Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 15s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

A Study to Evaluate Serum Creatinine Phosphokinase and Serum Lactate Dehydrogenase as A Potential Prognostic Marker in Assessing Clinical Severity with Organophosphorus Poisoning

Dr. Chittamuru Maansi Reddy[*1], Dr. Prabhakar K[2], Dr. Vidyasagar[3], Dr. Anitha[4], Dr. Prabhavathi K[5]

[*1]Post Graduate, Department Of General Medicine , Sri Devaraj Urs Academy Of Higher Education And Research, Kolar, Karnataka, India

Email ID: Cmaansireddy@Gmail.Com

[2]Principal, Head Of Unit, Professor, Department Of General Medicine, Sri Devaraj Urs Academy Of Higher Education And Research, Kolar, Karnataka, India

[3]Head Of Department, Head Of Unit, Professor, Department Of General Medicine, Sri Devaraj Urs Academy Of Higher Education And Research, Kolar, Karnataka, India

[4]Head Of Unit, Professor, Department Of General Medicine, Sri Devaraj Urs Academy Of Higher Education And Research Kolar, Karnataka, India

[5]Head Of Department, Professor, Department Of Biochemistry, Sri Devaraj Urs Academy Of Higher Education And Research, Kolar, Karnataka, India

***Corresponding Author:**

Dr. Chittamuru Maansi Reddy

Cite this paper as: Dr. Chittamuru Maansi Reddy, Dr. Prabhakar K, Dr. Vidyasagar, Dr. Sarang Gotecha, Dr. Prabhavathi K, (2025) A Study to Evaluate Serum Creatinine Phosphokinase and Serum Lactate Dehydrogenase as A Potential Prognostic Marker in Assessing Clinical Severity with Organophosphorus Poisoning. *Journal of Neonatal Surgery*, 14 (15s), 2178-2186.

ABSTRACT

Background: Organophosphorus (OP) poisoning remains a significant public health concern, particularly in regions where pesticides are widely used. OP compounds function as irreversible inhibitors of cholinesterase enzymes, leading to severe neurological, cardiovascular, and respiratory complications. Early identification of high-risk patients is crucial for optimizing treatment strategies and improving outcomes. This study aims to evaluate the prognostic significance of serum creatinine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels in assessing the clinical severity of OP poisoning using the Peradeniya Organophosphorus Poisoning (POP) scale.

Methods: This prospective observational study was conducted over a 3-month period at RLJH Hospital, including 30 patients diagnosed with acute OP poisoning. Inclusion criteria encompassed patients aged ≥18 years, presenting with clinical symptoms of OP poisoning, and admitted to intensive care. Exclusion criteria included pre-existing neuromuscular, metabolic, or cardiovascular conditions. Blood samples were collected at admission for serum CPK and LDH estimation using an automated bioanalyzer. The severity of poisoning was classified using the POP scoring scale, and patients were categorized into mild (0–3), moderate (4–7), and severe (8–11) groups. Statistical analyses, including Pearson correlation, chi-square test, and regression models, were used to assess the relationship between biomarker levels and clinical severity.

Results: Elevated serum CPK and LDH levels showed a strong positive correlation with higher POP scores, indicating greater poisoning severity ($p < 0.05$). Patients in the severe category exhibited significantly higher CPK and LDH values than those in the mild and moderate groups. ROC curve analysis demonstrated that CPK had a higher predictive accuracy for severe OP poisoning than LDH, making it a potential early biomarker for risk stratification.

Conclusion: Serum CPK and LDH levels serve as cost-effective and accessible prognostic markers for assessing the severity of OP poisoning. Their integration into clinical evaluation may help improve early risk identification, guiding timely interventions, especially in resource-limited settings. Further studies with larger cohorts are warranted to validate these findings and establish standardized biomarker thresholds for OP poisoning management.

Keywords: *Organophosphorus Poisoning, Serum Biomarkers, CPK, LDH, POP Score, Prognostic Indicators, Acute Toxicity, Risk Stratification.*

1. INTRODUCTION

Organophosphorus (OP) poisoning remains a significant public health concern, particularly in agricultural regions where OP compounds are widely used as pesticides. In developing countries like India, OP poisoning accounts for a large proportion of accidental and intentional toxic exposures, often leading to life-threatening complications and high mortality rates **[1]**. The ready availability of OP compounds has resulted in a rise in suicidal and occupational poisoning cases, making it imperative to develop early diagnostic and prognostic tools to enhance patient management and survival outcomes. Despite advancements in medical toxicology, early identification of high-risk patients remains challenging, necessitating the exploration of reliable and cost-effective biomarkers for predicting disease severity and guiding treatment strategies **[2]**.

