Section: Miscellaneous

Original Research Article

ASSOCIATION OF SERUM CREATINE PHOSPHOKINASE LEVELS WITH CLINICAL OUTCOME IN ACUTE ORGANOPHOSPHORUS POISONING

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ABSTRACT

Background: Organophosphorus compound poisoning is a common toxicological emergency and is associated with significant morbidity and mortality, particularly in settings where pesticide compounds are easily accessible. Early assessment of severity is essential for timely referral, close monitoring and intensive care preparedness. Serum cholinesterase is commonly used in organophosphorus poisoning, but it may not always be readily available in peripheral or resource-limited centres. Serum creatine phosphokinase is an inexpensive and widely available biochemical marker that may reflect neuromuscular injury, fasciculations, respiratory muscle involvement and systemic toxicity in acute organophosphorus poisoning. The aim is to assess the prognostic significance of serum creatine phosphokinase levels in patients with acute organophosphorus poisoning.

Materials and Methods: This hospital-based observational study included 100 patients admitted with acute organophosphorus poisoning. Demographic details, poisoning-related characteristics, type of exposure, reason for poisoning, compound consumed and mode of consumption were recorded. Serum creatine phosphokinase levels were measured initially and during follow-up. Patients were categorised into mild, moderate and severe groups based on serum CPK levels: <390 IU/L, 391–600 IU/L and >600 IU/L, respectively. Patient outcome was assessed in terms of survival or death. The association between serum CPK category and outcome was analysed, and a p-value of less than 0.05 was considered statistically significant.

Results: Among the 100 patients studied, the highest proportion of cases was observed in the 19–30 years age group, accounting for 36% of the study population. Males constituted 64% and females constituted 36%. Intentional poisoning was the predominant type of exposure, observed in 93% of patients. Familial problems were the most common reported reason for poisoning, contributing to 65% of cases. Methyl parathion was the most frequently identified compound, accounting for 51% of cases, and consumption with water was the most common mode of intake, observed in 49% of patients.

Serum CPK level showed a significant association with clinical outcome. Among patients with CPK levels <390 IU/L, 77 out of 78 patients survived, with a survival rate of 98.72% and mortality of 1.28%. Among patients with CPK levels between 391 and 600 IU/L, 2 out of 7 patients survived and 5 died, giving a mortality rate of 71.43%. In patients with CPK levels >600 IU/L, mortality was 100%, with all 15 patients dying. The association between serum CPK category and outcome was statistically significant with $p<0.001$. Serial CPK assessment showed a fall in mean CPK levels in the mild group from 229.46 ± 58.12 IU/L to 124.37 ± 29.08 IU/L. In contrast, the moderate group showed an increase from 488.72 ± 73.41 IU/L to 1048.56 ± 526.34

IU/L, and the severe group showed an increase from 764.29 ± 96.18 IU/L to 1162.47 ± 259.73 IU/L.

Conclusion: Serum creatine phosphokinase showed a significant association with outcome in acute organophosphorus poisoning. Lower and falling CPK levels were associated with survival and biochemical recovery, whereas higher and rising CPK levels were associated with increased mortality. Serum CPK may therefore serve as a simple, inexpensive and clinically useful prognostic marker in acute organophosphorus poisoning, particularly in peripheral and resource-limited settings. It should, however, be interpreted along with clinical findings and after excluding other causes of CPK elevation.

Keywords: Organophosphorus poisoning, Creatine phosphokinase, Serum CPK, Prognostic marker, Mortality, Poisoning severity, Pesticide poisoning, Biochemical marker.

INTRODUCTION

Organophosphorus compound poisoning remains one of the major causes of acute toxicological emergencies, particularly in countries where agricultural pesticides are widely used and are easily accessible. These compounds are commonly employed in farming, pest control and domestic insecticide preparations. Because of their easy availability, they are frequently involved in both accidental and intentional poisoning. In rural and semi-urban regions, organophosphorus poisoning continues to contribute substantially to hospital admissions, intensive care requirement and poisoning-related mortality.[1]

The public health importance of organophosphorus poisoning is linked not only to its frequency but also to the rapidity with which severe clinical deterioration can occur. Poisoning may follow ingestion, inhalation or dermal exposure, but oral ingestion remains common in intentional cases. Several studies have shown that organophosphate poisoning commonly affects young and economically productive individuals, making it a significant medical and social problem.[2] In India, pesticide poisoning has remained an important contributor to lethal self-poisoning, and organophosphorus compounds form a major part of this burden because of their availability and toxicity.[3] Similar concerns have also been reported internationally, where poisoning-related deaths continue to represent an important preventable cause of mortality.[4]

Organophosphorus compounds produce toxicity mainly through inhibition of acetylcholinesterase. This leads to accumulation of acetylcholine at muscarinic, nicotinic and central nervous system synapses. As a result, patients may present with salivation, lacrimation, sweating, vomiting, diarrhoea, bronchorrhea, bronchospasm, bradycardia, miosis, fasciculations, muscle weakness, altered sensorium, seizures and respiratory failure. The clinical course may vary from mild cholinergic manifestations to severe poisoning requiring ventilatory support and intensive care.

