J Renal Inj Prev. 2023; x(x): e32045.

https://journalrip.com doi: 10.34172/jrip.2023.32045

Journal of Renal Injury Prevention

Paraquat poisoning; an experience from a tertiary care center in India

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ARTICLE INFO

Article Type:
Original

Article History:
Received: 18 March 2022
Accepted: 8 June 2023
ePublished: 23 September 2023

Keywords:
Paraquat
Poisoning
India
Outcome
Treatment options
Acute kidney injury

ABSTRACT

Introduction: Paraquat poisoning is a commonly used toxin within the Indian sub-continent. Despite several advances in medical care, mortality in cases of paraquat poisoning continues to be high with controversies in the ideal management.
Objectives: The aim of this study was to throw light on the clinical course of these patients in the current setting and evaluate some of the novel therapeutic options such as hemoperfusion in the setting of paraquat poisoning.
Patients and Methods: This study is a retrospective observational study examining all adult patients with paraquat poisoning over the last five years from a tertiary care centre in Southern India. Demographic characteristics, clinical and laboratory profile, treatment options, and outcome were analysed from online hospital records.
Results: Of 13 patients, all patients, except one, had ingested paraquat deliberately. Acute kidney injury (AKI) occurred in all patients (100%). Dialysis was required in 7 patients (54%) with AKI. Lung injury was seen in 63.5% of patients (n = 8) and most patients i.e. 92.3% (12 patients) had multi-organ dysfunction syndrome (MODS). Hemoperfusion was provided to 30.8% of patients without much benefit in terms of duration of hospitalisation or time to death. Hemodialysis and continuous renal replacement therapy (CRRT) were offered for conventional indications. Dialysis was found to have significant bearing on time to death.
Conclusion: Paraquat is a highly lethal herbicide with few effective therapeutic options. The majority of patients develop multi-organ dysfunction and succumb to death owing to circulatory collapse. Dialysis may be able to delay mortality in paraquat poisoning.

Original

Implication for health policy/practice/research/medical education:
Paraquat poisoning is a common method of accidental or deliberate self-harm in the Indian subcontinent. It carries with its poor prognosis and delivers a death sentence to most patients who consume it. Multiple therapies have been tried treat patients with poisoning. The efficacy of hemo-perfusion, anti-inflammatory therapy and dialysis in paraquat poisoning is not known. This study elaborates the different clinical manifestations, outcomes and evaluates therapies currently available for paraquat poisoning today.

Please cite this paper as: Jose , Elayaperumal I, Arumugam D, Matcha J. Paraquat poisoning; an experience from a tertiary care center in India. J Renal Inj Prev. 2023; x(x): e32045. doi: 10.34172/jrip.2023.32045.

Introduction

Paraquat (1, 1′-Dimethyl-4, 4′-bipyridinium dichloride) is a rapidly acting non-selective herbicide used extensively in Asia-pacific, Papua New Guinea, and the Caribbean countries. Because of the high toxicity paraquat has restricted use in the USA and withdrawn from European Union (1). Within the Indian sub-continent, paraquat has been used extensively in agricultural farms. In many instances, it has resulted in toxicity to humans both accidentally and deliberately. The mechanism of paraquat toxicity involves the generation of superoxide anion which leads to the formation of more toxic reactive oxygen species and oxidation of nicotinamide adenine dinucleotide phosphate (NADPH). With the depletion of NADPH and lipid peroxidation, cell membrane damage occurs leading to cell death (2).

Paraquat has a predilection for the lungs which have the highest oxygen tension. In the lung, it gets concentrated in type 1 and type 2 alveolar epithelial cells, causing pulmonary edema then subsequently results

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in an infiltration of inflammatory cells with damage to alveolar epithelium. Eventually this results in respiratory failure and pulmonary fibrosis. Paraquat is primarily excreted through kidneys where it causes acute tubular necrosis. The acute tubular necrosis then prevents further excretion of the toxin. Mortality is related to the quantity of the chemical ingested. With severe poisoning (when the ingested dose is more than 20 mL) multi-organ dysfunction syndrome (MODS), renal, liver, cardiac as well central nervous system dysfunction occurs.

