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Original Article

Internal Medicine Section

Spectrum of Biochemical Presentations in Patients with Paraquat Poisoning at a Tertiary Care Hospital in West Bengal, India: A Retrospective Cohort Study

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ABSTRACT

Introduction: Paraquat has been one of the most widely used herbicides in developing countries, particularly in Asia, and is notorious for its extremely high fatality rate due to multi-organ toxicity. In India, most available data are limited to small series or case reports, with very little systematic evidence emerging from Eastern India. Understanding the biochemical spectrum of paraquat poisoning is therefore crucial for guiding early diagnosis, management, and prevention strategies in this vulnerable population.

Aim: To analyse the biochemical spectrum of organ dysfunction in patients with paraquat poisoning admitted to a tertiary care hospital in Eastern India.

Materials and Methods: A retrospective cohort study was conducted at Jagannath Gupta Institute of Medical Sciences and Hospital, West Bengal, India, from November 2023 to July 2025. Twenty-five patients aged 19-45 years with intentional paraquat ingestion were included. Data on demographics, ingested doses of the poison, and liver and renal function tests were collected from the patients. Renal impairments were assessed by measuring serum creatinine at the time of admission and 48 hours later. Hepatic injuries were evaluated using Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), and bilirubin levels. Pulse oximetry was performed to measure oxygen saturation. Statistical analysis was conducted using Pearson's correlation coefficient and p-values.

Results: Most patients (14, 56%) were female, and 21 patients (84%) were aged 20-40 years. Twenty patients (80%) consumed more than 40 grams of paraquat, and 88% presented within six hours of ingestion. Serum creatinine showed a strong positive correlation with the ingested dose at admission (r=0.93) and at 48 hours (r=0.92). Oxygen saturation (SpO_2) levels demonstrated a strong negative correlation with the ingested dose (r=-0.91). Correlations of liver enzymes with the ingested dose were weak and non-significant. Male patients had significantly higher creatinine levels.

Conclusion: Paraquat poisoning causes severe renal and moderate hepatic dysfunction, with a high mortality rate. Early intervention is critical, although haemodialysis did not halt renal deterioration. The present study provides region-specific biochemical insights from Eastern India.

Keywords: Liver dysfunction, Renal failure, Respiratory dysfunction

INTRODUCTION

Paraquat has been one of the most commonly used herbicides in agriculture since 1955, particularly in developing countries such as Thailand [1,2]. In Thailand, the most common cause of acute poisoning is pesticides, followed by herbicides among which glyphosate and paraquat are predominant [3]. In Asia-Pacific countries, self-poisoning with paraquat is a leading cause of mortality and morbidity [4,5].

In India, paraquat poisoning is reported less frequently than pesticide (organophosphate) poisoning; however, it remains an emerging public health concern. Most available literature comprises small case series from Southern India with high case fatality rates, while systematic data from Eastern India are scarce highlighting the need for region-specific studies [6].

Paraquat generates reactive oxygen species through redox cycling, leading to disruption of the mitochondrial electron transport chain. This results in mitochondrial damage, lipid peroxidation, and cellular toxicity involving multiple organs such as the gastrointestinal tract, kidneys, liver, and others [1,3,5,6]. Paraquat can be absorbed through the gastrointestinal tract, skin, and inhalation. It has a large volume of distribution (1.2-1.6 L/kg), accumulating mainly in the lungs, kidneys, and liver. Its half-life is approximately 84 hours, and it is excreted through urine [3,7].

It is a highly toxic compound with a case fatality rate of up to 80%, primarily due to the absence of a specific antidote [8]. Ingestion of less than 20 mg/kg causes mild symptoms, while doses exceeding 40 mg/kg can lead to acute multiorgan failure and death within a few hours to days. Ingestion of 20-40 mg/kg typically results in mucosal injury followed by progressive organ failure [9-12]. The highest tissue concentration is found in the lungs [13]. Since the first reported case of paraquat poisoning, several studies have been conducted to explore its mechanisms and evaluate combination therapies aimed at modifying its toxicokinetics- either by enhancing elimination or reducing absorption [14-16].

With the exception of a few large studies from India, there is currently no published literature on paraquat poisoning from Eastern India, and most available data consist of isolated case reports [17,18].

Therefore, the present study was planned to analyse the biochemical spectrum of organ dysfunction in patients with paraquat poisoning admitted to a tertiary care hospital in Eastern India.

