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Original Research Paper

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PARAQUAT POISONING WITH RAPIDLY EVOLVING MULTIORGAN TOXICITY: A FOUR-PATIENT CASE SERIES FROM WESTERN INDIA

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ABSTRACT Paraquat poisoning can produce rapidly progressive oxidative injury involving the kidneys, liver, and lungs. We describe four patients with acute paraquat ingestion managed at a tertiary-care teaching hospital in Western India. Clinical trajectories differed at presentation but converged toward severe multiorgan toxicity. A 21-year-old woman developed marked acute kidney injury, hepatotoxicity, and respiratory deterioration within approximately 48 hours. A 20-year-old woman in early pregnancy developed oliguric acute kidney injury and rapidly progressive hypoxemic respiratory failure requiring hemodialysis, non-invasive ventilation, intubation, and mechanical ventilation, and died approximately 25 hours after tertiary admission. A 22-year-old man initially maintained room-air oxygen saturation but subsequently developed severe renal and hepatic injury and progressive pulmonary involvement; renal indices improved after renal replacement therapy while hepatotoxicity progressed. A 19-year-old man was initially conscious, hemodynamically stable, and maintaining an oxygen saturation of 98% on room air, but developed rapidly worsening renal, hepatic, and respiratory dysfunction and died the following day. All four patients had fatal outcomes. These cases emphasize the limited prognostic value of a single early assessment and support close serial multiorgan monitoring and timely organ support in severe paraquat poisoning.

KEYWORDS : Paraquat; Herbicide Poisoning; Acute Kidney Injury; Hepatotoxicity; Respiratory Failure; Case Series

1 | INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-bipyridinium) is a highly toxic herbicide in which systemic absorption can trigger redox cycling, reactive oxygen species generation, lipid peroxidation, mitochondrial dysfunction, inflammation, and cell death. The kidney, liver, and lung are prominent target organs. Pulmonary accumulation may lead to pneumonitis and later fibrosis, while acute kidney injury can impair paraquat elimination and potentially increase systemic exposure [1,2].

No specific antidote has established definitive efficacy. Current management is therefore based on early assessment, gastrointestinal decontamination when appropriate and sufficiently early, toxicological testing when available, cautious oxygen administration, and organ support. Extracorporeal and immunomodulatory strategies have been used in selected patients, but the quality and consistency of evidence remain limited [3].

Recent observational data associate adverse outcomes with renal and hepatic dysfunction, impaired oxygenation, and pulmonary infiltrates [4]. We present four fatal cases to illustrate how paraquat toxicity may evolve along different temporal pathways, including initially preserved physiology, fulminant respiratory failure in early pregnancy, and discordant organ responses during supportive treatment.

2 | Case Series

2.1 | Case I

2.1.1 | Case History and Examination

A 21-year-old woman presented on 28 July 2026 after an alleged ingestion of approximately 100 mL of paraquat at an agricultural field at around 2:00 PM. She developed multiple episodes of vomiting and reached the tertiary-care emergency department at approximately 4:00 PM. She had no known comorbidities. On admission, she was conscious and oriented (GCS 15/15), with a pulse rate of 92 beats/min, blood pressure of 122/80 mmHg, and SpO2 of 98% on room air.

2.1.2 | Investigations and Treatment

Admission investigations showed serum creatinine 1.08 mg/dL, total bilirubin 0.80 mg/dL, direct bilirubin 0.21 mg/dL, sodium 143 mEq/L, potassium 3.04 mEq/L, and leukocyte count 23,200/μL with 87% neutrophils. Over approximately 48 hours, serum urea increased to 128.40 mg/dL, creatinine to 5.87 mg/dL, total/direct bilirubin to 9.95/7.61 mg/dL, and ALT to 688 IU/L. Leukocyte count increased to 28,900/μL with 90% neutrophils. Management included close monitoring, intravenous fluids, strict intake-output monitoring, N-acetylcysteine, sodium bicarbonate, gastroprotective and antiemetic therapy, antimicrobials, methylprednisolone, and cyclophosphamide with mesna. Progressive respiratory deterioration required escalation to BiPAP.

2.1.3 | Outcome and Follow-Up

Hepatorenal toxicity and respiratory failure progressed despite intensive supportive management. The patient died on 30 July 2026 at approximately 8:00 PM.

2.2 | Case II

2.2.1 | Case History and Examination

A 20-year-old woman, G2P1L1A0, with approximately 2 months of amenorrhea and a positive urine pregnancy test, presented after an alleged ingestion of approximately 10 mL of paraquat at an agricultural field in Morbi, Gujarat. She developed four episodes of vomiting, abdominal pain, and progressive breathlessness. After initial care at a primary healthcare facility, she was referred to the tertiary-care centre. At presentation, she was conscious but acutely ill, with sinus tachycardia (120-140 beats/min), blood pressure 140/90 mmHg, respiratory rate 24-28 breaths/min, and SpO2 approximately 95% on room air. Respiratory distress and hypoxemia subsequently worsened.

