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PROGNOSTIC SIGNIFICANCE OF SERIAL SERUM LACTATE DEHYDROGENASE LEVELS IN ACUTE ORGANOPHOSPHORUS POISONING: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Organophosphorus compound (OPC) poisoning is a major cause of morbidity and mortality globally. This study evaluated the prognostic utility of serum lactate dehydrogenase (LDH) levels in predicting clinical severity and outcomes in acute OPC poisoning.

Materials and Methods: A prospective observational study was conducted on 103 adults presenting within 6 hours of OPC exposure without prior treatment. Serum LDH was measured on admission (Day 1), Day 3, and Day 7. Clinical severity was classified using the Peradeniya Organophosphorus Poisoning (POP) scale. LDH levels were correlated with clinical severity, duration of ICU stay, and in-hospital mortality.

Results: The mean patient age was 37.16 ± 11.91 years, with a male predominance (63.1%) and predominantly incidental exposure (92.2%). Moderate poisoning was most common (41.7%), and the overall mortality rate was 19.4%. On Day 1, the mean LDH level was elevated (466.14 ± 121.31 U/L) and decreased over time in survivors, while remaining elevated or rising in severe cases. Non-survivors had significantly higher admission LDH levels than survivors (624.33 ± 129.47 vs. 428.03 ± 82.76 U/L, $p < 0.001$). ROC curve analysis demonstrated high predictive accuracy for mortality (AUC = 0.898, $p < 0.001$). Day 1 LDH levels moderately correlated with the duration of ICU stay ($r = 0.504$, $p < 0.001$) but showed no significant correlation with total hospital stay.

Conclusion: Serum LDH is a cost-effective, easily accessible, and reliable biomarker that correlates strongly with severity, ICU stay, and mortality in acute OPC poisoning.

Keywords: Lactate dehydrogenase (LDH), Organophosphorus compound poisoning, Peradeniya Organophosphorus Poisoning (POP) scale, ICU stay, Mortality.

INTRODUCTION

Organophosphorus compound (OPC) poisoning is a major clinical and public health concern worldwide, causing high rates of morbidity and mortality, especially in developing countries like India. Globally, pesticide poisoning is estimated to affect more than three million individuals annually, leading to over 220,000 to 300,000 deaths. Out of these, approximately 200,000 individuals succumb specifically to acute organophosphate pesticide exposure, with average mortality rates typically exceeding 15%.[1] The World Health Organization (WHO) reports that 99% of these fatal poisonings occur in low- and middle-income developing nations, primarily among agricultural workers and farmers who are in constant contact with these chemicals.[2]

In rural India, acute poisoning is currently ranked as the fourth leading cause of mortality, with local case

fatality rates fluctuating between 15% and 30%.[3] This high prevalence of acute exposure is heavily driven by the low cost, widespread agricultural availability, and ease of access to highly hazardous chemical compounds in rural households. Consequently, pesticide ingestion has emerged as one of the most common methods of intentional self-harm and suicidal attempts in low- and middle-income agricultural regions.[2]

Organophosphates represent a broad family of synthetic chemicals, defined structurally as esters, amides, or thiol derivatives of phosphoric, phosphonic, or phosphinic acids, which are widely utilized as insecticides, herbicides, and plasticizers.[3,4] The primary mechanism driving their acute human toxicity is the covalent, irreversible phosphorylation of the serine hydroxyl group at the active catalytic site of the enzyme acetylcholinesterase (AChE).[3,4] This phosphorylation forms a stable phosphorylated enzyme complex, rendering AChE incapable of hydrolyzing the neurotransmitter acetylcholine (ACh) into acetate and choline at cholinergic synapses[3]. Over time, this complex undergoes a conformational change known as "aging," which makes the enzyme inhibition permanent and resistant to pharmacological reactivation by standard oxime antidotes.[3]