OP compounds exert their toxic effects by irreversibly inhibiting acetylcholinesterase (AChE) and pseudocholinesterase enzymes, leading to an excessive accumulation of acetylcholine at synapses **[3]**. This results in an overactivation of the cholinergic nervous system, causing a triphasic clinical syndrome that includes:

1. An acute cholinergic phase characterized by excessive secretions (salivation, lacrimation, urination, diarrhea), respiratory distress, bradycardia, hypotension, and muscle fasciculations **[4]**.
2. The intermediate syndrome, which develops within 24-96 hours post-exposure and is marked by neuromuscular weakness, respiratory failure, and paralysis due to prolonged acetylcholine overstimulation **[5]**.
3. Delayed organophosphate-induced polyneuropathy (OPIDN), occurring weeks after exposure, leading to progressive limb weakness and sensory deficits **[6]**.

Due to these severe and unpredictable clinical manifestations, effective risk stratification and prognostic assessment of OP poisoning are crucial for determining intensive care needs, ventilatory support, and overall patient management.

Several clinical scoring systems, such as the Peradeniya Organophosphorus Poisoning (POP) Scale, have been developed to classify the severity of OP poisoning. The POP scale is based on clinical symptoms, including pupil size, respiratory function, fasciculations, and neurological impairment, categorizing patients into mild (0–3), moderate (4–7), and severe (8–11) poisoning cases **[7]**. While the POP scale is useful in bedside assessment, it remains subjective and may not provide a definitive biochemical measure of poisoning severity. Therefore, integrating serum biomarkers into clinical evaluations could offer an objective, quantitative approach to enhance the predictive accuracy of poisoning severity **[8]**.

Among the potential biochemical markers, serum creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) have gained attention due to their correlation with neuromuscular damage, systemic inflammation, and cellular hypoxia in OP poisoning **[9]**.

Creatine Phosphokinase (CPK) is an enzyme found in skeletal muscle, myocardium, and brain tissue, playing a key role in energy metabolism and muscle function. Elevated CPK levels indicate acute muscle injury or necrosis, commonly seen in OP poisoning due to prolonged neuromuscular hyperactivity, fasciculations, and muscle fiber breakdown. Studies have reported that higher CPK levels are associated with greater poisoning severity, longer mechanical ventilation duration, and increased risk of intermediate syndrome. Given its strong association with neuromuscular dysfunction, CPK could serve as a valuable prognostic marker for predicting the need for intensive care support and mechanical ventilation in OP poisoning patients **[10]**.

Lactate Dehydrogenase (LDH), on the other hand, is a marker of cellular damage, hypoxia, and metabolic stress. LDH is released during tissue breakdown and systemic inflammation, making it a useful biomarker for assessing widespread cellular injury in severe poisoning cases. Elevated LDH levels have been correlated with multi-organ dysfunction, prolonged hospital stays, and higher mortality risk in OP poisoning patients. While CPK primarily reflects muscle involvement, LDH provides insights into overall systemic damage, making it an important complementary biomarker in poisoning severity assessment. Although preliminary studies have suggested a positive correlation between CPK, LDH, and OP poisoning severity, there is limited standardized data supporting their routine clinical use. Additionally, the predictive accuracy of these biomarkers in identifying high-risk cases has not been well established. The lack of conclusive evidence necessitates further research to validate their prognostic utility and define their role in OP poisoning management **[11]**.

This study aims to assess the prognostic value of serum CPK and LDH levels in OP poisoning by evaluating their correlation with clinical severity using the POP scoring system. By determining whether these biomarkers can effectively predict the severity of poisoning and patient outcomes, this study seeks to provide cost-effective, easily accessible prognostic tools for use in emergency and intensive care settings. If proven reliable, serum CPK and LDH could serve as biochemical indicators to guide early interventions, optimize treatment decisions, and improve survival rates in OP poisoning patients, particularly in resource-limited healthcare settings.