Respiratory failure is one of the most serious complications of organophosphorus poisoning and is a major determinant of mortality. It may occur due to excessive bronchial secretions, bronchospasm, central respiratory depression and weakness of respiratory muscles. In critically ill patients, associated fluid and electrolyte disturbances may further complicate clinical management and outcome.[5] Cardiac manifestations, including rhythm disturbances and electrocardiographic abnormalities, have also been reported in organophosphorus poisoning and may add to clinical severity.[6,7] Although organophosphorus poisoning is more frequently discussed in adults, paediatric poisoning has also been reported and requires careful clinical assessment because presentation and severity may vary with age and exposure pattern.[8]

Assessment of severity at admission is essential because early identification of high-risk patients can guide monitoring, referral, atropine therapy, oxime use, ventilatory preparedness and intensive care management. Traditionally, clinical features, atropine requirement, ventilator requirement and serum cholinesterase levels have been used to assess severity. However, cholinesterase estimation may not be immediately available in all hospitals, especially in peripheral and resource-limited centres. Moreover, the degree of cholinesterase inhibition may not always parallel clinical severity in every patient. Therefore, there is a continuing need for simple, inexpensive and easily measurable biochemical markers that can support clinical judgement.

Serum creatine phosphokinase is an enzyme released into the circulation following skeletal muscle injury. In organophosphorus poisoning, persistent fasciculations, neuromuscular overactivity, respiratory muscle weakness, hypoxia and muscle fibre injury may contribute to elevation of serum CPK levels. Therefore, serum CPK may reflect the extent of neuromuscular involvement and systemic toxicity. Since CPK estimation is widely available, inexpensive and repeatable, it has potential value as an adjunctive marker for assessing severity and predicting outcome in acute organophosphorus poisoning.

Serial measurement of serum CPK may also provide clinically useful information. A falling CPK level during treatment may suggest recovery and absence of ongoing muscle injury, whereas persistently elevated or rising levels may indicate worsening muscle involvement, respiratory compromise or poor prognosis. Hence, evaluation of serum CPK at admission and during follow-up may help in early risk stratification and timely intervention.

The present study was undertaken to assess the prognostic significance of serum creatine phosphokinase levels in patients with acute organophosphorus poisoning and to determine its association with clinical outcome.

Aim

To assess the prognostic significance of serum creatine phosphokinase levels in patients with acute organophosphorus poisoning.

Objectives

1. To evaluate serum creatine phosphokinase levels in patients admitted with acute organophosphorus poisoning.
2. To determine the association between serum creatine phosphokinase levels and clinical outcome in patients with acute organophosphorus poisoning.

MATERIALS AND METHODS

Study Design: This was a hospital-based observational study conducted among patients admitted with acute organophosphorus poisoning.

Study period: The study was conducted over a period of one year From April 2024 –May 2025

Study setting: The study was conducted in the Department of General Medicine at Govt Siddhartha Medical College, Vijayawada, Andhra Pradesh, India.

Study population: The study population included patients admitted with a history and clinical diagnosis of acute organophosphorus compound poisoning during the study period.

Sample size: A total of 100 patients with acute organophosphorus poisoning were included in the study.

Inclusion criteria

Patients with a history of acute organophosphorus compound poisoning who were admitted during the study period were included in the study. Patients of either sex were considered eligible for inclusion. Patients in whom serum creatine phosphokinase levels were measured during admission and follow-up were included.

Exclusion criteria

Patients with conditions known to independently elevate serum creatine phosphokinase levels were excluded from the study. These included patients with recent trauma, seizures unrelated to poisoning, myocardial infarction, known myopathy, renal failure, recent intramuscular injections, prolonged immobilisation or any other condition that could cause elevation of serum CPK independent of organophosphorus poisoning. Patients with incomplete clinical or laboratory data were also excluded.

Data collection: After admission, demographic details such as age and sex were recorded. Details related to poisoning, including type of exposure, reason for poisoning, compound consumed and mode of consumption, and were documented. Clinical examination findings and relevant treatment details were recorded. Serum creatine phosphokinase levels were measured initially and during follow-up. Patients were categorised according to serum CPK levels into mild, moderate and severe groups.

Categorisation of serum CPK levels

Serum CPK levels were categorised as follows:

- Mild: <390 IU/L
- Moderate: 391–600 IU/L
- Severe: >600 IU/L

Outcome assessment: The primary outcome assessed was patient outcome in terms of survival or death. The relationship between serum CPK levels and outcome was analysed. Mortality and survival rates were calculated across different serum CPK categories. Serial changes in serum CPK levels were also analysed to assess biochemical improvement or deterioration.

Statistical analysis

The collected data were entered and analysed using appropriate statistical methods. Categorical variables were expressed as frequency and percentage. Continuous variables were expressed as mean and standard deviation. The association between serum CPK category and patient outcome was analysed using appropriate tests of significance. A p-value of less than 0.05 was considered statistically significant.