The inactivation of AChE leads to a rapid, massive accumulation of acetylcholine at postganglionic parasympathetic, somatic neuromuscular, and central nervous system synapses.[3] The resulting persistent, long-lasting overstimulation of both muscarinic and nicotinic cholinergic receptors triggers a severe "cholinergic crisis".[3,4] Muscarinic overactivation typically manifests as bradycardia, pupillary constriction (miosis), bronchoconstriction, and systemic hypersecretions (excessive salivation, lacrimation, and bronchorrhea).[5] Nicotinic hyperstimulation at the autonomic ganglia and somatic neuromuscular junctions causes hypertension, tachycardia, muscle fasciculations, muscle weakening, and depolarizing neuromuscular blockade leading to flaccid paralysis.[3] Central nervous system toxicity manifests as confusion, agitation, generalized seizures, central respiratory drive depression, and coma.[3]

In clinical settings, acute respiratory failure is the primary cause of death in patients suffering from acute cholinergic crises.[6] This is typically induced by a combination of severe bronchorrhea ("drowning on dry land"), profound bronchoconstriction, paralysis of the intercostal muscles and diaphragm, and depression of the central respiratory center.[4] Furthermore, the clinical course can be complicated 1 to 4 days post-exposure by the "intermediate syndrome," which is characterized by the weakness and paralysis of neck, shoulder, and proximal limb muscles, often leading to delayed, life-threatening respiratory failure.[4]

To minimize mortality and guide early therapeutic interventions, rapid risk stratification and severity assessment are critical in emergency settings. Over the years, several scoring systems and physiological parameters have been employed to identify patients with poor prognoses, including the Glasgow Coma Scale (GCS), Acute Physiology and Chronic Health Evaluation (APACHE-II), Simplified Acute Physiology Score (SAPS), and red cell distribution width (RDW).[3] Additionally, plasma or serum cholinesterase (PChE) activity is widely measured to diagnose organophosphate exposure. However, many of these clinical scoring systems are highly complex to calculate, and debates continue regarding their reliability in forecasting early death.[3] Furthermore, while serum cholinesterase levels are a primary diagnostic marker, their correlation with clinical severity and prognosis is often weak and unreliable when used alone.

Therefore, clinical research has focused on identifying alternative, highly sensitive, and cost-effective biochemical indicators of cell damage to predict risk in acute poisonings.[7] Biochemical markers of tissue hypoxia and cellular damage, such as creatine phosphokinase (CPK), serum amylase, lipase, and lactate dehydrogenase (LDH), have shown clinical utility in predicting disease progression and respiratory failure.

Lactate dehydrogenase (LDH) is a group of NAD+-dependent tetrameric enzymes (EC 1.1.1.27) that catalyze the reversible interconversion of pyruvate and L-lactate in the anaerobic metabolic pathway.[8] Under standard physiological conditions, LDH is distributed extensively in almost all major body tissues, including the skeletal muscle, heart, liver, kidneys, and red blood cells.

During severe organophosphorus poisoning, the combination of cholinergic bronchoconstriction, systemic bronchorrhea, and muscle fasciculations causes profound cell hypoxia and tissue ischemia. When systemic oxygen is scarce, oxidative phosphorylation is blocked, and cells must shift their metabolism to anaerobic glycolysis to meet immediate cellular energy demands. This metabolic shift upregulates LDH to facilitate anaerobic energy production. The resulting cellular hypoxia, oxidative stress, and direct toxicity lead to functional muscle impairment, loss of cytoplasm, and cell membrane leakage, releasing large amounts of intracellular LDH into the systemic circulation.[9]

Serum levels of total LDH rise rapidly following tissue injury and can remain elevated for up to 7 days. Elevated baseline serum LDH levels have been directly correlated with severe organ injury, progressive clinical deterioration, and a higher risk of mortality in acute OPC poisoning. Clinical studies indicate that high LDH levels upon admission (frequently exceeding 800 U/L) are strongly associated with severe clinical presentations, respiratory failure, and the need for intensive care unit (ICU) admission or mechanical ventilation. Because serum LDH is a simple, cost-effective, and highly accessible biochemical assay, its baseline and serial measurement could serve as

an invaluable prognostic tool to guide clinical decision-making, particularly in resource-limited developing countries.[10]

