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Case Series

Paraquat poisoning: a grim prognosis and urgent need for policy action – a case series

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ABSTRACT

Paraquat is a highly toxic, rapidly acting, non-selective herbicide with widespread use in India despite being banned in many countries. It is associated with a high mortality rate and lacks a specific antidote. Aim and objective were to present a case series of paraquat poisoning, highlighting clinical presentation, complications, treatment modalities, and outcomes. Emphasizing the importance of referral of patients to centers with facilities where lifesaving treatment like renal replacement therapy (haemodialysis, hemoperfusion, continuous Veno-venous hemofiltration) can be done on time to reduce the high mortality rate associated with it. Retrospective chart review of 10 patients admitted with paraquat poisoning at AIIMS Bhopal between March 2022 and March 2023. All patients presented 2-22 days post-ingestion, with amounts ranging from 10-100 ml. Common features included oral and gastrointestinal ulcers, hepatitis, acute kidney injury (AKI), and acute respiratory distress syndrome (ARDS). Hemodialysis was performed in two patients presenting within 3 days. Despite supportive therapy (steroids, N-acetylcysteine, renal replacement therapy), mortality was 100%, with ARDS being the universal cause of death. Paraquat poisoning carries a dismal prognosis with no survivors in our cohort. Early referral to tertiary care centers and timely initiation of renal replacement therapy may improve outcomes. Urgent public health measures and consideration of a ban are warranted.

Keywords: Paraquat, Acute poisoning, ARDS, Renal replacement therapy, Mortality

INTRODUCTION

Paraquat, a bipyridyl compound first marketed in 1962 under the brand name Gramoxone, is a widely available herbicide in India despite being banned in 67 countries. Its low cost and unrestricted use have led to frequent poisoning cases, particularly in suicide attempts.[1] Even small ingestions (10-30 ml of 20-24% solution) can be fatal. After ingestion paraquat actively uptake into pulmonary alveolar epithelial cells via the polyamine transport system, where it generates reactive oxygen species through redox cycling, leads to extensive oxidative stress, mitochondrial dysfunction, lipid peroxidation, cellular necrosis, which gradually progress and subsequently develop pulmonary fibrosis. In addition to pulmonary injury, paraquat also causes significant damage to other organs including kidneys, liver, gastrointestinal tract, and cardiovascular system. Respiratory symptoms typically appear within 3-4 days, and a PaO_2<60 mmHg or >50% ground-glass opacities by day 7 of ingestion are associated with very high fatality.[2,3] With no specific antidote, treatment is limited to supportive care and renal

replacement therapy (RRT), which shows benefit only if initiated within hours of ingestion.[4]

Given the continued use of paraquat in India and the scarcity of awareness regarding high futility of paraquat poison. This case series describes the clinical course organ involvement and outcomes of patients with paraquat poisoning managed at a tertiary center in central India, it also highlights the devastating mortality that was associated with delayed referral and underscores the urgent need for heightened awareness, early transfer to tertiary care center with extracorporeal therapy facility, and stronger regulatory measures.

CASE SERIES

A retrospective review was conducted of all paraquat poisoning cases admitted to AIIMS Bhopal between March 2022 and March 2023. Data collected included demographic characteristics, estimated amount ingested, day of presentation, clinical features, investigations, treatment modalities, and outcomes. Autopsy findings were reviewed when available.

A total of 10 patients with confirmed paraquat poisoning were admitted to the intensive care unit of AIIMS Bhopal during the study period (March 2022 to March 2023). The cohort consisted of 5 males and 5 females with ages ranging from 18-44 years (mean age: 28 years). All patients had a history of oral ingestion of paraquat, with the estimated quantity consumed ranging from 10 ml to 100 mL of a 24% paraquat formulation. Interval between ingestion and presentation to our tertiary care center varied considerably, ranging from 2-22 days. Notably, none of patients presented within the first 24 hours following ingestion. This delay significantly limited the opportunity for early extracorporeal toxin removal and may have contributed to uniformly poor outcomes observed. The largest ingestion was 100 ml of 24% paraquat solution.

The figure depicts the distribution of major clinical manifestations observed among patients with paraquat poisoning. Oral ulceration, hepatitis, AKI, and ARDS were observed in all patients (100%). Gastrointestinal ulceration occurred in 40% of cases, while pneumothorax/pneumomediastinum developed in 30%. Hypoglycemia and hypotension were observed in one patient each (10%) indicating widespread systemic toxicity. Respiratory involvement was universal and represented the most severe complication, characterized by progressive hypoxemia, bilateral pulmonary infiltrates on chest imaging, and increasing oxygen requirements (ARDS). Three patients (30%) developed additional pulmonary complications, including pneumomediastinum and/or pneumothorax, suggestive of advanced alveolar injury and severe pulmonary toxicity of paraquat poison.

All patients received supportive intensive care management including corticosteroids and N-acetylcysteine. Renal replacement therapy in the form of hemodialysis was performed in three patients who presented relatively earlier compared to the remainder of the cohort. The longest survival was 30 days after ingestion. Despite these interventions, progressive deterioration occurred in all cases and ARDS with refractory hypoxemic respiratory failure was the final cause of death in all patients.

Table 1 summarizes the demographic characteristics, estimated amount ingested, treatment received, and clinical outcomes of all patients.

