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SERUM POTASSIUM LEVEL IN ORGANOPHOSPHORUS POISONING AND ITS IMPACT ON CLINICAL OUTCOME: A PROSPECTIVE OBSERVATIONAL STUDY FROM NORTHWESTERN RAJASTHAN

General Medicine

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ABSTRACT

Background: Organophosphorus compound (OPC) poisoning is a leading cause of morbidity and mortality in developing nations. Hypokalemia is a recognised but incompletely characterised complication of OPC poisoning, and its prognostic implications remain understudied particularly in the northwestern Indian region. **Methods:** This prospective observational study enrolled 150 consecutive patients with confirmed OPC poisoning admitted between March 2024 and February 2025 at the Department of General Medicine, Sardar Patel Medical College & PBM Hospital, Bikaner. Serum potassium was measured on admission. Patients were stratified as hypokalemic (<3.5 mmol/L) or normokalemic (≥3.5 mmol/L), and clinical outcomes ICU admission, ventilator use, oxygen requirement, intermediate syndrome (IMS), and mortality were compared between groups. **Results:** The mean age was 27.88 ± 7.47 years; 69.33% were male. Monocrotophos (38.67%) and chlorpyriphos (32%) were the predominant compounds. Hypokalemia was present in 38 (25.33%) patients; mean serum potassium was 4.01 ± 1.11 mmol/L. Hypokalemic patients had significantly higher rates of ICU admission (89.47% vs. 33.93%), mechanical ventilation (76.32% vs. 30.36%), oxygen requirement (84.21% vs. 51.79%), and mortality (57.89% vs. 19.64%) compared with normokalemic patients (all $p = 0.0001$). Mean serum potassium was significantly lower in those who died (2.8 ± 0.5 mmol/L) than in survivors (3.9 ± 0.5 mmol/L; $p = 0.001$). Serum albumin and acetylcholinesterase differed significantly between groups. **Conclusion:** Hypokalemia at admission is a significant predictor of adverse outcomes in OPC poisoning, including increased ICU utilisation, ventilatory support requirements, and mortality. Serum potassium an inexpensive and widely available investigation should be measured routinely in all OPC poisoning patients at admission and during hospitalisation to guide timely correction and improve prognosis.

KEYWORDS

Organophosphorus Poisoning, Serum Potassium, Hypokalemia, Cholinesterase Inhibition, Mortality, Northwestern Rajasthan

1. INTRODUCTION

Organophosphorus compounds (OPCs) are a chemically diverse group of phosphoric acid esters used extensively as agricultural pesticides, household insecticides, and in weaponised form as nerve agents. The first organophosphorus compound, tetraethyl pyrophosphate (TEPP), was synthesised in 1854, but large-scale use began post-World War II when parathion and malathion were commercialised. Today, India hosts one of the highest burdens of OPC poisoning globally, reflecting the compound's low cost, unrestricted rural availability, and the country's heavy agricultural dependence.

The World Health Organization estimates approximately three million cases of acute OPC poisoning annually worldwide, with 300,000 deaths two-thirds attributable to intentional self-ingestion. In India, poisoning ranks as the fourth leading cause of death. OPC poisoning predominates in rural agricultural zones; in North India, it competes with aluminium phosphide as the commonest poisoning agent.

Potassium is the principal intracellular cation, maintained in serum within the narrow range of 3.5–5.0 mmol/L by intricately regulated renal, hormonal, and cellular mechanisms. Even minor deviations can compromise neuromuscular and cardiac function. Hypokalemia in OPC poisoning is well recognised but mechanistically poorly understood. Proposed contributors include gastrointestinal potassium losses through vomiting, catecholamine-driven intracellular potassium shift secondary to sympathetic ganglionic stimulation, metabolic acidosis-driven transcellular shifts, and hypomagnesaemia-induced renal potassium wasting.

When hypokalemia complicates OPC poisoning, it may exacerbate already-compromised neuromuscular function, prolong respiratory muscle weakness, and precipitate potentially fatal cardiac dysrhythmias. The present study was designed to systematically evaluate serum potassium levels at admission in a defined OPC-poisoning cohort from Bikaner, Rajasthan, and to correlate these levels with major clinical parameters and outcomes.

2. Objectives

1. To assess serum potassium level in the setting of acute organophosphorus compound poisoning.
2. To correlate serum potassium level with clinical parameters including ICU admission, need for ventilator support, oxygen requirement, development of intermediate syndrome, and patient outcome (survival vs. mortality).

