# Clinical profile and outcome of paraquat poisoning at a tertiary care centre in North Karnataka, a retrospective observational study

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## Abstract

**Background:** Paraquat is a herbicide commonly used for controlling weeds in India. The primary target organs for paraquat poisoning are the lungs and the kidneys. Acute cases of poisoning with paraquat are admitted to the hospital with various stages of acute kidney injury.

**Aim:** To study the clinical presentation and outcome of paraquat poisoning in a tertiary care setting.

**Materials and Methods:** A retrospective observational study was conducted at SDM College of Medical Sciences and Hospital, Dharwad. In this study we included the data of all patients admitted to the hospital with paraquat poisoning for a period of three-year and four months between January 2018 to April 2021.

**Results:** A total of 12 participants were included in the final analysis**.** All the patients consumed paraquat with the suicidal intention only. The quantity of paraquat ingested was quite varied, ranging from as low as 5ml to as high as 200ml. Acute Kidney Injury (AKI) was diagnosed in 58.3% of patients. Among which three patients were in stage 1, one was in stage 2, and three were in stage 3. The mortality rate was 58.33%. The major cause of death for these patients was multiple organ dysfunction syndromes (71.42 %).

**Conclusion:** Acute kidney injury is the major clinical outcome of paraquat poisoning other than lung injury. This may result in multiple organ dysfunction syndrome (MODS) and mortality. Paraquat poisoning is due to consumption with suicidal intent. Most of them were young. Early management with hemoperfusion may have a positive effect on reducing mortality.

**Key words:** Paraquat poisoning, Acute Kidney Injury, multiple organ dysfunction syndromes (MODS), Hemoperfusion.

## Introduction

Paraquat is a herbicide commonly used to control weeds in India[1]. Paraquat is classified as "class II chemical or moderately hazardous by the World Health Organization and as 'hazardous' by other agencies"[2,3]. The active ingredient in paraquat is 1,1'-dimethyl-4,4'-bipyridinium which is toxic to organs in humans and has been the cause of acute poisoning by ingestion and also by skin exposure. The primary target organ for paraquat poisoning are the lungs and kidneys. Due to its structural similarity to naturally occurring polyamines it is absorbed by alveolar cells and its accumulation leads to damage in the form of alveolitis and fibrosis of the lungs[4]. Paraquat is secreted by the kidneys because of which there is accumulation in the proximal tubular cells at higher concentrations and which in-turn causes vacuolation of epithelial cells leading to renal tubular necrosis[5]. The cause of death in most of the cases are due to lung injury or multiorgan failure[6].

Exposure to paraquat (PQ) is mainly by ingestion with suicidal intent. Consumption of PQ >40mg/kg causes multiple organ dysfunction with death within 48 hours of consumption, whereas consumption of < 20 mg/kg of PQ will result in mild symptoms with high survival rates[6]. There are no specific antidotes available

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to block the effects of PQ. Majority of patients receive systemic steroids or cyclophosphamide or antioxidants which reduces free radical damage [7].

It has been observed that acute cases of poisoning with paraquat are admitted to the hospital with various stages of acute kidney injury. Often specific diagnosis of paraquat poisoning was missing. Few studies are available observing the outcome of paraquat poisoning and the efficacy of hemoperfusion. Hence this study was conducted to describe the clinical profile (symptoms, signs, and biochemical profile) and also the outcome of patients who presented with paraquat poisoning.

**Materials and methods:**

A retrospective observational study was conducted at SDM College of Medical Sciences and Hospital, Dharwad. The data collection was done retrospectively from the hospital records from January 2018 to April 2021. Patients aged more than 18 years of age admitted to the hospital in whom the history of paraquat poisoning was obtained from history, reference letters and the bottle produced. Patients aged more than 18 years of age who presented with paraquat poisoning while patients with pre-existing renal or liver diseases and other poisonings were excluded from the study.