Ethical considerations: The study was conducted after obtaining approval from the Institutional Ethics Committee. Patient confidentiality was maintained throughout the study. Data were used only for academic and research purposes.

RESULTS

The study included 100 patients with organophosphorus poisoning. The demographic profile, poisoning characteristics, serum creatine phosphokinase levels, patient outcomes, and serial CPK changes are presented in the following tables.

Table 1: Demographic and poisoning-related profile of the study population

Variable	Category	Number (%)
Age group	19–30 years	36 (36%)
	31–40 years	22 (22%)

	41–50 years	24 (24%)
	51–60 years	15 (15%)
	>60 years	3 (3%)
Sex	Male	64 (64%)
	Female	36 (36%)
Type of exposure	Accidental	7 (7%)
	Intentional	93 (93%)
Reason for poisoning	Familial problems	65 (65%)
	Financial problems	16 (16%)
	Ill health	7 (7%)
	Job stress	5 (5%)
	Others	7 (7%)
Poisoning agent	Bug killer liquid	14 (14%)
	Chlorpyrifos	8 (8%)
	Dichlorvos	5 (5%)
	Fenthion	6 (6%)
	Monocrotophos	9 (9%)
	Methyl parathion	51 (51%)
	Quinalphos	7 (7%)
Mode of consumption	With milk	33 (33%)
	With water	49 (49%)
	Others	18 (18%)

Among the 100 patients included in the study, the highest proportion of cases was observed in the 19–30 years age group, accounting for 36% of the study population. Males formed the majority, with 64% of cases, while females accounted for 36%. Intentional poisoning was the predominant type of exposure, observed in 93% of patients. Familial problems were the most common reported reason for poisoning, contributing to 65% of cases, followed by financial problems in 16%. Among the organophosphorus compounds, methyl parathion was the most frequently identified agent, accounting for 51% of cases. Consumption with water was the most common mode of intake, observed in 49% of patients.

Table 2: Association between serum creatine phosphokinase level and patient outcome

Serum CPK level	Survival	Death	Total (%)	p-value
<390 IU/L	77	1	78 (78%)	
391–600 IU/L	2	5	7 (7%)	<0.001
>600 IU/L	0	15	15 (15%)	
Total	79	21	100 (100%)	

A clear association was observed between serum CPK level and patient outcome. Among patients with serum CPK levels <390 IU/L, 77 out of 78 patients survived, while only 1 patient died. In the group with CPK levels between 391–600 IU/L, survival decreased markedly, with only 2 survivors and 5 deaths. In patients with CPK levels >600 IU/L, mortality was 100%, with all 15 patients in this category dying. The association between serum CPK level and outcome was statistically significant with a p-value <0.001, indicating that higher serum CPK levels were strongly associated with increased mortality.

Table 3: Comparison of initial and final serum creatine phosphokinase levels of patients

Serum CPK category	n	Initial CPK level, IU/L	Final CPK level, IU/L	p-value
Mild (<390 IU/L)	78	229.46 ± 58.12	124.37 ± 29.08	<0.001
Moderate (390–600 IU/L)	7	488.72 ± 73.41	1048.56 ± 526.34	0.031
Severe (>600 IU/L)	15	764.29 ± 96.18	1162.47 ± 259.73	<0.001

Serial assessment of serum CPK levels showed different trends across the three severity categories. In the mild group, mean serum CPK decreased from 229.46 ± 58.12 IU/L initially to 124.37 ± 29.08 IU/L finally, and this reduction was statistically significant. In contrast, patients in the moderate group showed an increase in mean CPK from 488.72 ± 73.41 IU/L to 1048.56 ± 526.34 IU/L, which was also statistically significant. Similarly, the severe group showed a rise in mean CPK from 764.29 ± 96.18 IU/L to 1162.47 ± 259.73 IU/L, with statistical significance. These findings indicate that patients with higher initial CPK levels had worsening biochemical progression and poorer clinical outcomes.

Table 4: Distribution of mortality according to serum CPK category

Serum CPK level	Total patients	Deaths	Mortality rate
<390 IU/L	78	1	1.28%
391–600 IU/L	7	5	71.43%
>600 IU/L	15	15	100.00%
Total	100	21	21.00%

Mortality increased progressively with increasing serum CPK levels. Patients with CPK levels <390 IU/L had a very low mortality rate of 1.28%. Mortality increased sharply to 71.43% among patients with CPK levels between 391–600 IU/L. The highest mortality was observed in patients with CPK levels >600 IU/L, where all patients died, resulting in a mortality rate of 100%. Overall mortality in the study population was 21%.

