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PROGNOSTIC SIGNIFICANCE OF SERUM CREATINE PHOSPHOKINASE LEVELS IN ORGANOPHOSPHOROUS POISONING IN A TERTIARY CARE HOSPITAL: A CROSS SECTIONAL STUDY

General Medicine

Dr. Harinath Reddy Karukula — 3rd Year Post Graduate Dept. Of General Medicine Santhiram Medical College & General Hospital Nandyal-518501

Dr Leela Sree Barabari* — MD Assistant Professor Dept. Of General Medicine Santhiram Medical College & General Hospital Nandyal-518501*Corresponding Author

ABSTRACT

Background: Acute organophosphorus (OP) compound poisoning is a major public health emergency and a leading cause of inpatient mortality in developing countries like India. While acetylcholinesterase inhibition confirms exposure, alternative, readily accessible, and cost-effective biomarkers are needed to predict clinical severity and outcome.**Objective:** To estimate serum creatine phosphokinase (CPK) levels in acute OP poisoning and correlate them with clinical severity, antidote requirements, and final outcomes. Materials and Methods: This hospital-based prospective observational study evaluated 100 adult patients admitted with a history of acute OP poisoning within 6 hours of consumption at Government Rajaji Hospital, Madurai, over a 6-month period. Serial serum CPK levels were estimated on days 1, 4, and 7, and correlated with clinical features, total dosages of atropine and pralidoxime, need for mechanical ventilation, and mortality. **Results:** Out of 100 cases, 63% were male and 94% were intentional ingestions. Methyl parathion (52%) was the most common compound consumed. Initial serum CPK levels showed a highly significant positive correlation ($P < 0.001$) with clinical severity and the total dosage requirements of atropine and pralidoxime. Patients with initial CPK values > 600 IU/L experienced a 100% mortality rate (16/16 cases), whereas those with initial CPK values < 390 IU/L had a 100% survival rate (78/78 cases) ($P = 0.012$). In recovering patients, serial CPK levels demonstrated a progressive decline toward normal values. **Conclusion:** Serum CPK is a highly valuable, inexpensive, and easily accessible prognostic biomarker in acute OP poisoning. Early tracking of elevated initial CPK levels effectively predicts respiratory failure, intensive care requirements, and mortality risk, enabling prompt risk stratification and timely referral.

KEYWORDS

Organophosphorus Poisoning, Creatine Phosphokinase, Prognosis, Biomarker, Respiratory Failure.

INTRODUCTION

Organophosphorus (OP) compounds have assumed considerable global importance as widely used agricultural insecticides. In developing nations like India, acute poisoning with these agents stands as the commonest cause of emergency department admissions and inpatient mortality, with an overall case fatality ratio hovering around 20%. The World Health Organization (WHO) estimates approximately 3 million pesticide exposures annually, resulting in nearly 200,000 deaths across developing regions. India bears one of the highest incidences worldwide, where nearly 90% of cases are suicidal in nature, involving a young and economically productive age group.

The classic toxic mechanism of OP compounds involves the phosphorylation and irreversible inhibition of acetylcholinesterase (AChE) at muscarinic and nicotinic synapses. This causes endogenous accumulation of acetylcholine at synaptic clefts, precipitating an acute cholinergic crisis characterized by bronchorrhea, bradycardia, muscle fasciculations, and altered sensorium. Early mortality is largely driven by central respiratory drive depression, severe bronchospasm, or cardiac arrhythmias caused by direct myocardial necrosis (myocardiotoxicity).

While plasma butyrylcholinesterase (BChE) and RBC acetylcholinesterase assays are traditionally requested to confirm exposure, their routine diagnostic use for predicting severity is restricted. BChE levels fluctuate widely based on underlying liver disease, malnutrition, and genetic variants, and the laboratory assays are largely unavailable in resource-poor, peripheral medical centers. Consequently, there is an urgent need for an easily quantifiable, inexpensive alternative biochemical parameter that can efficiently track systemic severity, muscle fiber necrosis, and high-risk respiratory outcomes at the time of admission.

AIM AND OBJECTIVE

AIM:

To evaluate the prognostic significance of serum creatine phosphokinase (CPK) levels in patients with organophosphorous poisoning.

OBJECTIVES:

1.To measure serum creatine phosphokinase (CPK) levels in patients with organophosphorous poisoning at the time of admission.
2. To assess the association between serum creatine phosphokinase levels and severity of organophosphorous poisoning.