Study Objectives

Renal dysfunction: To assess kidney involvement by monitoring serum creatinine at admission and after 48 hours.

Hepatic dysfunction: To evaluate liver injury through AST, ALT, and bilirubin levels.

Pulmonary dysfunction: To assess lung involvement using pulse oximetry (SpO_2).

Dose correlation: To determine the correlation between the ingested dose of paraquat and biochemical derangements.

Sex predilection: To identify any sex-based differences in biochemical presentation.

MATERIALS AND METHODS

This retrospective cohort study was conducted at Jagannath Gupta Institute of Medical Sciences & Hospital, Budge Budge, West Bengal, India, over a period of one year and nine months (November 2023 to July 2025) among patients admitted with intentional paraquat poisoning. The study was approved by the Institutional Ethics Committee (IEC approval no. JIMSH/IEC/2023/11).

Inclusion and exclusion criteria: Patients aged between 19 and 45 years with a confirmed history of intentional paraquat ingestion were included. Cases with accidental exposure, uncertain diagnosis, or incomplete medical records were excluded.

A total of 25 patients fulfilling the eligibility criteria during the study period were included by complete enumeration, as paraquat poisoning cases are relatively uncommon. No formal sample size calculation was performed due to the retrospective nature of the study.

Study Procedure

An anonymised proforma was used to collect demographic and clinical details, including the dose of paraquat ingested and time to hospital admission.

- Renal involvement was assessed using serum creatinine values at admission and after 48 hours.
- Hepatic dysfunction was evaluated through AST, ALT, and bilirubin levels.
- Pulmonary function was assessed using pulse oximetry (SpO_2).

All data were retrieved from medical records and analysed using the Pearson correlation coefficient and p-values to determine associations between the ingested dose and biochemical derangements.

STATISTICAL ANALYSIS

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 31. Results were presented in graphs and tables for clarity. The Pearson correlation coefficient (r) was used to evaluate associations between the ingested dose and biochemical parameters. A p-value <0.05 was considered statistically significant.

RESULTS

The [Table/Fig-1] demonstrates that the majority of patients, 21 (84%), were between 20 and 40 years of age, reflecting the vulnerability of young adults to intentional paraquat ingestion. Females constituted a slight majority (56%), suggesting possible gender-related psychosocial factors in this region. Most patients (80%) consumed more than 40 grams of paraquat, and the vast majority (88%) presented within six hours of ingestion indicating both high-dose exposure and relatively early hospital presentation.

Despite early arrival, severe biochemical derangements were observed: nearly all patients (96%) had elevated serum creatinine, over half had markedly raised AST and ALT, and 44% had SpO_2 <80%, indicating significant renal, hepatic, and pulmonary involvement at admission. All patients succumbed within seven days of hospitalisation.

The [Table/Fig-2] demonstrates serum creatinine at admission and after 48 hours was significantly higher in males than females (p=0.04 and p=0.012, respectively). The ingested dose of paraquat was also significantly higher in males (p=0.03), suggesting more rapid and severe acute kidney injury due to higher ingestion doses.

Variables	Values
Sex	
Male	11 (44%)
Female	14 (56%)
Age (years)	
Less than 20	2 (8%)
20-40	21 (84%)
More than 40	2 (8%)
Amount consumed	
Less than 20 gram	0
20 to 40 gram	5 (20%)
More than 40 gram	20 (80%)
Time of admission	
Less than 6 hours	22 (88%)
6 to 24 hours	3 (12%)
More than 24 hours	0
Serum AST level (normal ≤40 IU/L)	
Less than 3 times normal	9 (36%)
3 to 4 times normal	2 (8%)
More than 4 time normal	14 (56%)
Serum ALT level (Normal ≤40 IU/L)	
Less than 3 times normal	8 (32%)
3 to 4 times normal	9 (36%)
More than 4 time normal	8 (32%)
Serum creatinine on admission (0.7 to 1.3 mg/dL for men and 0.6 to 1.1 mg/dL for women)*	
More than 2	24 (96%)
SpO_2	
More than 80%	14 (56%)
Less than 80%	11 (44%)
Serum bilirubin (Normal 0.2-1 mg/dL)	
Less than 1	4 (16%)
1.1-2.5	9 (36%)
2.6-4	7 (28%)
More than 4	5 (20%)