3. MATERIAL AND METHODS

3.1 Study Design and Setting

This was a prospective observational study conducted in the Department of General Medicine, Sardar Patel Medical College and PBM Associated Group of Hospitals, Bikaner, Rajasthan — a tertiary-care referral centre serving Northwestern Rajasthan. The study duration was one year, from March 2024 to February 2025.

3.2 Sample Size

A sample size of 150 patients was calculated assuming an OPC poisoning mortality of 25% (per published reference), at 80% power and 5% alpha error, using the formula $N = 2Z^2pq/l^2$. All consecutive patients fulfilling eligibility criteria were enrolled by consecutive sampling.

3.3 Inclusion and Exclusion Criteria

Included: patients >18 years with confirmed OPC poisoning admitted to medicine wards or ICU, who provided informed consent (or whose guardian consented in case of altered sensorium). OPC poisoning was diagnosed on the basis of: (i) history of exposure to organophosphate insecticide; (ii) characteristic cholinergic clinical signs/symptoms; (iii) symptomatic improvement with atropine and oximes; and (iv) serum AChE activity <50% of laboratory minimum normal (<3,600 IU/L).

Excluded: patients who refused consent, had received pre-hospital treatment, were pregnant, had pre-existing hypokalaemia, hyponatraemia, hypophosphataemia, seizures during hospitalisation, known thyroid or hepatic, renal, or muscle disease, or were receiving drugs known to alter potassium homeostasis (diuretics, beta-blockers, statins, ACE inhibitors, ARBs).

3.4 Data Collection and Clinical Assessment

All patients underwent standardised history-taking and physical examination. Severity of OPC intoxication at first contact was graded using the Peradeniya Organophosphorus Poisoning (POP) Scale. Ten millilitres of venous blood were collected at admission (Day 1) without

tourniquet application, into plain and EDTA vials, for the following investigations: complete blood count; liver function tests (SGOT, SGPT, ALP, total bilirubin, serum albumin); renal function tests (serum creatinine, blood urea); serum electrolytes (potassium, sodium, calcium, magnesium); serum acetylcholinesterase; CPK-NAC; serum vitamin D; serum vitamin B12; CRP; and serum uric acid. Patients were followed daily during hospitalisation; ICU admission, ventilator use, oxygen requirement, development of intermediate syndrome, and final outcome (discharged vs. expired) were documented.

3.5 Statistical Analysis

Data were analysed using Epi Info Version 7.2.6. Categorical variables were presented as frequencies and percentages; continuous variables as mean ± standard deviation (SD). Chi-square test was applied for comparison of proportions between groups; independent samples t-test for continuous variables. A p-value of <0.05 was considered statistically significant.

4. RESULTS

4.1 Demographic Profile

One hundred and fifty patients with confirmed OPC poisoning were enrolled. The majority (44%) were in the 26–35 year age group, followed by the 18–25 year group (43.33%), reflecting predominance in young adults. The mean age was 27.88 ± 7.47 years (range 18–55 years). Males constituted 69.33% of cases. A majority (62%) were from rural areas, consistent with the agricultural character of the region. Farmers accounted for 55.33% of the cohort, followed by students (27.33%) and housewives (12%). Detailed demographic characteristics are presented in Table 1.

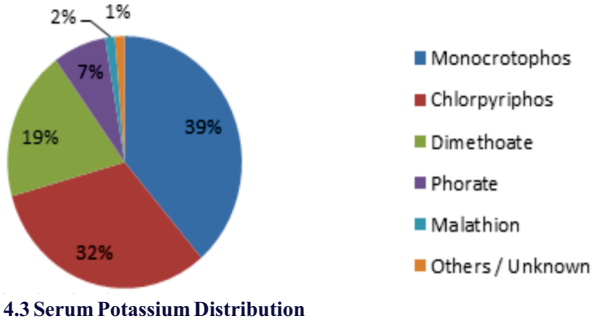
Table 1: Demographic and Clinical Profile of Study Subjects (n=150)

Variable	Category	Number (n)	Percentage (%)
Age (years)	18 – 25	65	43.33
	26 – 35	66	44.00
	36 – 45	17	11.33
	46 – 55	02	1.33
Mean ± SD age	27.88 ± 7.47 years		
Sex	Male	104	69.33
	Female	46	30.67
Residence	Rural	93	62.00
	Urban	57	38.00
Occupation	Farmer	83	55.33
	Student	41	27.33
	Housewife	18	12.00
	Auto Driver / Labourer	6	4.00
	Veterinary Doctor	2	1.33

4.2 Organophosphorus Compound Profile

Monocrotophos was the most commonly ingested compound (38.67%), followed by chlorpyriphos (32.00%) and dimethoate (19.33%). Phorate accounted for 7.33%, while malathion and other/unknown compounds each constituted 1.33% (Figure 1).