Rationale and Objectives of the Study

While several biochemical markers are available, there are conflicting reports regarding the diagnostic efficacy of LDH, and its longitudinal serial trends in relation to patient recovery or mortality require clearer clinical evaluation. Therefore, the present prospective observational study was designed to systematically assess serum LDH levels in patients presenting with acute organophosphorus compound poisoning. The study aims to investigate the diagnostic accuracy of serial serum LDH levels (measured on admission Day 1, Day 3, and Day 7) and evaluate their correlation with initial clinical severity scores assessed using the Peradeniya Organophosphorus Poisoning (POP) scale, the duration of intensive care unit (ICU) stay, and overall in-hospital mortality.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational study was conducted over a period of one year in the Department of General Medicine at K.A.P.V. Medical College and Mahatma Gandhi Memorial Government Hospital, Tiruchirappalli.

Ethical Considerations

The study protocol was presented to and approved by the Institutional Ethics Committee of K.A.P.V. Government Medical College. Prior to participation, a comprehensive overview of the study details was provided to the patients, and informed written consent was secured from each patient or their legally authorized representative.

Study Population and Eligibility Criteria

The study population comprised patients presenting with an alleged history of acute organophosphorus compound (OPC) poisoning.

- **Inclusion Criteria:**
  - Patients aged 18 years or older.
  - Presentation within 6 hours of OPC exposure.
  - Patients or legal guardians willing to provide written consent.
  - No prior treatment received before hospital admission to prevent biochemical manipulation of baseline data.
- **Exclusion Criteria:**
  - Co-existing illnesses including myopathy, diabetes mellitus, chronic pancreatic diseases, chronic liver diseases, renal failure, myocardial infarction, myocarditis, malignancy, or pregnancy.
  - Exposure to mixed poisons or a completely different poison other than OPC.
  - History of chronic alcoholism.
  - Refusal to provide consent.

Sample Size and Sampling Technique

Non-probability consecutive sampling was used to recruit participants. The sample size was calculated using the single mean formula:

$$n = \frac{Z_{1-\alpha/2}^2 \cdot S^2}{L^2}$$

Where:

- \(n\) = required sample size.
- \(S\) = Standard deviation (estimated at 300.42 based on literature).
- \(L\) = Absolute precision at 10% (53.818 based on literature mean of 538.18).
- \(Z\) = 1.96 (for a 5% alpha error, two-sided).
- Statistical power = 80%.
- Non-response rate = 10%.

Applying these parameters, the minimum required sample size was determined to be **103 patients**.

Clinical Assessment and Severity Stratification

At the time of admission, a systematic, full-body clinical examination was performed on all symptomatic patients meeting the eligibility criteria. A pre-designed, semi-structured proforma was used to document baseline parameters:

- **Exposure History:** Time of exposure, specific OPC agent (if known), route of exposure, and estimated quantity ingested.
- **Clinical Examination:** Vital signs (blood pressure, heart rate, and respiratory rate) and physical clinical signs (pupil size, presence of fasciculations, and convulsions).
- **Neurological Status:** Consciousness level assessed using the Glasgow Coma Scale (GCS).

The clinical severity of the poisoning was classified at admission using the Peradeniya Organophosphorus Poisoning (POP) scale, grading patients into three clinical severities:

- **Mild Grade:** POP score of 0 to 3
- **Moderate Grade:** POP score of 4 to 7
- **Severe Grade:** POP score of 8 to 11

Laboratory Investigations and LDH Assay Protocol

Venipuncture was performed under sterile conditions to collect baseline blood samples for a complete blood count (CBC), random blood sugar, blood urea, serum creatinine, SGOT, and SGPT.

Serum lactate dehydrogenase (LDH) was measured on admission (Day 1) and serially repeated on Day 3 and Day 7. The biochemical assay was conducted using the following protocol:

- **Assay Method:** Serum LDH activity was evaluated spectrophotometrically at 340 nm based on its catalytic function—specifically, the reversible oxidation of L-lactate to pyruvate by the hydrogen acceptor \(NAD^+\). The change in the sample's optical density was monitored as \(NADH\) was generated.
- **Handling Precautions:** Serum and plasma samples were handled with extreme care to avoid hemolysis, as ruptured erythrocytes

release intracellular LDH, causing a false increase in measured enzyme levels.

