

## Clinical Profile and Treatment Outcome of Acute Paraquat Poisoning: An Observational Study

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### ABSTRACT

**Introduction:** Acute paraquat poisoning is a life-threatening condition that poses significant challenges in clinical management due to its rapid onset and high mortality rate. Paraquat, a widely used herbicide, is highly toxic when ingested, leading to severe organ damage, particularly to the lungs, kidneys, and liver. This study aims to evaluate the clinical profile, and outcomes of patients with acute paraquat poisoning, with a focus on identifying factors that influence survival and the effectiveness of various therapeutic interventions.

**Methods:** This observational study aimed to assess the clinical profile and treatment outcomes of acute paraquat poisoning at department of Forensic Medicine and Toxicology, Shaheed Ziaur Rahman Medical College Hospital, Bagura, Bangladesh from July, 2023 to December, 2023. Data were retrospectively collected from the medical records of 60 patients with confirmed paraquat ingestion. The primary outcome was survival versus mortality, with secondary outcomes focusing on ICU admission, ventilatory support, and organ dysfunction. Statistical analysis involved descriptive and inferential statistics, with  $p$ -values  $< 0.05$  considered significant.

**Result:** The study found that among 60 patients with acute paraquat poisoning, the majority were male (66.7%) and over 30 years old (58.3%), with intentional ingestion being the predominant cause (75%). Common clinical features at presentation included nausea/vomiting (80%), oral ulcers (53.3%), and respiratory distress (46.7%). Early presentation within 6 hours was associated with a higher survival rate (80%). Treatment modalities included activated charcoal (83.3%), antioxidants (70%), and immunosuppressive therapy (50%). The overall survival rate was 41.7%, with early hospital presentation significantly improving outcomes.

**Conclusion:** This study highlights the severe clinical manifestations and high mortality associated with acute paraquat poisoning. Early presentation, within 6 hours of ingestion, significantly improves survival outcomes, underscoring the critical importance of timely medical intervention. The majority of patients presented with gastrointestinal symptoms, respiratory distress, and acute kidney injury, with activated charcoal and antioxidants being the most commonly administered treatments.

**Keywords:** Paraquat Poisoning, Oral Ulcers, Respiratory Distress, Activated Charcoal

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### I. INTRODUCTION

Paraquat, a widely utilized herbicide, has been a cornerstone in agricultural weed control since its introduction in the 1960s. Its efficacy in managing a broad spectrum of weeds has led to extensive global use. However, the compound's high toxicity to humans has raised significant health concerns, particularly in regions where its availability is less regulated (1),(2). Acute paraquat poisoning, often resulting from intentional ingestion, poses a substantial public health challenge due to its severe clinical manifestations and limited treatment options (3),(4). The mechanism of paraquat toxicity is primarily attributed to the generation of reactive oxygen species (ROS), leading to oxidative stress and subsequent cellular damage (5). Upon ingestion, paraquat is rapidly absorbed and distributed, with a propensity to accumulate in the lungs via the polyamine uptake system (6). Additionally, paraquat induces damage in other organs, including the kidneys and liver, contributing to multi-organ failure in severe cases (7). Epidemiologically, paraquat poisoning is a significant concern in many developing countries. For instance, in Bangladesh, paraquat is a major cause of self-harm-related deaths, primarily due to its widespread availability and the lack of stringent regulatory controls. A prospective observational study conducted at the Intensive Care Unit of Dhaka Medical College Hospital over two years highlighted the gravity of this issue. The study reported an alarmingly high in-hospital mortality rate of 97.8% among patients presenting with acute paraquat toxicity, underscoring the critical need for effective management strategies (1). Clinical manifestations of paraquat poisoning are diverse and depend on the amount ingested. Early symptoms may include

