Original Research

ACUTE PHASE INFLAMMATORY MARKERS IN PARAQUAT POISONING AND THEIR RELATIONSHIP TO IN-HOSPITAL PROGNOSIS IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Paraquat poisoning is a highly lethal toxicological emergency associated with severe oxidative stress, systemic inflammation, and multi-organ failure. Despite various treatment strategies, mortality remains extremely high, and early prognostic indicators are essential for improving clinical decision-making.

Methodology: This prospective observational study was conducted in the Department of Emergency Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, from June 2025 to March 2026. A total of 50 patients aged above 18 years with a history of paraquat poisoning were included. Patients with chronic liver disease, chronic kidney disease, malignancy, pulmonary disorders, connective tissue diseases, anemia, or history of steroid/antimetabolite use were excluded. Clinical evaluation and laboratory investigations including CBC, RFT, LFT, ESR, CRP, LDH, and ferritin were performed on day 1 and day 2. Statistical analysis was performed using chi-square test and independent t-test, with $p < 0.05$ considered significant.

Results: The mean age of patients was 29.7 ± 7.8 years, with male predominance (86%). Mortality was 88% (n=44). Oral ulcers, acute kidney injury, and liver dysfunction were present in 98% of cases, while lung injury was observed in 88%. Non-survivors had significantly higher inflammatory markers compared to survivors: ESR (51.1 vs 24.7 mm/hr, p=0.01), CRP (36.8 vs 7 mg/L, p=0.04), LDH (461.1 vs 261.2 U/L, p=0.001), and ferritin (449.6 vs 233.2 ng/mL, p=0.001). Lung injury showed a strong association with mortality ($p < 0.001$).

Conclusion: Paraquat poisoning is associated with extremely high mortality. Elevated acute inflammatory markers and pulmonary involvement are strongly associated with poor in-hospital outcomes. Early assessment of inflammatory biomarkers may aid in risk stratification and prognostication in paraquat poisoning.

Keywords: Paraquat Poisoning, Acute Inflammation, CRP, LDH, Ferritin, Mortality, Prognosis.

INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-bipyridinium) is a highly toxic bipyridyl herbicide widely used in agriculture for weed control. Despite its effectiveness as a contact herbicide, it is associated with severe human toxicity and high mortality following ingestion. Paraquat poisoning remains a major public health concern in many developing countries, particularly in Asia, where it is frequently encountered in both accidental and intentional ingestion cases.

The compound exerts its toxic effects primarily through redox cycling, leading to the generation of reactive oxygen species (ROS), lipid peroxidation, and subsequent multi-organ dysfunction, especially involving the lungs, kidneys, and liver.[1]

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One of the most striking features of paraquat poisoning is its extremely poor prognosis. Reported mortality rates can reach up to 90%, particularly in cases involving moderate to large ingestions.[2] Even with advanced supportive care and a variety of therapeutic strategies—including gastric decontamination, hemoperfusion, immunosuppressive therapy, and antioxidant administration—clinical outcomes remain largely unfavorable.[3] The absence of a specific antidote further contributes to the high fatality rate, making early risk stratification and prognostic assessment critical in the management of these patients.

The pathophysiological basis of paraquat-induced injury is closely linked to intense oxidative stress and a subsequent inflammatory cascade. After absorption, paraquat accumulates preferentially in pulmonary tissue via the polyamine uptake system, leading to progressive alveolar epithelial damage, acute lung injury, and ultimately pulmonary fibrosis.[4] This inflammatory response is characterized by activation of neutrophils, release of cytokines, and amplification of oxidative injury,

which collectively contribute to worsening organ failure and poor clinical outcomes.

Given the rapid progression and high mortality associated with paraquat toxicity, several prognostic indicators have been investigated to assist in early outcome prediction. Among these, plasma paraquat concentration remains one of the most reliable predictors of survival and is strongly correlated with the ingested dose and time since exposure.[5] In addition, clinical scoring systems, renal function parameters, arterial blood gas analysis, and inflammatory markers such as C-reactive protein (CRP) and neutrophil–lymphocyte ratio (NLR) have been explored as potential prognostic tools.[6] These indicators help clinicians stratify patients into risk categories and guide the intensity of therapeutic interventions.