Table 5: Distribution of survival according to serum CPK category

Serum CPK level	Total patients	Survivors	Survival rate
<390 IU/L	78	77	98.72%
391–600 IU/L	7	2	28.57%
>600 IU/L	15	0	0.00%
Total	100	79	79.00%

Survival showed an inverse relationship with serum CPK levels. The highest survival rate was seen in patients with CPK levels <390 IU/L, where 98.72% survived. Survival declined markedly to 28.57% in patients with CPK levels between 391–600 IU/L. No survival was observed among patients with CPK levels >600 IU/L. These findings further support the role of serum CPK as a prognostic marker in organophosphorus poisoning.

Table 6: Change in serum CPK levels from initial to final measurement

Serum CPK category	n	Initial CPK level, IU/L	Final CPK level, IU/L	Mean change in CPK	Direction of change
Mild (<390 IU/L)	78	229.46 ± 58.12	124.37 ± 29.08	−105.09	Decreased
Moderate (390–600 IU/L)	7	488.72 ± 73.41	1048.56 ± 526.34	+559.84	Increased
Severe (>600 IU/L)	15	764.29 ± 96.18	1162.47 ± 259.73	+398.18	Increased

The mean change in serum CPK levels showed improvement in the mild group and worsening in the moderate and severe groups. In patients with CPK levels <390 IU/L, the mean CPK decreased by 105.09 IU/L, suggesting biochemical recovery. In the moderate group, CPK increased by 559.84 IU/L, indicating significant biochemical deterioration. In the severe group, CPK increased by 398.18 IU/L, further supporting progressive muscle injury and poor outcome among patients with higher CPK levels.

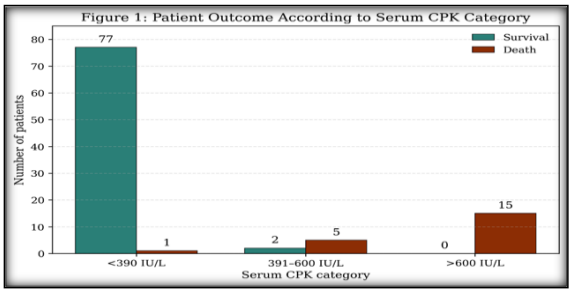

Figure 1: Patient Outcome According to Serum CPK Category

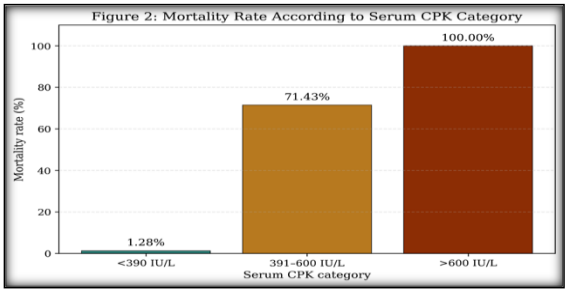

Figure 2: Mortality Rate According to Serum CPK Category

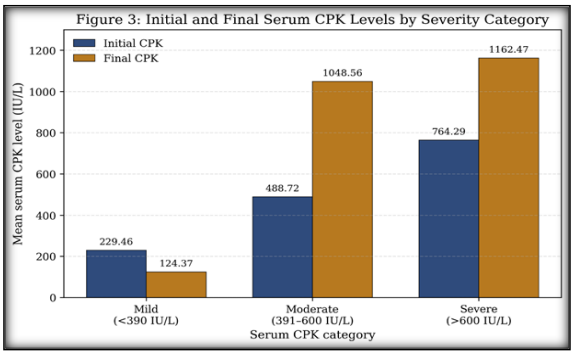

Figure 3: Initial and Final Serum CPK Levels by Severity Category

In this study of 100 patients with organophosphorus poisoning, most patients were young adults, with the highest frequency seen in the 19–30 years age group. Males were more commonly affected than females. Intentional poisoning was the predominant type of exposure, and familial problems were the most common underlying reason. Methyl parathion was the most commonly detected organophosphorus compound, and consumption with water was the most frequent mode of intake.

Serum creatine phosphokinase showed a strong association with clinical outcome. Patients with lower CPK levels had better survival, while increasing CPK levels were associated with sharply rising mortality. Mortality was only 1.28% in patients with CPK levels below 390 IU/L, but increased to 71.43% in the moderate CPK group and reached 100% in patients with CPK levels above 600 IU/L. The association between serum CPK category and outcome was statistically significant with a p-value <0.001.

Serial CPK comparison also showed a meaningful biochemical pattern. Patients in the mild group

showed a decline in final CPK levels, whereas patients in the moderate and severe groups showed increasing final CPK levels. These findings suggest that serum CPK may serve as a useful prognostic biochemical marker in organophosphorus poisoning, particularly for identifying patients at higher risk of mortality.

DISCUSSION

Demographic pattern, poisoning profile and association of serum CPK with outcome: Organophosphorus compound poisoning continues to remain a clinically important emergency, particularly in regions where pesticide compounds are easily available for agricultural and domestic use. The outcome of such poisoning depends on several factors, including the type and quantity of compound consumed, delay in reaching hospital, severity of cholinergic manifestations, respiratory involvement, need for ventilatory support, and the biochemical response of the patient. Among the biochemical parameters, serum creatine phosphokinase has gained attention as a possible marker of severity because it reflects muscle injury, fasciculations, respiratory muscle involvement and systemic toxicity.