After identifying the cases based on the inclusion and exclusion criteria, the records were analysed, and the details about the demographic characteristics, route and amount of paraquat ingestion, whether suicidal/ accidental, clinical features, laboratory parameters, organ involvement and response to treatment and final outcome of the patients was recorded. Mortality details with the cause of death following paraquat poisoning were recorded. The diagnosis of Acute Kidney Injury (AKI) was made as per Acute Kidney Injury Network (AKIN) criteria, based on serum creatinine and urine output[8].

**Ethics statement:**

Institutional review board approval was obtained [SDMIEC/2021/88-A]. The authors followed the tenets of declaration of Helsinki.

**Statistical methods**

The results are obtained by descriptive analysis using mean with standard deviation (SD) or median and interquartile range (IQR) for numeric data, depending on the distribution. Categorical variables were described using frequency and proportion. SPSS software, V.22 was used for analysis.

**Results:**

**Table 1: Summary of demographic parameters (N=12)**

| Parameters | Summary |
|---|---|
| Age (in years) | 30.08 ± 10.59 |
| **Gender** | |
| Female | 4 (33.33%) |
| Male | 8 (66.67%) |

A total of 12 patients were included. The clinical data of the all patients were analysed. The mean age of the patients with paraquat poisoning was 30 ± 10.59 years. The youngest patient was 20 years, and the oldest was 55 years of age. Eight patients were male, and four were female. (Table 1)

**Table 2: Clinical presentation of Paraquat poisoning (N=12)**

| **Intention of Ingestion** | |
|---|---|
| Suicidal | 12 (100%) |
| Amount of paraquat ingested (in ml) | 50 ± 57.64 (range 5 to 200 ml) |
| **Duration (hours) from ingestion to admission** | |
| <6 hours | 6 (50%) |
| 6 to 24 hours | 2 (16.67%) |
| > 24 hours | 4 (33.33%) |
| **Symptoms on presentation** | |
| Vomiting | 6 (50%) |
| Altered sensorium | 4 (33.33%) |
| Abdominal pain | 1 (8.33%) |
| Vomiting, pain abdomen, sore throat | 1 (8.33%) |
| GCS at presentation | |
| 3 to 9 | 4 (33.33%) |
| 9 to 12 | 8 (66.67%) |
| Oxygen Saturation | 96.42 ± 5.35 |

All the patients consumed paraquat with the suicidal intention. There was a wide range of quantities ingested, ranging from 5ml to 200ml. 50% (N=6) got admitted within 6 hrs of ingestion, and 33 % (N=4) were admitted after 24 hours of ingestion. 50% of patients had vomiting as a major complaint, followed by altered sensorium in 33%. 4 patients presented with GCS of 3 to 9, and 8 patients had GCS of 9 to 12. (Table 2)

**Table 3: Biochemical parameters of Paraquat poisoning (N=12)**

| **ABG** | |
|---|---|
| Metabolic acidosis | 7 (58.33%) |
| Normal | 5 (41.67%) |
| **Creatinine (Quantitative day 1)** | **3.01±4.27 (range 0.69 to 15.89)** |
| **Liver Function Test** | **Median (IQR)** |
| Abnormal Total Bilirubin | 3 (25%) |
| Direct bilirubin (N=10) | 3 (25%) |
| SGOT (N=10) | 3 (25%) |
| SGPT (N=10) | 2 (16.66%) |
| INR (N=9) | 6 (50 %) |

With respect to the biochemical parameters of the patients, 58.3% of the patient presented with metabolic acidosis. With respect to liver function tests, increased total and direct bilirubin were seen in 3 (25%) of the patients. Increased SGOT in 3 (25%) and increased SGPT in 2 (16.6%) of the patients. Increased INR was present in 6 (50%) of the patients. (Table 3)

Out of 12 patients, 58.3% (N=7) patients had Acute Kidney Injury (AKI) (Figure 1).