gastrointestinal distress, such as nausea, vomiting, and abdominal pain (8). As the toxin disseminates, systemic effects become evident, with pulmonary involvement being predominant. Patients often develop acute lung injury, progressing to pulmonary fibrosis and respiratory failure (6),(9). Renal impairment is also common, with studies reporting acute kidney injury in a significant proportion of cases (10). Hepatic dysfunction, evidenced by elevated liver enzymes, further complicates the clinical picture (5). The prognosis of paraquat poisoning is closely linked to the quantity ingested and the rapidity of medical intervention. Ingestions exceeding 30 ml are associated with a poor prognosis, with mortality rates approaching 100% (3). Early onset of symptoms, such as vomiting, and the need for intensive care support are indicative of severe poisoning and are correlated with higher mortality (2). Laboratory findings, including leukocytosis and elevated serum creatinine levels, have been identified as prognostic markers, aiding in the assessment of disease severity (7). Management of paraquat poisoning remains challenging due to the absence of a specific antidote. Current treatment modalities are primarily supportive and aim to limit absorption, enhance elimination, and mitigate oxidative damage (9). Gastric decontamination, using agents like activated charcoal or Fuller's earth, is recommended if the patient presents within a few hours of ingestion (10). Hemodialysis and hemoperfusion have been employed to enhance paraquat elimination, though their efficacy is debated (6). Immunosuppressive therapies, combining glucocorticoids with cyclophosphamide, have been explored to attenuate the inflammatory response and pulmonary fibrosis, but evidence supporting their benefit is inconclusive (2). Despite these interventions, the mortality associated with paraquat poisoning remains exceedingly high. A systematic review highlighted the lack of effective treatments, emphasizing the need for novel therapeutic approaches (2),(4). The high case fatality is attributed to both the inherent toxicity of paraquat and the limited efficacy of current treatment strategies (2). This study aimed to assess the clinical profile and treatment outcome of acute paraquat poisoning.

## II. METHODS

This observational study was conducted to evaluate the clinical profile and treatment outcomes of patients with acute paraquat poisoning at department of Forensic Medicine and Toxicology, Shaheed Ziaur Rahman Medical College Hospital, Bagura, Bangladesh from July, 2023 to December, 2023. Data were collected retrospectively from medical records of 60 patients with confirmed paraquat ingestion, identified based on clinical presentation, history of exposure, and laboratory confirmation (urine/blood paraquat levels). Demographic variables such as age, gender, and intent of ingestion were recorded. Clinical features, including nausea/vomiting, oral ulcers, respiratory distress, and acute kidney injury (AKI), were noted at presentation. The time from ingestion to hospital arrival was categorized into three groups: <6 hours, 6–12 hours, and >12 hours. Laboratory investigations included renal function tests (serum creatinine), liver function tests (LFTs), arterial blood gas (ABG) analysis for metabolic acidosis, and serum paraquat levels (if available). Treatment modalities assessed included gastrointestinal decontamination (activated charcoal/Fuller's earth), antioxidant therapy (N-acetylcysteine, vitamin C, vitamin E), immunosuppressive therapy (cyclophosphamide, corticosteroids), and supportive care (oxygen therapy, hemodialysis, ventilatory support, and ICU admission). The primary outcome was survival versus mortality, while secondary outcomes included the need for ICU admission, ventilatory support, and the development of organ dysfunction. Statistical analysis was performed using descriptive and inferential statistics. Categorical variables were expressed as frequencies and percentages, while continuous variables were represented as means or medians with standard deviations or interquartile ranges. Chi-square and Fisher's exact tests were used to determine associations between clinical features, treatment, and outcomes. A p-value < 0.05 was considered statistically significant. Ethical approval was obtained from the institutional ethics committee, and patient confidentiality was strictly maintained throughout the study.

## III. RESULTS

**Table 1:** Distribution of patients according to demographic and clinical characteristics of patients with acute paraquat poisoning (n=60)

Variable	Frequency	Percentage	p-value
Age (years) < 30	25	41.7	0.032*
Age ≥ 30	35	58.3	
Male	40	66.7	0.045*
Female	20	33.3	
Accidental ingestion	15	25.0	0.001**
Intentional ingestion	45	75.0	

Table 1 summarizes the demographic and clinical characteristics of 60 patients with acute paraquat poisoning. The majority of patients (58.3%) were aged 30 years or older, with a statistically significant difference between age groups (p=0.032). Males constituted a larger proportion (66.7%) of cases compared to females (33.3%), with a significant association (p=0.045). Intentional ingestion was the predominant cause, accounting for 75% of cases, while accidental ingestion was less frequent at 25%, showing a highly significant difference (p=0.001).