In the present study, 100 patients with organophosphorus poisoning were evaluated. The maximum number of patients belonged to the 19–30 years age group, which constituted 36% of the study population. This was followed by the 41–50 years age group with 24% and the 31–40 years age group with 22%. Only 15% of patients were in the 51–60 years age group, and 3% were above 60 years. This distribution shows that organophosphorus poisoning was more frequent among young and middle-aged adults. The higher involvement of this age group may be due to greater psychosocial burden, occupational responsibilities, domestic stress, financial pressure and easier physical access to organophosphorus compounds.

A male predominance was observed in the present study, with males accounting for 64% of cases and females accounting for 36%. This may be explained by greater occupational exposure among males, especially in agricultural or outdoor work, along with increased access to pesticide compounds. The type of exposure showed that intentional poisoning was overwhelmingly common, accounting for 93% of cases, while accidental poisoning was seen in only 7%. This finding indicates that most cases in the present study were related to deliberate self-poisoning rather than accidental exposure.

The reasons for poisoning further support the role of psychosocial factors. Familial problems were the most common reason, contributing to 65% of cases. Financial problems accounted for 16%, ill health for 7%, job stress for 5% and other reasons for 7%. These findings suggest that organophosphorus poisoning in this study was not merely a toxicological event but was closely linked to social, emotional and domestic circumstances. Hence, management of organophosphorus poisoning should not end with acute medical care alone; it should also include psychosocial assessment and preventive counselling wherever possible.

With regard to the poisoning agent, methyl parathion was the most frequently identified compound, accounting for 51% of cases. This was followed by bug killer liquid in 14%, monocrotophos in 9%, chlorpyrifos in 8%, quinalphos in 7%, fenthion in 6% and dichlorvos in 5%. The predominance of methyl parathion may reflect local availability, usage pattern and ease of access. Oral ingestion was the main route of exposure. Consumption with water was the most common mode, seen in 49% of patients, followed by consumption with milk in 33% and other modes in 18%. This pattern is consistent with the high proportion of intentional poisoning observed in the study.

The demographic and exposure profile of the present study is broadly comparable with the study by Ahamed Kalil et al., who also evaluated 100 patients with organophosphorus poisoning. Their study reported that the 19–30 years age group was most commonly affected, with male predominance, familial problems as the leading cause and methyl parathion as the most frequently detected compound. They also observed that patients with serum CPK values above 600 IU/L had 100% mortality, and the association between CPK level and outcome was statistically significant with $p=0.012$.[20] In the present study, the 19–30 years age group accounted for 36% of cases, males constituted 64%, familial problems accounted for 65%, methyl parathion was identified in 51%, and patients with CPK levels above 600 IU/L also had 100% mortality. However, the association between CPK level and outcome in the present study was stronger, with $p<0.001$.

The central finding of the present study was the significant association between serum CPK levels and patient outcome. Among patients with serum CPK levels below 390 IU/L, 77 out of 78 patients survived, while only one patient died. This group had a survival rate of 98.72% and a mortality rate of 1.28%. In contrast, among patients with serum CPK levels between 391 and 600 IU/L, only 2 out of 7 patients survived, while 5 patients died. The mortality rate in this moderate CPK group was 71.43%. The most severe outcome was observed among patients with serum CPK levels above 600 IU/L, where all 15 patients died, resulting in 100% mortality. Overall, the survival rate in the present study was 79%, while the mortality rate was 21%. The association between serum CPK category and outcome was statistically significant with $p<0.001$.

This graded increase in mortality with rising CPK levels suggests that serum CPK may be a useful prognostic marker in acute organophosphorus poisoning. A lower CPK level was associated with

favourable outcome, whereas moderate and severe elevations were associated with a sharp decline in survival. The finding that all patients with CPK levels above 600 IU/L died indicates that severe elevation of CPK may identify a very high-risk group requiring intensive monitoring and aggressive supportive care.

The findings of the present study are supported by Das et al., who reported that higher serum CPK levels were significantly associated with increased poisoning severity, atropine requirement, need for ventilatory support and poor outcome in acute organophosphorus poisoning.[21] This is consistent with the present study, where mortality increased progressively from 1.28% in the mild CPK group to 71.43% in the moderate CPK group and reached 100% in the severe CPK group. These observations indicate that serum CPK reflects not only biochemical injury but also clinical severity and prognosis.

Bhattacharyya et al. reported that serum CPK showed a strong relationship with the Peradeniya Organophosphorus Poisoning scale, serum cholinesterase levels, arterial pH and total atropine requirement. They proposed serum CPK as a probable marker of severity in organophosphorus poisoning.[14] The present study supports this view because progressively increasing CPK levels were associated with falling survival and rising mortality. The strong association observed in the present study reinforces the usefulness of CPK as an accessible prognostic marker, particularly when interpreted along with clinical assessment.

