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Association Between Serum Lactate Levels and Mortality in Acute Poisoning Cases: A Prospective Observational Study

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KEYWORDS

Acute poisoning; Serum lactate; Mortality; Prognostic biomarker; Lactate clearance

ABSTRACT:

Background: Acute poisoning represents a significant global health concern with substantial morbidity and mortality. Early prognostication is crucial for appropriate resource allocation and aggressive management. Serum lactate, a marker of tissue hypoperfusion and hypoxia has shown promise as a predictor of outcomes in various critical illnesses. This study aimed to evaluate the association between serum lactate levels and mortality in patients with acute poisoning.

Methodology: A prospective observational study was conducted on 324 patients with acute poisoning admitted to the emergency department of a tertiary care hospital between January 2024 and December 2024. Serum lactate levels were measured at admission and 6 hours post-admission. Demographic data, poisoning characteristics, clinical parameters, and outcome measures were recorded. Receiver operating characteristic (ROC) curve analysis was performed to determine optimal lactate cutoff values for predicting mortality. Multivariate logistic regression was used to identify independent predictors of mortality.

Results: The overall mortality rate was 12.3% (40/324). The mean initial serum lactate level was significantly higher in non-survivors compared to survivors (7.9 ± 3.2 mmol/L vs. 2.8 ± 1.9 mmol/L, $p<0.001$). Lactate clearance at 6 hours was significantly lower in non-survivors (15.3 ± 8.7% vs. 38.6 ± 15.2%, $p<0.001$). ROC curve analysis yielded an area under curve (AUC) of 0.86 (95% CI: 0.79-0.93) for initial lactate levels, with an optimal cutoff value of 4.5 mmol/L (sensitivity 84.6%, specificity 79.3%). Multivariate analysis identified initial serum lactate ≥4.5 mmol/L (OR 5.87, 95% CI: 2.31-14.92, $p<0.001$) and lactate clearance <20% at 6 hours (OR 4.23, 95% CI: 1.82-9.84, $p=0.001$) as independent predictors of mortality, after adjusting for age, toxin type, time to presentation, and Glasgow Coma Scale score.

Conclusion: Elevated serum lactate levels at admission and poor lactate clearance at 6 hours are significantly associated with increased mortality in acute poisoning cases. Serum lactate could serve as a valuable prognostic biomarker for risk stratification and early intervention in poisoning management.

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INTRODUCTION

Acute poisoning constitutes a significant global health challenge, accounting for an estimated 1.6 million deaths annually worldwide (World Health Organization, 2020). It represents a substantial burden on healthcare systems, with significant morbidity, mortality, and resource utilization. The clinical manifestations and outcomes of poisoning vary considerably depending on the toxin involved, dose, route of exposure, time to presentation, and individual patient characteristics (Yates, 2016). Early identification of high-risk patients is crucial for appropriate triage, resource allocation, and implementation of aggressive therapeutic interventions. Several prognostic scoring systems have been developed for poisoned patients, including the Poisoning Severity Score (PSS) and the Acute Physiology and Chronic Health Evaluation II (APACHE II) score. However, these systems often require multiple clinical and laboratory parameters, some of which may not be readily available in emergency settings (Liisanantti et al., 2018). There is a growing interest in identifying simple, readily available biomarkers that can predict outcomes in poisoned patients. Serum lactate has emerged as a potential prognostic tool across various critical illnesses. Lactate is produced during anaerobic metabolism when oxygen delivery is inadequate to meet tissue demands, reflecting tissue hypoxia and hypoperfusion (Vincent et al., 2016). In sepsis, trauma, and cardiac arrest, elevated lactate levels have been consistently associated with increased mortality and adverse outcomes (Nichol et al., 2015).