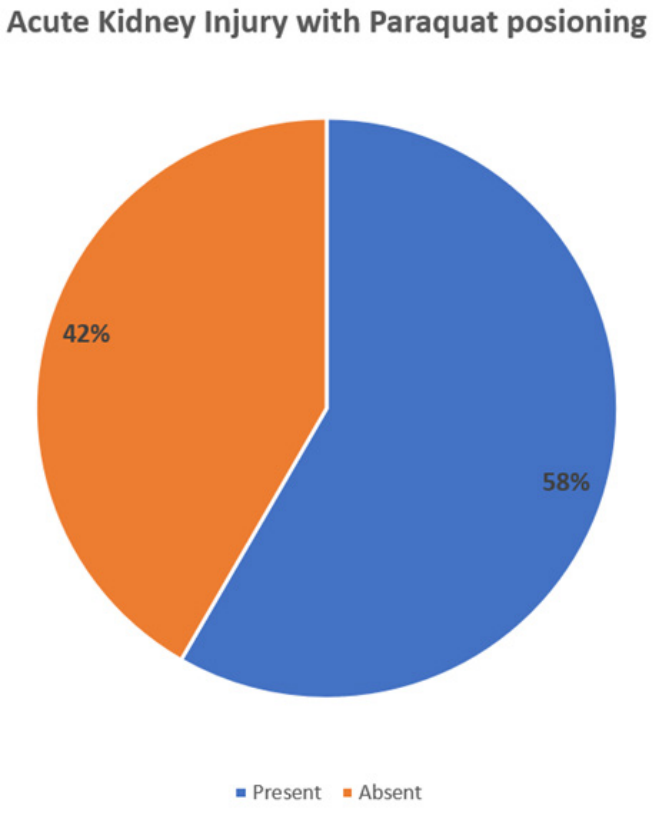


**Figure 1: Acute kidney injury with paraquat poisoning.**

Among which 3 were in stage 1, 1 was in stage 2, and 3 were in stage 3. On chest Xray, 10 patients had normal findings and 2 patients showed lung involvement. (Figure 2)

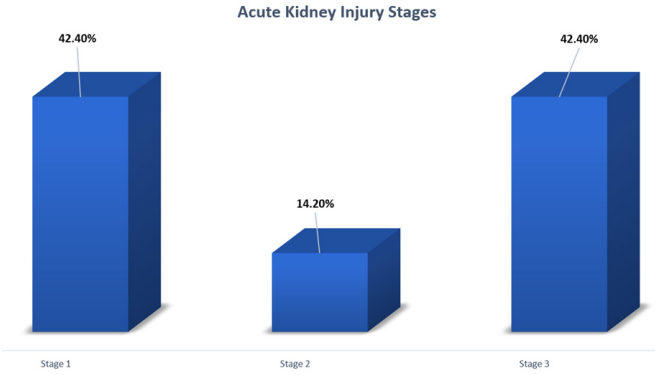


**Figure 2: Acute kidney injury stages.**

Regarding the clinical management of the cases, all patients were treated with gastric lavage and activated charcoal. Hemoperfusion was done in 3 patients. One patient received 5 sessions of hemoperfusions, second patient received 4 session and third patient received 1 hemoperfusion. In other patients hemoperfusion was not done as the presentation was beyond the time period required for hemoperfusion and patients were not affordable. 62.5 % (N=5) required ventilatory support.

**Table 4: Outcome of paraquat poisoning**

| **Outcome** | **N(%)** |
|---|---|
| Discharged Against Medical Advice -DAMA | 4 (33.3%) |
| Recovery | 1 (8.33) |
| Death | 7 (58.33%) |
| Mortality occurrence time (N=7) | |
| 24 to 72 hours | 3 (42.86%) |
| >72 hours | 4 (57.14%) |
| Cause of death (N=7) | |
| MODS | 5 (71.42%) |
| Others | 2 (28.58%) |

The clinical outcome of patients admitted with paraquat poisoning was analysed. 7 out of 12 patients died with mortality rate of 58.33%. One patient recovered fully. 33.3% (N=4) went discharge against medical advice (DAMA), and their final outcome was not assessed. Out of the 7 patients who died, 3 patients died within 72 hours and 4 patients after 72 hours. The major cause of death for these patients, 71.42 % (N=5), was multiple organ dysfunction syndromes (MODS) and 2 patients died of other causes.(Table 4)