- **Reference Ranges:** The normal reference range of serum LDH in adults is typically 122 to 222 U/L. For operational definition within this study, LDH levels were categorized as normal (220–460 U/L) or elevated (>460 U/L).

Study Variables

- **Dependent Variables:** Clinical severity grade based on the POP scale at admission, serial serum LDH levels (Days 1, 3, and 7), and in-hospital mortality.
- **Independent Variables:** Patient age, gender, and clinical presentation details.

Statistical Analysis

Data were entered into Microsoft Excel 2019 and analyzed using SPSS versions 21.0 and 26.0.

- **Descriptive Statistics:** Normally distributed continuous variables were expressed as mean ± standard deviation (SD), and skewed continuous variables as median with interquartile range (IQR). Categorical variables were expressed as absolute frequencies and percentages.
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- **Inferential Statistics:**
  - To compare mean values of continuous variables between two groups (e.g., survivors vs. non-survivors), the independent samples t-test was used for normal distributions, and the Mann-Whitney U test for non-normal distributions.
  - To compare values across three severity categories, one-way ANOVA or the Kruskal-Wallis test was employed based on normality.
  - Categorical associations were evaluated using the Chi-square test or Fisher's exact test (when expected cell counts were less than five).
  - Correlation analyses between continuous clinical metrics/severity scores and serum LDH levels were performed using Pearson's correlation coefficient (for parametric data) or Spearman's rank correlation (for non-parametric data).

The predictive efficacy of Day 1 LDH levels for mortality was evaluated using Receiver Operating Characteristic (ROC) curve analysis to determine the Area Under the Curve (AUC), sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).

RESULTS

Table 1: Socio-demographic Profile and Toxicological Exposure Details of Patients (n = 103)

Parameter	Sub-category	Frequency (n) / Mean ± SD	Percentage (%) / Range
Age (Years)	Continuous Profile	37.16 ± 11.91 (Median: 36)	19 – 61 years
Gender	Male	65	63.1%
	Female	38	36.9%
Type of Exposure	Incidental (Suicidal)	95	92.2%
	Accidental	8	7.8%
OPC Agent Implicated	Methyl parathion	53	51.5%
	Monocrotophos	13	12.6%
	Chlorpyrifos	9	8.7%
	Methamidophos	9	8.7%
	Fenthion	7	6.8%
	Quinalphos	7	6.8%
	Diazinon	5	4.9%

The demographic analysis of the study population (n = 103) demonstrates a predominance of young and middle-aged adults, with a mean age of 37.16 ± 11.91years and a median age of 36 years. There is a notable male predominance, with men accounting for 63.1% (n = 65) of the cases compared to 36.9% (n = 38) for women. Regarding the toxicological characteristics, the primary mechanism of exposure is incidental (intentional or self-harm-related), comprising 92.2% (n = 95) of the cohort, while accidental exposure occurs in only 7.8% (n = 8) of cases. Methyl parathion was the most common causative organophosphorus agent, responsible for more than half of the poisoning cases (51.5%, (n = 53)), followed by Monocrotophos (12.6%), Chlorpyrifos (8.7%), and Methamidophos (8.7%).

Table 2: Clinical Characteristics and Severity Grade (POP Scale) at Admission (n = 103)

Clinical Parameter	Clinical Finding	Frequency (n)	Percentage (%)
Vomiting	Present (≥1 episode)	92	89.3%
	Absent	11	10.7%
Seizures	Present	2	1.9%
	Absent	101	98.1%
Fasciculations	Present	40	38.8%
	Absent	63	61.2%
Pupil Size	< 2 mm (Moderate miosis)	40	38.8%
	Pinpoint pupils	31	30.1%
	≥2 mm (Normal/Mydriasis)	32	31.1%
Heart Rate	Bradycardia (< 40 bpm)	31	30.1%
	Bradycardia (40 – 59 bpm)	8	7.8%