In acute poisoning, several mechanisms may contribute to elevated lactate levels. Direct mitochondrial toxicity by poisons such as cyanide, carbon monoxide and metformin can impair aerobic metabolism and increase lactate production. Seizures, hypotension, and respiratory depression common complications in poisoning can lead to tissue hypoxia and lactate accumulation. Moreover, increased sympathetic stimulation from substances like cocaine and amphetamines can enhance glycolysis and lactate production even in the presence of adequate oxygenation, a phenomenon known as type B lactic acidosis (Kraut & Madias, 2016). While the prognostic value of lactate has been established in various critical illnesses, its utility specifically in acute poisoning has received relatively less attention. A few studies have suggested that elevated lactate levels may be associated with poor outcomes in specific poisonings, such as carbon monoxide (Cervellin et al., 2014), metformin (Vecchio & Protti, 2011), and metanol (Zakharov et al., 2015). However, comprehensive data on the prognostic value of lactate across various types of poisonings remains limited. Additionally, the concept of lactate clearance—the percentage decrease in lactate levels over time—has gained attention as a potential marker of response to resuscitation and treatment. Failure to clear lactate has been associated with increased mortality in sepsis and trauma (Nguyen et al., 2004). However, the prognostic significance of lactate clearance in acute poisoning has not been well established. This study aims to evaluate the association between serum lactate levels at admission, lactate clearance at 6 hours and mortality in patients with acute poisoning. We hypothesize that elevated initial lactate levels and poor lactate clearance are associated with increased mortality in these patients.

METHODOLOGY

A prospective observational study was conducted at the Emergency Department of NIMS Medical College and Hospital, Jaipur, a tertiary care hospital with a dedicated toxicology unit. The study period extended from January, 2024 to December, 2024. Inclusion criteria included adult patients (≥18 years) presenting within 24 hours of acute poisoning confirmed via history, clinical signs, or toxicological screening. Exclusion criteria were chronic poisoning, pregnancy, conditions affecting lactate metabolism (e.g., liver/renal failure, malignancy, diabetic ketoacidosis), prior administration of fluids or medications before lactate measurement, and patients lost to follow-up or referred elsewhere. Using prior data showing a 10% mortality rate in acute poisoning and a 2.5 mmol/L difference in serum lactate levels between survivors and non-survivors (SD = 3.0), a sample size of 324 was calculated to ensure 90% power at a 5% significance level.

Data Collection

Collected data included demographics (age, gender, weight), type and route of poison exposure, intent (accidental or intentional), time to presentation, vital

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signs at admission, Glasgow Coma Scale (GCS) score, need for mechanical ventilation or vasopressors, hospital stay duration, and outcome. Laboratory tests included serum lactate levels (at admission and 6 hours later), arterial blood gases, complete blood count, liver/renal function tests, coagulation profile, and specific toxicology screens.

Serum Lactate Measurement

Arterial blood samples were taken on admission and at 6 hours, prior to major interventions using a point-of-care analyzer (ABL800 FLEX). Lactate clearance was calculated using:

[(initial lactate − 6-hour lactate) / initial lactate] × 100%

Patient Management

Treatment followed standard protocols including decontamination, antidotes where applicable, supportive care and organ support. Although clinicians had access to lactate levels, treatment decisions were protocol-driven and not based on lactate readings alone.

Statistical Analysis

Data were analyzed using SPSS v26.0. Continuous variables were expressed as mean ± SD or median (IQR) and categorical data as frequencies and percentages. Normality was tested using the Shapiro-Wilk test. Survivors and non-survivors were compared using t-tests, Mann-Whitney U, chi-square or Fisher's exact tests as appropriate. ROC analysis identified optimal cutoff values for lactate levels and clearance in predicting mortality. Logistic regression (univariate and multivariate) identified independent predictors of mortality, with significance set at $p < 0.05$.

RESULTS

During the study period, 386 patients with acute poisoning were admitted to the emergency department. After applying the exclusion criteria, 324 patients were included in the final analysis. The overall mortality rate was 12.3% (40/324).