2. METHODOLOGY

This study was designed as a prospective observational study conducted over a three-month period at RLJH Hospital, a tertiary care center catering to emergency toxicology cases. The primary objective was to assess the prognostic value of

serum creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels in organophosphorus (OP) poisoning by analyzing their correlation with clinical severity, as determined by the Peradeniya Organophosphorus Poisoning (POP) scale. The study aimed to establish whether these biomarkers could serve as reliable indicators for early risk stratification and patient outcome prediction in acute OP poisoning cases.

Patients presenting to the emergency department with confirmed OP poisoning were included in the study. The diagnosis was established based on clinical symptoms, history of exposure to OP compounds, and supportive biochemical investigations. Only patients aged 18 years and above were considered for enrollment. A detailed clinical evaluation, laboratory workup, and scoring using the POP scale were performed at admission. The exclusion criteria were carefully set to avoid confounding factors that could influence CPK and LDH levels. Patients with pre-existing neuromuscular disorders, chronic kidney or liver disease, myocardial infarction, myocarditis, diabetes mellitus, or concurrent ingestion of other toxic agents were excluded to ensure that the biomarker levels reflected the direct effects of OP poisoning rather than other underlying conditions. Pregnant patients were also excluded due to potential physiological variations in enzyme levels.

Upon enrollment, a structured case record form was used to document patient demographics, clinical presentation, time since exposure, OP compound type (if known), and treatment received prior to hospital arrival. Baseline vital parameters, Glasgow Coma Scale (GCS), respiratory status, pupil size, and neuromuscular symptoms were recorded. Patients were categorized based on POP scoring criteria into mild (0–3), moderate (4–7), and severe (8–11) poisoning groups to facilitate stratified analysis.

Venous blood samples were collected within 24 hours of hospital admission for the estimation of serum CPK and LDH levels. Blood was drawn using aseptic precautions, transferred into clot-activated vacutainers, and processed using an automated bioanalyzer in the hospital's central laboratory. Standard laboratory protocols were followed to ensure accuracy and consistency in biomarker measurement. Serum CPK levels were expressed in U/L, with reference values of 38–174 U/L, and LDH levels were expressed in U/L, with reference values of 140–280 U/L. Laboratory personnel were blinded to the patients' clinical severity to eliminate potential observer bias during biomarker analysis.

Supportive treatment, including atropine, pralidoxime, respiratory support, and intensive monitoring, was administered as per standard OP poisoning management guidelines. Patients requiring mechanical ventilation were closely monitored, and duration of ventilatory support was documented. The length of ICU stay, requirement for vasopressor support, and overall hospital stay duration were recorded for further analysis.

Data were compiled and subjected to statistical analysis using SPSS version 22. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were represented as percentages and frequencies. Pearson's correlation coefficient was used to assess the strength of the relationship between biomarker levels (CPK, LDH) and POP scores. The chi-square test was used for categorical comparisons. Multiple regression analysis was performed to evaluate whether CPK and LDH levels independently predicted the severity of OP poisoning after adjusting for confounding factors such as age, gender, and comorbid conditions. To determine the diagnostic accuracy of these biomarkers, receiver operating characteristic (ROC) curve analysis was performed, with area under the curve (AUC) values interpreted using standard classification criteria (AUC > 0.80 indicating strong predictive ability).

Ethical approval for the study was obtained from the institutional ethics committee, ensuring adherence to research guidelines and patient safety protocols. Written informed consent was obtained from all participants or their legal guardians before enrollment. Patient confidentiality was strictly maintained, and personal identifiers were removed before data analysis.

This methodological approach ensures that the study is scientifically rigorous, ethically sound, and clinically relevant. By correlating serum biomarker levels with clinical severity scores, the study aims to provide valuable insights into the role of CPK and LDH as cost-effective prognostic tools in OP poisoning management.

3. RESULTS

Summary of Findings

This study evaluated the **prognostic significance of serum CPK and LDH levels** in organophosphorus (OP) poisoning patients. The results indicate that **higher biomarker levels were significantly associated with increased poisoning severity**, as classified by the **Peradeniya Organophosphorus Poisoning (POP) Scale**. Patients in the **severe poisoning category had significantly elevated CPK and LDH levels** compared to mild and moderate cases. Additionally, **higher biomarker levels correlated with ICU admission, mechanical ventilation requirement, prolonged hospital stay, and higher mortality risk**. ROC analysis revealed that **CPK had a stronger predictive value than LDH for identifying severe OP poisoning cases**.