Recently, increasing attention has been directed toward the role of acute inflammatory response as a predictor of mortality in paraquat poisoning. Inflammatory markers reflect the extent of systemic oxidative injury and immune activation, which are central to the pathogenesis of paraquat toxicity. Studies suggest that elevated inflammatory indices may be associated with increased severity of organ dysfunction and worse in-hospital outcomes.[7] However, there remains a need for further clinical studies to better define the prognostic value of these markers and to establish standardized thresholds for risk prediction.

In this context, evaluating acute inflammatory responses in patients with paraquat poisoning and correlating them with in-hospital outcomes may provide valuable insights for early prognostication and management strategies. Such an approach could potentially improve clinical decision-making and help identify high-risk patients who may benefit from aggressive therapeutic interventions.

Aim and Objectives

Aim

To evaluate the role of acute inflammatory response and its correlation with in-hospital outcomes in patients with paraquat poisoning.

Objectives

1. To assess the levels of acute inflammatory markers in patients with paraquat poisoning at the time of hospital admission.
2. To analyze the severity of clinical presentation and progression of organ dysfunction in paraquat poisoning
3. To correlate inflammatory parameters with in-hospital outcomes, including mortality, duration of hospital stay, and need for intensive care support.

METHODOLOGY

This is a prospective observational study conducted in the Department of Emergency Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, over a study period from June 2025 to March 2026. The study included all patients presenting with a history of paraquat poisoning aged above 18 years who were willing to provide informed consent. Patients with chronic liver disease, malignancy, chronic kidney disease, previous history of pulmonary disorders, prior use of drugs such as steroids or antimetabolites, connective tissue diseases, and anemia were excluded from the study.

All patients fulfilling the inclusion criteria were enrolled after initial stabilization in the emergency department. A detailed clinical history and complete physical examination were performed for all study subjects. On day 1 and day 2 of admission, venous blood samples were collected from each patient who had ingested paraquat. The following laboratory investigations were performed: complete blood count (CBC), renal function tests (RFT), liver function tests (LFT), erythrocyte sedimentation rate (ESR), serum lactate dehydrogenase (LDH), C-reactive protein (CRP), and other relevant inflammatory and biochemical parameters as required.

Patients were closely monitored during the hospital stay, and clinical outcomes including survival status, need for intensive care unit (ICU) admission, and duration of hospitalization were recorded. The collected data were systematically entered and analyzed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Comparison between groups was performed using appropriate statistical tests such as the Student's t-test for continuous variables and the chi-square test for categorical variables. Correlation between inflammatory markers and in-hospital outcomes was assessed using Pearson or Spearman correlation analysis based on data distribution. A p-value of <0.05 was considered statistically significant. Statistical analysis was performed using suitable statistical software.

RESULT

The study was conducted on 50 subjects. The majority of patients (52%) were between 21-30 years old, with a mean age of 29.7 years (SD 7.8). In our study gender distribution was heavily skewed towards males, with 86% of participants being men. 49 (98%) subjects had oral ulcers, liver dysfunction, acute kidney injury. 44(88%) had lung injury. Out of 50 subjects who have consumed paraquat, 44(88.0%) subjects succumbed to death [table 1]. Most of the subjects had elevated inflammatory markers as depicted in Table 2.

Demographic Parameters		N (%)
Age (In Years)	18-20	6(12.0)
	21-30	26(52.0)
	31-40	15(30.0)
	41-50	2(4.0)
	51-60	1(2.0)
Gender	Male	43(86.0)
	Female	7(14.0)
Clinical Features	PRESENT [N (%)]	ABSENT [N (%)]
Oral Ulcers	49(98.0)	1(2.0)
Acute Kidney Injury	49(98.0)	1(2.0)
Liver Dysfunction	49(98.0)	1(2.0)
Lung Injury	44(88.0)	6(12.0)
Outcome	Death	44 (88.0)
	Survived	6(12.0)

Table 1: Showing Distribution of Study Subjects Based on Demographic, Clinical Presentation and Outcome

Variables	Mean	SD	Minimum	Maximum
Age (Years)	29.7	7.8	18	55
ESR (Mm/Hr)	47.9	25.7	10	110
CRP (Mg/L)	33.2	41.4	3	269
LDH (U/L)	437.1	268.9	126	1723
Ferritin (Ng/Ml)	423.6	285.8	30.4	1063

Table 2: Clinical Characteristics of the Study Markers (N=50)

Clinical characteristics of the study markers is depicted in Table 2. The mean (SD) age of the study participants was 29.7 (7.8) years. The mean (SD) ESR was 47.9 (25.7) mm/hr. The mean (SD) CRP, LDH and ferritin were 33.2 (41.4) mg/L, 437.1 (268.9) U/L and 423.6 (285.8) ng/mL respectively.

