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Clinical Profile and Outcomes of Acute Organophosphorus Poisoning Correlated with Serum Cholinesterase Levels And Peradeniya Organophosphorus Poisoning Score: A Cross-Sectional Observational Study At A Tertiary Care Hospital

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ABSTRACT

Background: Acute organophosphorus (OP) compound poisoning continues to be an important public health emergency in agricultural India accounting for a large proportion of poisoning deaths. The Peradeniya Organophosphorus Poisoning (POP) score and serum pseudocholinesterase (PChE) are the two indices developed to assess severity of OP poisoning but need to be systematically evaluated for combining the two and predicting the need for mechanical ventilation, intermediate syndrome and death. Methods: This is a cross-sectional observational study that recruited 180 adults with acute OP poisoning who were admitted to a tertiary care hospital during 18-month period. At admission, the POP score, the serum PChE, clinical features, treatment parameters (atropine dose, pralidoxime use), the need for mechanical ventilation, the presence of intermediate syndrome, the length of stay in the ICUs and in-hospital mortality were recorded. Results: The most prevalent compound was chlorpyrifos which accounted for 38.3%. Thirty-two.2% had severe POP score (≥8). Intermediate (14.4%) and mechanical ventilation (36.1%) required and in hospital mortality (9.4%). A negative correlation was shown between serum PChE and severity of POPs ($r=-0.62$; $p<0.001$). Independent predictors of mortality were delayed presentation >6 hours, delayed PChE <400 U/L, mechanical ventilation, and POP score ≥8 (all aOR>2.0). Conclusion: The use of the combination of both the POP score and serum PChE at admission in acute OP poisoning allows for good severity stratification. Early intensive care, appropriate atropinisation and oxime therapy, where applicable, are the mainstay of management. There is an emergency need for policy-level regulation of highly toxic OP compounds.

KEYWORDS: Organophosphorus poisoning; POP score; serum pseudocholinesterase; cholinergic toxidrome; intermediate syndrome; atropine; mechanical ventilation; India.

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INTRODUCTION

Pesticide poisoning is a worldwide public health problem that has been estimated to cause 300,000 to 385,000 deaths each year, mostly in Asia and Africa [1]. The most common class of pesticide associated with poisoning deaths throughout the world is organophosphorus (OP) compounds, which inhibit acetylcholinesterase (AChE) causing the accumulation of acetylcholine at muscarinic and nicotinic synapses [2]. In India, OP poisoning cases make up around 30-40% of all pesticide poisoning cases in hospital-based series [3,4] and cases involving OP compounds are highly fatal due to easy availability, low cost, and highly toxic nature of OP class 1a or 1b compounds by WHO classification (monocrotophos, methyl parathion, dichlorvos).

The clinical syndrome of acute OP poisoning is characterised by cholinergic excess — muscarinic features (SLUDGE: salivation, lacrimation, urination, defaecation/diarrhoea, GI cramps, emesis; bronchospasm, bradycardia, miosis) and nicotinic features (tachycardia, hypertension, muscle fasciculations, weakness, paralysis). Central acetylcholinesterase inhibition causes central nervous system (CNS) features, such as anxiety, confusion, seizures, and coma [5]. Treatment involves high dose atropine to dry secretions, oximes – AChE reactivators (effective first 24–48 hrs of OP poisoning before enzyme ageing), and early ventilatory support for respiratory failure – the major cause of death in acute OP toxicity [6].

There are two validated clinical tools to assess the severity: (1) The Peradeniya Organophosphorus Poisoning (POP) score is a 12 point composite which incorporates miosis, bradycardia, fasciculations, level of consciousness, GCS and seizures, that stratifies patients into mild, moderate and severe poisoning (0–3, 4–7, and ≥8 respectively) [7]; and (2) Serum pseudocholinesterase (PChE; butyrylcholinesterase; plasma ChE) activity, which correlates with the degree of systemic OP exposure and enzyme inhibition, provides a quantitative biochemical severity marker [8]. The usefulness of both of these tools in predicting outcomes is in isolation, but their complementary use and the inverse relationship between the POP and PChE in the context of a South Indian hospital, has not been systematically characterised.