[Table/Fig-1]: Baseline demographic, exposure, and biochemical characteristics of 25 patients.
* The value in other patient is 1.2 in male which was within the normal limit

Parameters	Mean+/- SD (Male)	Mean+/- SD (Female)	p-value	Mean+/-SD (Overall)
Age (years)	30.86±7.79	30.27±6.78	0.84	30.6±7.22
Serum creatinine on admission	5.48±1.29	4.27±1.47	0.04	4.95±1.48
Serum creatinine (48h)	6.22±1.41	4.46±1.71	0.012	5.45±1.76
ALT	151.60±60.91	132.36±56.36	0.42	143.08±58.78
AST	174.64±89.48	163.47±81.66	0.74	169.72±84.54
SpO_2	80.14±5.1	83±5.69	0.206	81.4±5.45
Bilirubin	2.21±1.22	2.69±1.27	0.34	2.42±1.24
Amount consumed (Gram)	68.21±19.18	50±20.62	0.03	60.2±21.48
Survival duration (days)	2.21±0.97	3.6±1.43	0.01	2.79±1.35
Time of admission (hours after ingestion)	2.93±1.44	4.18±1.4	0.03	3.48±1.53

[Table/Fig-2]: Comparison of clinical and laboratory parameters between males and females.

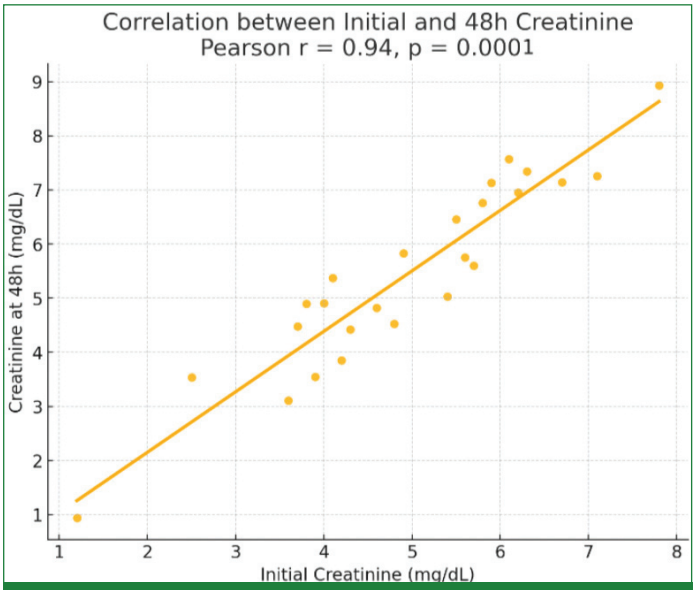
In [Table/Fig-3] the correlation analysis demonstrated a very strong positive correlation between the ingested dose of paraquat and serum creatinine levels, both at admission (r=0.93, p <0.001) and after 48 hours (r=0.92, p <0.001), indicating dose-dependent renal dysfunction.

Parameter	Correlation Coefficient (r)	p-value
Serum Creatinine (Admission)	0.93	<0.001
Serum Creatinine (48h)	0.92	<0.001
ALT	0.16	0.4401
AST	-0.22	0.3016
SpO_2	-0.91	<0.001
Bilirubin	-0.227	0.276

[Table/Fig-3]: Correlation between ingested dose and biochemical parameters.

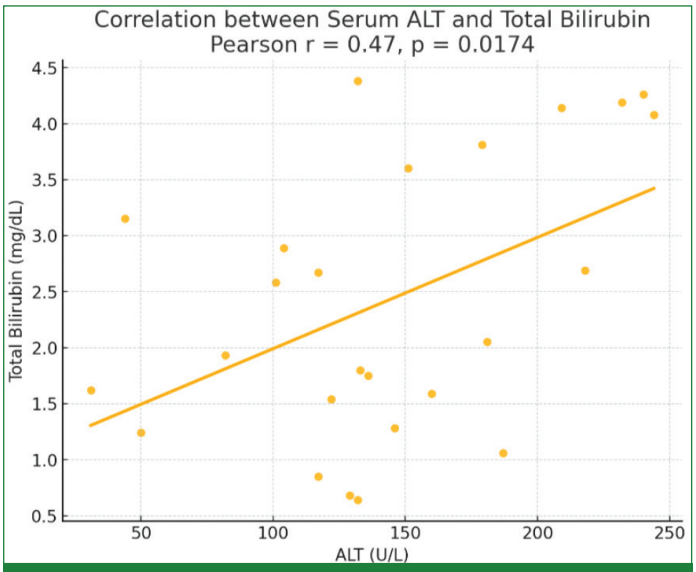
In contrast, hepatic enzymes (ALT and AST) showed weak and statistically non-significant correlations with the ingested dose, suggesting that hepatic involvement was variable and less directly dose-related. A strong negative correlation was observed between the ingested dose and SpO_2 (r= -0.91, p <0.0001), highlighting the severe pulmonary compromise associated with higher doses.

