Original Article

A Study of Correlation of CPK MB Levels with Prognosis in Organophosphorous Poisoning

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Abstract

Background: Organophosphorus compound (OPC) poisoning remains a significant public health concern, particularly in developing countries. Early identification of prognostic indicators is essential for improving clinical outcomes. The objective is to evaluate demographic characteristics, clinical severity, electrocardiographic changes, and cardiac enzyme levels in patients with OPC poisoning and to assess their association with functional outcomes. **Material and Methods:** This prospective observational study included 217 patients presenting with OPC poisoning to the Department of General Medicine, District Hospital, Tumakuru, Karnataka, over an 18-month period (February 2019 to August 2020). Data on demographics, Peradeniya Organophosphorus Poisoning (POP) score, ECG findings, pseudocholinesterase, and CPK-MB levels were collected and analyzed in relation to ICU stay and clinical outcomes. **Results:** The majority of patients were aged <30 years, with male predominance. Most cases were mild (53.92%), followed by moderate and severe poisoning. QT prolongation was the most common ECG abnormality and showed a significant association with severity and adverse outcomes. Pseudocholinesterase levels increased, while CPK-MB levels decreased over time, both demonstrating significant trends ($p<0.001$). Cardiac enzyme levels correlated with severity, ICU stay, and mortality. The overall recovery rate was 90.32%, with a mortality rate of 9.68%. **Conclusion:** QT prolongation and cardiac biomarkers, particularly CPK-MB, serve as important prognostic indicators in OPC poisoning. Their integration into clinical assessment may improve risk stratification and patient management. Further studies are warranted to validate these findings.

Keywords: Organophosphorus poisoning, QT prolongation, CPK-MB, pseudocholinesterase, prognosis, POP score.

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Introduction

India is the world's most populous country, accounting for about 16% of the global population, with agriculture serving as a primary occupation for a significant portion of its people. Organophosphorus compounds have been extensively used for decades in agriculture as pesticides to protect crops and improve yield.[1] OPCs are heterogenous compounds designed for pests and weed control. OPCs poisoning is an important preventable public health problem in developing countries like India. Though accidental poisoning can occur following exposure or inhalation, Serious poisoning often follows suicidal ingestion which is more common than former.[2]

OPCs poisoning is the most common cause of poisoning in rural India and worldwide. According to WHO, 3 million cases are due to pesticide poisoning every year, in which, about 1 million are accidental and 2 million are suicidal poisonings, leads to more than 0.25 million deaths per year. OPCs pesticide exposure occurs through inhalation, ingestion and dermal contact.[3]

Organophosphate compounds are irreversible inhibitors of the enzyme acetyl cholinesterase, binding to the esteric site of the enzyme. They inhibit both cholinesterase and pseudo-cholinesterase activity. This inhibition cause's accumulation of acetylcholine at synapses with resultant overstimulation of neurotransmission thus leads to initial stimulation and eventual exhaustion of cholinergic synapses. Respiratory paralysis and cardiac arrest are considered as the most common causes of death in these patients. Cardiac complications commonly associated with organophosphorus compound (OPC) poisoning can be severe and potentially fatal. However, these complications are largely preventable if identified early and managed promptly. In cardiac muscle basic pathology is acetyl choline binds with its receptors and release calcium ions in to cytosol. In OPCs poisoning due to excessive availability of acetylcholine, which binds to receptors, induces a mass inflow of calcium ions in to cytosol, which leads to depolarization of muscle end plates leading to muscular damage. Many factors are responsible for respiratory failure such as respiratory muscle weakness, bronchospasm with bronchorrhea, and respiratory tract infection.[4] OPCs poisoning leads to three main syndromes:

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Acute cholinergic syndrome, intermediate syndrome (IMS), and OP induced delayed neuropathy (OPIDN).[5] The complications of OP poisoning are acidosis, respiratory paralysis, acute renal failure, seizures, arrhythmia, aspiration etc. 2 Raised CPK-MB levels have been associated with acute OPCs poisoning patients mainly due to cardiac muscle and skeletal muscle involvement. CPK MB isozyme predominates in cardiac muscle and lesser in skeletal muscle. High levels of CPK-MB have been associated with mortality in acute OPCs poisoning patients.[6] Diagnosis of OPCs poisoning is based on characteristic symptoms and signs, and history of exposure to a known OPCs compound. Most helpful signs are miosis and muscle fasciculations. Estimation of serum or RBC cholinesterase level and electrodiagnostic tests are helpful in confirming the diagnosis. Clinical features of OPCs poisoning appear when RBC cholinesterase activity isof 0-3 is graded as mild, 4-7 is graded as moderate and 8-11 is graded as severe poisoning.[8] Recent evidence indicates that intensive management can significantly reduce mortality in organophosphorus compound (OPC) poisoning. This study aims to assess serum CPK-MB levels in affected patients, alongside their epidemiological characteristics, clinical features, and severity grading, and to correlate these findings with outcomes. It further seeks to determine the prognostic value of serum CPK-MB, with the goal of supporting early detection and improving outcome prediction in OPC poisoning cases.

Materials and Methods

Study Design and Setting: This prospective, hospital-based cross-sectional study was conducted in the Department of General Medicine at District Hospital, Tumakuru, Karnataka. Patients presenting to the casualty with organophosphorus compound (OPC) poisoning and meeting the inclusion criteria were enrolled in the study. Total sample size of 217 patients was included in the study. All eligible patients and/or their caregivers were informed about the nature and purpose of the study. Written informed consent was obtained from those who agreed to participate prior to enrollment.

Eligible patients were enrolled after thorough screening based on the inclusion and exclusion criteria. Relevant clinical, biochemical, and epidemiological data were collected using a structured proforma. Ethical approval for the study was obtained from the Institutional Ethics and Research Committee of Sri Siddhartha Medical College, Tumakuru.

Study Population: The study included patients of both sexes aged >18 years admitted with OPC poisoning to the casualty and General Medicine department.

Study Duration: The study was carried out over a period of 18 months, from February 2019 to August 2020.

Inclusion Criteria: All symptomatic patients with a history of organophosphorus compound (OPC) ingestion, categorized as mild, moderate, or severe according to the Peradeniya Organophosphorus Poisoning (POP) scale, were included in the study.

Exclusion Criteria: Patients were excluded if they had ingested substances other than OPCs or had received treatment outside prior to admission. Patients with pre-existing cardiac conditions such as rheumatic heart disease or ischemic heart disease were also excluded. Additional exclusions included chronic alcoholics, patients with chronic kidney disease, those aged below 18 years, and patients on medications such as statins, fibrates, aspirin, anticoagulants, or steroids. Patients who had received intramuscular injections, had recent trauma, postoperative status, or were diagnosed with chronic inflammatory muscle diseases such as polymyositis or dermatomyositis were also excluded.

Data Collection: Patients fulfilling the inclusion criteria and admitted to the casualty in the Department of General Medicine at District Hospital, Tumakuru, Karnataka were enrolled in the study. Detailed demographic data, including age and sex, were recorded. Information regarding the type of organophosphorus (OP) compound and route of ingestion was obtained. A thorough clinical examination was performed, and severity of poisoning was assessed using the Peradeniya Organophosphorus Poisoning (POP) scale.

Biochemical evaluation included measurement of serum CPK-MB levels at the time of admission using the modified IFCC method in the hospital laboratory. Follow-up measurements were carried out on day 3 and day 5. In cases of mortality, detailed documentation of clinical and laboratory findings was maintained.

Study Procedure: Patients with a history of OPC poisoning who had not received prior treatment and were admitted to the casualty department were included as study subjects and managed according to standard treatment protocols.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SAS version 9.2 for Windows. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation. Chi-square test or Fisher's exact test was used for comparison of categorical variables. Independent sample t-test and one-way ANOVA were applied for comparison of continuous variables. Normality assumptions were assessed prior to analysis. Correlation between serum CPK-MB, pseudocholinesterase levels, and patient prognosis was evaluated. A p-value of <0.05 was considered statistically significant.

Results

A total of 217 patients with organophosphorus compound (OPC) poisoning were included in the analysis. The mean age was 42.76 ± 15.06 years, with the highest incidence observed in individuals aged ≤30 years (28.11%), followed by 31–40 years (20.28%) ($p<0.001$). Males constituted a higher proportion of cases (56.68%) compared to females (43.32%), demonstrating a statistically significant gender distribution ($p=0.049$).