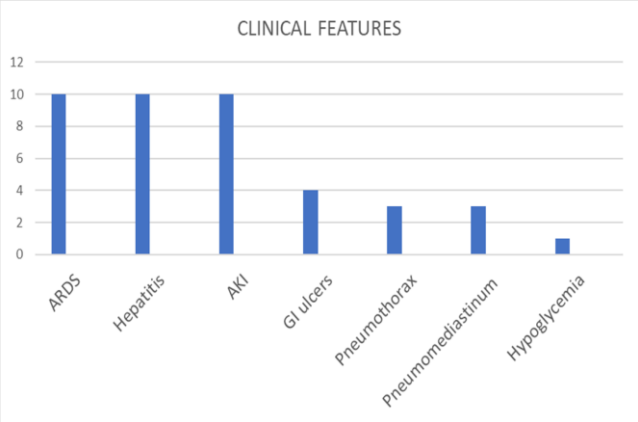

Figure 1: Frequency of clinical manifestations and organ involvement in patients with paraquat poisoning, (n=10).

Table 1: Clinical characteristics of 10 patients with paraquat poisoning.

Cases	Age (in years)/sex	Day of presentation	Amount ingested	Treatment	Outcome
1	22 M	Day 8	80-100 ml	Steroids, NAC	Died (ARDS, Day 21)
2	22 M	Day 6	Undiluted 2 mouthfuls	Steroids, NAC	Died (ARDS, Day 7)
3	42 F	Day 3	10 ml	Steroids, NAC, HD	Died (ARDS, Day 3)
4	24 F	Day 7	100 ml	Steroids, NAC	Died (ARDS, Day 8)
5	18 F	Day 8	50 ml	Steroids, NAC	Died (ARDS, Day 15)
6	21 M	Day 10	10 ml	Steroids, NAC	Died (ARDS, Day 15)
7	27 F	Day 22	20 ml	Steroids, NAC	Died (ARDS, Day 30)
8	18 F	Day 3	50 ml diluted	Steroids, NAC, HD	Died (ARDS, Day 6)
9	44 M	Day 2	30 ml undiluted	HD, Steroids, NAC	Died (ARDS, Day 25)
10	42 M	Day 10	10 ml	Steroids, NAC	Died (ARDS, Day 15)

DISCUSSION

This case series underscores the extremely high mortality associated with paraquat ingestion, consistent with previous reports from India and abroad.

The 100% mortality observed in our case series highlights the devastating nature of paraquat poisoning and it is consistent with contemporary literature reporting mortality rates ranging from 50% to over 80%, depending on the quantity ingested, severity of organ dysfunction, and timing of presentation.[5] Recent prospective studies have identified paraquat poisoning as one of the most lethal toxicological emergencies that was encountered in critical care unit.[6]

The pathophysiology involves initial mucosal injury, systemic organ dysfunction, and delayed but irreversible pulmonary fibrosis and ARDS being the universal causes of death, similar finding was noted in study done by Subhajit et al.[7]

Early renal replacement therapy may help reduce mortality if initiated within 4-6 hours. However, in our series, delayed presentation (≥2 days) nullified its potential benefits. Corticosteroids and antioxidants (e.g., NAC, vitamins C and E) were administered, but no survival benefit was observed. Previous studies from South India have documented survival in patients who reached tertiary centers early and received hemoperfusion and continuous Veno venous hemofiltration.[4]

Similarly finding was also noted in case series by Rajur et al.[8] Early initiation of renal replacement therapy i.e. within 4 hours of consumption can have a better prognosis.[8] Hemodialysis was the only mode of treatment that can delayed time to death was also noted in study done by Nisha et al.[9] Similarly Emerging evidence suggests that earlier initiation of hemodialysis or other extracorporeal therapies may improve survival and delay progression to irreversible pulmonary injury.[10] Study done by Yang et al also support the use of extracorporeal therapies in paraquat poisoning appears to be a promising modality to reduce the mortality, especially when initiated as early as after ingestion.[11]

Lack availability of specific antidote mortality rates remains high, similar finding was also noted in study done by Kotal et al however initiation of immunosuppressive therapy in early may improve survival in these patients.[12,13]

Unfortunately, most patients first seek care at primary centers, where nonspecific early symptoms (oral ulcers, mild GI upset) often lead to conservative management and delayed referral similar finding was noted in case series done by Manu Prakash et al showed that study reveals that the mortality rate can be reduced, if the child presented to the hospital within 8 hours of ingestion.[14]

It is also evident in recent study from Northen India that paraquat poisoning has exceptionally high mortality rates, particularly among patients presenting after the first 24 hours, emphasizing the importance of early referral and removal of toxin through extracorporeal strategies.[15]

Our findings also high lighten the need for increased awareness among frontline healthcare providers about the lethality of paraquat, early referral to tertiary centers, and policy-level interventions to implementing stringent regulations on its sale and distribution may be more effective than relying on treatment after exposure has occurred as also noted in study done by Guntheti et al.[16]

CONCLUSION

Paraquat poisoning remains almost universally fatal once significant ingestion occurs, particularly when presentation is delayed and it continues to represent one of the most lethal forms of pesticide intoxication encountered in critical care practice. In this case series, all patients developed multiorgan dysfunction culminating in severe ARDS and death despite receiving contemporary supportive management. Delayed presentation to specialized centers emerged as a major contributor to poor outcomes, limiting the effectiveness of extracorporeal toxin removal strategies.

Our findings highlight the need for increased awareness among frontline healthcare providers about the lethality of paraquat, early referral to tertiary centers with renal replacement therapy available, and policy-level interventions including consideration of a nationwide ban so mortality can be reduced and promoting other non-toxic or less-toxic alternatives.

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