The [Table/Fig-4] demonstrates that the correlation coefficient was 0.94 (p <0.0001), indicating a strong positive correlation between initial serum creatinine and 48-hour values. This suggests progressive renal damage despite haemodialysis.



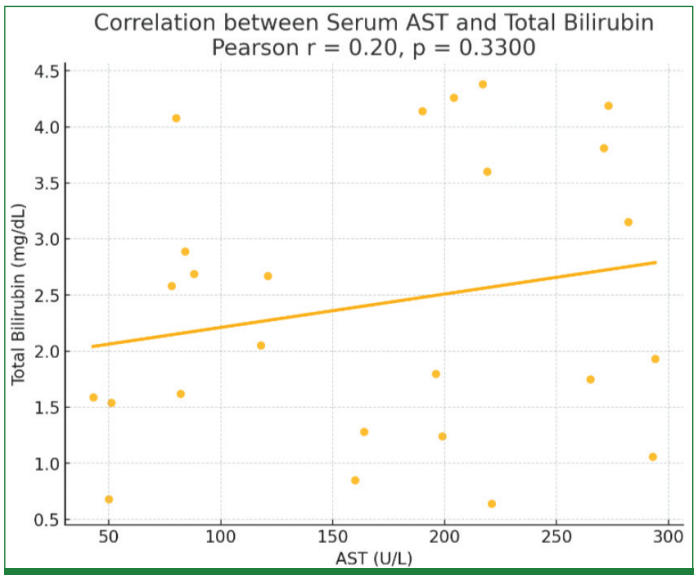
[Table/Fig-4]: Correlation between serum creatinine at admission and at 48 hours after admission.

The [Table/Fig-5] demonstrates the correlation coefficient was 0.47 (p <0.05), suggesting a moderate and statistically significant positive correlation between serum bilirubin and ALT levels.



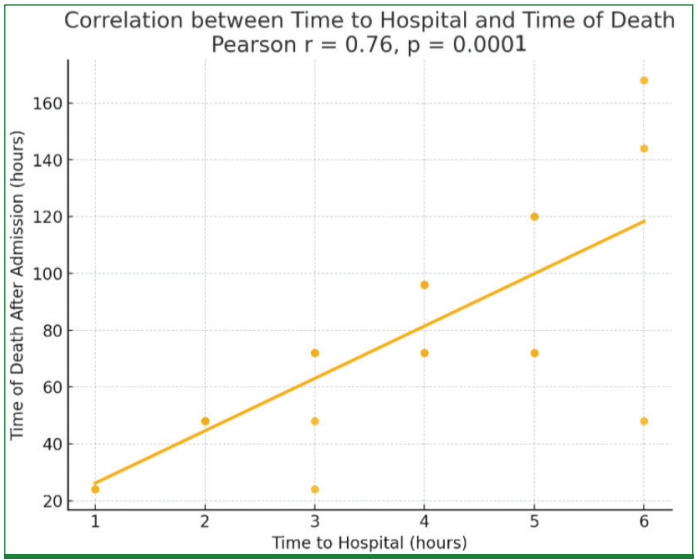
[Table/Fig-5]: Correlation between serum ALT and bilirubin levels.

The [Table/Fig-6] demonstrates that the correlation coefficient was 0.20 (p >0.05), indicating a weak and non-significant positive correlation between serum AST and bilirubin levels.



[Table/Fig-6]: Correlation between serum AST and bilirubin levels.

The [Table/Fig-7] demonstrates that the correlation coefficient was 0.76 (p <0.0001), showing a strong positive correlation between delay in hospital admission and duration of survival after admission. This may be explained by lower ingestion doses in patients who presented later, resulting in relatively prolonged survival.



[Table/Fig-7]: Correlation between time to hospital admission and survival duration.