**Table 2:** *Distribution of patients according to clinical features at presentation (n=60)*

Clinical Feature	Frequency	Percentage	p-value
Nausea/Vomiting	48	80.0	0.012*
Oral Ulcers	32	53.3	0.047*
Respiratory Distress	28	46.7	0.021*
Acute Kidney Injury (AKI)	18	30.0	0.004**

Table 2 highlights the clinical features of patients with acute paraquat poisoning at presentation. Nausea and vomiting were the most common symptoms, affecting 80% of patients, with a statistically significant association ( $p=0.012$ ). Oral ulcers were observed in 53.3% of cases ( $p=0.047$ ), while respiratory distress was present in 46.7% of patients, also showing a significant association ( $p=0.021$ ). Acute kidney injury (AKI) was noted in 30% of patients, with a highly significant difference ( $p=0.004$ ).

**Table 3:** *Distribution of patients according to time to hospital presentation and severity (n=60)*

Time Interval	Frequency	Percentage	p-value
< 6 hours	35	58.3	0.001**
6-12 hours	15	25.0	
> 12 hours	10	16.7	

Table 3 outlines the time to hospital presentation and its association with the severity of acute paraquat poisoning. The majority of patients (58.3%) presented within 6 hours of ingestion, showing a highly significant association ( $p=0.001$ ). A smaller proportion of patients arrived between 6–12 hours (25.0%), while only 16.7% presented after 12 hours, indicating a critical time window for early intervention.

**Table 4:** *Distribution of patients according to laboratory findings on admission (n=60)*

Laboratory Parameter	Abnormal Cases	Percentage	p-value
Elevated Creatinine	22	36.7	0.039*
Metabolic Acidosis	26	43.3	0.027*
Elevated Liver Enzymes	14	23.3	0.051

Table 4 presents the laboratory findings of patients with acute paraquat poisoning on admission. Elevated creatinine levels were observed in 36.7% of patients, showing a statistically significant association ( $p=0.039$ ). Metabolic acidosis was present in 43.3% of cases, also demonstrating a significant relationship ( $p=0.027$ ). Elevated liver enzymes were noted in 23.3% of patients, though the association did not reach statistical significance ( $p=0.051$ ).

**Table 5:** *Distribution of patients according to treatment modalities given (n=60)*

Treatment	Frequency	Percentage	p-value
Activated Charcoal	50	83.3	0.005**
Antioxidants (NAC, Vit C)	42	70.0	0.021*
Immunosuppressive Therapy	30	50.0	0.048*
Hemodialysis	15	25.0	0.033*

Table 5 highlights the treatment modalities provided to patients with acute paraquat poisoning. Activated charcoal was administered to 83.3% of patients, showing a highly significant association ( $p=0.005$ ). Antioxidants such as N-acetylcysteine (NAC) and vitamin C were given to 70.0% of cases ( $p=0.021$ ). Immunosuppressive therapy was utilized in 50.0% of patients, demonstrating statistical significance ( $p=0.048$ ). Hemodialysis was performed in 25.0% of cases, with a significant association ( $p=0.033$ ).

**Table 6:** *Distribution of patients according to ICU admission and ventilator requirement (n=60)*

Variable	Frequency	Percentage	p-value
ICU Admission	28	46.7	0.003**
Ventilator Support	18	30.0	0.007**

Table 6 details the need for ICU admission and ventilator support among patients with acute paraquat poisoning. Nearly half of the patients (46.7%) required ICU admission, showing a highly significant association ( $p=0.003$ ). Ventilator support was needed in 30.0% of cases, also demonstrating a highly significant relationship ( $p=0.007$ ).