Currently there are no specific guidelines available for the management of paraquat poisoning. Decontamination with activated charcoal and elimination of the toxin from blood by hemoperfusion is used when patients with paraquat poisoning present within 2 to 4 hours of ingestion. However, studies have shown varied results on the efficacy of this approach. As paraquat induces inflammation and injury, immunosuppressants such as steroids, and cyclophosphamide have also been tried in few randomized controlled trials. Only few studies have been conducted to date looking at the clinical profile of patients, their course in hospital and their outcomes.

Objectives

This study aims to throw light on less studied aspects of paraquat poisoning such as the clinical profile of patients, their course in hospital, utility of various therapeutic options that have been used to treat paraquat and provide a comprehensive review on paraquat poisoning.

Patients and Methods

Study design

Data for this study was collected in a retrospective manner from the dates of January 2015 up to January 2020 from the online database of a large tertiary level hospital in Southern India. All adult patients with a diagnosis of "paraquat poisoning" were evaluated. Other herbicides and toxin ingestions were excluded. All patients with paraquat poisoning in this hospital are treated according to the following protocol.

Standard operating protocol for paraquat poisoning

Assessment and management of airway, breathing, and circulation is performed on arrival to the emergency room. Patients who reach the hospital within 4 hours of paraquat ingestion are given activated charcoal of 1gm/kg through a nasogastric tube to reduce the gastrointestinal absorption. Fluid resuscitation was carried out with 15–20 mL/kg/h. Oxygen supplementation is given to patients with PO_2 of less than 70 mm Hg. Daily monitoring of intake-output chart, cardiac, respiratory status, and level of consciousness was conducted in all patients. Acute kidney injury is diagnosed based on Kidney Disease Improving Global Outcomes (KDIGO) criteria and hemodialysis is offered to patients based on conventional indications. Patients with circulatory failure requiring dialysis are managed with continuous renal replacement therapy (CRRT). Hemoperfusion was conducted for patients who arrive at the hospital within 4 hours of ingestion. Antioxidants such as vitamin C tablets 500 mg twice daily and vitamin E 400 mg twice daily is given to all patients. Dexamethasone injection 4 mg twice daily is given to patients who consume more than 20 mL of paraquat as anti-inflammatory therapy.

Statistical analysis

For this study, baseline characteristics such as the quantity of paraquat toxin ingested the time interval between ingestion and arrival to the hospital, laboratory data including complete blood count, renal function tests, liver function tests, X-ray, and computed tomography (CT) of the chest was noted from hospital online database. The details of treatment administered were also noted and outcome data retrieved from hospital records. The data obtained was then analysed using SPSS version 21. Odds ratio and Pearson's chi-square testing were conducted to check the efficacy of interventions in paraquat poisoning.

Results

From hospital records there were 13 patients in all who had consumed paraquat over a period of five years. The baseline characteristics of the study population are shown in Table 1. The mean age of the study population was 26.4 years with 8 males (61.5%). Most patients consumed the toxin with intent to deliberately harm themselves (n = 12, 92.3%) and only one patient (7.7%) consumed the toxin accidentally. Most of the patients (70%) had consumed a large quantity of the concentrate (>20 mL).

Table 1. Baseline characteristics of the study population

Baseline characteristics	N= 13 patients (percent/2 standard deviations from mean)
Mean age	26.46 years (±7.96)
Gender	
Male: Female	8: 5
Reason for consumption	
Accidental	1 (7.6%)
Deliberate self-harm	12 (92.3%)
Type of poison	
Only paraquat	11 (84.6%)
Mixed content	2 (15.3%)
Quantity consumed	
≤10 mL	2 (15.3%)
10-20 mL	1 (7.6%)
≥20 mL	7 (53.8%)
Mean quantity consumed	83.5 mL (± 71.33)
Co-morbidities in the study population	None
Mean time to arrival at the hospital	17.13 hours (± 30.77)

Table 2 shows the baseline laboratory data of patients who had consumed paraquat. Leucocytosis was seen in 91.7% of patients. Thrombocytopenia was less often seen (25%). All patients eventually developed acute kidney injury (AKI) and most patients had elevated liver enzymes and bilirubin along with prolonged prothrombin time on admission.