Among the identified compounds, dimethoate was the most common agent (29.95%), followed by chlorpyriphos (24.42%) and methyl parathion (19.35%) ($p<0.001$). Based on the Peradeniya Organophosphorus Poisoning (POP) score, the majority of patients presented with mild poisoning (53.92%), while 35.48% and 10.60% had moderate and severe poisoning, respectively ($p<0.001$).

Treatment modalities included atropine with pralidoxime (PAM) in 53.92% of cases and combination therapy with magnesium sulfate in 46.08%, with no statistically significant difference between groups (p=0.248). A shorter ICU stay (<7 days) was observed in 56.22% of patients, whereas 43.78% required prolonged hospitalization (≥7 days) (p<0.001). The overall recovery rate was 90.32%, while mortality was 9.68% (p<0.001).

Severity-wise analysis demonstrated a strong association between POP score and clinical outcomes. Patients with mild poisoning had minimal ICU requirements and no mortality, whereas severe poisoning was associated with prolonged ICU stay (100%) and significantly higher mortality (65.22%) (p<0.001). ECG abnormalities increased with severity, particularly QT prolongation and ST elevation, indicating progressive cardiac involvement.

Biochemical analysis revealed a significant temporal trend in biomarkers. Pseudocholinesterase levels increased progressively from day 1 to day 5 across all severity groups, while CPK-MB levels showed a declining trend over the same period (p<0.001). Importantly, normalization of pseudocholinesterase and CPK-MB levels by day 5 was significantly associated with favorable clinical outcomes (p<0.001), highlighting their prognostic utility.

Table 1: Baseline Characteristics and Exposure Profile

Variable	Category	n (%)	p-value
Age	≤30	61 (28.11)	<0.001
	31–40	44 (20.28)	
	41–50	40 (18.43)	
	51–60	35 (16.13)	
	61–70	35 (16.13)	
	≥70	2 (0.92)	
Gender	Male	123 (56.68)	0.049
	Female	94 (43.32)	
OP Compound	Dimethoate	65 (29.95)	<0.001
	Chlorpyriphos	53 (24.42)	
	Methyl parathion	42 (19.35)	
	Others/Unknown	57 (26.27)	

Table 2: Clinical Severity, Treatment, and Outcomes

Variable	Category	n (%)	p-value
POP Severity	Mild	117 (53.92)	<0.001
	Moderate	77 (35.48)	
	Severe	23 (10.60)	
Treatment	Atropine + PAM	117 (53.92)	0.248
	Atropine + PAM + MgSO4	100 (46.08)	
ICU Stay	<7 days	122 (56.22)	<0.001
	≥7 days	95 (43.78)	
Outcome	Recovered	196 (90.32)	<0.001
	Death	21 (9.68)	

Table 3: Severity-wise Clinical Correlates

Parameter	Mild	Moderate	Severe	p-value
ICU stay ≥7 days	4 (3.42%)	68 (88.31%)	23 (100%)	<0.001
Mortality	0	6 (7.79%)	15 (65.22%)	<0.001
QT Prolongation	8.55%	24.68%	65.22%	<0.001
ST Elevation	7.69%	18.18%	60.87%	<0.001
Sinus Tachycardia	17.09%	15.58%	17.39%	<0.01
Sinus Bradycardia	11.97%	7.79%	21.74%	<0.01

Table 4A: Biomarker Trends (Mean ± SD)

Biomarker	Severity	Day 1	Day 3	Day 5	p-value
Pseudocholinesterase	Mild	2376.9 ± 639.9	4056.4 ± 1258.9	8410.3 ± 1936.0	<0.001
	Moderate	1013.0 ± 213.0	3087.0 ± 772.6	7328.6 ± 2019.4	
	Severe	595.7 ± 87.8	1547.8 ± 1204.9	3119.1 ± 3184.3	
CPK-MB	Mild	40.8 ± 9.8	26.4 ± 7.2	13.2 ± 4.3	<0.001
	Moderate	52.7 ± 9.2	30.7 ± 10.2	17.6 ± 12.7	
	Severe	56.5 ± 8.2	44.8 ± 15.9	37.4 ± 21.9	

Table 4B: Biomarkers vs Outcome

Biomarker	Day	Normal (Recovered)	Abnormal (Death)	p-value
Pseudocholinesterase	Day 3	18	21	0.228
	Day 5	196	20	<0.001
CPK-MB	Day 3	73	21	0.0001
	Day 5	185	21	<0.001

Discussion

In this study patient in the age group ranging from 19 to 75 years were included with mean age of 42.76±15.06 years. The highest number of patients were in the age group ≤ 30 years (28.11%), followed by 31-40 years (20.28%), which is similar to that in other studies.[9,10]

The incidence of poisoning was more in males (56.68%) when compared to females (43.32%). This study correlated well with study done by Dash et al.'s study,[11] which showed an incidence of 67% in males and 23% in females.