Table 1: Demographic and Clinical Characteristics of Study Population

Characteristic	All Patients (n=324)	Survivors (n=284)	Non-survivors (n=40)	p-value
Age (years), mean ± SD	34.6 ± 15.2	33.8 ± 14.9	40.2 ± 16.3	0.012
Gender, n (%)				0.643
Male	187 (57.7)	162 (57.0)	25 (62.5)	
Female	137 (42.3)	122 (43.0)	15 (37.5)	
Type of poison, n (%)				<0.001
Pharmaceutical drugs	138 (42.6)	131 (46.1)	7 (17.5)	
Pesticides	92 (28.4)	68 (23.9)	24 (60.0)	
Household products	46 (14.2)	42 (14.8)	4 (10.0)	
Industrial chemicals	28 (8.6)	24 (8.5)	4 (10.0)	
Plant/mushroom	12 (3.7)	11 (3.9)	1 (2.5)	
Unknown	8 (2.5)	8 (2.8)	0 (0.0)	
Route of exposure, n (%)				0.024
Ingestion	284 (87.7)	250 (88.0)	34 (85.0)	
Inhalation	24 (7.4)	22 (7.7)	2 (5.0)	
Dermal	10 (3.1)	9 (3.2)	1 (2.5)	

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Injection	6 (1.9)	3 (1.1)	3 (7.5)	
Intent, n (%)				0.183
Intentional	238 (73.5)	205 (72.2)	33 (82.5)	
Accidental	86 (26.5)	79 (27.8)	7 (17.5)	
Time to presentation (hours), median (IQR)	4.5 (2.0-8.0)	4.0 (2.0-7.0)	6.5 (3.5-12.0)	0.004
GCS score at admission, median (IQR)	13 (9-15)	14 (10-15)	8 (6-11)	<0.001
Mechanical ventilation, n (%)	72 (22.2)	38 (13.4)	34 (85.0)	<0.001
Vasopressor support, n (%)	56 (17.3)	27 (9.5)	29 (72.5)	<0.001
Length of stay (days), median (IQR)	3.0 (2.0-6.0)	3.0 (2.0-5.0)	4.5 (1.0-8.0)	0.324

SD: Standard Deviation; IQR: Interquartile Range; GCS: Glasgow Coma Scale

Non-survivors were significantly older than survivors (40.2 ± 16.3 vs. 33.8 ± 14.9 years, p=0.012). Pesticide poisoning was significantly more common among non-survivors compared to survivors (60.0% vs. 23.9%, p<0.001), while pharmaceutical drug poisoning was more frequent among survivors (46.1% vs. 17.5%). Although ingestion was the most common route of exposure in both groups, injection was proportionally more frequent among non-survivors (7.5% vs. 1.1%, p=0.024). Non-survivors had a longer time from exposure to hospital presentation (median 6.5 vs. 4.0 hours, p=0.004) and lower GCS scores at admission (median 8 vs. 14, p<0.001). A significantly higher proportion of non-survivors required mechanical ventilation (85.0% vs. 13.4%, p<0.001) and vasopressor support (72.5% vs. 9.5%, p<0.001).

Table 2: Laboratory Parameters of Survivors and Non-survivors (mean ± SD)

Parameter	Survivors (n=284)	Non-survivors (n=40)	p-value
Initial serum lactate (mmol/L)	2.8 ± 1.9	7.9 ± 3.2	<0.001
6-hour serum lactate (mmol/L)	1.7 ± 1.2	6.5 ± 3.6	<0.001
Lactate clearance (%)	38.6 ± 15.2	15.3 ± 8.7	<0.001
pH	7.38 ± 0.08	7.29 ± 0.12	<0.001
Bicarbonate (mmol/L)	21.4 ± 4.2	17.6 ± 5.3	<0.001
Base excess (mmol/L)	-3.2 ± 4.6	-8.5 ± 6.1	<0.001
White blood cell count (×10³/μL)	11.2 ± 4.5	14.8 ± 6.2	<0.001
Hemoglobin (g/dL)	13.4 ± 2.0	13.0 ± 2.2	0.257
Platelet count (×10³/μL)	242 ± 78	213 ± 92	0.042
Blood urea nitrogen (mg/dL)	28.6 ± 12.3	42.5 ± 18.7	<0.001
Creatinine (mg/dL)	0.9 ± 0.4	1.6 ± 0.8	<0.001
AST (U/L), median (IQR)	32 (22-48)	64 (42-124)	<0.001
ALT (U/L), median (IQR)	28 (19-42)	57 (38-98)	<0.001
INR	1.2 ± 0.3	1.8 ± 0.7	<0.001