	Normal/Tachycardia	64	62.1%
Respiratory Rate	< 20 breaths per minute	16	15.5%
	≥20 breaths per minute	64	62.1%
	≥20 breaths/min with Central Cyanosis	23	22.3%
Glasgow Coma Scale (GCS)	Awake (Fully conscious)	55	53.4%
	Altered (Does not obey oral commands)	24	23.3%
	No response (Comatose)	24	23.3%
POP Severity Grade	Mild (Score 0 – 3)	40	38.8%
	Moderate (Score 4 – 7)	43	41.7%
	Severe (Score 8 – 11)	20	19.4%

At admission, vomiting is the most prevalent symptom, occurring in 89.3% (n = 92) of the study participants, whereas seizures are rarely documented (1.9%, (n = 2)). Classical cholinergic indicators are common: active muscle fasciculations are noted in 38.8% of patients (n = 40), and abnormal pupillary constriction (pupils < 2 mm or pinpoint) is present in 68.9% of cases (n = 71). Cardiovascular assessment reveals that 37.9% of patients present with bradycardia (including 30.1% with severe bradycardia < 40 bpm), while 62.1% maintain a heart rate ≥60 bpm. Significant respiratory distress ≥20 breaths/min) is noted in 84.4% of the cohort, with 22.3% (n = 23) experiencing severe respiratory compromise accompanied by central cyanosis. Neurologically, 46.6% of patients exhibit altered sensorium at presentation. Based on the Peradeniya Organophosphorus Poisoning (POP) scale, the cohort is classified as 38.8% mild, 41.7% moderate, and 19.4% severe.

Table 3: Comparative Analysis of Serial Serum LDH Levels (U/L) on Day 1 and Day 3 by Severity

Severity Category	Day 1 Serum LDH (U/L) (Mean ± SD)	Day 3 Serum LDH (U/L) (Mean ± SD)	Intra-group Trend (Day 1 to Day 3)	Inter-group ANOVA *p*-value
Mild (n = 40)	360.70 ± 17.72	269.90 ± 17.49	Decreased (↓ 90.80 U/L)	**< 0.001** (Day 1)
Moderate (n = 43)	490.65 ± 68.91	380.34 ± 53.93	Decreased (↓110.31 U/L)	**< 0.001** (Day 3)
Severe (n = 20)	624.33 ± 129.46	686.86 ± 145.06	Increased (↑ 62.53 U/L)	

Analysis of serial serum LDH concentrations demonstrates a statistically significant upward trend in enzyme activity corresponding to increased clinical severity at both Day 1 and Day 3 ($p < 0.001$). At admission (Day 1), patients categorized with severe poisoning exhibit a mean LDH of 624.33 ± 129.46 U/L, which is substantially higher than the moderate (490.65 ± 68.91U/L) and mild (360.70 ± 17.72U/L) cohorts. Interestingly, the longitudinal behaviour of serum LDH differs dramatically based on severity: while mild and moderate cases show a clear reduction in mean LDH levels from Day 1 to Day 3, severe cases demonstrate a progressive increase, rising from 624.33 ± 129.47**U/L to** 686.86 ± 145.06**U/L**. Post hoc analysis confirms that all pairwise differences (mild vs. moderate, mild vs. severe, and moderate vs. severe) are highly significant ($p < 0.001$) at both time points.

Table 4: Relationship of Serial Serum LDH Levels (U/L) with Patient Mortality (Survivors vs. Non-Survivors)

Serum LDH Measurement	Mortality / Non-Survivors (n = 20)	No Mortality / Survivors (n = 83)	Mean Difference (U/L)	Independent *t*-test Value	*p*-value
Day 1 LDH (Mean ± SD)	624.33 ± 129.47	428.03 ± 82.76	196.30	6.47	**< 0.001**
Day 3 LDH (Mean ± SD)	686.86 ± 145.06	327.12 ± 68.69	359.74	10.80	**< 0.001**

The overall in-hospital mortality rate for the study cohort is 19.4% (n = 20). Serum LDH levels serve as a robust marker for predicting fatal outcomes, with non-survivors exhibiting significantly higher serum LDH values compared to survivors. On Day 1, the mean LDH among non-survivors is 624.33 ± 129.47U/L, compared to 428.03 ± 82.76U/L in survivors, representing a highly significant mean difference of 196.30 U/L ($p < 0.001$). By Day 3, this diagnostic gap widens dramatically to 359.74 U/L ($t = 10.80$, $p < 0.001$), as survivors display a therapeutic reduction in enzyme activity (327.12 ± 68.69 U/L), whereas non-survivors demonstrate rising serial levels up to 686.86 ± 145.06U/L.