**Table 5: Clinical parameters of patients who died**

| Parameter | |
|---|---|
| Mean Age | 30 ± 10.7 years |
| Mean volume of consumption | 74.28 ± 65.79 ml |
| GCS | All patients had a GCS score of 3 on admission |
| Chief Complaint | Altered sensorium - 5, Vomiting - 2 |
| Acute Kidney Injury (AKI) | Stage 1 - 2, Stage 3 - 2 |

Outcome is available only for 8 patients as 4 patients underwent discharge against medical advice (DAMA) and outcome was not assessed. Among these 8 patients, 7 died and 1 patient recovered fully. With respect to the presence of Acute Kidney Injury and its stages, 1 patient who recovered had Stage 2 AKI. 2 patients who had Stage 1 AKI died and 2 patients who had Stage 3 AKI died. With respect to performed hemoperfusions on patients, 1 patient died, 1 patient recovered and 1 patient went discharge against medical advice (DAMA). All 6 patients who did not receive hemoperfusion died. No statistical significance test was performed because of the small number of patients.

## Discussion

Paraquat poisoning has vital toxicological importance, especially in the southern part of India, as it is used widely. The mortality rate in our study was 58.33% which is significantly higher. This rate is comparable to the study by Raghavendra Rao et al. in India, which showed a mortality rate of 61%[9]. This is similar to the hospital mortality in other countries, too, which ranges from 35-62%[10,11]. Majority of the deaths in our study were due to multiple organ dysfunction syndromes (MODS). The finding is similar to another study conducted by M. Indira et al. in South India, in which all the deaths (N=6) were due to MODS[12].

In this study, all patients consumed paraquat with suicidal intention, and there was no accidental consumption of paraquat. This is comparable to a study conducted by Kanchan et al. in South India, which shows 92.9% of paraquat poisoning was with suicidal intention[13]. In our study, the mean age was 30 years, and the majority of the patients were male. This is comparable to the study by Kanchan et al., which showed paraquat poisoning mainly occurs in young males[13].

Three patients underwent hemoperfusion. Rao et al. reported that early hemoperfusion might be of benefit[9]. Other studies conducted by Li et al. and Koo et al. showed that there is moderate benefit when hemoperfusion is combined with continuous venovenous hemofiltration[14,15].

The respiratory system was analysed through Chest X-ray and the requirement of ventilatory support. X-ray showed involvement of the left lower lobe. Similar findings were observed by Kanchanet al. in South India, in which major involvement was seen in the lower lobe of the lungs[13].

In our study, Acute Kidney Injury has been seen in 58.3 % (N=7) of patients with paraquat poisoning. Three patients had stage 3 AKI. The association of paraquat poisoning with AKI is explained by Wang et al. where it has been explained that paraquat-induced oxidative stress affects the renal tubules[16]. A study by Gao et al. showed that the incidence of AKI could reach 71.7% with paraquat poisoning[17]. The causative link between paraquat and acute kidney injury has been explained in a systematic review conducted by Vadovar D et al[18].

Retrospective single centre study with no long term follow up data and non-availability of PQ blood levels are few limitations of the study. Four patients got discharged against medical advice, and the prognosis of those patients was not recorded.

## Conclusion

Paraquat is an herbicide, when consumed, has a high mortality rate. It mainly affects the lung and kidneys. Acute kidney injury is the major clinical outcome of paraquat poisoning. It causes multiple organ dysfunction syndromes (MODS) as the main cause of death. Paraquat poisoning is due to consumption with suicidal intent. Most of the patients were of young age. Early management with hemoperfusion may have a positive effect on reducing mortality. There is a need for evidence-based management for a successful outcome from paraquat poisoning. Hence this study provides vital evidence for the development of management protocols in the future where there may be a role of hemoperfusion even in delayed presentation

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