Fig 1: Distribution of Cases by Organophosphorus Compound



4.3 Serum Potassium Distribution

Of 150 patients, 38 (25.33%) had serum potassium <3.5 mmol/L (hypokalemia) at the time of admission, while 112 (74.67%) were normokalemic. The overall mean serum potassium was 4.01 ± 1.11 mmol/L.

4.4 Biochemical Parameters: Comparison Between Groups

Serum acetylcholinesterase was significantly lower in the hypokalemic group (mean 1848.5 ± 34.4 IU/L) compared with the normokalemic group (1819.9 ± 45.25 IU/L; p = 0.0001), indicating greater enzymatic inhibition in hypokalaemic patients. Serum albumin was also significantly lower in the hypokalemic group (3.72 ± 0.43 g/dL vs. 3.94 ± 0.43 g/dL; p = 0.007). Liver enzymes (SGOT, SGPT), serum bilirubin, creatinine, and blood urea did not differ significantly between the two groups. These findings are detailed in Table 2.

Table 2: Comparison of Biochemical Parameters in Hypokalemic vs. Normokalemic Groups

Parameter	Hypokalemia (Mean ± SD)	Normokalemia (Mean ± SD)	P Value
SGOT (IU/L)	43.50 ± 18.3	43.23 ± 16.57	0.933 (NS)
SGPT (IU/L)	34.86 ± 20.48	31.84 ± 20.26	0.430 (NS)
Serum Albumin (g/dL)	3.72 ± 0.43	3.94 ± 0.43	0.007*
Serum Bilirubin (mg/dL)	0.62 ± 0.2	0.70 ± 0.3	0.128 (NS)
Serum Creatinine (mg/dL)	0.61 ± 0.36	0.68 ± 0.35	0.292 (NS)
Blood Urea (mg/dL)	23.63 ± 12.25	26.56 ± 12.53	0.212 (NS)
Acetylcholinesterase (IU/L)	1848.5 ± 34.4	1819.9 ± 45.25	0.0001**
CPK-NAC (U/L)	686.52 ± 464.2	635.2 ± 96.5	NS

NS = Not Significant; *p < 0.05; **p < 0.0001

4.5 Clinical Outcomes by Potassium Status

The hypokalemic group showed significantly worse outcomes across all major clinical endpoints, as summarised in Table 3. ICU admission was required in 89.47% of hypokalemic patients versus 33.93% of normokalemic patients (p = 0.0001). Mechanical ventilation was needed in 76.32% of hypokalemic patients versus 30.36% of normokalemic patients (p = 0.0001). Supplemental oxygen was required in 84.21% of hypokalemic versus 51.79% of normokalemic patients (p = 0.0001).

Overall mortality was 29.33% (44/150). Among patients with hypokalemia, 22 of 38 (57.89%) died, compared with 22 of 112 (19.64%) in the normokalemic group — a statistically highly significant difference (p = 0.0001). Intermediate syndrome (IMS) occurred in 4.67% overall (7 patients); it was more frequent in hypokalemic patients (10.52% vs. 2.67%), though this difference did not reach statistical significance (p = 0.124).

Table 3: Clinical Outcomes According to Serum Potassium Status

Outcome Parameter	Normokalemia n=112 (%)	Hypokalemia n=38 (%)	Total n=150 (%)	P Value
ICU Admission	38 (33.93%)	34 (89.47%)	72 (48%)	0.0001**
Ventilator Support	34 (30.36%)	29 (76.32%)	63 (42%)	0.0001**
Oxygen Requirement	58 (51.79%)	32 (84.21%)	90 (60%)	0.0001**
Intermediate Syndrome	3 (2.67%)	4 (10.52%)	7 (4.67%)	0.124 (NS)
Mortality (Death)	22 (19.64%)	22 (57.89%)	44 (29.33%)	0.0001**
Recovered	90 (80.36%)	16 (42.11%)	106 (70.67%)	0.0001**

**p < 0.0001 (highly significant); NS = Not Significant

4.6 Serum Potassium and Survival

Mean serum potassium was substantially lower in patients who died (2.8 ± 0.5 mmol/L) compared with those who survived to discharge (3.9 ± 0.5 mmol/L), and this difference was statistically significant (p = 0.001), underscoring the value of admission serum potassium as a prognostic marker.