Knowing the clinical-toxicological profile, severity predictors and outcomes of OP poisoning in a tertiary care emergency hospital setting has direct implications for a triage protocol, allocation of resources in a tertiary hospital or public health advocacy for the restriction of its use of highly toxic OP compounds, which is a WHO approved policy and is variably regulated across Asian

countries, but not in India [9].

MATERIALS AND METHODS

2.1 Study Design and Population

This was a cross-sectional, observational study done in emergency department, medical ICU and general medical wards of a tertiary care hospital during 18 months. Inclusion: adults ≥18 years admitted with suspected acute OP poisoning with confirmed history of exposure by ingestion/inhale/dermal exposure with characteristic cholinergic symptoms, AND serum PChE <80% of the lower limit of normal (LLN) (range 3,100 – 6,500 U/L by colorimetric assay, Ellman method). Exclusion: carbamate poisoning (PChE will recover in hours, RBC AChE is normal), organochlorine or pyrethroid poisoning, unknown poison, incomplete records. Approval received from IEC, written informed consent (legal guardian consent). STROBE guidelines *followed.*

2.2 Clinical Assessment and POP Scoring

Structured proforma – age, sex, weight, manner of poisoning (ingestion/inhalation/dermal) intent (suicidal/accidental/occupational) specific OP compound, estimated dose, onset to hospital time (OT), pre-hospital decontamination, vital signs (HR, RR, BP, SpO2, temperature), GCS, AVPU, clinical features (SLUDGE symptoms scored, fasciculations, miosis, bronchospasm). Score obtained at the time of admission by the physician attending the child (standardised by the training workshop and scoring card). Serum PChE by Ellman's colorimetric assay (Randox Laboratories kit). RBC AChE where available (n=60 subset). Serial PChE at admission, 24h, 48h, and 72h. Atropine total dose to secretion dryness documented (mg); pralidoxime (2-PAM) use; need for endotracheal intubation and mechanical ventilation (MV), duration of MV; intermediate syndrome (IS) defined per Senanayake criteria (flaccid proximal limb weakness, neck flexor weakness, cranial nerve palsies developing 24–96 hours after acute crisis resolution); ICU LOS; total hospital LOS; in-hospital mortality.

2.3 Laboratory Investigations

Serum PChE (Ellman), ABG/VBG (respiratory failure defined as PaO2 <60 mmHg on room air or PaCO2 >50 mmHg), ECG (QTc, arrhythmias), CBC, RFT, electrolytes, LFT, blood glucose, urine toxicology screen (dipstick OP test; positive qualitative confirmatory OP immunoassay).

2.4 Statistical Analysis

SPSS v26. Descriptive statistics. POP severity categories (mild 0–3, moderate 4–7, severe ≥8) compared by Kruskal-Wallis/ANOVA and chi-square. Spearman correlation for POP score vs serum PChE. ROC analysis for PChE and POP score cut-offs for mortality and MV requirement. Multivariable binary logistic regression (outcome: in-hospital mortality); aOR (95% CI); $p<0.05$ significant.

RESULTS

3.1 Clinical and Toxicological Profile

180 patients enrolled (66.7% male; mean age 32.6 ± 12.4 years; mean weight 56.8 ± 9.2 kg). Intent: suicidal 72.2%, accidental (occupational/agricultural) 22.8%, unknown 5.0%. Compound distribution: chlorpyrifos 38.3%, monocrotophos 23.9%, dichlorvos 18.3%, phorate 6.7%, methyl parathion 5.0%, others 7.8%. Mean OHT was 4.8 hours (median 3.5 hours; IQR 2–7 hours); 37.2% arrived >6 hours post-exposure. Full clinical profile is presented in Table 1.