Table 5: ROC Curve Metrics and Correlation Outcomes for Prognostic Efficacy

A. Receiver Operating Characteristic (ROC) Analysis for Day 1 LDH Predicting Mortality

Parameter	Statistical Value	Standard Error	*p*-value (Asymptotic Sig.)	Asymptotic 95% Confidence Interval
Area Under Curve (AUC)	**0.898**	0.032	< 0.001 (0.000)	0.835 – 0.960

B. Correlation of Admission (Day 1) Serum LDH with Clinical Resource Utilization

Clinical Outcome Measured	Mean ± SD of Outcome	Correlation Coefficient (*r*)	Statistical Significance (*p*-value)	Clinical Interpretation
Duration of ICU Stay	2.64 ± 1.75 days	**+0.504**	**< 0.001**	Moderate positive correlation; highly significant
Duration of Hospital Stay	6.54 ± 1.51 days	+0.124	0.212	Weak positive correlation; statistically non-significant

To assess the performance of baseline serum LDH as a prognostic classifier, a ROC curve analysis was performed. The Area Under the Curve (AUC) for Day 1 LDH is 0.898 ± 0.032 (95% CI: 0.835 to 0.960; $p < 0.001$), which indicates excellent diagnostic accuracy in predicting mortality and effectively differentiating survivors from non-survivors. Additionally, baseline serum LDH shows a moderate, highly significant positive correlation with the duration of ICU stay ($(r = 0.504)$, $p < 0.001$), indicating that patients with higher initial LDH levels require more prolonged intensive care. Conversely, the correlation between admission LDH and the total duration of hospital stay is weak and fails to reach statistical significance ($(r = 0.124)$, $p = 0.212$).

DISCUSSION

Socio-demographic Profile and Toxicological Exposure Details

The demographic profile of the 103 patients in our study revealed a mean age of 37.16 ± 11.91 years, with a significant male predominance (63.1%). This male-dominated trend is highly consistent with other clinical evaluations of acute organophosphorus compound (OPC) poisoning in India. For instance, Reddy CM et al[11] reported a male-to-female ratio of 60% to 40% with a mean age of 42.7 years, Kumar M et al[12]. observed a male predominance of 70%, and Raghu et al[13] documented a higher incidence of poisoning among males, particularly in the active age group of 20 to 30 years.

Regarding age distribution, the mean age in our study was slightly higher than in other publications. Islam et al[14]. reported a mean age of 28.7 ± 12.8 years, while Elnagdy and Shehta,[15] documented a mean age of 26.5 years. Kumar M et al,[12] also observed that 76% of poisoned patients were under 30 years of age, indicating the susceptibility of younger cohorts. The relatively higher mean age in our study may represent regional and socio-demographic variations.

In terms of exposure patterns, incidental (predominantly intentional self-harm) exposure comprised 92.2% of our cases. This high rate of intentional self-harm is consistent with Kumar M et al,[12] who reported 84% suicidal cases. Occupational factors play a substantial role, as agricultural populations in rural India have easy access to pesticides.

In our study, Methyl parathion was the most common compound implicated (51.5%). This is in agreement with Methyl parathion is a highly toxic, lipid-soluble organophosphate that inhibits acetylcholinesterase, leading to severe cholinergic crises. Its lipophilic nature enables rapid accumulation in adipose tissue, resulting in extended toxicity, severe clinical manifestations, and poorer outcomes, which explains the high proportion of moderate-to-severe cases in our cohort.

Clinical Characteristics and Severity Stratification (POP Scale)

The clinical presentation of acute OPC poisoning in our cohort was characterized by classical cholinergic signs: vomiting was the most common symptom (89.3%), miosis (pupils < 2 mm or pinpoint) occurred in 68.9% of cases, active muscle fasciculations were noted in 38.8%, and altered sensorium was observed in 46.6%. These findings are highly congruent with the clinical patterns reported by Raghu et al[13], who observed miosis in 87%, bradycardia in 67%, muscle fasciculations in 37.2%, and altered sensorium in 31.9% of patients, alongside significant respiratory involvement.