Dimethoate (29.95%) was the most common compound implicated in the poisoning. It was followed by Chlorpyriphos (24.42%) and Methyl parathion (19.35%). This was different from the study done by P Karki et al.[12] Who found the most common compound as Methyl parathion (23%) followed by Propoxur (5%), which can be explained by the difference in availability of compound in a geographic location. In 16 patients (7.37%) the compound was not brought and the patient was diagnosed and treated on the basis of clinical features.

The ECG reflects the widespread cardiac toxicity of organophosphate compounds. Ludromirsky et al,[13] had described three phases of cardiotoxicity after organophosphate compound poisoning. Phase 1 – brief period of increased sympathetic tone; phase 2- prolonged period of parasympathetic activity; phase 3 – Q- T prolongation followed by torsade de pointes, ventricular tachycardia and the ventricular fibrillation. Both sympathetic and parasympathetic overactivity are known to cause cardiotoxicity.

In the study done by Sadeesh et al and. Balouch et al.[14,15] QT prolongation was the most common ECG abnormality, which was observed and supported by our study. QT prolongation was seen only in 20.27% of the patients in this study as compared to 67% in study by Sadeesh et al,[14] but it was comparable with the study done by Balouch et al.[15] ST segment elevation was seen 17.05% of the patients, which was similar to the study by Sadeesh et al. (24%). The ECG changes like Atrial fibrillation, prolonged PR interval and ventricular tachycardia found in the study by Sadeesh et al,[14] were not found in the present study.

QT prolongation was seen in 8.55% of patients with mild poisoning, 24.68% of the patients with moderate poisoning and 65.22% patient with severe poisoning (p-value <.0001) indicating that prolonged QT interval may be an indicator of severity.

In the present study cardiac enzymes (CPK-MB) which are used as an indicator of cardiac injury were negative and positive respectively in 21 of 217 patients (10%) on day 1, 3 and 5 of admission. This was similar to the study done by Sadeesh et al. which also showed enzyme positivity in 10% of the cases. Most of the patients who showed enzyme positivity also had QT prolongation on ECG, both of which reverted back at the time of discharge suggesting a transient ischemic process as described by Singh G.[16] On day 1, the observed pseudocholinesterase levels were completely lesser than the normal range (4850 to 12000 IU/L). On day 3, out of 217 subjects, 18 subjects were observed with normal pseudocholinesterase levels and for the remaining 199 subjects, the levels were lesser than the normal range. On day 5, 196 subjects were observed with normal pseudocholinesterase levels and for the remaining 21 subjects. The distribution of pseudocholinesterase levels with patient's outcome were statistically significant (p-value <.0001).

Raised CPK-MB levels have been associated with acute OPC poisoning patients mainly due to cardiac muscle, skeletal muscle and respiratory muscle involvement. High levels of CPK-MB have been associated with mortality in acute OPC poisoning patients. The researchers revealed that CPK-MB levels more than 40U/L have 10 to 20 times of odds of death within three days of admission. Compared to Chharba ML et al,[17] study the mortality in patient with elevated creatine kinase was 39.47% as against 4.76% in patients with normal creatine kinase.

Conclusion

A total of 217 patients with organophosphorus poisoning were analyzed over 18 months. Most patients were aged <30 years and predominantly male. The majority presented with mild poisoning, with no significant gender difference in severity. QT prolongation was the most common ECG abnormality and was strongly associated with severity and adverse outcomes, indicating prognostic value. Cardiac enzyme levels, particularly CPK-MB, correlated with severity, ICU stay, and outcome, supporting their role as prognostic markers.

Further studies are required to validate the prognostic significance of cardiac biomarkers in organophosphorus poisoning.

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Conflicts of interest

There are no conflicts of interest.

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