Table 1. Clinical and Toxicological Profile of Acute OP Poisoning Patients by POP Score Category (n=180)

Characteristic	Mild POP 0–3 (n=62)	Moderate POP 4–7 (n=60)	Severe POP ≥8 (n=58)	p-value
Age, years (mean ±SD)	30.4±11.8	33.2±12.6	34.4±12.8	0.24
Male sex, n (%)	42 (67.7%)	40 (66.7%)	38 (65.5%)	0.98
Suicidal intent, n (%)	44 (71.0%)	42 (70.0%)	44 (75.9%)	0.72
Chlorpyrifos ingestion, n (%)	30 (48.4%)	22 (36.7%)	17 (29.3%)	0.09
Monocrotophos ingestion, n (%)	6 (9.7%)	16 (26.7%)	21 (36.2%)	0.002
Mean OHT, hours (±SD)	3.2±1.8	4.8±2.6	6.6±3.4	<0.001
OHT >6 hours, n (%)	12 (19.4%)	22 (36.7%)	34 (58.6%)	<0.001
Serum PChE, U/L median (IQR)	2180 (1640–2840)	1140 (680–1760)	420 (210–680)	<0.001
GCS <12, n (%)	0 (0.0%)	18 (30.0%)	44 (75.9%)	<0.001
Miosis, n (%)	28 (45.2%)	48 (80.0%)	56 (96.6%)	<0.001
Bronchospasm/excess secretions	14 (22.6%)	40 (66.7%)	54 (93.1%)	<0.001
Mechanical ventilation, n (%)	2 (3.2%)	22 (36.7%)	41 (70.7%)	<0.001

3.2 POP Score Vs Serum PChE Correlation and Outcomes

There was a strong inverse correlation between POP score at admission and serum PChE (Spearman $r=-0.62$, $p<0.001$): as POP score increased, PChE decreased correspondingly. Intermediate syndrome occurred in 26/180 (14.4%), predominantly in the moderate (18.3%) and severe (22.4%) POP groups. Mechanical ventilation was required in 65/180 (36.1%), with mean ventilation duration 4.2 ± 2.8 days. In-hospital mortality was 17/180 (9.4%). Total atropine dose ranged from 4 mg (mild) to 480 mg (severe). Outcomes are presented in Table 2.

Table 2. Treatment Parameters and Outcomes by POP Score Category (n=180)

Outcome / Treatment	Mild POP 0–3 (n=62)	Moderate POP 4–7 (n=60)	Severe POP ≥8 (n=58)	p-value
Intermediate syndrome, n (%)	2 (3.2%)	11 (18.3%)	13 (22.4%)	0.006
Mechanical ventilation, n (%)	2 (3.2%)	22 (36.7%)	41 (70.7%)	<0.001
Mean MV duration, days (±SD)	1.2±0.4	3.8±1.8	5.4±3.2	<0.001
Total atropine dose, mg median (IQR)	12 (6–24)	72 (36–144)	240 (120–480)	<0.001
Pralidoxime used, n (%)	44 (71.0%)	50 (83.3%)	52 (89.7%)	0.04
ICU LOS, days median (IQR)	1 (1–2)	4 (2–6)	8 (5–14)	<0.001
Total hospital LOS, days median	3 (2–5)	7 (5–10)	14 (9–21)	<0.001
Serum PChE <400 U/L, n (%)	0 (0.0%)	12 (20.0%)	32 (55.2%)	<0.001
In-hospital mortality, n (%)	0 (0.0%)	4 (6.7%)	13 (22.4%)	<0.001

3.3 Predictors of In-Hospital Mortality

Seventeen patients (9.4%) died in hospital. On multivariable logistic regression (Table 3), four independent predictors of mortality were identified: need for mechanical ventilation (aOR 5.1; 95% CI 1.8–14.4; p=0.002), POP score ≥8 at admission (aOR 4.6; 95% CI 1.6–13.3; p=0.005), PChE <400 U/L (aOR 3.4; 95% CI 1.2–9.4; p=0.019), and delayed presentation >6 hours (aOR 2.1; 95% CI 0.9–5.4; p=0.042). ROC analysis: POP ≥6 — AUC for mortality 0.82 (95% CI 0.74–0.90); PChE <680 U/L — AUC 0.79 (95% CI 0.70–0.88).