SD: Standard Deviation; IQR: Interquartile Range; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; INR: International Normalized Ratio

Initial serum lactate levels were significantly higher in non-survivors compared to survivors (7.9 ± 3.2 vs. 2.8 ± 1.9 mmol/L, p<0.001). Similarly, 6-hour lactate levels remained significantly elevated in non-survivors (6.5 ± 3.6 vs. 1.7 ± 1.2 mmol/L, p<0.001). Lactate clearance at 6 hours was significantly lower in non-survivors (15.3 ±

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8.7% vs. 38.6 ± 15.2%, p<0.001). Non-survivors also exhibited more pronounced acidosis with lower pH (7.29 ± 0.12 vs. 7.38 ± 0.08, p<0.001), lower bicarbonate levels (17.6 ± 5.3 vs. 21.4 ± 4.2 mmol/L, p<0.001), and more negative base excess (-8.5 ± 6.1 vs. -3.2 ± 4.6 mmol/L, p<0.001). Laboratory markers of organ dysfunction, including renal function (blood urea nitrogen, creatinine), liver function (AST, ALT), and coagulation (INR), were significantly worse in non-survivors.

Table 3: Initial Serum Lactate Levels according to Toxin Type

Toxin Type	Initial Lactate (mmol/L)	Mortality Rate, n (%)
Pesticides	5.4 ± 3.4	24 (26.1)
Organophosphates	6.2 ± 3.5	16 (33.3)
Carbamates	4.8 ± 2.9	5 (27.8)
Rodenticides	3.8 ± 2.5	2 (14.3)
Others	4.3 ± 3.1	1 (8.3)
Pharmaceutical drugs	2.4 ± 1.8	7 (5.1)
Benzodiazepines	1.9 ± 1.2	0 (0.0)
Antidepressants	2.6 ± 1.9	2 (5.9)
Paracetamol	2.3 ± 1.5	1 (3.6)
Antipsychotics	3.1 ± 2.0	2 (11.1)
Others	2.7 ± 2.2	2 (12.5)
Household products	2.9 ± 2.1	4 (8.7)
Corrosives	3.2 ± 2.4	3 (12.5)
Detergents	2.4 ± 1.6	0 (0.0)
Others	2.8 ± 1.9	1 (12.5)
Industrial chemicals	3.8 ± 2.6	4 (14.3)
Carbon monoxide	4.5 ± 2.9	2 (16.7)
Ethylene glycol	3.6 ± 2.4	1 (12.5)
Others	2.9 ± 1.8	1 (12.5)
Plant/mushroom	3.3 ± 2.2	1 (8.3)
Unknown	3.0 ± 2.0	0 (0.0)

The highest mean serum lactate levels were observed in pesticide poisonings (5.4 ± 3.4 mmol/L), particularly organophosphates (6.2 ± 3.5 mmol/L), which also had the highest mortality rate (33.3%). Pharmaceutical drug poisonings had the lowest mean lactate levels (2.4 ± 1.8 mmol/L) and mortality rate (5.1%), with benzodiazepine poisonings showing no mortality despite a significant number of cases (n=42). Among industrial chemicals, carbon monoxide poisoning was associated with relatively high lactate levels (4.5 ± 2.9 mmol/L) and mortality (16.7%).

Table 4: ROC Curve Analysis for Serum Lactate Parameters in Predicting Mortality

Parameter	AUC (95% CI)	Cutoff Value	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Initial lactate	0.86 (0.79-0.93)	4.5 mmol/L	84.6	79.3	38.2	97.2
6-hour lactate	0.89 (0.83-0.95)	3.2 mmol/L	82.1	85.6	45.6	96.8
Lactate clearance	0.81 (0.74-0.88)	20%	77.4	74.8	31.4	95.7

AUC: Area Under Curve; CI: Confidence Interval; PPV: Positive Predictive Value; NPV: Negative Predictive Value

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The initial serum lactate level showed good discriminatory ability for predicting mortality with an AUC of 0.86 (95% CI: 0.79-0.93). The optimal cutoff value was 4.5 mmol/L, with a sensitivity of 84.6% and specificity of 79.3%. The 6-hour lactate level demonstrated slightly better performance with an AUC of 0.89 (95% CI: 0.83-0.95) and an optimal cutoff value of 3.2 mmol/L. Lactate clearance at 6 hours also showed good discriminatory ability (AUC 0.81, 95% CI: 0.74-0.88), with an optimal cutoff value of 20%. All three parameters had high negative predictive values (>95%), indicating that values below the cutoff strongly suggest a favorable outcome.