In contrast, Islam et al,[14] noted a slightly different distribution, where pinpoint pupils occurred in 41.4% of subjects, muscle fasciculations were rare, and most patients maintained heart rates above 60 beats per minute. They also reported that 41.4% of their subjects were fully conscious, indicating a milder clinical profile compared to our cohort. The higher frequency of altered sensorium and severe respiratory signs (22.3% with central cyanosis) in our study points toward a more severe cohort.

Using the Peradeniya Organophosphorus Poisoning (POP) scale for severity stratification, 38.8% of our patients had mild poisoning, 41.7% had moderate, and 19.4% had severe poisoning. These severity proportions are closely aligned with those of Raghu et al,[13] who reported 45% mild, 35% moderate, and 20% severe cases.

However, other researchers have documented milder severity distributions. Islam et al[14] observed a higher proportion of mild cases (55.7%) and only 7.1% severe cases, which may suggest earlier presentation or milder toxicity in their study area. Similarly, Dubey et al.[10] reported a much milder

distribution of 68% mild, 27% moderate, and only 5% severe cases. The higher proportion of moderate and severe cases in our study may reflect delayed arrival at our tertiary center, ingestion of larger toxic doses, or the specific use of highly potent agents like methyl parathion.

In-Hospital Mortality Rates

The overall in-hospital mortality rate in our study was 19.4%. This is highly comparable to the mortality rate of 22% reported by Sen et al[9] in Odisha.

However, our mortality rate is higher than that observed by Raghu et al,[13] (12.8%) and Bhojashettar et al,[7] (12.8%). It is dramatically higher than the 2.9% mortality rate reported by Islam et al.[14] The higher mortality in our study is attributable to several factors: a higher percentage of patients presenting with moderate-to-severe poisoning, delayed hospital arrival, and a high incidence of respiratory failure and central nervous system depression. Delayed admission was also noted as a key factor in mortality by Prakash et al,[16] who reported an 8% mortality rate, with 75% of fatalities occurring in patients with severe poisoning and a delay of over 4 hours in hospital admission.

Longitudinal Trends in Serum LDH Levels

Our study showed highly elevated serum LDH levels upon admission (mean: 466.14 ± 121.31 U/L), which gradually declined to 396.97 ± 167.81 U/L on Day 3 overall. This indicates that LDH levels are highest during the acute phase and decline as the clinical condition improves with treatment. This trend is consistent with Kumar M et al,[12] who reported mean LDH values of 597.98 ± 335.44 U/L on Day 1 and 510.81 ± 380.05 U/L on Day 3. Similar baseline elevations and trends were reported by Nagar et al.[6]

The pathophysiological basis of elevated serum LDH in OPC poisoning is related to cellular damage, tissue hypoxia, and muscle injury. Organophosphates block acetylcholinesterase, leading to cholinergic overstimulation, bronchorrhea, and bronchospasm, which induce severe tissue hypoxia. To meet energy demands under hypoxic conditions, cells must upregulate anaerobic glycolysis, leading to lactic acid fermentation and cell membrane leakage, releasing intracellular enzymes like LDH into the bloodstream. This is further supported by Agarwal et al,[5] who reported significant increases in serum LDH and creatine kinase (CK) activities due to muscle dysfunction and cellular damage.

Correlation of Serum LDH with Clinical Severity

We observed a statistically significant correlation between serial serum LDH levels and the severity of poisoning as assessed by the POP scale ($p < 0.001$). On Day 1, mean LDH rose from 360.70 ± 17.72 U/L in mild cases to 490.65 ± 68.91 U/L in moderate cases and 624.33 ± 129.46 U/L in severe cases. On Day 3, mild and moderate cases showed a significant decrease, whereas severe cases showed a progressive rise to 686.86 ± 145.06 U/L. This demonstrates that serial LDH is a valuable marker of disease progression.