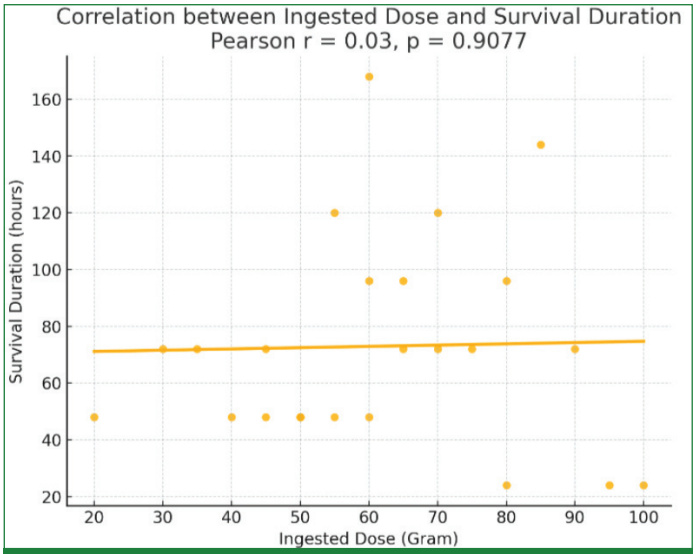
The [Table/Fig-8] demonstrates that the correlation coefficient was 0.03 (p >0.05), indicating no significant correlation between the ingested dose and survival duration, suggesting that dose alone did not determine survival time.

The [Table/Fig-9] demonstrates that the correlation coefficient was 0.42 (p <0.001), indicating a moderately positive relationship between patient age and serum creatinine at admission.

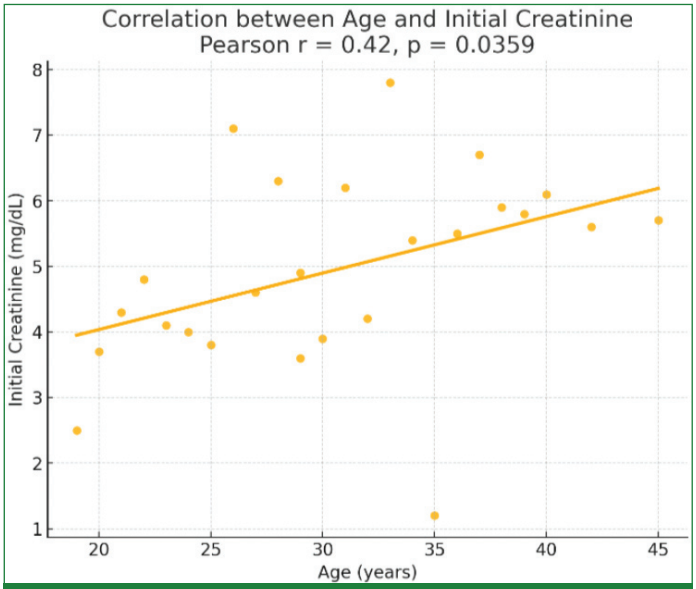
The [Table/Fig-10] demonstrates that the correlation coefficient was -0.91 (p <0.0001), demonstrating a strong negative correlation between the ingested dose and SpO_2 level- indicating that as the ingested dose increased, oxygen saturation decreased.

DISCUSSION

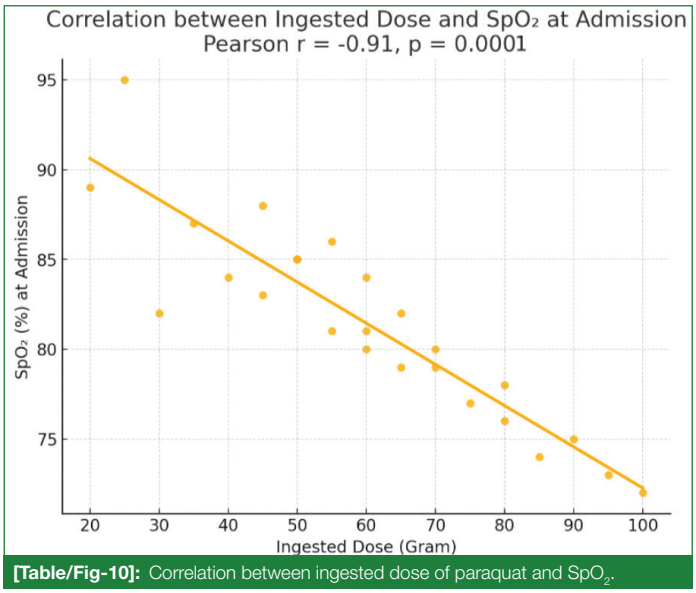
In the present retrospective cohort study from Eastern India, a dose-dependent and strong rise in serum creatinine was observed at admission and at 48 hours, indicating persistent paraquat-associated renal injury despite haemodialysis. A strong inverse



[Table/Fig-8]: Correlation between ingested dose and survival duration.