2.2.2 | Investigations and Treatment

Initial hemoglobin was 11.3 g/dL, leukocyte count 12,400/μL with 87% neutrophils, and platelet count 200,000/μL. Within approximately one day, urea was 109.10 mg/dL and creatinine 5.30 mg/dL. Total/direct bilirubin was 3.69/2.96 mg/dL, ALT 152 IU/L, sodium 143 mEq/L, potassium 3.12 mEq/L, total protein 4.78 g/dL, and albumin 2.54 g/dL. ECG showed sinus tachycardia. Management included intensive monitoring, intravenous fluids, strict intake-output monitoring, methylprednisolone, N-acetylcysteine, sodium bicarbonate, pantoprazole, ondansetron, and broad-

spectrum antimicrobials. Emergency hemodialysis was initiated for oliguric severe acute kidney injury. Respiratory support escalated to BiPAP; approximately 5 hours after admission, SpO2 fell to 72% despite BiPAP. Gasping respiration and impending arrest approximately 30 minutes later prompted endotracheal intubation and invasive mechanical ventilation.

2.2.3 | Outcome and Follow-Up

Progressive multiorgan failure and refractory hypoxemia developed despite intensive organ support. Cardiac arrest occurred, and death was documented approximately 25 hours after tertiary-care admission.

2.3 | Case III

2.3.1 | Case History and Examination

A 22-year-old man presented on 13 January 2025 after acute paraquat ingestion reported at approximately 11:30 PM. He was conscious and oriented on arrival. Initial pulse was approximately 80 beats/min and blood pressure approximately 100/70 mmHg. During subsequent observations, pulse ranged from approximately 72 to 98 beats/min, blood pressure from approximately 120-142/80-96 mmHg, and room-air SpO2 remained approximately 96%. Bilateral air entry was documented

2.3.2 | Investigations and Treatment

Initial investigations demonstrated severe acute kidney injury and hepatotoxicity: blood urea 184.04 mg/dL, creatinine 9.94 mg/dL, total/direct bilirubin 5.20/4.40 mg/dL, and SGPT 237 IU/L. Following renal replacement therapy, urea and creatinine improved to 141.24 mg/dL and 3.32 mg/dL, respectively, whereas total/direct bilirubin increased to 15.10/12.00 mg/dL, indicating progressive hepatic injury. With worsening hypoxemia, HRCT thorax showed bilateral consolidation and ground-glass opacities interpreted as aspiration pneumonitis with subpleural fibrotic changes. Management included renal replacement therapy, intravenous fluids, N-acetylcysteine, sodium bicarbonate, corticosteroids, antimicrobials, and escalating respiratory support.

2.3.3 | Outcome and Follow-Up

Despite renal replacement therapy and intensive supportive care, progressive multiorgan dysfunction and respiratory failure developed. The patient died on 23 January 2025 at approximately 2:40 PM.

2.4 | Case IV

2.4.1 | Case History and Examination

A 19-year-old man was admitted to the tertiary-care centre on 3 February 2025 at approximately 2:18 PM, approximately one day after paraquat ingestion. He had received initial supportive care at a private hospital before referral. At tertiary admission, he was conscious, oriented, and hemodynamically stable (GCS 15/15), with pulse 78 beats/min, blood pressure 120/78 mmHg, normal body temperature, and SpO2 98% on room air. No major respiratory abnormality was evident initially.

2.4.2 | Investigations and Treatment

Investigations available from 2 February 2025 showed serum creatinine 2.70 mg/dL, total bilirubin 1.0 mg/dL (direct 0.8 mg/dL; indirect 0.2 mg/dL), and SGPT approximately 16 IU/L. The clinical course subsequently deteriorated rapidly. By 4 February, serum urea was 115.56 mg/dL, creatinine 6.31 mg/dL, total bilirubin 6.70 mg/dL (direct 5.50 mg/dL), SGPT 122 IU/L, and potassium 2.70 mEq/L, with progressive renal, hepatic, and respiratory involvement. Intensive supportive management and organ-directed care were provided according to clinical requirements.

2.4.3 | Outcome and Follow-Up

The patient developed progressive multiorgan failure despite intensive supportive management and died on 4 February 2025.

Table 1 | Comparative Clinical Characteristics of the Four Cases

Variable	Case I	Case II	Case III	Case IV
Age/sex	21/F	20/F	22/M	19/M
Clinical context	Large reported ingestion	Early pregnancy	Initially preserved oxygenation	Initially stable physiology
Approx. time to tertiary care	~2 h	After initial primary care	Hours	~1 day
Early presentation	Vomiting	Vomiting, abdominal pain, dyspnea	SpO2 96%-99% on room air	GCS 15/15; SpO2 98% on room air
Renal course	Creatinine 1.08 -> 5.87 mg/dL	Creatinine 5.30 mg/dL; oliguric AKI; HD	Creatinine 9.94 -> 3.32 mg/dL after RRT	Creatinine 2.70 -> 6.31 mg/dL
Hepatic course	Bilirubin 0.80 -> 9.95 mg/dL; ALT 688 IU/L	Bilirubin 3.69 mg/dL; ALT 152 IU/L	Bilirubin 5.20 -> 15.10 mg/dL; SGPT 237 IU/L documented initially	Bilirubin 1.0 -> 6.70 mg/dL; SGPT 16 -> 122 IU/L
Respiratory course	Progressive deterioration; BiPAP	BiPAP -> intubation/MV	Later hypoxemia; bilateral consolidation/GGO on HRCT	Initially normal oxygenation -> respiratory deterioration
Outcome	Death, 30 Jul 2026	Death ~25 h after tertiary admission	Death, 23 Jan 2025	Death, 4 Feb 2025