5. DISCUSSION

The principal finding is that hypokalemia at admission is a significant and independent predictor of adverse outcomes in OPC poisoning, including greater need for ICU care, mechanical ventilation, supplemental oxygen, and higher mortality.

The preponderance of young adult males (mean age 27.88 ± 7.47

years; 69.33% male) mirrors published data from similar agricultural settings. Nimsarkar et al. (2023) reported that 74% of their series were male, and the 21–30 year age group predominated. Basnet et al. (2024) reported a mean age of 26.96 years.

Monocrotophos (WHO Class I/Ib extremely hazardous) was the most common agent (38.67%), followed by chlorpyriphos (32%). This is consistent with findings by Pathik Patel et al. (2025), who also identified monocrotophos as the leading agent. Regional variation is notable: Kothiwale et al. (2019) reported malathion and chlorpyrifos as primary compounds in their cohort. The predominance of highly toxic OPC compounds in our series likely contributes to the relatively high overall mortality of 29.33%.

Hypokalemia was identified in 25.33% of our patients, lower than reported by some centres: Vandana Dandekar et al. (2019) found 68.4% hypokalemia; Narasimhamurthy et al. (2017) reported 56%; and Kundavaram et al. (2025) reported 43%. These differences may reflect heterogeneity in compound types, severity of poisoning at the time of presentation, pre-hospital management, or exclusion criteria applied. The mechanisms underlying hypokalemia in OPC poisoning are multifactorial. Excessive vomiting causes gastrointestinal potassium loss. Sympathetic ganglionic stimulation, driven by accumulation of acetylcholine at nicotinic ganglionic receptors, results in massive catecholamine release, which in turn drives intracellular potassium shift via stimulation of Na+/K+-ATPase. Additionally, metabolic acidosis frequently present in severe OPC poisoning may initially be associated with extracellular potassium shifts, while subsequent renal correction contributes to net potassium loss. Hypomagnesaemia compounds this via impaired tubular potassium conservation.

The striking difference in ICU admission rates between hypokalemic (89.47%) and normokalemic (33.93%) groups highlights the clinical severity associated with potassium depletion. These rates are higher than those reported by George et al. (2023; 11.8% ICU rate) and Nimsarkar et al. (2023; 14% ICU rate), which likely reflects differences in case-mix severity. Ventilator support was required by 76.32% of hypokalemic patients — significantly more than normokalemic patients (30.36%). This is biologically coherent: potassium is essential for the maintenance of resting membrane potential across skeletal muscle fibres, including the diaphragm and accessory respiratory muscles.

Serum acetylcholinesterase activity was significantly lower in the hypokalemic group ($p = 0.0001$), suggesting that greater inhibition of cholinesterase reflecting more severe poisoning accompanies hypokalemia.

Mortality and Prognostic Utility of Serum Potassium

Mortality was 57.89% in the hypokalemic group versus 19.64% in the normokalemic group ($p = 0.0001$) among the strongest associations reported. The mean serum potassium in non-survivors was 2.8 ± 0.5 mmol/L versus 3.9 ± 0.5 mmol/L in survivors ($p = 0.001$), further strengthening the prognostic value of this parameter. Kishore Kumar et al. (2025) reported that all deaths in their series occurred in the hypokalemic group ($p = 0.0077$).

6. SUMMARY AND CONCLUSION

This prospective observational study of 150 patients with confirmed OPC poisoning from Northwestern Rajasthan demonstrates that hypokalemia present in 25.33% of patients at admission is strongly and significantly associated with adverse clinical outcomes including higher ICU admission, mechanical ventilation requirements, oxygen dependency, and mortality. Serum potassium was significantly lower in non-survivors (2.8 ± 0.5 mmol/L) than in survivors (3.9 ± 0.5 mmol/L), establishing it as a clinically meaningful prognostic marker.

Given that serum potassium measurement is inexpensive, universally available, and rapidly reportable even in resource-limited settings, we recommend its routine measurement in all patients with OPC poisoning at admission and at regular intervals during hospitalisation. Timely detection and correction of hypokalemia should be integrated into the standard protocol for managing these patients.

7. Strengths and Limitations

Strengths: Prospective design; standardised diagnostic criteria; large regional cohort from an underrepresented demographic; comprehensive outcome tracking.

Limitations: Single-centre study; serial potassium measurements not performed; some confounders (pre-hospital vomiting duration, precise time-to-atropinisation) not uniformly quantifiable; generalisability to other Indian regions may be limited given compound-use patterns vary geographically.

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