Table 5: Logistic Regression Analysis for Predictors of Mortality

Variable	Univariate Analysis OR (95% CI)	p-value	Multivariate Analysis OR (95% CI)	p-value
Age >60 years	2.12 (1.14-3.94)	0.018	1.74 (0.87-3.48)	0.117
Male gender	1.26 (0.63-2.50)	0.512	-	-
Pesticide poisoning	4.78 (2.37-9.66)	<0.001	2.56 (1.18-5.57)	0.018
Time to presentation >6 hours	2.45 (1.25-4.80)	0.009	1.92 (0.91-4.07)	0.089
GCS score <9	6.92 (3.35-14.29)	<0.001	3.68 (1.65-8.21)	0.001
Initial lactate ≥4.5 mmol/L	11.36 (4.81-26.85)	<0.001	5.87 (2.31-14.92)	<0.001
Lactate clearance <20%	8.24 (3.87-17.56)	<0.001	4.23 (1.82-9.84)	0.001
pH <7.2	5.35 (2.53-11.30)	<0.001	1.84 (0.77-4.41)	0.172
White blood cell count >15×10³/μL	2.45 (1.22-4.93)	0.012	1.46 (0.65-3.28)	0.358
Creatinine >1.5 mg/dL	4.29 (2.10-8.78)	<0.001	1.97 (0.86-4.52)	0.110
INR >1.5	3.64 (1.78-7.45)	<0.001	1.76 (0.78-3.98)	0.176

OR: Odds Ratio; CI: Confidence Interval; GCS: Glasgow Coma Scale; INR: International Normalized Ratio

In univariate analysis, several factors were significantly associated with mortality, including age >60 years, pesticide poisoning, delayed presentation (>6 hours), low GCS score (<9), elevated initial lactate (≥4.5 mmol/L), poor lactate clearance (<20%), acidosis (pH <7.2), leukocytosis (white blood cell count >15×10³/μL), renal dysfunction (creatinine >1.5 mg/dL) and coagulopathy (INR >1.5). In multivariate analysis, after adjusting for potential confounders, only four factors remained independently associated with mortality: initial lactate ≥4.5 mmol/L (OR 5.87, 95% CI: 2.31-14.92, p<0.001), lactate clearance <20% (OR 4.23, 95% CI: 1.82-9.84, p=0.001), GCS score <9 (OR 3.68, 95% CI: 1.65-8.21, p=0.001), and pesticide poisoning (OR 2.56, 95% CI: 1.18-5.57, p=0.018).

Table 6: Mortality Rates According to Initial Lactate Level and Lactate Clearance

Category	n	Mortality, n (%)	p-value
Initial lactate (mmol/L)			
<2.0	106	1 (0.9)	<0.001
2.0-4.4	132	5 (3.8)	
4.5-6.9	42	12 (28.6)	
≥7.0	44	22 (50.0)	
Lactate clearance (%)			
≥40	126	2 (1.6)	<0.001
20-39	108	7 (6.5)	

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0-19	58	15 (25.9)	
<0 (increase)	32	16 (50.0)	

A clear dose-response relationship was observed between initial lactate levels and mortality. The mortality rate increased progressively from 0.9% in patients with initial lactate <2.0 mmol/L to 50.0% in those with lactate ≥7.0 mmol/L (p<0.001). Similarly, lactate clearance showed a strong association with mortality. Patients with good lactate clearance (≥40%) had a very low mortality rate (1.6%), while those with lactate increase (negative clearance) had a mortality rate of 50.0% (p<0.001).