Figure 1 shows the organ involvement in paraquat poisoning where the majority of patients (92.3%) had multi-organ dysfunction. Liver and renal involvement was seen in all patients. Lung involvement was the next most commonly seen in 61.5% of patients. The circulatory collapse was seen in 46.2% of people. Neurological involvement was the least common clinical manifestation of paraquat poisoning in this study.

Little over half the study population (54%) presented early to the hospital within 4 hours of paraquat consumption. Table 3 shows the renal involvement in paraquat poisoning. The mean creatinine level at presentation was 3.8 mg/dL. AKI occurred in all 13 patients (100%) with 10 (76.7%) of them developed within 24 hours and the remaining (n = 3, 23.1%) in 48 hours. Dialysis was required in 7 patients (54%) with AKI. Moreover, CRRT was offered to one patient, the rest received conventional hemodialysis.

The treatment administered to patients is represented

Table 2. Baseline Laboratory characteristics

Lab parameter at presentation	N =13 (percent/(2 standard deviations from mean - 2SD)	Missing data
Total counts elevated (>11 000 cells/cmm)		
Number of patients with high count	11 (91.7%)	1 Missing
Average total count	20 475 cells/cmm (±12208.27)	
Mean hemoglobin	14.30 g/dL (± 2.10)	
Thrombocytopenia (<1.5 lak/cmm)		
Number of patients with thrombocytopenia	3 (25%)	1 Missing
Average platelet count	1.76 lak/cmm (±0.75)	
Elevated creatinine (>1.4 mg/dL)		
Number of patients with elevated creatinine	11 (84.6%)	None
Average creatinine at presentation	3.8 mg/dL (±2.84)	
Serum Potassium levels		
Hypokalemia <3.5 mEq/L	6 (46.2%)	None
Hyperkalemia>5.5 mEq/L	1 (7.6%)	
Mean potassium	3.80 (±0.88)	
Serum Bicarbonate levels		
<10 mEq/L	1 (7.6%)	
10–22 mEq/L	11 (84.6%)	None
>22 mEq/L	1 (7.6%)	
Mean bicarbonate levels	17.23 (±4.12)	
Elevated total bilirubin (>1 mg/dL)		
Number of patients with elevated bilirubin	9 (81.8%)	2 Missing
Average bilirubin	5.07 mg/dL (±4.20)	
SGPT (>40 U/L)		
Number of patients with abnormal SGOT	11 (91.7%)	1 Missing
Mean SGOT value	307.50 U/L (± 347.791)	
SGOT (>40U/L)		
Number of patients with abnormal SGPT	12 (100%)	1 Missing
Mean SGPT value	553.67 U/l (±718.64)	
Mean time to fulminant hepatic failure	40.42 h (±35.62)	None
Prothrombin time (>11.5 s)		
Number of patients with prolonged PT	11 (91.7%)	1 Missing
Mean PT value	18.70 s (±16.05)	
INR value (>1.2 abnormal)		
Number of patients with elevated INR	4 (33.3%)	1 Missing
Mean INR value	1.66 (±1.38)	
APTT value (>40 s abnormal)		
Number of patients with elevated APTT	3 (25%)	1 Missing
Mean APTT value	39.51 s (± 31.05)	