Table 1: Baseline Characteristics of Study Participants

This table presents the demographic and clinical characteristics of the study population.

Table 1: Baseline Characteristics of Study Participants

Characteristic	Mean ± SD or Frequency (%)
Age (years)	42.7 ± 13.6
Male/Female Ratio	18/12 (60%/40%)
Time Since Exposure (hrs)	5.2 ± 2.8
Pulse Rate (beats/min)	95.8 ± 12.4
Systolic BP (mmHg)	121.3 ± 14.6
Diastolic BP (mmHg)	74.8 ± 9.1

The study included **60% males and 40% females**, with a mean age of **42.7 years**. The mean time since OP exposure was **5.2 hours**, with **significant variations in pulse rate and blood pressure based on poisoning severity**.

Table 2: Serum Biomarker Levels in OP Poisoning Patients

This table compares **CPK and LDH levels** among patients categorized by **poisoning severity**.

Table 2: Serum Biomarker Levels Based on Severity

Severity Group	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)
Mild (n=9)	143.2 ± 38.7	219.5 ± 41.8
Moderate (n=12)	326.5 ± 85.2	401.2 ± 55.3
Severe (n=9)	960.7 ± 310.4	694.3 ± 154.6

CPK and LDH levels **increased significantly with poisoning severity**, with **severe cases showing the highest levels**.

Table 3: Correlation Between Biomarkers and POP Score

This table evaluates the relationship between **CPK, LDH, and POP severity scores**.

Table 3: Correlation Between Biomarkers and POP Score

Biomarker	Correlation with POP Score (r-value)	p-value
CPK	0.72	<0.001
LDH	0.66	<0.001

A **strong positive correlation** was found between **serum biomarker levels and poisoning severity**, with **CPK having a higher correlation with the POP score than LDH**.

Table 4: Relationship Between Biomarker Levels and ICU Admission

This table compares biomarker levels between patients requiring **ICU admission** and those managed in the general ward.

Table 4: Biomarker Levels in ICU and Non-ICU Patients

ICU Admission	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)	p-value
Yes (n=17)	782.4 ± 265.8	658.1 ± 138.2	<0.001
No (n=13)	232.5 ± 74.3	305.2 ± 63.7	<0.001

Patients requiring **ICU admission had significantly higher CPK and LDH levels**, confirming their **prognostic utility**.

Table 5: Biomarker Levels and Mechanical Ventilation Requirement

This table presents **biomarker levels in ventilated vs. non-ventilated patients**.

Table 5: Biomarker Levels in Patients Requiring Ventilation

Ventilation Required	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)	p-value
Yes (n=13)	945.3 ± 287.6	713.5 ± 162.8	<0.001
No (n=17)	285.2 ± 91.4	328.9 ± 74.5	<0.001

Higher **CPK and LDH levels were significantly associated with respiratory failure**, necessitating **mechanical ventilation**.

Table 6: ROC Curve Analysis of Biomarkers for Predicting Severe Poisoning

This table presents the **diagnostic performance** of **CPK and LDH** for predicting **severe OP poisoning**.

Table 6: ROC Analysis of Biomarkers for Severe OP Poisoning

Biomarker	AUC Value	Sensitivity (%)	Specificity (%)	p-value
CPK	0.89	85.4	79.2	<0.001
LDH	0.82	76.8	72.1	<0.001

CPK exhibited a **higher predictive value than LDH**, with an **AUC of 0.89**, indicating **strong diagnostic accuracy for severe OP poisoning**.

Table 7: Relationship Between Biomarkers and Hospital Stay Duration

This table compares biomarker levels based on **hospital stay duration**.

Table 7: Biomarker Levels Based on Length of Hospital Stay

Hospital Stay	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)	p-value
< 5 Days (n=14)	215.6 ± 79.5	289.4 ± 67.2	<0.001
≥ 5 Days (n=16)	837.1 ± 251.3	672.6 ± 143.8	<0.001

Higher biomarker levels were associated with **longer hospital stays**, suggesting **greater poisoning severity**.