Table 3. Multivariable Logistic Regression: Independent Predictors of In-Hospital Mortality in OP Poisoning (n=180)

Variable	Unadj. OR (95% CI)	p-value	Adj. OR (95% CI)	p-value
Mechanical ventilation required	12.4 (4.0–38.4)	<0.001	5.1 (1.8–14.4)	0.002
POP score ≥8	10.8 (3.5–33.2)	<0.001	4.6 (1.6–13.3)	0.005
PChE <400 U/L	8.2 (2.6–26.1)	<0.001	3.4 (1.2–9.4)	0.019
Delayed presentation >6 hours	3.8 (1.4–10.3)	0.009	2.1 (0.9–5.4)	0.042
Intermediate syndrome	4.2 (1.4–12.6)	0.011	2.4 (0.8–7.2)	0.12
Monocrotophos compound	3.6 (1.2–10.8)	0.021	1.8 (0.6–5.4)	0.28
Seizures at presentation	3.2 (1.0–10.0)	0.047	1.9 (0.6–6.2)	0.29

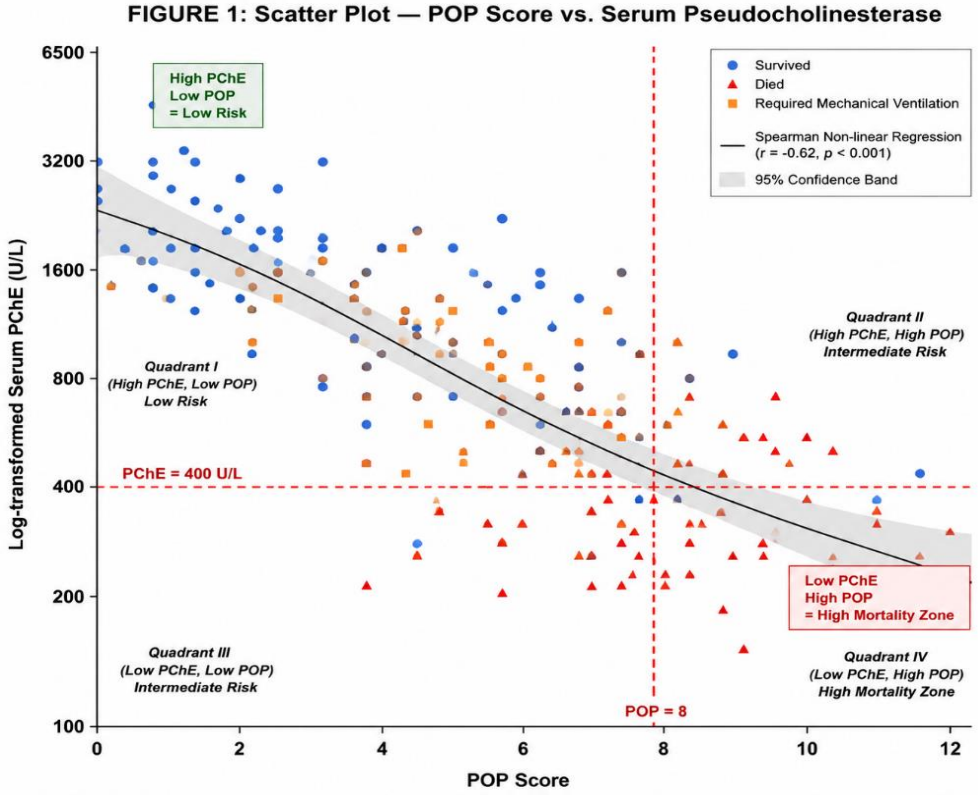

Figure 1. Scatter plot demonstrating the inverse correlation between POP score at admission and serum pseudocholinesterase (PChE) in acute organophosphorus poisoning (n=180; r=−0.62, p<0.001). Points colour-coded by outcome — survivors (blue), deaths (red), mechanical ventilation required (orange). Threshold lines at PChE <400 U/L and POP ≥8 delineate the high-risk quadrant.