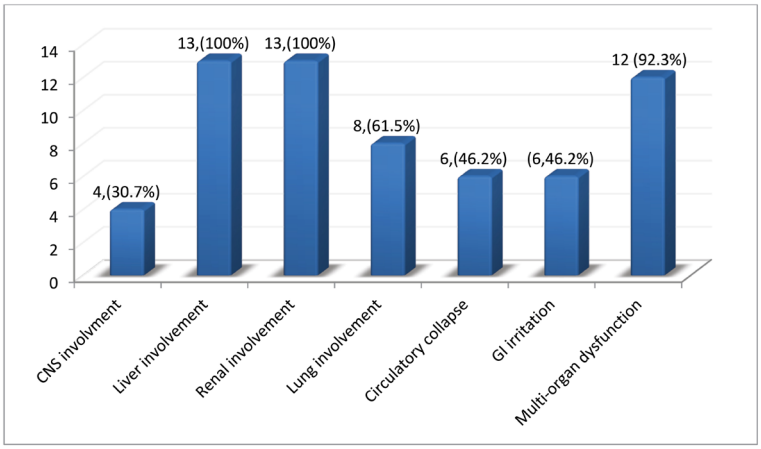

Figure 1. Organ involvement in paraquat poisoning (N =13).

in Table 4. Decontamination with activated charcoal was carried out in 69.27% of patients. Anti-oxidant therapy (vitamin C 500 mg twice daily and vitamin E 400 mg twice daily) was administered to three patients (23.1%). Dexamethasone injection 4 mg twice daily was given to seven patients (70%) who consumed more than 20 ml of paraquat as anti-inflammatory therapy. Hemoperfusion was conducted for four patients (30.8%) of the study population who reached the hospital within 4 hours of consumption. Plasma exchange was done for two patients with fulminant hepatic failure.

Paraquat poisoning resulted in death in 69% of patients while the remaining 31% of patients were lost to follow up after they were discharged against medical advice. The most common cause of death was MODS with circulatory failure (56%) followed by cardiac toxicity (22%) and acute respiratory distress syndrome (ARDS 11%). The mean time to death was 4.9 days with 55.5% of patients dying after 72 hours of hospital admission. The mean duration of hospitalization was 4.5 days. Of all the interventions used to treat paraquat poisoning only hemodialysis (conventional or CRRT) was found to have a significant bearing on the time to death as shown in Table 5.

Discussion

Paraquat poisoning can occur through contact, inhalation, or ingestion. The most common route of poisoning is ingestion. Contact with paraquat usually results in a mild clinical course limited to local irritation whereas, the inhalation of paraquat is highly lethal since it not only causes local inflammation to the lungs but also gets absorbed into the bloodstream through the alveolar capillaries (3). When ingested, it is absorbed rapidly and incompletely from the gut with only1-5% of an oral dose absorbed through the intestine. Paraquat has a low volume of distribution of 1-2 L/kg (4). It has a half-life of roughly 85 hours and 90% of ingested paraquat is rapidly removed by the kidneys (5).

Paraquat is a bright green corrosive which has a pungent odour. It is usually available as a liquid formulation of a

Table 3. Renal involvement in paraquat poisoning (N=13)

Renal parameters	N=13 (percentage/ 2 standard deviations from mean)	Missing
Proteinuria		
None	2 (18.2%)	2 Missing
2+ proteinuria	4 (36.4%)	
3+ proteinuria	5 (45.5%)	
RBC		
<2 cells/cmm	3 (27.3%)	2 Missing
>2 cells/cmm	8 (72.7%)	
Time to AKI involvement		
<24 hours	10 (76.9%)	None
>48 hours	3 (23.1%)	
Mean time to involvement	31.65 hours (±39.02) (1.3 days)	
Mean creatinine value at presentation	3.80 mg/dL (±2,84)	None
Peak creatinine value	5.12 mg/dL (±2.37)	None
Requirement for RRT	7 (53.8%)	None
Mean time to HD requirement	63.42 hours (±47.35) (2.6 days)	None

Table 4. Treatment options received by the patient (N=13)

Type of treatment	No. (%)
Decontamination with activated charcoal	9 (69.27)
Dexamethasone	3 (23.1)
Antioxidants	3 (23.1)
Dialysis	7 (53.8)
Conventional hemodialysis	6 (46.2)
CVVHDF	1 (7.7)
Hemoperfusion	4 (30.8)
Mean number of hemoperfusion	1.50 (±0.57) sessions per patient
Plasma exchange	2 (15.4)
Mean number of plasma exchange cycles	2.50 (±2.12)

