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A Case Series of Paraquat Poisoning: Clinical Presentation, Management, and Outcomes

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Abstract

Background: Paraquat, a highly toxic herbicide, causes severe organ damage and has a high mortality rate. Though banned in few states in India, it remains accessible in Kerala and is increasingly used for self-poisoning.
Methods: A retrospective review of medical records from October to December 2024 of patients who presented within 48 hours of ingestion of Paraquat, diagnosis confirmed by history and urine dithionate test was identified. Data on clinical presentation, investigations, management, and outcomes were collected.
Results: Ten patients, five men and five women, aged 20–60 years, appeared within 48 hours of ingestion. All patients had impaired liver function tests (LFT), and 9 had impaired renal function tests (RFT). Six patients developed lung involvement with desaturation. Management included hemoperfusion (7 patients), immunosuppressive therapy (8 patients), and hemodialysis (5 patients). Nine patients expired, primarily due to renal and lung failure, while one survived with resolved organ dysfunction.
Conclusions: Despite intensive treatment, paraquat poisoning has a high death rate, underscoring the need for efficient preventive measures and more research into therapeutic approaches.

Keywords: Paraquat poisoning, case series, clinical presentation, management, hemoperfusion, outcomes.

INTRODUCTION

Paraquat is a commonly used herbicide and effective at controlling weeds, but is highly toxic to humans. Ingestion of paraquat can result in pulmonary fibrosis, which is a common cause of death and multi-organ failure. Although paraquat is prohibited in some states in India, it is still available in Kerala, India, and contributes to a high number of poisoning cases [1]. Paraquat poisoning develops in three stages: an early stage characterized by gastrointestinal symptoms like vomiting, and oral ulcers within 36 hours of ingestion, followed by a second phase that lasts 2–5 days and is characterized by hepatic and renal failure, and a third phase that is dominated by pulmonary fibrosis, which is frequently fatal. Hepatic damage is characterized by centrilobular necrosis and cholestasis, whereas renal failure is marked by hypovolemia and proximal nephropathy. While doses of 20–40 mg/kg cause multi-organ failure, doses above 40 mg/kg can cause acute pneumonitis, acute respiratory distress syndrome (ARDS), and metabolic acidosis within days, doses below 20 mg/kg may cause milder, reversible damage [2,3].
Ten patients with paraquat poisoning who were admitted to a tertiary care center in Kerala between October and December 2024 are described in this case series, with an emphasis on the clinical presentation, treatment approaches, and results. In an area where paraquat poisoning is common, the series seeks to raise awareness of the issue by highlighting the need for stronger laws and better treatment options. As it documents the real world results in a particular area, this case series contributes to the body of literature and emphasizes how urgent it is to address the accessibility and treatment issues with paraquat.

METHODS

A three-month retrospective review of medical records from October to December 2024 was conducted. A history of paraquat consumption, the presence of mucosal lesions indicative of paraquat poisoning or dithionate in the urine, and the availability of complete clinical data were among the inclusion criteria. Data was collected and information was

analyzed on the patient's demographics, clinical presentation, laboratory tests, imaging reports, upper gastrointestinal endoscopy reports, treatment administered, and clinical results. The Institutional authorities and the Scientific Review Board gave their approval. Throughout the entire study, patient confidentiality was upheld.

RESULTS

This case series consists of ten patients with paraquat poisoning who were admitted to a tertiary care center in Kerala, India, between October and December 2024. It included five men and five women between ages 20 to 60 years. The most frequent symptom described by them was vomiting, that appeared in all patients within 48 hours of ingestion. Eight of the ten patients gave a positive history of ingestion and for two patients whose history was initially ambiguous, a positive urine dithionate test were used to confirm the diagnosis.

All ten patients had impaired LFT, and nine patients had impaired RFT.OGD revealed different levels of esophageal damage in multiple patients, categorized using the Zargar grading system, and six patients had lung involvement with desaturation (Table 1). One patient with a Zargar IIIB esophageal injury underwent feeding jejunostomy, and another with a Zargar IIIA injury was scheduled to receive it but expired prior to the procedure.

Seven patients received hemoperfusion, eight patients received immunosuppressive treatment with cyclophosphamide (15 mg/kg/day for two days) and methylprednisolone (1 g/day for three days, followed by dexamethasone (20 mg/kg for fourteen days), and five patients with worsening renal failure received maintenance hemodialysis. Nine of the ten patients succumbed to death. Three expired secondary to deteriorating lung function and respiratory failure, four from renal failure, and two from both renal failure and respiratory failure. All deaths, with the exception of two patients who lived for 14 days, occurred within 5–7 days of consumption. One patient survived and is under follow-up, with resolution of renal and liver function abnormalities. (Table 2)