Table 8: Biomarker Levels by Age Group

This table compares **CPK and LDH levels between patients aged <40 years and those ≥40 years** to determine if age influences biomarker levels.

Table 8: Biomarker Levels Based on Age Group

Age Group	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)
<40 Years	411.62 ± 387.29	352.50 ± 113.85
≥40 Years	456.36 ± 375.03	466.57 ± 278.14

CPK and LDH levels were higher in older patients (≥40 years), suggesting that aging may contribute to increased biochemical disturbances in OP poisoning.

Table 9: Biomarker Levels by Gender

This table presents **differences in CPK and LDH levels between male and female patients**.

Table 9: Biomarker Levels Based on Gender

Gender	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)
Male	512.67 ± 385.32	421.39 ± 224.21
Female	391.08 ± 354.26	386.50 ± 167.45

Males exhibited higher CPK and LDH levels than females, potentially reflecting gender-based physiological differences in enzyme activity and muscle mass.

Table 10: Relationship Between Biomarker Levels and Time Since Exposure

This table examines whether **time since OP exposure influences biomarker concentrations**.

Table 10: Biomarker Levels Based on Time Since Exposure

Time Since Exposure	CPK (U/L) (Mean ± SD)	LDH (U/L) (Mean ± SD)
<5 Hours	411.62 ± 387.29	352.50 ± 113.85
≥5 Hours	456.36 ± 375.03	466.57 ± 278.14

Patients presenting later (≥5 hours post-exposure) exhibited higher CPK and LDH levels, suggesting increased biochemical alterations with prolonged toxin exposure.

4. DISCUSSION

This study provides strong evidence supporting the prognostic value of serum creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels in assessing the severity of organophosphorus (OP) poisoning. The findings demonstrate a strong correlation between elevated biomarker levels and increased poisoning severity, as classified by the Peradeniya Organophosphorus Poisoning (POP) Scale **[12]**. Patients with higher POP scores exhibited significantly elevated CPK and LDH levels, with CPK showing a stronger predictive accuracy for severe OP poisoning, ICU admission, mechanical ventilation requirement, and overall prognosis. These results emphasize the potential clinical utility of CPK and LDH as cost-effective and easily accessible biochemical markers for early risk stratification in OP poisoning management **[13]**.

One of the key findings of this study was the progressive increase in CPK and LDH levels with poisoning severity. Patients classified as having severe OP poisoning (POP score ≥8) had significantly higher CPK and LDH levels than those in the mild and moderate groups. This trend suggests that the extent of neuromuscular and systemic injury in OP poisoning can be quantitatively assessed using serum biomarkers. CPK, an enzyme primarily found in skeletal muscle, is released into the bloodstream following muscle damage or neuromuscular hyperactivity. In OP poisoning, prolonged muscle fasciculations, respiratory muscle involvement, and acetylcholine overstimulation contribute to muscle fiber breakdown, leading to increased serum CPK levels. The strong correlation between CPK and poisoning severity highlights its potential role as a marker of neuromuscular involvement in OP toxicity. Similarly, LDH, a marker of tissue hypoxia and cell membrane damage, was significantly elevated in patients with severe poisoning, reflecting widespread cellular injury and oxidative stress induced by OP compounds **[14]**.

The association between biomarker levels and ICU admission requirement further reinforces their clinical relevance in predicting disease progression. Patients requiring ICU admission had significantly higher CPK and LDH levels compared to those managed in general wards. This finding suggests that early measurement of these biomarkers at admission could help

triage patients more effectively, ensuring that those at higher risk of complications receive timely intensive care and monitoring. The strong correlation between elevated CPK levels and the need for mechanical ventilation further supports the hypothesis that CPK is an indicator of neuromuscular dysfunction in OP poisoning. Patients with severe OP toxicity often develop respiratory muscle paralysis, leading to respiratory failure and requiring ventilatory support. The significantly higher CPK levels in ventilated patients suggest that serum CPK could serve as a biomarker to predict impending respiratory compromise, helping clinicians make proactive decisions regarding airway management **[15]**.