Table 5. Odds ratio for time for early death (<72 hours) for various factors in paraquat poisoning

	Odds ratio (95% confidence interval)	*P* value
Dose of paraquat more than 20 mL	5 (0.27-91.51)	0.26
Time to arrival at the hospital less than 4 hours	2.5 (0.25-24.71)	0.42
Anti-oxidant therapy	0.33 (0.02-5.02)	0.41
Anti-inflammatory therapy	2 (0.13-29.80)	0.61
Hemodialysis	0.08 (0.05-1.19)	0.04
Hemoperfusion	0.80 (0.07-8.47)	0.85
Plasma therapy	0.83 (0.04-16.99)	0.90

20% solution (6). It may be sold in an isolated formulation or mixed with other compounds (sodium chlorate or 2, 4-dimethylamine). Severe intoxication is seen with more than 20 mL of consumption of the toxin. The majority of individuals in this study, up to 70%, consumed a high dose of paraquat and this might account for the high mortality rate in this study. The dose of paraquat consumed has been shown to correlate with mortality in other studies as well. In literature, urine concentration of paraquat of less than 1 mg/L within 24 hours of consumption is associated with a good prognosis (7). Urine is usually treated with 2ml of 1% solution of sodium dithionate in 1 N sodium hydroxide. A blue colour is usually indicative of the presence of paraquat. In this study, serum and urine levels of the toxin were not done due to logistic reasons.

In this study, the mean age of paraquat consumption was 26.46 years. There was a male predominance in the consumption of paraquat and most cases of paraquat intake was with intention of deliberate self-harm. This is in keeping with other studies on herbicide poisoning where the mean age at consumption was 25.38 years, male: female ratio for consumption is 3: 2, and 95% of all cases were due to intentional consumption of the poison (8).

Existing data from the Indian subcontinent shows AKI in 76.5%, hepatic involvement in 47%, ARDS in 53%, circulatory collapse in 30%, and MODS in 47% of all patients with paraquat poisoning (9). However, in the current study, organ involvement was more severe with 92.3% of patients developing multi-organ dysfunction. All patients showed liver and renal involvement while 61.5% had lung involvement. Up to 46.2%had circulatory collapse while CNS involvement was less often noted in only 30.7% of individuals.

The "golden hours" of paraquat poisoning refers to the immediate hours after consumption when certain therapeutic options such as hemoperfusion may be effective (10). In this study, 53.8% of cases had arrived early to the hospital within the therapeutic window. Owing to the early time of arrival in this cohort, hemoperfusion was done in 30.8% of patients. The patients who underwent hemoperfusion did not show any improvement in outcome (in terms of mortality or time to death) compared to those who did not undergo hemoperfusion.

Renal involvement is commonly seen in paraquat poisoning since the kidneys are the primary means of the excretion of paraquat. In this study, the incidence of AKI was 100%. The mean creatinine at presentation was higher than expected. RRT was required in 53.8% of the entire patient cohort. In other cohorts' up to 51.4% of patients with paraquat suffered AKI (11). AKI is attributed to multiple causes such as direct toxicity of paraquat on renal tubular cells via free radicle generation, mitochondrial injury, nuclear kappa factor b activation, or secondary to shock, volume depletion, and sepsis. Fanconi syndrome has been described with paraquat toxicity (12). In data published by Kim et al, most patients developed AKI during the hospital stay and not at admission with peak creatinine of 4.38mg/dl achieved at 5 days. Dialysis was

required in 48.3% of individuals 3.8 days after admission. The requirement for hemodialysis was a significant predictor of mortality (13). Most patients in this cohort developed AKI within 24 hours of presentation to the hospital and peak creatinine was 5.1 mg/dL. The mean time to dialysis requirement was 2.6 days and 53.8% of those with AKI in this cohort required HD. This presents a much shorter time to HD requirements than previously described. Hemodialysis was able to delay time to death significantly but was unable to show any mortality benefit.