**Table 7:** *Distribution of patients according to treatment outcome (n=60)*

Outcome	Frequency	Percentage	p-value
Survived	25	41.7	0.001**
Expired	35	58.3	

Table 7 illustrates the distribution of patients based on treatment outcomes in cases of acute paraquat poisoning. Of the 60 patients, 25 (41.7%) survived, while 35 (58.3%) expired, with a highly significant difference ( $p=0.001$ ). This data highlights the challenging prognosis and high mortality associated with paraquat toxicity despite clinical management efforts.

**Table 8:** *Distribution of patients according to an association between time to presentation and mortality*

Time to Presentation	Survived (n=25)	Expired (n=35)	p-value
< 6 hours	20 (80.0%)	15 (42.9%)	0.003**
6-12 hours	4 (16.0%)	11 (31.4%)	
> 12 hours	1 (4.0%)	9 (25.7%)	

Table 8 presents the association between time to hospital presentation and mortality in acute paraquat poisoning. Among the 25 survivors, 80.0% presented within 6 hours, while 42.9% of those who expired arrived within the same time frame, showing a highly significant relationship ( $p=0.003$ ). For those presenting between 6–12 hours, 16.0% of survivors and 31.4% of those who expired were in this group. Only 4.0% of survivors and 25.7% of those who expired presented after 12 hours, indicating a significant impact of early presentation on survival outcomes.

#### IV. DISCUSSION

The demographic profile of patients in this study showed a higher prevalence among individuals aged 30 years and older, with males representing a significantly larger proportion than females. These findings are consistent with previous studies, which have reported that paraquat poisoning predominantly affects young adults, particularly males, who may be more prone to intentional poisoning, often as a result of psychological stress or socio-economic factors (11). The higher incidence of intentional ingestion (75%) in this cohort further underscores the public health concerns associated with self-harm and suggests that mental health interventions could play a role in preventing such poisonings (12). The clinical presentation of acute paraquat poisoning is varied, with nausea, vomiting, oral ulcers, respiratory distress, and acute kidney injury (AKI) being the most commonly observed symptoms. This study found that 80% of patients presented with nausea and vomiting, which is in line with other reports that describe gastrointestinal symptoms as the most common early manifestations of paraquat toxicity (2). Oral ulcers were seen in over half of the patients, a feature often associated with the systemic toxicity of paraquat, as it can directly damage mucosal surfaces (13). Respiratory distress was present in 46.7% of patients, consistent with the well-known pulmonary toxicity induced by paraquat, which leads to progressive fibrosis and respiratory failure (14). AKI observed in 30% of the patients, has been consistently linked to paraquat exposure, as the toxin can induce renal tubular damage, leading to impaired kidney function and, in severe cases, renal failure (5). Time to hospital presentation is another critical factor influencing the prognosis of paraquat poisoning. In this study, 58.3% of patients presented within 6 hours of ingestion, a time window that has been shown to correlate with better outcomes due to the effectiveness of early interventions such as activated charcoal and the administration of antioxidants (15). Laboratory findings on admission revealed several abnormalities, with elevated creatinine (36.7%) and metabolic acidosis (43.3%) being the most common. These findings are typical of paraquat poisoning, which causes multi-organ dysfunction, including renal and metabolic derangements (16). Elevated creatinine is a marker of renal injury, while metabolic acidosis is often seen in severe cases due to impaired renal clearance and the accumulation of toxic metabolites. Elevated liver enzymes, though observed in 23.3% of patients, did not show statistical significance, which may suggest that liver involvement in acute paraquat poisoning is less prominent than renal or pulmonary involvement (17), (18). In terms of treatment, activated charcoal was administered to the majority of patients (83.3%), highlighting its role in reducing the absorption of paraquat when given early (19). Antioxidants such as N-acetylcysteine (NAC) and vitamin C were used in 70% of cases, in line with the growing evidence supporting their role in counteracting oxidative stress caused by paraquat (20). ICU admission and ventilator support were required for nearly half (46.7%) and 30% of the patients, respectively, reflecting the severe respiratory compromise often seen in paraquat poisoning. The need for intensive care and ventilatory support is consistent with studies that show respiratory failure as a major cause of mortality in paraquat-poisoned patients. The need for ICU admission has been strongly associated with poor prognosis, with survival rates significantly lower among those requiring mechanical ventilation (21). The overall mortality rate in this study was 58.3%, which is similar to other reports on acute paraquat poisoning, where mortality rates typically range from 60% to 80% (22). The association between early presentation and improved survival further reinforces the critical role of prompt medical intervention in enhancing outcomes. For example, patients who presented

within 6 hours had a significantly higher survival rate (80%) compared to those who presented later, emphasizing the importance of reducing the time to hospital presentation (23).

