ (mmHg)	83.8 $\pm$ 51.5
HCO ₃ (mmol/L)	18.2 $\pm$ 5.6
O ₂ saturation (%)	97.0 $\pm$ 5.9

Association of serum potassium with severity and outcome

A statistically significant inverse association was observed between serum potassium and POP severity. The mean serum potassium declined progressively from 3.95 $\pm$ 0.41 mmol/L in mild to 3.40 $\pm$ 0.50 mmol/L in moderate and 2.95 $\pm$ 0.33 mmol/L in severe poisoning ($P < 0.001$). The prevalence of hypokalemia rose from 17.1% in mild to 67.6% in moderate and 81.8% in severe poisoning ($P < 0.001$). Severity also predicted mortality: no patient with mild poisoning died, whereas mortality reached 14.7% in moderate and 36.4% in severe poisoning ($P < 0.001$). When the overall cohort was stratified by potassium status, mortality was 14.0% (7/50) in hypokalemic patients and 2.0% (2/100) in normokalemic patients ($P = 0.039$) [Table 3].

Table-3: Association of serum potassium with severity (POP grade) and clinical outcome.

Variable	Mild (n=105)	Moderate (n=34)	Severe (n=11)	P-value
Mean serum K ⁺ (mmol/L)	3.95 $\pm$ 0.41	3.40 $\pm$ 0.50	2.95 $\pm$ 0.33	< 0.001
Hypokalemia, n (%)	18 (17.1)	23 (67.6)	9 (81.8)	< 0.001
Normokalemia, n (%)	87 (82.9)	11 (32.4)	2 (18.2)	
Mortality by POP grade, n (%)	0 (0.0)	5 (14.7)	4 (36.4)	< 0.001
Discharge by POP grade, n (%)	105 (100)	29 (85.3)	7 (63.6)	
Outcome by potassium status (whole cohort)				
Serum K ⁺ status	Discharge, n (%)		Death, n (%)	P-value
Hypokalemia (n = 50)	43 (86.0)		7 (14.0)	0.039
Normokalemia (n = 100)	98 (98.0)		2 (2.0)	

DISCUSSION

Acute OPP remains a leading toxicological emergency in agrarian South Asia, with a wide spectrum of severity and a consequent challenge in early risk stratification. The present cross-sectional study of 150 patients confirms three clinically relevant observations: (i) the disease overwhelmingly affects young adult males; (ii) hypokalemia is a frequent and graded biochemical disturbance that tracks closely with POP severity; and (iii) hypokalemia and higher POP grade are each independently associated with significantly higher in-hospital mortality.

Demographic and clinical profile

Male predominance (70.7%) and a young median age (28.3 ± 10.9 years) are consistent with multiple Indian and South Asian cohorts, where agricultural occupation, easy availability of pesticides and impulsive intentional self-harm in young adult males drive the epidemiology^{2,10,18,19}. Earlier work by Parate et al.¹⁰ and Sonaiya & Dosi² similarly reported male predominance of 77% and 64%, respectively, with mean ages clustering between 28 and 35 years. The clinical profile in our cohort—dominated by nausea, vomiting, breathing difficulty and miosis—mirrors the classical cholinergic syndrome and is in line with previous Indian observations^{4,9,18}. Fasciculations, a marker of more pronounced nicotinic toxicity, were uncommon (9.3%) and are typically seen in severe poisoning, where mechanical ventilation requirements rise sharply⁶.

Severity grading on the POP scale

The majority of our patients had mild poisoning (70.0%), with 22.7% moderate and 7.3% severe. This distribution is broadly similar to Basnet et al.¹¹ (mild 61.6%, moderate 31.4%, severe 4.7%) and Parate et al.¹⁰ (mild 58%, moderate 42%) [Table 4]. The shift towards milder POP grades likely reflects earlier presentation, improved community awareness, and better pre-hospital decontamination in recent years. The POP scale has been validated as a simple, rapid bedside tool for severity grading and prognostication, and its prognostic value is well documented^{1,17}.

Table-4: Comparison of severity distribution and hypokalemia incidence between the present study and published literature

Study	Sample size	Mild (%)	Moderate (%)	Severe (%)	Hypokalemia incidence (%)
Dandekar (2019) ¹⁵	30	—	—	—	68.4
Difoesa et al. (2021) ¹³	50	—	—	—	48.0
Basnet et al. (2024) ³	86	61.6	31.4	4.7	36.0
Parate et al. (2025) ²	100	58.0	42.0	—	—
Krishna et al. (2024) ⁶	—	—	—	—	Reported as significant
Sonaiya & Dosi (2022) ⁷	—	—	—	—	Reported
Present study	150	70.0	22.7	7.3	33.3