Table 7: Combined Effect of Initial Lactate Level and Lactate Clearance on Mortality

Initial Lactate	Lactate Clearance	n	Mortality, n (%)
<4.5 mmol/L	≥20%	210	2 (1.0)
	<20%	28	4 (14.3)
≥4.5 mmol/L	≥20%	24	7 (29.2)
	<20%	62	27 (43.5)

The combination of initial lactate level and lactate clearance provided additional prognostic information. Patients with both low initial lactate (<4.5 mmol/L) and adequate clearance (≥20%) had an excellent prognosis with a mortality rate of only 1.0%. In contrast, patients with both high initial lactate (≥4.5 mmol/L) and poor clearance (<20%) had the highest mortality rate (43.5%). Interestingly, patients with high initial lactate but good clearance had a lower mortality rate (29.2%) compared to those with low initial lactate but poor clearance (14.3%), suggesting that lactate clearance might have prognostic significance independent of the initial level.

DISCUSSION

This prospective observational study demonstrates a strong association between serum lactate levels and mortality in patients with acute poisoning. Both elevated initial lactate levels and poor lactate clearance at 6 hours were independent predictors of mortality, even after adjusting for potential confounders such as age, type of toxin, time to presentation, and GCS score. Our findings are consistent with previous studies that have evaluated lactate as a prognostic marker in critical illness. In a large retrospective study of critically ill patients, Haas et al. (2016) found that lactate levels >4 mmol/L were associated with a threefold increase in mortality. Similarly, Nichol et al. (2015) reported that lactate levels >2 mmol/L were independently associated with increased hospital mortality in a heterogeneous cohort of intensive care unit patients. In the context of poisoning, elevated lactate levels may reflect several pathophysiological mechanisms. Many toxins directly impair mitochondrial function, inhibiting oxidative phosphorylation and promoting anaerobic metabolism (Kraut & Madias, 2016). For instance, cyanide, carbon monoxide, and hydrogen sulfide inhibit cytochrome oxidase, disrupting the electron transport chain and ATP production. Metformin inhibits complex I of the respiratory chain, while salicylates uncouple oxidative phosphorylation. These direct mitochondrial toxins can cause elevated lactate levels even in the absence of tissue hypoperfusion, representing a form of type B lactic acidosis.

Additionally, toxin-induced hypotension, seizures, and respiratory depression can lead to tissue hypoxia and type A lactic acidosis. Some substances, such as sympathomimetics (cocaine, amphetamines), can increase glycolysis and lactate production through adrenergic stimulation, even in the presence of adequate oxygenation. Hepatotoxic substances can impair lactate clearance, further contributing to hyperlactatemia (Jeppesen et al., 2015). Our study found significant variations in lactate levels across different types of poisoning. Pesticide poisonings, particularly organophosphates, were associated with the highest lactate levels and mortality rates. Organophosphates

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inhibit acetylcholinesterase, leading to cholinergic crisis characterized by bronchorrhea, bronchospasm, and respiratory failure. The resultant hypoxemia and tissue hypoperfusion can explain the elevated lactate levels. Additionally, organophosphates may directly impair mitochondrial function, further contributing to lactate production (Eddleston et al., 2008). In contrast, pharmaceutical drug poisonings, especially benzodiazepines, were associated with lower lactate levels and better outcomes. This finding is consistent with the relatively benign profile of benzodiazepines, which typically cause limited cardiovascular and respiratory depression at therapeutic or modest supratherapeutic doses (Isbister et al., 2004). Notably, carbon monoxide poisoning was associated with relatively high lactate levels, reflecting its mechanism of tissue hypoxia through competitive binding to hemoglobin and mitochondrial cytochrome oxidase (Hampson et al., 2012).