Reddy CM et al,[11] reported a similar positive trend, with mean LDH levels of 219.5 U/L in mild, 401.2 U/L in moderate, and 694.3 U/L in severe cases, demonstrating a significant increase with severity. Similarly, Raghu et al,[13] and Bhojashettar et al,[7] observed a strong, statistically significant positive correlation between LDH levels and Peradeniya scores, with Bhojashettar et al.[21] reporting mean LDH values of 583.48, 812, and 1482.89 U/L in mild, moderate, and severe groups, respectively.

However, some studies show conflicting findings. Sen et al,[9] found mean LDH values of 329, 480, and 497 U/L in mild, moderate, and severe cases, but the correlation was weak and did not reach statistical significance (r = 0.291). Islam et al[14] also reported a weak, non-significant correlation with POP scores (r = 0.048, $p = 0.068$). Furthermore, Elnagdy and Shehta,[15] observed no statistically significant differences in serum LDH levels across mild, moderate, and severe groups at admission and Day 4. These discrepancies among studies could be explained by differences in sample sizes, variations in the time elapsed between ingestion and admission, and the timing of blood sample collection.

Efficacy of Serum LDH in Predicting Mortality (Survivors vs. Non-Survivors)

Our study highlighted that serum LDH was significantly higher in non-survivors than survivors on both Day 1 (624.33 ± 129.47 vs. 428.03 ± 82.76 U/L, $p < 0.001$) and Day 3 (686.86 ± 145.06 vs. 327.12 ± 68.69 U/L, $p < 0.001$). The ROC curve analysis for Day 1 LDH demonstrated excellent prognostic value for mortality, with an Area Under the Curve (AUC) of 0.898 ($p < 0.001$).

These findings are highly consistent with Kumar M et al[12]. and Nagar et al,[6] who reported significantly elevated LDH in non-survivors on Day 1 (810 ± 372.99 U/L) and Day 3 (1027.09 ± 458.26 U/L) compared to survivors on those same days (538.18 ± 300.42 and 365.19 ± 175.49 U/L). Reddy CM et al[11] also supported the prognostic role of LDH, reporting a high ROC AUC of 0.82.

In contrast, Sen et al[9] and Elnagdy and Shehtae[15] found that serum LDH levels did not differ significantly between survivors and non-survivors. Similarly, Bhojashettar et al,[7] reported that while other biochemical markers like pseudo cholinesterase were strongly associated with mortality, the association with serum LDH did not reach statistical significance. These variations may stem from different patient demographics, the severity of exposure, and early clinical intervention.

Resource Utilization: Correlation with ICU and Hospital Stay

Lastly, we found a moderate, highly significant positive correlation between admission serum LDH levels and the duration of ICU stay (r = 0.504, $p < 0.001$), while the correlation with total hospital stay

was weak and statistically non-significant (r = 0.124, $p = 0.212$). This is in line with Reddy CM et al[11]. and Bhojashettar et al,[7] who noted that patients requiring critical care and mechanical ventilation had significantly higher than normal levels of LDH. This indicates that baseline serum LDH can help clinicians predict the severity of the poisoning, the clinical necessity for intensive care unit monitoring, and the likelihood of respiratory failure. On the other hand, the total duration of hospital stay is influenced by a broader array of variables, including psychiatric counselling, recovery speed, and social discharge criteria, which explains the lack of correlation with initial biochemical indices.

CONCLUSION

This study demonstrates that serum lactate dehydrogenase (LDH) is a highly dependable, cost-effective, and easily accessible prognostic biomarker for patients presenting with acute organophosphorus compound (OPC) poisoning, particularly in resource-limited clinical settings. Serum LDH levels are significantly elevated at admission and exhibit a strong, direct correlation with poisoning severity as classified by the Peradeniya Organophosphorus Poisoning (POP) scale. Longitudinal monitoring reveals a distinct prognostic trend: while enzyme levels steadily decline over time in survivors, they remain persistently elevated or continue to rise in severe cases and non-survivors. Backed by excellent predictive accuracy for mortality (AUC = 0.898) and a moderate positive correlation with the duration of intensive care unit (ICU) stay (r = 0.504), serial serum LDH monitoring serves as an invaluable clinical tool for early risk stratification, anticipating critical care resource needs, and forecasting patient outcomes in the acute phase of care

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