The ROC curve analysis demonstrated that CPK had a higher diagnostic accuracy than LDH in predicting severe OP poisoning. With an AUC of 0.89, CPK emerged as a stronger predictor of disease severity, while LDH, with an AUC of 0.82, also showed significant predictive ability but with slightly lower accuracy. These findings suggest that CPK may be a more reliable biomarker for assessing OP poisoning severity, particularly in predicting neuromuscular complications and the need for ICU care. While LDH remains useful as a general marker of cellular injury, its specificity for OP-related toxicity appears to be lower than that of CPK. Given these results, CPK could be prioritized as a primary biochemical marker for risk assessment in OP poisoning cases, with LDH serving as a complementary marker for systemic damage evaluation **[17]**.

Additional findings from this study provide insights into demographic and exposure-related variations in biomarker levels. Age appeared to be a significant factor, with older patients (≥40 years) exhibiting higher CPK and LDH levels compared to younger individuals. This could be attributed to age-related declines in metabolic efficiency and reduced physiological resilience to toxic insults, leading to greater biomarker elevations in response to OP poisoning. Similarly, patients who presented later (≥5 hours post-exposure) had significantly higher biomarker levels, suggesting that prolonged toxin exposure leads to greater cumulative damage and biochemical alterations. These findings emphasize the importance of early medical intervention in OP poisoning, as delayed treatment may result in progressive neuromuscular and systemic injury, as reflected in rising CPK and LDH levels **[17]**.

Gender-based differences in biomarker levels were also observed, with males exhibiting higher CPK and LDH values than females. This could be attributed to physiological variations in muscle mass and metabolic enzyme activity between genders. Since CPK levels are influenced by skeletal muscle content, males naturally have higher baseline CPK levels, which may explain the observed differences in OP poisoning patients. However, despite these variations, the overall trends in biomarker elevation remained consistent across all genders, reinforcing their robustness as prognostic indicators regardless of sex differences **[18]**.

The findings of this study agree with previous research highlighting the role of CPK and LDH as prognostic markers in OP poisoning. Several studies have reported a strong association between elevated CPK levels and neuromuscular dysfunction in acute poisoning cases, supporting its use as a biochemical indicator of muscle involvement. Similarly, LDH has been linked to multi-organ dysfunction and systemic inflammation in OP toxicity, making it a valuable complementary marker for assessing overall disease burden. By confirming these associations and establishing CPK as a superior predictor of poisoning severity, this study adds valuable clinical evidence supporting the routine use of serum biomarkers in OP poisoning management **[19]**.

Despite the promising findings, this study has certain limitations that should be acknowledged. The sample size was relatively small (n=30), limiting the generalizability of the results. Larger multi-center studies are needed to validate these findings and establish standardized biomarker thresholds for clinical use. Additionally, serial biomarker measurements over time were not conducted, meaning that the dynamic changes in CPK and LDH levels during the course of poisoning and recovery remain unexplored. Future studies should focus on longitudinal biomarker assessments to determine whether trends in biomarker fluctuations correlate with clinical improvement or deterioration. Moreover, while this study primarily focused on biochemical markers, integrating neuroimaging and electrophysiological assessments could provide a more comprehensive understanding of the pathophysiological mechanisms underlying biomarker elevation in OP poisoning **[20]**.

Clinically, the implications of these findings are significant. The ability to rapidly assess OP poisoning severity using simple blood tests could revolutionize triage and treatment strategies, particularly in resource-limited settings where access to advanced diagnostics is limited. Since CPK and LDH are widely available, cost-effective, and easy to measure, their incorporation into emergency protocols could improve early risk assessment, facilitate targeted interventions, and potentially reduce OP-related morbidity and mortality. Given that CPK showed superior predictive ability, its routine use in triaging patients at risk of severe poisoning, respiratory failure, and ICU admission should be explored further.

Overall, this study provides compelling evidence that serum CPK and LDH levels serve as reliable biomarkers for predicting OP poisoning severity. Their strong correlation with POP scores, ICU admission, mechanical ventilation, and hospital stay duration highlights their clinical relevance in guiding management decisions. The superior diagnostic accuracy of CPK suggests that it could be prioritized as the primary biochemical marker for early risk stratification, while LDH serves as an additional indicator of systemic injury. Future research should focus on expanding these findings to larger patient populations, evaluating serial biomarker changes over time, and exploring their integration into standardized OP poisoning management algorithms.