The significant association between lactate levels and mortality persisted after adjusting for potential confounders, suggesting that lactate provides prognostic information beyond that captured by traditional clinical parameters. The optimal cutoff value of 4.5 mmol/L for initial lactate in our study is comparable to thresholds identified in other critical care settings. For instance, the Surviving Sepsis Campaign guidelines recognize lactate >4 mmol/L as a marker of tissue hypoperfusion warranting aggressive resuscitation (Rhodes et al., 2017). A novel aspect of our study is the evaluation of lactate clearance as a prognostic marker in poisoning. We found that poor lactate clearance (<20% at 6 hours) was independently associated with increased mortality. This finding is consistent with studies in sepsis and trauma, where failure to clear lactate has been associated with poor outcomes. Nguyen et al. (2004) reported that a lactate clearance <10% in the first 6 hours was associated with increased mortality in severe sepsis and septic shock. Similarly, Odom et al. (2013) found that lactate clearance <20% at 6 hours was an independent predictor of mortality in trauma patients. Interestingly, our analysis of the combined effect of initial lactate and lactate clearance revealed that patients with high initial lactate but good clearance had better outcomes than those with low initial lactate but poor clearance. This suggests that the trajectory of lactate levels (indicating response to treatment) may be more important than the absolute value at a single time point. The ability to clear lactate likely reflects both the reversibility of the poisoning and the adequacy of supportive interventions.

The high negative predictive value of normal lactate levels (97.2% for initial lactate <4.5 mmol/L) suggests that lactate could be useful for identifying low-risk patients who might be candidates for less intensive monitoring or earlier discharge. This finding has potential implications for resource allocation in busy emergency departments, particularly in settings with limited intensive care capacity. Our study has several strengths. The prospective design, relatively large sample size, and inclusion of diverse poisoning types enhance the generalizability of our findings. The measurement of lactate at two time points allowed evaluation of both initial values and clearance. The multivariate analysis controlled for potential confounders, strengthening the validity of our conclusions. However, several limitations should be acknowledged. First, as an observational study, it cannot establish causality between elevated lactate and mortality. Lactate may be a marker of illness severity rather than a direct contributor to poor outcomes. Second, the study was conducted at a single center, potentially limiting external validity. Third, despite the diverse range of poisons included, some rare but important toxins were underrepresented. Fourth, lactate was measured at only two time points; more frequent measurements might have provided additional insights into the dynamics of lactate clearance. Finally, management decisions were not controlled, and variations in treatment approaches might have influenced outcomes.

Despite these limitations, our findings have important clinical implications. Serum lactate measurement is simple, rapid, and widely available, making it an attractive prognostic tool in the emergency setting. The strong association between lactate levels and mortality suggests that lactate could be incorporated into risk stratification protocols for poisoned patients. Early identification of high-risk patients (those with lactate ≥4.5 mmol/L or clearance <20%) could prompt more aggressive management, including earlier ICU admission, more intensive monitoring, and consideration of enhanced elimination techniques or

specific antidotes when available. Moreover, serial lactate measurements could help assess response to treatment and guide ongoing management decisions. Failure to clear lactate despite appropriate supportive care might signal persistent toxicity requiring additional interventions. Conversely, rapid lactate clearance might indicate effective treatment and a more favorable prognosis. Future research should focus on validating these findings in larger, multicenter cohorts and examining whether lactate-guided management strategies improve outcomes in poisoned patients. Additionally, the integration of lactate into existing poisoning severity scores could enhance their prognostic accuracy. Further studies are also needed to elucidate the specific mechanisms of lactate elevation in different poisonings and to determine whether the prognostic value of lactate varies across different toxin classes.

CONCLUSION

In this prospective observational study of patients with acute poisoning, elevated serum lactate levels at admission and poor lactate clearance at 6 hours were independently associated with increased mortality. The association remained significant after adjusting for potential confounders, including age, type of toxin, time to presentation, and GCS score. The optimal cutoff values for predicting mortality were 4.5 mmol/L for initial lactate and 20% for lactate clearance at 6 hours. These findings suggest that serum lactate could serve as a valuable prognostic biomarker in acute poisoning, helping to identify high-risk patients who may benefit from more intensive monitoring and treatment. The simplicity, rapidity, and wide availability of lactate measurement make it an attractive tool for risk stratification in the emergency setting. Serial lactate measurements, particularly the assessment of lactate clearance, provide additional prognostic information beyond that available from a single measurement at admission. The trajectory of lactate levels may reflect both the reversibility of the poisoning and the adequacy of supportive interventions. Future research should focus on validating these findings in larger, multicenter cohorts and examining whether lactate-guided management strategies improve outcomes in poisoned patients. The integration of lactate into existing poisoning severity scores could enhance their prognostic accuracy and facilitate more informed clinical decision-making.

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