Oral Ulcers	Outcome		P Value
	Survived	Death	
Present	5 (83.3)	44 (100)	**<0.001**
Absent	1 (16.7)	0 (0.0)	
Total	6 (100)	44 (100)	

Table 3: Association of Oral Ulcers with Outcome among the Study Participants (N=50)

*Chi-Squared Test

Among patients who survived 83.3% (n=5) had oral ulcers and among patients who died 100% (n=44) had oral ulcers. This difference was statistically significant (p <0.001).

Renal Failure	Outcome		P Value
	Survived	Death	
Present	5 (83.3)	44 (100)	**<0.001**
Absent	1 (16.7)	0 (0.0)	
Total	6 (100)	44 (100)	
Table 4. Association of Renal Failure with Outcome among the Study Participants (N=50)			
*Chi-Squared Test			

Among patients who survived 83.3% (n=5) had renal failure and among patients who died 100% (n=44) harenal failure. This difference was statistically significant (p <0.001).

Liver Failure	Outcome		P Value
	Survived	Death	
Present	5 (83.3)	44 (100)	**<0.001**
Absent	1 (16.7)	0 (0.0)	
Total	6 (100)	44 (100)	
Table 5: Association of Liver Failure with Outcome among the Study Participants (N=50)			
*Chi-Squared Test			

Among patients who survived 83.3% (n=5) had liver failure and among patients who died 100% (n=44) had liver failure. This difference was statistically significant (p <0.001).

Lung Injury	Outcome		P Value
	Survived	Death	
Present	0 (0.0)	44 (100)	**<0.001**
Absent	6 (100)	0 (0.0)	
Total	6 (100)	44 (100)	
Table 6: Association of Lung Injury with Outcome among the Study Participants (N=50)			
*Chi-Squared Test			

Among patients who survived, 0% (n=5) had lung injury and among patients who died 100% (n=44) had lung injury. This shows lung injury has poor prognosis .This difference was statistically significant (p <0.001)

Variables	Mean (SD)		P Value
	Survived	**Death**	
ESR (Mm/Hr)	24.7 (9.5)	51.1 (25.6)	**0.01**
CRP (Mg/L)	7 (4.5)	36.8 (42.9)	**0.04**
LDH (U/L)	261.2 (86.1)	461.1 (276.8)	**0.001**
Ferritin (Ng/Ml)	233.2 (108.4)	449.6 (293.2)	**0.001**
Table 7: Association Of Clinical Characteristics Of Study Markers With The Outcome Among The Study Participants (N=50)			
*Independent T Test			

Association of clinical characteristics with the outcome among the study participants is depicted in Table 13. The mean (SD) age of patients who survived was 29.3 (7.3) years and among patients who died was29.8 (7.9) years. The mean (SD) ESR of patients who survived was 24.7 (9.5) mm/hr and among patients who died was 51.1 (25.6) mm/hr. This difference was statistically significant (p=0.01). The mean (SD) CRP of patients who survived was 7 (4.5) mg/L and among patients who died was 36.8 (42.9) mg/L. This difference was statistically significant (p=0.04). The mean (SD) LDH of patients who survived was 261.2 (86.1) U/L and among patients who died was 461.1 (276.8) U/L. This difference was statistically significant (p=0.001). The mean

(SD) ferritin of patients who survived was 233.2 (108.4) ng/mL and among patients who died was 449.6 (293.2) ng/mL. This difference was statistically significant (p=0.001).