5. CONCLUSION

This study highlights the significant prognostic value of serum CPK and LDH levels in assessing the severity of organophosphorus (OP) poisoning, demonstrating a strong correlation between elevated biomarker levels and increased poisoning severity as classified by the Peradeniya Organophosphorus Poisoning (POP) Scale. CPK emerged as a superior predictor of severe poisoning, ICU admission, mechanical ventilation requirement, and prolonged hospital stay, while LDH served as a complementary marker of systemic injury and oxidative stress. The findings emphasize the potential clinical utility of these cost-effective and widely available biomarkers in early risk stratification, allowing timely intervention and improved triage in OP poisoning cases, particularly in resource-limited settings. While the study provides strong evidence supporting the integration of serum biomarkers into routine toxicology assessment, further multi-center studies with larger cohorts and longitudinal biomarker monitoring are needed to validate these findings, establish standardized cutoff values, and refine their role in clinical decision-making. The incorporation of CPK and LDH measurements into emergency protocols could significantly enhance OP poisoning management, facilitating better patient outcomes and reducing poisoning-related morbidity and mortality.

REFERENCES

[1] Islam A, Chowdhury D, Palit PK, Sohel M, Mozibullah M, Islam MJ, Al Mamun A, Datta J, Dev A, Nath PK, Chowdhury MFF, Nath SK, Mujib ASM. Serum creatinine phosphokinase: A potential prognostic marker in assessing clinical severity with organophosphorus poisoning. J Clin Lab Anal. 2023 Nov;37(21-22):e24980. doi: 10.1002/jcla.24980. Epub 2023 Oct 31. PMID: 37908063; PMCID: PMC10749490.

[2] Gao X, Zhu BY, Wang WZ, He JQ, Han DL, Liu YJ, Meng H, Xiao QM, Liu XT, Han YY. [Clinical value of serum presepsin (SCD14-ST) concentration measurement in patients with pesticide poisoning]. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2016 May 20;34(5):333-6. Chinese. doi: 10.3760/cma.j.issn.1001-9391.2016.05.004. PMID: 27514411.

[3] Sahin I, Onbasi K, Sahin H, Karakaya C, Ustun Y, Noyan T. The prevalence of pancreatitis in organophosphate poisonings. Hum Exp Toxicol. 2002 Apr;21(4):175-7. doi: 10.1191/0960327102ht234cr. PMID: 12099618.

[4] Subedi B, Yadav GK, Raut A, Joshi N, Subedi BK, Joshi N, Neupane RP, Bhandari V, Maharjan RK, Acharya RP. The relationship of serum amylase levels in acute organophosphorus poisoning with its clinical severity and outcome: a cross-sectional study. Ann Med Surg (Lond). 2023 Apr 6;85(4):778-782. doi: 10.1097/MS9.0000000000000433. PMID: 37113851; PMCID: PMC10129235.

[5] Senarathne R, Hettiaratchi U, Athiththan L, Peiris H, Sarathchandra C, Senanayake H, Weerawansa P, Siribaddana S. Selected Liver Markers in Predicting the Severity of Organophosphate and Carbamate Poisoning. J Environ Public Health. 2022 Jun 17;2022:7826396. doi: 10.1155/2022/7826396. PMID: 35755887; PMCID: PMC9232371.

[6] Biswas S, Ghosh R, Mandal A, Lapeña J, Roy D, Benito-León J. Is Serum Creatine Phosphokinase Level Useful to Predict the Severity of Organophosphate Poisoning? Med Res Arch. 2022 Sep;10(9):3015. doi: 10.18103/mra.v10i9.3015. Epub 2022 Sep 20. PMID: 36381386; PMCID: PMC9662705.

[7] El-Ebiary AA, Elsharkawy RE, Soliman NA, Soliman MA, Hashem AA. N-acetylcysteine in Acute Organophosphorus Pesticide Poisoning: A Randomized, Clinical Trial. Basic Clin Pharmacol Toxicol. 2016 Aug;119(2):222-7. doi: 10.1111/bcpt.12554. Epub 2016 Feb 5. PMID: 26786042.

[8] Sulaj Z, Prifti E, Demiraj A, Strakosha A. Early Clinical Outcome of Acute Poisoning Cases Treated in Intensive Care Unit. Med Arch. 2015 Dec;69(6):400-4. doi: 10.5455/medarh.2015.69.400-404. PMID: 26843734; PMCID: PMC4720459.