[Table/Fig-9]: Correlation between age and serum creatinine at admission.



[Table/Fig-10]: Correlation between ingested dose of paraquat and SpO_2.

correlation between the ingested dose and SpO_2 reflected pulmonary compromise, while hepatic enzyme changes showed weak or variable associations. Female predominance and 100% fatality were notable findings. Collectively, these data identify renal dysfunction as the most consistent biochemical hallmark, with pulmonary involvement closely tracking the ingested dose, while hepatic injury appears idiosyncratic and less dose-related.

Paraquat toxicity is driven by redox cycling and Reactive Oxygen Species (ROS) generation, leading to mitochondrial damage and lipid peroxidation. These mechanisms cause multiorgan injury- particularly in the lungs, kidneys, and liver- which aligns with the organ-specific patterns observed in this study [1,3,5,7]. Its absorption, distribution, and prolonged elimination kinetics (predominantly involving the lung, kidney, and liver, with a long half-life) explain the progressive tissue injury despite supportive care [3,7]. The high case fatality rate observed in this cohort is consistent with previous studies, underscoring the absence of a specific antidote and the narrow margin between exposure and multiorgan failure [7-13].

In contrast to earlier Indian studies most of which were limited to small case series, apart from the larger cohorts of Rao R et al., and Ravichandran R et al., (n=55), the present study uniquely provides region-specific biochemical correlation data from Eastern India, based on 25 systematically analysed cases [17,18]. Comparable studies are summarised in [Table/Fig-11] [6,17-24]. The female predominance noted here contrasts with the male-skewed cohorts reported from southern India, although the age distribution (20-40 years) mirrors previously identified high-risk groups [18]. Reported mortality rates from paraquat poisoning vary widely from 22-55% in some studies to as high as 92% in others [17,21]. The findings in the present study lie at the more severe end of this spectrum, highlighting the extreme lethality of paraquat.

The present study also demonstrated that higher ingested doses were associated with elevated serum creatinine and lower oxygen saturation, showing a clear dose-response relationship. These observations corroborate earlier toxicological studies by Kim HJ et al., [7] and the clinical prognostic analyses by Gawarammana IB and Buckley NA [5].

The observation that serum creatinine worsened despite haemodialysis aligns with heterogeneous outcomes reported in the literature on extracorporeal interventions [17]. A multicentre study by Yeh YT et al., suggested that haemoperfusion may improve overall survival [25]; however, timely initiation appears crucial, with earlier intervention associated with better outcomes [17,24]. The continued renal deterioration in this cohort suggests that delayed or suboptimal timing of haemodialysis is unlikely to reverse established tubular injury.

Adjunctive pharmacologic therapies- such as pulse cyclophosphamide with corticosteroids or antioxidant regimens- have shown variable or context-dependent benefits and require further validation before routine use [11,16,26-28]. Operationally, the findings emphasise that ultra-early triage, risk stratification based on ingested dose, and prompt initiation of dialysis or haemoperfusion may improve outcomes in real-world settings [27,29].

The weak and inconsistent hepatic correlations (ALT/AST/bilirubin vs dose) suggest that hepatic injury is heterogeneous and less dose-deterministic than renal or pulmonary damage. This is consistent with prior reports describing a wide spectrum of hepatic responses from transient enzyme elevations to toxic hepatitis [2,9-11]. Clinically, this implies that renal and respiratory parameters should serve as the primary indicators for early prognostication, while liver function tests should be interpreted in context rather than as principal severity markers.

For clinicians, the ingested dose at presentation, creatinine trajectory (0-48 hours), and SpO_2 trends should guide urgent triage and management decisions. For researchers, these findings underscore the need for prospective studies evaluating time-to-haemoperfusion, standardised antioxidant and immunosuppressive protocols, and the development of practical prognostic tools integrating dose with early renal- pulmonary indices. From a public health perspective, preventive measures and restricted access to paraquat remain critical in rural settings, where intentional self-poisoning is a persistent problem [4,5].