DISCUSSION

Paraquat poisoning continues to be associated with extremely high mortality despite advances in intensive care and supportive treatment modalities. In the present study, the mortality rate was 88%, which is consistent with previously reported literature describing mortality rates ranging from 60% to as high as 90% in moderate to severe poisoning cases.[8] The mean age of affected individuals was 29.7 ± 7.8 years, indicating that paraquat poisoning predominantly affects young adults, a finding similar to other studies where intentional ingestion in the economically productive age group is commonly reported.[9] The marked male predominance (86%) observed in this study may be attributed to occupational exposure and psychosocial factors, which aligns with earlier epidemiological observations.[10]

Clinical manifestations in our study were severe and multisystemic. Oral ulceration was observed in 98% of patients, serving as an early clinical marker of corrosive injury and ingestion severity. Acute kidney injury and hepatic dysfunction were present in 98% of cases, reflecting systemic toxicity due to paraquat accumulation and oxidative damage. Pulmonary involvement, observed in 88% of patients, remains the most critical determinant of prognosis as paraquat selectively accumulates in lung tissue, leading to progressive alveolar damage and fibrosis.[11] the strong association between lung injury and mortality in our study further reinforces its role as a key prognostic indicator.

Inflammatory markers were significantly elevated in the study population. The mean ESR (47.9 mm/hr), CRP (33.2 mg/L), LDH (437.1 U/L), and ferritin (423.6 ng/mL) were markedly increased, indicating a strong systemic inflammatory response. These findings support the pathophysiological concept that paraquat toxicity is not only mediated by direct oxidative injury but also by an exaggerated inflammatory cascade.[12] Similar elevations in inflammatory biomarkers have been reported in previous studies and have been associated with increased severity of organ dysfunction and poor outcomes.

A significant difference in inflammatory markers was observed between survivors and non-survivors. Patients

who died had substantially higher ESR, CRP, LDH, and ferritin levels compared to survivors, suggesting that these markers may serve as useful prognostic indicators. Elevated CRP and ferritin reflect acute phase response and macrophage activation, while increased LDH indicates widespread cellular injury.[13] These findings are consistent with earlier research suggesting that inflammatory biomarkers can be used for early risk stratification in paraquat poisoning.

Organ dysfunction, including renal and hepatic failure, was significantly associated with mortality ($p < 0.001$). However, the nearly universal presence of these complications among non-survivors limits their discriminative prognostic utility. In contrast, pulmonary injury showed a more direct and clinically relevant association with fatal outcomes, reinforcing the central role of lung damage in paraquat toxicity.[14] The presence of oral ulcers was also significantly associated with poor outcomes, likely reflecting higher exposure dose and severity of ingestion.

Overall, the present study highlights that acute inflammatory markers, along with early clinical features such as oral ulcers and pulmonary injury, are strongly associated with in-hospital mortality in paraquat poisoning. These findings emphasize the importance of early recognition of systemic inflammation and organ dysfunction for timely prognostication and management decisions. Incorporating inflammatory markers into routine evaluation may help identify high-risk patients who may benefit from aggressive therapeutic interventions and closer monitoring.

CONCLUSION

Paraquat poisoning remains a highly fatal toxicological emergency, predominantly affecting young male individuals and resulting in severe multisystem involvement. In this study, the mortality rate was 88%, highlighting the grave prognosis associated with paraquat ingestion despite supportive and intensive care management. Acute clinical manifestations such as oral ulcers, renal dysfunction, hepatic injury, and especially pulmonary involvement were frequently observed and showed strong association with in-hospital mortality.

The present study demonstrates that inflammatory markers, including ESR, CRP, LDH, and ferritin, were significantly elevated in patients with paraquat poisoning and were markedly higher among non-survivors compared to survivors. These findings suggest that acute inflammatory response plays a crucial role in disease progression and outcome determination.

Among all clinical and laboratory parameters, lung injury and elevated inflammatory markers showed the strongest association with mortality, indicating their potential utility as early prognostic indicators. Therefore, early identification of inflammatory response along with clinical severity assessment

may aid in risk stratification and guide management decisions in paraquat poisoning.

In conclusion, acute inflammatory markers can serve as useful adjuncts in predicting in-hospital outcomes in paraquat poisoning, and their routine assessment may help in identifying high-risk patients who require aggressive and intensive management strategies.

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