Activated charcoal has been described as one of the best methods of decontamination for paraquat poisoning particularly if done within 2-4 hours after ingestion. In this cohort, 69.2% of patients underwent decontamination with activated charcoal. Gastric lavage is contraindicated in paraquat poisoning owing to the caustic nature of the poison. Hemoperfusion and hemodialysis particularly CRRT has been tried in the setting of paraquat toxicity with data showing effective removal of paraquat. Although initially hemoperfusion was considered to be required only as a one-time therapy for toxin removal over the first few hours of consumption, recent studies have continued hemoperfusion titrating it according to plasma levels of paraquat (14). In this study cohort, CRRT was done in only 1 patient (owing to logistic constraints) and hemoperfusion was done in 4 patients. There was no difference in time to mortality among those who received hemoperfusion vs those who did not. Plasma exchange was also attempted for fulminant hepatic failure in 2 patients with no benefit. Haemodialysis was the only treatment modality that delayed death in paraquat poisoning in this study.

Some studies have shown that the use of the "Caribbean scheme" was associated with lesser mortality in subjects who ingested 45 mL of 20% paraquat. This Caribbean scheme consisted of a combination of cyclophosphamide, dexamethasone, furosemide, and vitamins B and C (15). In this study, although the Caribbean scheme was not used, antioxidants such as vitamin C and E were used in patients and anti-inflammatory agent was used in 3 patients (23.1%) each. These however did not reduce mortality or delay time to death significantly. Mortality in this cohort was found to be 69%. The most common cause of mortality in the study cohort was MODS with circulatory failure. Only one patient died of ARDS.

The strengths of this study are the number of patients with paraquat poisoning and the range of therapies that was used as treatment. This is an uncommon disease and most cohorts are very limited in number. The limitations however are that this is a retrospective study and urine and plasma levels of the toxin were not available. Since this is a rare poisoning, the data presented is likely to help clinicians in treatment of individual cases. This is also the first study to show that HD was able to delay death due to poisoning.

Conclusion

Paraquat poisoning carries a dismal prognosis despite several new therapies for the same. Mortality rates remain high among those treated despite early hospitalisation. This cohort represents a patient population with a large quantity of paraquat consumption. The main cause of death appears to be circulatory collapse. Hemodialysis was the only treatment modality that delayed time to death in this cohort. Further studies with quantification of the serum and urine paraquat levels may be useful in guiding treatment decisions.

Limitations of the study

This is a retrospective analysis of patient outcomes and hence is subject to errors of absent and missing information and observer bias.

Authors' contribution

Conceptualisation: Indhumathi Elayaperuma, Jayakumar Matcha.
Data curation: Nisha Jose, Indhumathi Elayaperuma, Devia Arumugam, Jayakumar Matcha.
Investigation: Nisha Jose, Indhumathi Elayaperuma, Devia Arumugam.
Methodology: Nisha Jose, Indhumathi Elayaperuma, Devia Arumugam, Jayakumar Matcha.
Project administration: Indhumathi Elayaperuma.
Resources: Nisha Jose, Devia Arumugam, Jayakumar Matcha.
Supervision: Indhumathi Elayaperuma, Jayakumar Matcha.
Validation: Nisha Jose, Indhumathi Elayaperuma, Jayakumar Matcha.
Visualization: Nisha Jose, Indhumathi Elayaperuma, Devia Arumugam, Jayakumar Matcha.
Writing–original draft: Nisha Jose.
Writing–review and editing: Nisha Jose, Indhumathi Elayaperuma, Devia Arumugam, Jayakumar Matcha.

Conflict of interest

The authors declare that there is no conflict of interest.

Ethical issues

The research followed the tents of the Declaration of Helsinki. The Ethics Committee of Sri Ramachandra Institutional Ethics committee approved this study (#ID number- CSP-MED/21/JAN/65/01). Guidelines set by the institutional review board have been strictly followed). Accordingly, written informed consent taken from all participants before any intervention. Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Funding/Support

There was no external source of funding for this project.

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