Table 1: Clinical and Laboratory Findings of Patients with Paraquat Poisoning

Patient No.	Age (Years)	Sex	Time to Presentation	History of Ingestion	Vomiting	Oral Ulcers	Urine Dithionate Test	Renal Failure	Hepatic Dysfunction	ABG Abnormalities	Chest Imaging Findings	OGD
1	24	M	<24 hrs	Yes	Yes	Yes	Positive	Yes	Yes	Metabolic acidosis	Bilateral infiltrates	-
2	60	F	<24 hrs	Yes	Yes	Yes	Positive	Yes	Yes	Hypoxia	Fibrotic changes	Zargar IIB esophagus
3	35	F	<48 hrs	Yes	Yes	Yes	Positive	No	Yes	Normal	Normal	-
4	42	M	<48 hrs	Yes	Yes	Yes	Not done	Yes	Yes	Metabolic acidosis	Diffuse alveolar damage	-
5	27	M	<24 hrs	Yes	Yes	Yes	Positive	Yes	Yes	Respiratory alkalosis	Bilateral opacities	Zargar IIIA esophagus
6	32	F	<24 hrs	No	Yes	Yes	Positive	Yes	Yes	Hypoxia	Consolidation + fibrosis	Zargar IIIA proximal stomach
7	49	M	<48 hrs	Yes	Yes	Yes	Not done	Yes	No	Metabolic acidosis	Interstitial edema	Zargar IIB esophagus

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Patient No.	Age (Years)	Sex	Time to Presentation	History of Ingestion	Vomiting	Oral Ulcers	Urine Dithionate Test	Renal Failure	Hepatic Dysfunction	ABG Abnormalities	Chest Imaging Findings	OGD
8	38	F	<48 hrs	Yes	Yes	Yes	Positive	No	Yes	Respiratory failure	Ground-glass opacities	Zargar IIIB esophagus
9	22	M	<24 hrs	Yes	Yes	Yes	Positive	Yes	Yes	Hypoxia	Early fibrosis	-
10	50	F	<24 hrs	No	Yes	Yes	Positive	No	No	Normal	Normal	Zargar IIA esophagus

Table 3- Interventions, Patient outcomes and cause of death

Patient No.	Hemoperfusion	Immunotherapy	Hemodialysis	Ventilatory Support	Feeding Jejunostomy	Outcome	Time to Death / Recovery	Primary Cause of Death
1	Yes	Yes	Yes	Yes	No	Expired	Day 5	Pulmonary fibrosis
2	Yes	Yes	Yes	Yes	Yes	Expired	Day 7	Renal failure
3	No	Yes	No	No	No	Survived	Ongoing follow-up	–
4	Yes	Yes	Yes	Yes	No	Expired	Day 6	Renal + pulmonary failure
5	Yes	Yes	Yes	Yes	No	Expired	Day 5	Pulmonary fibrosis
6	No	Yes	Yes	Yes	No	Expired	Day 7	Renal failure
7	Yes	Yes	Yes	Yes	Planned (expired)	Expired	Day 14	Pulmonary fibrosis
8	Yes	Yes	No	Yes	No	Expired	Day 14	Renal failure
9	Yes	Yes	Yes	Yes	No	Expired	Day 6	Renal + pulmonary failure
10	No	No	No	No	No	Expired	Day 7	Renal failure

DISCUSSION

This case series shows 10 patients who presented with paraquat poisoning, nine of whom died. The laboratory results, management methods, and clinical presentation are consistent with those reported in the literature. The most common symptoms of paraquat poisoning include vomiting, which is followed by hepatic and renal dysfunction, and finally pulmonary fibrosis, which is the main cause of death. Seven patients in our series developed lung involvement, nine had impaired RFT, and all patients had impaired LFT [1,2]. The lack of a specific antidote makes it difficult to treat paraquat poisoning. Supportive care, which is the cornerstone of treatment, includes hemodialysis, immunosuppressive therapy, and hemoperfusion. Eight patients were treated with methylprednisolone and cyclophosphamide in our series, while seven patients underwent hemoperfusion. The limited effectiveness of the available treatment methods is highlighted by the fact that only one patient survived despite these measures. A recent case series from South India found that 15 paraquat poisoning survivors were treated with hemodialysis, N-acetyl cysteine, corticosteroids, and symptomatic therapy. Our series did not produce comparable results, possibly due to differences in patient characteristics, such as the amount of paraquat used or the time of treatment initiation, but it suggests that early and aggressive treatment may improve outcomes.

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In line with previous reports, our series had a high mortality rate, with nine out of ten patients dying. For example, all seven of the pediatric patients who had paraquat poisoning died despite treatment, according to a case series [4]. Autopsy investigations have also revealed widespread organ damage in fatal cases, especially in the kidneys, liver, and lungs [5]. Recovery may occasionally result from milder cases or early intervention, as the only survivor in our series had resolved renal and liver function.

The small sample size and the retrospective nature of data collection may lead to bias. The exact amount of paraquat ingested was not quantified in most cases, making it difficult to correlate dose with outcome. Additionally, the study did not include detailed histopathological data, which could provide additional insights into the mechanisms of organ damage.

Despite these limitations, this series adds to the literature on paraquat poisoning and emphasizes the need for stricter regulations on the availability of this herbicide to avoid paraquat misuse [6]. Future research should focus on developing more effective treatments for paraquat poisoning, such as novel antioxidants or improved immunosuppressive regimens. Additionally, preventive strategies, including public education campaigns and enforcement of bans, are essential to reduce the incidence of paraquat poisoning.

CONCLUSION

This case series of 10 patients with paraquat poisoning demonstrates a severe clinical course and high mortality associated with this toxicity. Only one patient survived, highlighting the limitations of current treatment strategies. Further preventive measures to limit access to paraquat and further research into novel treatments to improve outcomes in patients affected need to be implemented.

Declarations-

Competing Interests: The authors declare no competing interests.

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