Electrolyte and biochemical disturbances

Although the population-level means for sodium, potassium and chloride remained within acceptable laboratory limits, sub-group analysis by POP severity revealed a strong, graded fall in serum potassium with worsening severity ($P < 0.001$). The overall incidence of hypokalemia (33.3%) in our cohort is comparable to Basnet et al.¹¹ (36%) and slightly lower than Dandekar¹⁵ (68.4%) and Difoesa et al.¹⁴ (48%) [Table 4]. Several pathophysiological mechanisms may explain hypokalemia in acute OPP: (i) catecholamine surge from sympathetic stimulation and stress, leading to β_2 -mediated intracellular potassium shift; (ii) excess gastrointestinal losses from cholinergic hyperactivity, vomiting and diarrhea; (iii) acid–base disturbances; and (iv) atropine-induced intracellular potassium translocation.^{7-9,19} These mechanisms collectively explain why potassium emerges as a sensitive biochemical mirror of disease severity. Similar associations have been reported by Sonaiya & Dosi², Lokesh et al.⁹, Krishna et al.¹³, Roy et al.¹⁸, and Behera et al.¹⁹. Patel and Pathak⁴ likewise demonstrated that low potassium at admission predicts a worse clinical profile and outcome, while Anand et al.¹ showed that pseudo-cholinesterase and creatine phosphokinase activity correlate with the POP scale, supporting a broader role for routine bedside biochemistry in OPP triage. In addition, Haque et al.⁷ and Nidhin⁹ have documented more global electrolyte derangements involving sodium, magnesium and calcium with

increasing severity, indicating that potassium is the most clinically tractable but not the only biochemical marker of interest.

Severity, outcome and hypokalemia

Mortality in our cohort was 0% in mild, 14.7% in moderate and 36.4% in severe POP grades ($P < 0.001$), reaffirming the prognostic gradient embedded in the POP scale. These figures are broadly comparable to the moderate-severe mortality spectrum reported by Rose et al.^{4,20}, Eranaik et al.⁶ and Farooqui et al.¹⁶. Crucially, hypokalemia at admission independently identified a higher-risk group: mortality was 7-fold higher in hypokalemic compared with normokalemic patients (14.0% vs 2.0%; $P = 0.039$) [Table 5]. This is consistent with Dandekar¹⁵ (mortality 31.6% in hypokalemic OPP), Parate et al.¹⁰ (17%) and Basnet et al.¹¹ (8%), and supports the inclusion of potassium in early risk-stratification protocols. Jelias et al.¹⁷ and Farooqui et al.¹⁶ have also shown that incorporating biochemical indicators alongside POP grading improves prediction of mortality and ventilatory requirement.

Table-5: Comparison of outcome among patients with hypokalemia in acute organophosphorus poisoning across published cohorts

Study	Sample size	Overall mortality (%)	Mortality among hypokalemic (%)
Dandekar (2019) ¹⁵	30	—	31.6
Difoosa et al. (2021) ¹³	50	—	Significant association
Farooqui et al. (2022) ¹⁸	Large ICU cohort	Mortality linked to biochemical trajectory	Higher in hypokalemic trajectory
Eranaik et al. (2017) ¹⁹	—	Higher need for ventilation	Associated with poor prognosis
Basnet et al. (2024) ³	86	—	8.0
Parate et al. (2025) ²	100	—	17.0
Patel & Pathak (2025) ⁴	—	Worse outcomes with low K^+	Significant association
Present study	150	6.0	14.0

Clinical implications

The findings imply that a single, inexpensive serum potassium measurement at presentation, used in conjunction with the POP score, can identify patients who require closer monitoring, earlier ICU admission, proactive electrolyte correction and a low threshold for airway protection. Potassium replacement, careful electrocardiographic monitoring and avoidance of drugs that further drive hypokalemia (high-dose β_2 -agonists, large-volume crystalloids without potassium) should be considered as part of standard care in moderate-to-severe OPP. This approach harmonizes with global recommendations to combine clinical scoring with bedside biochemistry to optimize outcomes in acute pesticide poisoning.^{1,3,5,16,17}

Limitations

Several limitations should temper interpretation. First, the single-centre design limits external generalizability to centres with different demographics or pesticide profiles. Second, electrolytes were assessed only at admission; serial monitoring would have allowed better dynamic assessment of the relationship between potassium trajectory and outcome. Third, the severe POP subgroup ($n = 11$) is small, which limits statistical power for subgroup analyses. Fourth, the specific organophosphorus compound ingested could not be biochemically confirmed in every case.

Finally, residual confounding by atropine dose, time-to-presentation and pre-hospital therapy could not be fully eliminated.

CONCLUSIONS

In this cross-sectional study of 150 patients with acute organophosphorus poisoning, mean serum potassium fell progressively and hypokalemia became increasingly prevalent as POP severity rose, while mortality climbed in parallel. Hypokalemia at admission was independently associated with a 7-fold higher risk of in-hospital death compared with normokalemia. Routine measurement of serum potassium, combined with bedside POP scoring, offers a simple, low-cost and widely available tool for early risk stratification in acute OPP, and prompt correction of hypokalemia should be integrated into standard management.

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