Recent studies have also strengthened the evidence for serum CPK as a severity marker. Chandraiah and Bentoor studied 73 patients with organophosphate poisoning and reported that 43.8% had mild poisoning, 39.7% had moderate poisoning and 16.4% had severe poisoning according to the POP scale. They observed a strong correlation between POP score and CPK levels, with $r=0.817$ and $p<0.001$, and reported an overall mortality rate of 15.1%.[23] In comparison, the present study showed an overall mortality rate of 21% and a statistically significant association between CPK category and outcome with $p<0.001$. Although the mortality rate was higher in the present study, both studies support the same conclusion that increasing CPK levels are associated with greater severity and adverse outcome.

Piyush et al. evaluated serum CPK and serum amylase as surrogate markers of severity and clinical outcome in 100 patients with organophosphate poisoning. Their study reported that higher POP scores were associated with elevated CPK and amylase levels and that these markers were useful in predicting severity, prolonged hospital stay and mortality, with diagnostic accuracy being particularly useful in serial measurements.[24] This finding is relevant to the present study because CPK levels not only showed association with mortality but also demonstrated meaningful changes on follow-up assessment.

Overall, the first part of the discussion shows that the present study is in agreement with both earlier and recent literature. The demographic profile was comparable to previous reports, and the association between serum CPK and outcome was clear. Patients with low CPK levels had better survival, while those with marked CPK elevation had poor outcome. Therefore, serum CPK appears to be a practical, inexpensive and clinically relevant biochemical marker for early risk stratification in patients with acute organophosphorus poisoning.

Serial serum CPK changes, pathophysiological basis, comparison with literature and clinical implications: Serial measurement of serum CPK provided additional information beyond the initial value. In the present study, patients in the mild CPK group showed a decline in serum CPK levels during follow-up. The mean initial CPK level in this group was 229.46 ± 58.12 IU/L, which decreased to 124.37 ± 29.08 IU/L on final measurement. The mean reduction was 105.09 IU/L, and the change was statistically significant with $p<0.001$. This decline in serum CPK suggests biochemical recovery and corresponds with the favourable clinical outcome observed in this group.

In contrast, patients in the moderate and severe CPK groups showed rising CPK values on follow-up. In the moderate group, mean CPK increased from 488.72 ± 73.41 IU/L initially to 1048.56 ± 526.34 IU/L finally. The mean increase was 559.84 IU/L, and the change was statistically significant with $p=0.031$. In the severe group, mean CPK increased from 764.29 ± 96.18 IU/L to 1162.47 ± 259.73 IU/L, with a mean rise of 398.18 IU/L and $p<0.001$. These findings indicate that persistent or rising CPK levels may reflect ongoing muscle injury, worsening biochemical severity and increased risk of mortality.

The serial pattern observed in the present study is clinically meaningful. Patients with falling CPK values were more likely to recover, whereas patients with increasing CPK values had poorer outcomes. This suggests that serial CPK estimation may be useful not only at admission but also during the course of treatment. A single initial value helps in early risk categorisation, while repeated measurements may help in assessing whether the patient is improving or deteriorating.

The usefulness of serial CPK measurement has also been reported by Thejaswini and Raveekumaran. In their study of 30 patients with acute organophosphorus poisoning, serum CPK levels correlated significantly with POP score on day 0, day 3 and day 5. The correlation coefficients were 0.763, 0.803 and 0.683 respectively, and all were statistically significant with $p<0.001$. They also reported mean serum CPK values of 1129.7 U/L on day 0, 1303.17 U/L on day 3 and 1288.73 U/L on day 5.[22] The present study supports this observation because serial CPK monitoring showed a clear difference between recovering and worsening

groups. Mild cases showed a fall in CPK, while moderate and severe cases showed an increase.

The biological basis for serum CPK elevation in organophosphorus poisoning is plausible. Organophosphorus compounds inhibit acetylcholinesterase, leading to accumulation of acetylcholine at muscarinic and nicotinic receptor sites. Persistent stimulation of nicotinic receptors at the neuromuscular junction may cause fasciculations, muscle fatigue, weakness and respiratory muscle involvement. Severe poisoning may also result in hypoxia, prolonged muscle activity and muscle fibre injury. These processes can lead to leakage of CPK from damaged skeletal muscle into the circulation.

Bhattacharyya et al. also suggested that elevated serum CPK in acute organophosphorus poisoning may be due to muscle fibre necrosis, particularly in severe poisoning.[14] This mechanism supports the findings of the present study, where higher CPK categories were associated with poor outcomes. The progressive rise in mortality from mild to severe CPK categories may therefore represent increasing neuromuscular and systemic involvement.

Respiratory compromise is another important factor linking elevated CPK with poor prognosis. Severe organophosphorus poisoning may lead to respiratory failure through multiple mechanisms, including central respiratory depression, respiratory muscle weakness, bronchorrhea and bronchospasm. Involvement of respiratory muscles may contribute to muscle injury and CPK elevation. In the present study, all patients with CPK levels above 600 IU/L died, suggesting that marked CPK elevation may identify patients with severe systemic toxicity and high risk of respiratory failure or other fatal complications.