Authors	Place of study	Sample size	Objectives	Parameters assessed	Conclusions
Kanchan T et al., [6] (2015)	Manipal, South India	Not specified (analysis of fatal cases)	Analyze uncommon cause of fatal poisoning due to paraquat	Clinical and forensic features	Paraquat poisoning though uncommon is often fatal; emphasizes awareness and preventive strategies.
Rao R, et al., [17] (2017)	South India	Not clearly mentioned	Assess role of early hemoperfusion therapy in paraquat poisoning	Timing of therapy, survival outcomes	Early hemoperfusion during 'golden hours' improves outcome in severe paraquat poisoning.
Ravichandran R, et al., [18] (2020)	South India (tertiary care center)	55 patients	Study retrospective outcomes of paraquat poisoning cases	Clinical features, survival, complications	High fatality; renal and pulmonary dysfunction common; emphasizes early recognition.
Narendra SS and Vinaykumar S [19] (2015)	South India	Case series (exact number not given)	Describe paraquat poisoning cases	Clinical presentation	Reported variable outcomes; underlines importance of early supportive therapy.
Prasad DRMM and Chennabasappa A [20] (2015)	Asia-Pacific (case series)	Not specified	Outcome of poisoned patients using therapeutic flowchart	Treatment response	Structured flowchart-based management may standardize care and improve prognosis.
Sandhu JS, et al., [21] (2003)	India	5-year study (number not clearly given)	Study outcome of paraquat poisoning over 5 years	Clinical course, survival	High mortality; paraquat poisoning remains a major challenge.
Pavan M [22] (2013)	India	Not specified	Study acute kidney injury following paraquat poisoning	Renal involvement	Acute kidney injury is a frequent complication of paraquat poisoning in India.
Banday TH et al., [23] (2014)	Rural Karnataka, India	Not specified	Assess manifestation, complications, and outcomes	Clinical course, complications	High incidence of complications; paraquat poisoning carries poor prognosis.
Jagadeesan M et al., [24] (2017)	Tertiary care hospital, India	Not specified	Study clinical profile of paraquat poisoning	Clinical features, biochemical derangements	Confirmed severe renal, hepatic, and pulmonary involvement; prognosis remains poor.
Present study	Jagannath Gupta Institute of Medical Sciences & Hospital, West Bengal, Eastern India	25 patients	Analyze biochemical spectrum of organ dysfunction in paraquat poisoning	Serum creatinine, AST, ALT, bilirubin, SpO_2, ingested dose correlation, sex differences	Renal and pulmonary dysfunction strongly dose-dependent and most prognostic; hepatic dysfunction variable; high mortality despite hemodialysis; emphasizes need for ultra-early detection and prevention strategies.

[Table/Fig-11]: Similar studies from the literature [6,17-24].

Limitation(s)

The limitations of this study include a small sample size, retrospective design, and the absence of multivariable adjustment. The initiation and modality of extracorporeal therapies were not standardised, limiting causal inference regarding the efficacy of haemodialysis or haemoperfusion. These limitations highlight the need for prospective, multicentre studies incorporating time-stamped treatment data and adjusted analyses (for age, sex, delay to admission, and ingested dose).

CONCLUSION(S)

This study from Eastern India demonstrates that paraquat poisoning causes severe renal impairment that correlates strongly with ingested dose, persists despite haemodialysis, and represents the most reliable biochemical predictor of outcome. Pulmonary involvement, reflected by decreasing SpO_2, also showed a strong dose-dependent relationship, whereas hepatic dysfunction was less consistent and variable. The young age profile and female predominance underscore the socio-demographic vulnerability of affected rural populations.

These findings suggest that early assessment of ingested dose, along with close renal and pulmonary monitoring, is essential for prognosis. Delayed or non-optimised haemodialysis alone is unlikely to alter disease progression. The extremely high fatality rate observed underscores the urgent need for early triage, preventive strategies, and further evaluation of alternative therapies such as haemoperfusion and antioxidant-immunosuppressive combinations.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Aug 13, 2025
- Manual Googling: Oct 16, 2025
- iThenticate Software: Oct 25, 2025 (8%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

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