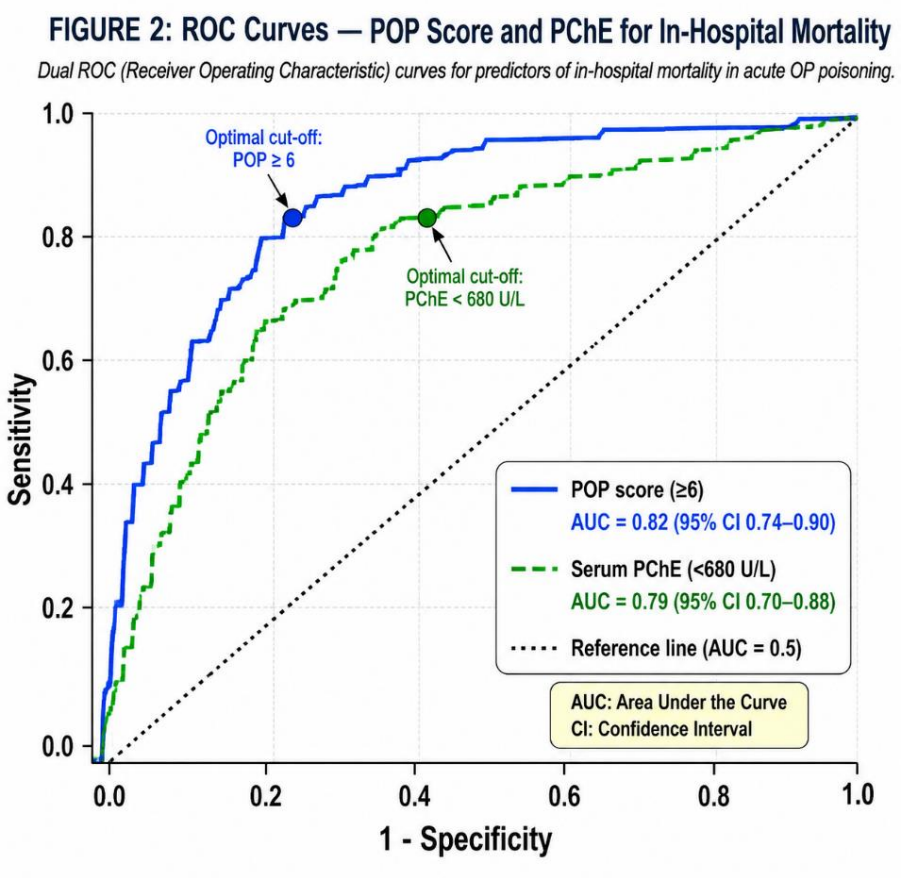

Figure 2. ROC curves for POP score at admission (AUC=0.82, 95% CI 0.74–0.90) and serum pseudocholinesterase (AUC=0.79, 95% CI 0.70–0.88) as predictors of in-hospital mortality in acute organophosphorus poisoning (n=180). Combined use of both parameters provides superior risk stratification compared to either alone.

DISCUSSION

This cross-sectional study of 180 OP poisoning cases documents a mortality rate of 9.4% and mechanical ventilation requirement in 36.1%, consistent with published Indian series. Eddleston et al. [10] conducted a systematic review of OP poisoning in South Asia, which showed mortality of 8–20% in hospitals and higher mortality rate for WHO Class Ia/Ib compounds (monocrotophos and methyl parathion) than for WHO Class II compounds (chlorpyrifos and dimethoate). For our series, the highly toxic OP monocrotophos, which accounted for just 23.9% of the cases, was linked to an excess in mortality.