Serum cholinesterase estimation has traditionally been used in the diagnosis and assessment of organophosphorus poisoning. However, cholinesterase testing may not be available in all centres, especially at peripheral hospitals. In addition, butyrylcholinesterase activity may differ according to the specific compound and may not always reflect clinical severity. Eddleston et al. reported that butyrylcholinesterase activity may not reliably predict outcome in all patients with organophosphorus poisoning and should be interpreted with caution.[17] In such settings, serum CPK may serve as a useful supportive biochemical marker because it is inexpensive, widely available and easy to repeat.

However, serum CPK is not specific to organophosphorus poisoning. It can be elevated in several other conditions, including trauma, seizures, myocardial injury, myopathies, prolonged immobilisation, intramuscular injections, renal dysfunction and other causes of skeletal muscle damage. Therefore, CPK should not be interpreted as an isolated diagnostic marker. Perreault et al. reported that CPK leaks into blood and urine after skeletal muscle injury and remains a useful marker for detecting and monitoring muscle damage.[19] This supports its role in organophosphorus poisoning, but only when other possible causes of CPK elevation are clinically excluded.

The time course of CPK elevation is also relevant. Sahjian and Frakes reported that when muscle injury is ongoing, CPK levels may remain elevated because the half-life of CPK is approximately 1.5 days, and values generally normalise within 5–6 days after a single muscle insult.[18] This observation is consistent with the present study. Patients in the mild group showed declining CPK levels, suggesting absence of ongoing muscle injury and response to treatment. In contrast, patients in the moderate and severe groups showed rising CPK levels, suggesting continued muscle injury and worsening severity.

The findings of the present study have important clinical implications. Serum CPK estimation can assist in early triage, especially in settings where patients present to peripheral centres before referral. Patients with CPK levels below 390 IU/L had excellent survival, with a survival rate of 98.72%. However, patients with CPK levels between 391 and 600 IU/L had a mortality rate of 71.43%, and those with CPK levels above 600 IU/L had 100% mortality. Therefore, patients with moderate or severe CPK elevation should be considered high-risk and should be monitored closely for respiratory failure, worsening cholinergic features and need for ventilatory support.

The practical value of serum CPK lies in its availability and repeatability. In resource-limited hospitals, where cholinesterase assays or advanced toxicological testing may not be immediately available, CPK can provide useful supportive information. It should not replace clinical assessment, POP scoring, respiratory monitoring or cholinesterase testing where available, but it can add important prognostic value. The present study, along with earlier and recent literature, supports the use of serum CPK as an adjunctive marker for assessing severity and outcome in acute organophosphorus poisoning.[14,20–24]

Thus, the serial and categorical analysis of serum CPK in the present study demonstrates that CPK is not merely an associated laboratory abnormality but has practical prognostic relevance. Lower and falling values were associated with survival and recovery, whereas higher and rising values were associated with clinical deterioration and mortality. These findings support the inclusion of serum CPK estimation in the routine assessment and follow-up of patients with acute organophosphorus poisoning, particularly in peripheral and resource-limited clinical settings.

CONCLUSION

The present study demonstrated a significant association between serum creatine phosphokinase

levels and clinical outcome in patients with acute organophosphorus poisoning. Among the 100 patients studied, the overall survival rate was 79%, while mortality was 21%. Patients with serum CPK levels below 390 IU/L showed a favourable outcome, with a survival rate of 98.72% and mortality of only 1.28%. In contrast, patients with CPK levels between 391 and 600 IU/L showed a marked decline in survival, with mortality rising to 71.43%. The poorest outcome was observed among patients with CPK levels above 600 IU/L, where mortality was 100%.

Serial serum CPK estimation further supported its prognostic value. Patients in the mild CPK group showed a significant fall in mean CPK levels from 229.46 ± 58.12 IU/L to 124.37 ± 29.08 IU/L, indicating recovery. In contrast, patients in the moderate and severe groups showed rising final CPK levels, suggesting worsening biochemical severity and poor prognosis. The association between serum CPK category and patient outcome was statistically significant, with a p-value of <0.001.

Thus, serum CPK can be considered a simple, inexpensive, easily available, and clinically useful prognostic marker in acute organophosphorus poisoning. Higher initial CPK levels and rising serial CPK values indicate increased severity and mortality risk. However, serum CPK should be interpreted along with clinical findings and after excluding other possible causes of CPK elevation. Early identification of patients with elevated CPK levels may help in timely referral, close monitoring, intensive care preparedness, and reduction of complications and mortality in organophosphorus poisoning.