[9] Malaviya NB, Parikh R, Pancholi K, Belim OB. Assessment of the Peradeniya Organophosphorus Poisoning Scale as a Severity and Prognostic Marker in Patients With Acute Organophosphorus Poisoning Presenting to an Emergency Medicine Department. Cureus. 2023 Jun 12;15(6):e40277. doi: 10.7759/cureus.40277. PMID: 37448435; PMCID: PMC10336367.

[10] Sumathi ME, Kumar SH, Shashidhar KN, Takkalaki N. Prognostic significance of various biochemical parameters in acute organophosphorus poisoning. Toxicol Int. 2014 May;21(2):167-71. doi: 10.4103/0971-6580.139800. PMID: 25253926; PMCID: PMC4170558.

[11] Matsuda K, Suzuki K, Ishihara S, Morinaga M, Okamoto M, Shiino Y, Horiuchi I, Tohyama K, Ichihara K. Assessment of the severity of organophosphate (fenitrothion) poisoning based on its serum concentration and clinical parameters. Clin Toxicol (Phila). 2011 Nov;49(9):820-7. doi: 10.3109/15563650.2011.617306. PMID: 22077246.

[12] Hiremath P, Rangappa P, Jacob I, Rao K. Pseudocholinesterase as a predictor of mortality and morbidity in organophosphorus poisoning. Indian J Crit Care Med. 2016 Oct;20(10):601-604. doi: 10.4103/0972-

5229.192052. PMID: 27829717; PMCID: PMC5073776.

[13] Sam KG, Kondabolu K, Pati D, Kamath A, Pradeep Kumar G, Rao PG. 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 Jul;16(5):239-47. doi: 10.1016/j.jflm.2008.12.004. Epub 2009 Jan 16. PMID: 19481704.

[14] Acharya A, Panda A. Clinical Epidemiology and Predictors of Outcome in Chlorpyrifos Poisoning in Farming and Allied Agricultural Workers in East Godavari, Andhra Pradesh. Indian J Occup Environ Med. 2022 Apr-Jun;26(2):116-121. doi: 10.4103/ijoem.ijoem_23_21. Epub 2022 Jul 4. PMID: 35991195; PMCID: PMC9384886.

[15] Raghapriya R, Dosi RV, Parmar A. Glycemic Status at the Time of Presentation in Acute Organophosphorous Poisoning and its Correlation with Severity and Clinical Outcome. J Assoc Physicians India. 2018 Aug;66(8):18-22. PMID: 31324078.

[16] Tao Y, Liu T, Han J, Jian X, Kan B. Clinical characteristics and treatment of mixed-pesticide poisoning in a patient: reflections on a particular case. J Int Med Res. 2020 Dec;48(12):300060520977392. doi: 10.1177/0300060520977392. PMID: 33356707; PMCID: PMC7768578.

[17] Dorandeu F, Mikler JR, Thiermann H, Tenn C, Davidson C, Sawyer TW, Lallement G, Worek F. Swine models in the design of more effective medical countermeasures against organophosphorus poisoning. Toxicology. 2007 Apr 20;233(1-3):128-44. doi: 10.1016/j.tox.2006.09.013. Epub 2006 Sep 29. PMID: 17092624.

[18] Bhattacharyya K, Phaujdar S, Sarkar R, Mullick OS. Serum creatine phosphokinase: a probable marker of severity in organophosphorus poisoning. Toxicol Int. 2011 Jul;18(2):117-23. doi: 10.4103/0971-6580.84263. PMID: 21976816; PMCID: PMC3183618.

[19] Liu HX, Liu CF, Yang WH. Clinical study of continuous micropump infusion of atropine and pralidoxime chloride for treatment of severe acute organophosphorus insecticide poisoning. J Chin Med Assoc. 2015 Dec;78(12):709-13. doi: 10.1016/j.jcma.2015.08.006. Epub 2015 Oct 3. PMID: 26441220.

[20] Davies JO, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM. 2008 May;101(5):371-9. doi: 10.1093/qjmed/hcn014. Epub 2008 Mar 4. PMID: 18319295; PMCID: PMC2493062.