MATERIAL AND METHODS

Type of study: Hospital based cross sectional study
Source of data:
Patients with ORGANOPHOSPHOROUS POISONING admitted in the wards and ICU of general medicine in Santhiram medical College and general hospital,Nandyal

Duration of study:From JUNE 2025 to MAY 2026
Sample size: 100 cases

Methodology:
This is a hospital based cross sectional study.
Sampling technique: Simple random sampling

INCLUSION AND EXCLUSION CRITERIA

INCLUSION CRITERIA:

1. Patients > 18 years
2. Patients with history of exposure to OP poisoning within 6 hours

EXCLUSION CRITERIA:

1. Patients with indication of exposure to an entirely different poison other than OPC Poison.
2. Patients with OP poisoning and mixed with any other poison
3. Patients who have consumed poison along with alcohol
4. Patients who are chronic alcoholics
5. Patients with history suggestive of chronic liver disease
6. History suggestive of myopathy
7. Patients with history of malignancy and autoimmune diseases
8. Patients with history of renal disease
9. History of intake of drugs like – Eg: Statins, Fibrates, Dexamethasone

INVESTIGATIONS

All patients underwent a standardized baseline and diagnostic workup, which included complete hemogram (Hb, TC, DC, ESR), metabolic profile (Random blood sugar, blood urea, and serum creatinine), Liver Function Tests (SGOT, SGPT), ECG monitoring for cardiac abnormalities, and Chest X-ray to monitor for pulmonary congestion.

RESULTS

Demographic analysis revealed that the majority of patients belonged to the young, economically productive age bracket of 19–30 years (37%). Males comprised 63% of the study cohort, while females accounted for 37%. In terms of intent, 94% of cases were suicidal, with

primary domestic/familial friction being the most prominent underlying reason (66%). Methyl parathion was identified as the most frequently ingested toxic agent (52%). The primary clinical symptoms observed at admission included vomiting and abdominal pain (100%), generalized hypersecretion (79%), depressed mental status (25%), pin-point pupils (24%), muscle fasciculations (21%), and respiratory failure (21%).

Table 1: Correlation Between Initial Serum CPK Levels and Patient Outcome

Initial CPK Range (IU/L)	Severity Grade	Survival (n=79)	Death (n=21)	Total Patients (N=100)	Percentage (%)
< 390	Mild	78	0	78	74.00%
391–600	Moderate	1	5	6	6.00%
> 600	Severe	0	16	16	16.00%
Total		79	21	100	100.00%

*P-value = 0.012 (Statistically Significant)

Table 2: Comparison of Baseline (Initial) and Serial Serum CPK Levels Based on Severity

Clinical Stratification	Baseline CPK Mean (IU/L)	Baseline CPK SD (±)	Final Follow-up CPK Mean (IU/L)	Final Follow-up CPK SD (±)	Significance Level (P-value)
Mild (< 390 IU/L)	225.59	56.30	121.01	27.61	< 0.001 (Highly Significant)
Moderate (390–600 IU/L)	492.67	70.95	1076.33	554.49	0.028 (Significant)
Severe (> 600 IU/L)	751.19	91.33	1138.19	248.65	< 0.001 (Highly Significant)

DISCUSSION

Acute organophosphorus compound poisoning induces a major systemic toxic state that results in multi-organ failure if left unmitigated. The foundational diagnostic hallmark relies on validating severe depression of baseline cholinesterase configurations. However, the routine utilization of butyrylcholinesterase (BChE) as an index of severity presents distinct diagnostic vulnerabilities, given that its synthesis is highly erratic in pregnant women, systemic infections, and underlying liver dysfunction.

This trial successfully highlighted the clinical application of using serum creatine phosphokinase (CPK) as an alternative, highly responsive marker for evaluating severity in acute OP toxic syndromes. Our observations demonstrate that initial serum CPK levels elevate drastically during the hyperacute phase of toxic compound loading, occurring well before the onset of secondary complications like intermediate syndrome (IMS). This hyper-CPKemia is structurally linked to severe, widespread muscle fiber necrosis and cellular membrane disruption induced by sustained acetylcholine accumulation at the motor end-plate.

Our results established that elevated baseline CPK parameters correlate strongly with higher requirements for atropine and pralidoxime, prolonged ICU stays, and a greater need for mechanical ventilation. Severe cases presenting with CPK values > 600 IU/L showed a close link to central respiratory drive failure, severe bronchorrhea, and cardiac arrest. Conversely, individuals maintaining initial CPK titers < 390 IU/L had mild clinical profiles that required low antidote volumes and resulted in standard survival rates.

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