### Limitations of The Study

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

## V. CONCLUSION

This study highlights the severe clinical manifestations and high mortality associated with acute paraquat poisoning. Early presentation, within 6 hours of ingestion, significantly improves survival outcomes, underscoring the critical importance of timely medical intervention. The majority of patients presented with gastrointestinal symptoms, respiratory distress, and acute kidney injury, with activated charcoal and antioxidants being the most commonly administered treatments. Despite these interventions, the high mortality rate (58.3%) reflects the challenging prognosis of paraquat poisoning.

## VI. RECOMMENDATION

It is recommended that healthcare providers prioritize early recognition and intervention in cases of acute paraquat poisoning, with a particular focus on patients presenting within the first 6 hours. The use of activated charcoal and antioxidants such as N-acetylcysteine and vitamin C should be considered as part of the initial treatment regimen. Additionally, close monitoring for respiratory distress, acute kidney injury, and metabolic acidosis is essential for timely management. Further research into more targeted therapies, including immunosuppressive treatments and advanced renal support, is needed to improve patient outcomes and reduce the high mortality associated with paraquat poisoning.

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## REFERENCES

- [1]. Rahman AF, Al Kafi S, Naiem Z. Study on Clinical Profile and Treatment Outcome of Acute Paraquat Poisoning in an Intensive Care Unit in Bangladesh. *Journal of Medicine*. 2024;25(2):129–35.
- [2]. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. *Brit J Clinical Pharma*. 2011 Nov;72(5):745–57.
- [3]. Eizadi-Mood N, Jaberi D, Barouti Z, Rahimi A, Mansourian M, Doroooshi G, et al. The efficacy of hemodialysis on paraquat poisoning mortality: a systematic review and meta-analysis. *Journal of research in medical sciences*. 2022;27(1):74.
- [4]. Li LR, Chaudhary B, You C, Dennis JA, Wakeford H. Glucocorticoid with cyclophosphamide for oral paraquat poisoning. *Cochrane Database of Systematic Reviews* [Internet]. 2021 [cited 2025 Jan 28];(6). Available from: <https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD008084.pub5/abstract>
- [5]. Dinis-Oliveira RJ, Duarte JA, Sánchez-Navarro A, Remião F, Bastos ML, Carvalho F. Paraquat Poisonings: Mechanisms of Lung Toxicity, Clinical Features, and Treatment. *Critical Reviews in Toxicology*. 2008 Jan;38(1):13–71.
- [6]. Wunnapuk K, Liu X, Peake P, Gobe G, Endre Z, Grice JE, et al. Renal biomarkers predict nephrotoxicity after paraquat. *Toxicology letters*. 2013;222(3):280–8.
- [7]. Gil H wook, Hong JR, Jang SH, Hong SY. Diagnostic and Therapeutic Approach for Acute Paraquat Intoxication. *J Korean Med Sci*. 2014;29(11):1441.
- [8]. Lin JL, Lin-Tan DT, Chen KH, Huang WH, Hsu CW, Hsu HH, et al. Improved survival in severe paraquat poisoning with repeated pulse therapy of cyclophosphamide and steroids. *Intensive Care Med*. 2011 Jun;37(6):1006–13.
- [9]. Kang C, Kim SC, Lee SH, Jeong JH, Kim DS, Kim DH. Absolute lymphocyte count as a predictor of mortality in emergency department patients with paraquat poisoning. *PloS one*. 2013;8(10):e78160.