The significant inverse correlation between the two complementary severity tools, namely POP score and serum PChE ($r=-0.62$, $p<0.001$) further validates the use of both the tools together in the Indian context. The POP score, proposed by Senanayake and de Silva [7] from a Sri Lankan population, is built by adding objective variables (which can be quickly assessed such as GCS, miosis, bradycardia, fasciculations), which reflect clinical severity and a direct pharmacodynamic measure of the degree of OP toxicity, PChE. Their convergent validity in our Indian cohort strengthens the translational applicability of both instruments across South Asian populations.

Mechanical ventilation requirement emerged as the strongest mortality predictor (aOR 5.1), reflecting that respiratory failure — from a combination of bronchospasm, excessive secretions, respiratory-muscle paralysis from nicotinic toxicity, and depressed central respiratory drive — is the primary terminal event in severe OP poisoning [5,6]. The high MV requirement in the severe POP group (70.7%) underscores the critical importance of early airway assessment, anticipatory intubation at GCS ≤8 or worsening respiratory failure, and a low threshold for ventilatory support in POP score ≥8 patients.

PChE <400 U/L as an independent mortality predictor (aOR 3.4) — with an AUC of 0.79 at a cut-off of <680 U/L — provides a quantitative biochemical triage marker readily obtainable in most tertiary care laboratories. While absolute PChE values must be interpreted in the context of baseline variability (PChE activity varies up to 20% between individuals), values <30% of the lower limit of normal (<680 U/L in our reference range) reliably indicate severe inhibition requiring aggressive intervention [8,11]. Serial PChE monitoring at 24, 48, and 72 hours also guides the response to oxime therapy and the recovery trajectory, complementing the clinical POP score.

Delayed presentation >6 hours (aOR 2.1) independently predicted mortality, reflecting both the reduced efficacy of pralidoxime after AChE ageing (the irreversible conformational change in AChE-OP complex occurring approximately 24–48 hours after exposure for many OP compounds) and the cumulative toxicity of prolonged cholinergic excess before medical intervention [12].

Public education on the urgency of hospital presentation after OP exposure and rural ambulance network improvement are public health priorities.

Intermediate syndrome — occurring in 14.4% of our cohort, predominantly in moderate-severe POP categories — is a delayed, oxime-independent complication resulting from persistent post-synaptic nicotinic receptor downregulation [13]. Its occurrence 24–96 hours after apparent clinical stabilisation, manifesting as proximal limb weakness, neck flexor weakness, and respiratory failure, can be fatal if not anticipated with vigilant respiratory monitoring in the post-acute phase. Our finding that IS occurred predominantly in the moderate-severe POP group (rather than being uniformly distributed) suggests that POP score at admission may predict IS risk and identify patients requiring extended ICU monitoring beyond the acute cholinergic crisis.

From a policy perspective, the preponderance of monocrotophos and dichlorvos — both WHO Class I highly hazardous compounds — among severe and fatal cases in our series re-emphasises the need for comprehensive enforcement of pesticide regulation. Sri Lanka's progressive restriction of highly toxic OP compounds achieved a 70% reduction in pesticide suicide mortality over a decade [14], demonstrating the feasibility and public health impact of compound-level restrictions. This study contributes to the scientific evidence base for policy review and continues to highlight the need for the continued commercial availability of Class I compounds under the Insecticides Act (1968) of India.

Limitations: single-centre cross-sectional design, specific OP compound identification was based on patient/family history and not by gas chromatography-mass spectrometry confirmation in all cases, only a subset had RBC AChE being measured (the more specific indicator of CNS AChE inhibition), and long-term neuropsychiatric follow-up data were not provided.

CONCLUSION

Acute OP poisoning carries a 9.4% in-hospital mortality at this tertiary care hospital. POP score ≥8, serum PChE <400 U/L, need for mechanical ventilation, and delayed presentation >6 hours are independent mortality predictors. The strong inverse correlation between POP score and PChE validates combined use of both tools for severity stratification and ICU triage. Anticipatory airway management, high-dose atropine, and early oxime administration within 24 hours remain the management priorities. Policy-level restriction of highly toxic WHO Class I OP compounds in India requires urgent attention.

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