REFERENCES

1. Banday T, Desai M, Naik V, Tathineni B. Predictors of morbidity and mortality in organophosphorus poisoning: a case study in a rural hospital in Karnataka, India. N Am J Med Sci. 2015;7:259.
2. Amir A, Raza A, Qureshi T, Mahesar GB, Jafferi S, Haleem F, et al. Organophosphate poisoning: demographics, severity scores and outcomes from national poisoning control centre, Karachi. Cureus. 2020.
3. Karunarathne A, Bhalla A, Sethi A, Perera U, Eddleston M. Importance of pesticides for lethal poisoning in India during 1999 to 2018: a systematic review. BMC Public Health. 2021;21.
4. Wang L, Wu Y, Yin P, Cheng P, Liu Y, Schwebel DC, et al. Poisoning deaths in China, 2006–2016. Bull World Health Organ. 2018;96:314–326A.
5. Besen BAMP. Fluid and electrolyte overload in critically ill patients: an overview. World J Crit Care Med. 2015;4:116.
6. Jayaprakash N, Geetha S. A study of the ECG changes associated with organophosphorus compound poisoning. IAIM. 2019;6:69–73.
7. Anand S, Singh S, Nahar Saikia U, Bhalla A, Sharma YP, Singh D. Cardiac abnormalities in acute organophosphate poisoning. Clin Toxicol (Phila). 2009;47:230–235.
8. Caba IC, Ștreangă V, Dobrin ME, Jităreanu C, Jităreanu A, Profire BȘ, et al. Clinical assessment of acute organophosphorus pesticide poisoning in pediatric patients admitted to the toxicology emergency department. Toxics. 2022;10:582.
9. Bodien YG, Barra A, Temkin NR, Barber J, Foreman B, Vassar M, et al. Diagnosing level of consciousness: the limits of the Glasgow Coma Scale total score. J Neurotrauma. 2021;38:3295–3305.
10. Akyildiz BN, Kondolot M, Kurtoğlu S, Akin L. Organophosphate intoxication presenting as diabetic ketoacidosis. Ann Trop Paediatr. 2009;29:155–158.
11. Thiermann H, Zilker T, Eyer F, Felgenhauer N, Eyer P, Worek F. Monitoring of neuromuscular transmission in organophosphate pesticide-poisoned patients. Toxicol Lett. 2009;191:297–304.
12. Rajapakse BN, Thiermann H, Eyer P, Worek F, Bowe SJ, Dawson AH, et al. Evaluation of the Test-mate ChE field kit in acute organophosphorus poisoning. Ann Emerg Med. 2011;58:559–564.e6.
13. Tsai JR, Sheu CC, Cheng MH, Hung JY, Wang CS, Chong IW, et al. Organophosphate poisoning: 10 years of experience in southern Taiwan. Kaohsiung J Med Sci. 2007;23:112–119.
14. Bhattacharyya K, Phaujdar S, Sarkar R, Mullick OS. Serum creatine phosphokinase: a probable marker of severity in organophosphorus poisoning. Toxicol Int. 2011;18:117–123.
15. Eddleston M, Mohamed F, Davies JOJ, Eyer P, Worek F, Sheriff MHR, et al. Respiratory failure in acute organophosphorus pesticide self-poisoning. QJM. 2006;99:513–522.
16. Sam KG, Kondabolu K, Pati D, Kamath A, Pradeep Kumar G, Rao PGM. Poisoning severity score, APACHE II and GCS: effective clinical indices for estimating severity and predicting outcome of acute organophosphorus and carbamate poisoning. J Forensic Leg Med. 2009;16:239–247.
17. Eddleston M, Eyer P, Worek F, Sheriff MHR, Buckley NA. Predicting outcome using butyrylcholinesterase activity in organophosphorus pesticide self-poisoning. QJM. 2008;101:467–474.
18. Sahjian M, Frakes M. Crush injuries: pathophysiology and current treatment. Adv Emerg Nurs J. 2007;29:145–150.
19. Perreault S, Birca A, Piper D, Nadeau A, Gauvin F, Vanasse M. Transient creatine phosphokinase elevations in children: a single-centre experience. J Pediatr. 2011;159:682–685.
20. Ahamed Kalil A, Karthik Pandian T, Priya VP, Sathiya P. A study on the prognostic significance of serum creatine phosphokinase levels in organophosphorus poisoning. Int J Acad Med Pharm. 2024;6(1):1522–1526.
21. Das P, Bhattacharjee P, Nath BK, et al. Serum creatine phosphokinase level: as a severity marker in acute organophosphate poisoning. J Evid Based Med Healthc. 2018;5(14):1262–1266.
22. Thejaswini VVSB, Raveekumaran V. A study of serum creatine phosphokinase levels as a prognostic indicator in acute organophosphorus poisoning. J Indian Soc Toxicol. 2024;20(2):3–10.
23. Chandraiah R, Bentoor SN. Comparison of serum amylase and creatine phosphokinase levels in assessing the severity of organophosphate poisoning. J Contemp Clin Pract. 2025;11(6):380–384.
24. Piyush P, Ajad NK, Meena DK. A study of serum creatine phosphokinase and amylase levels as surrogate markers of severity and clinical outcome in organophosphate poisoning. European Journal of Cardiovascular Medicine. 2025;15(12):310–315.