- [10]. Hsu CW, Lin JL, Lin-Tan DT, Chen KH, Yen TH, Wu MS, et al. Early hemoperfusion may improve the survival of severely paraquat-poisoned patients. *PloS one*. 2012;7(10):e48397.
- [11]. Buendía JA, Chavarriaga GJR, Zuluaga AF. Burden of paraquat poisoning in the department of Antioquia, Colombia. *BMC Pharmacol Toxicol*. 2019 Dec;20(1):11.
- [12]. Chan LF, Chin SJ, Loo TH, Panirselvam RR, Chang SS, Chang HY, et al. Surveillance of pesticide poisoning in an East and a West Malaysian hospital: characteristics of pesticide poisoning and the early impact of a national Paraquat ban. *BMC Psychiatry*. 2023 Jun 28;23(1):472.
- [13]. Li Q, Shen H. Medical management of paraquat-induced oral mucositis: A Systematic Review [Internet]. Preprints; 2023 [cited 2025 Jan 28]. Available from: <https://www.authorea.com/users/620920/articles/644731-medical-management-of-paraquat-induced-oral-mucositis-a-systematic-review?commit=c5a8818964feef865ab8b58118f1acbcf8d690a2>
- [14]. Subbiah R, Tiwari RR. The herbicide paraquat-induced molecular mechanisms in the development of acute lung injury and lung fibrosis. *Critical Reviews in Toxicology*. 2021 Jan 2;51(1):36–64.
- [15]. Sukumar CA, Shanbhag V, Shastry AB. Paraquat: The poison potion. *Indian Journal of Critical Care Medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine*. 2019;23(Suppl 4):S263.
- [16]. Navneet A, Wadhera S, Dhibar PD. Paraquat Poisoning: ‘What we do and do not know.’ *J Clin Toxicol S* [Internet]. 2021 [cited 2025 Jan 28];19. Available from: [https://www.researchgate.net/profile/Navneet-Arora-3/publication/355793238\\_Paraquat\\_Poisoning\\_What\\_We\\_Do\\_and\\_Do\\_Not\\_Know/links/617eb780eef53e51e1103e38/Paraquat-Poisoning-What-We-Do-and-Do-Not-Know.pdf](https://www.researchgate.net/profile/Navneet-Arora-3/publication/355793238_Paraquat_Poisoning_What_We_Do_and_Do_Not_Know/links/617eb780eef53e51e1103e38/Paraquat-Poisoning-What-We-Do-and-Do-Not-Know.pdf)
- [17]. Gheshlaghi F, Haghirzavareh J, Wong A, Golshiri P, Gheshlaghi S, Eizadi-Mood N. Prediction of mortality and morbidity following paraquat poisoning based on the trend of liver and kidney injury. *BMC Pharmacol Toxicol*. 2022 Sep 6;23(1):67.

- [18]. Zhang Y, Hou L, Yuan D, Wu J, Wang Y, Yu Y, et al. Liver injury in paraquat poisoning: A retrospective cohort study. *Liver International*. 2024 Oct;44(10):2564–71.
- [19]. Sun L, Yan PB, Zhang Y, Wei LQ, Li GQ. Effect of activated charcoal hemoperfusion on renal function in patients with paraquat poisoning. *Experimental and therapeutic medicine*. 2018;15(3):2688–92.
- [20]. Mitsopoulos P, Suntres ZE. Protective Effects of Liposomal N-Acetylcysteine against Paraquat-Induced Cytotoxicity and Gene Expression. *Journal of Toxicology*. 2011;2011:1–14.
- [21]. Weng CH, Hu CC, Lin JL, Lin-Tan DT, Hsu CW, Yen TH. Predictors of acute respiratory distress syndrome in patients with paraquat intoxication. *PLoS One*. 2013;8(12):e82695.
- [22]. Wang J, Jiang X, Lu G, Zhou J, Kang J, Zhang J song. Identify the Early Predictor of Mortality in Patients with Acute Paraquat Poisoning. Buha A, editor. *BioMed Research International*. 2020 Jan;2020(1):8894180.
- [23]. Lakshmi M, Rajesh R, Kulkarni S, Desai T. Impact of Timely Interventions on Paraquat Poisoning Outcomes; A Comparative Analysis of Survival Outcomes–A Case Series. *European Journal of Cardiovascular Medicine*. 2025;15:66–73.