3 | DISCUSSION

This series illustrates a clinically important feature of paraquat poisoning: the initial bedside picture may substantially underestimate subsequent toxicity. Although the four patients differed in reported exposure, timing of referral, and early physiological findings, each ultimately developed severe multiorgan dysfunction and died. The shared pattern was not identical severity at presentation, but rapid evolution across renal, hepatic, and respiratory systems.

Case I showed a steep biochemical trajectory, with creatinine rising from 1.08 to 5.87 mg/dL and bilirubin from 0.80 to 9.95 mg/dL over approximately 48 hours, followed by respiratory deterioration. Case II represented the most fulminant early respiratory course: severe acute kidney injury and hepatic dysfunction were accompanied by rapidly worsening hypoxemia, escalation from BiPAP to invasive ventilation, and death approximately 25 hours after tertiary admission. The coexistence of early pregnancy is clinically notable, but this small series cannot determine whether pregnancy modified toxicity or outcome.

Case III demonstrates the potential for discordant organ trajectories. Renal indices improved after renal replacement therapy, yet bilirubin continued to rise and pulmonary disease progressed. This observation is important because biochemical improvement in kidney function after extracorporeal support should not be interpreted as evidence that systemic paraquat toxicity has resolved. Case IV provides a complementary warning: normal consciousness, stable

hemodynamics, and SpO2 of 98% on room air at tertiary admission were followed by marked renal and hepatic deterioration and evolving respiratory involvement within a short interval.

These trajectories are biologically plausible in the context of paraquat redox cycling and oxidative tissue injury. Pulmonary vulnerability reflects active paraquat accumulation in alveolar tissue, whereas acute kidney injury may both represent direct toxicity and reduce paraquat elimination [1,2]. In a prospective observational cohort of 103 patients, nonsurvivors had greater renal and hepatic dysfunction, poorer oxygenation, and more frequent pulmonary infiltrates; hepatic dysfunction and pulmonary infiltrates were independently associated with mortality [4]. An Indian case series of survivors also documented substantial variability in exposure history and renal, hepatic, and pulmonary involvement, underscoring the heterogeneity of clinical evolution [5].

Management remains challenging because no intervention has established a universally effective antidotal role. Contemporary guidance emphasizes early decontamination when appropriate, toxicological assessment where available, careful oxygen strategy, and prompt organ support. Hemoperfusion or renal replacement therapy and immunomodulatory regimens such as corticosteroids with cyclophosphamide have been used in selected patients, but observational case-series data cannot establish treatment efficacy [3]. Accordingly, the therapies described here should be interpreted as components of clinical management rather than evidence of benefit.

The principal strength of this series is the case-by-case chronology showing different routes to fatal multiorgan toxicity, including initially preserved oxygenation and discordant renal and hepatic responses. Important limitations are the small retrospective sample, reliance on routine clinical records, incomplete documentation of exact paraquat formulation and ingested dose in some cases, and absence of plasma paraquat concentration or urine dithionite testing in the records available for this manuscript. Treatment details were not uniformly documented across all four patients. These limitations restrict causal or comparative conclusions and preclude assessment of treatment effectiveness.

4 | CONCLUSION

Severe paraquat poisoning can evolve rapidly from apparently stable early physiology to renal, hepatic, and respiratory failure. Across these four fatal cases, serial trends were more informative than a single early oxygen saturation or laboratory panel. Close multiorgan surveillance, timely supportive care, and cautious interpretation of transient improvement in individual organ parameters are essential, while the effectiveness of paraquat-directed adjunctive therapies remains uncertain.

Key Clinical Message

Paraquat poisoning may progress to fatal multiorgan failure despite initially preserved hemodynamics and oxygenation. Serial renal, hepatic, and respiratory assessment is essential, because deterioration can be abrupt and improvement in one organ system after supportive therapy does not necessarily indicate overall recovery

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Ethics Statement

This retrospective case series was conducted using data obtained from existing medical records. The requirement for formal ethical approval was waived by the Institutional Ethics Committee of P.D.U. Government Medical College and Hospital, Rajkot, Gujarat, India. All patient information was de-identified to maintain confidentiality.

Consent

Written informed consent for publication of the clinical details was obtained from the legally authorized representatives of all patients. All patient information has been de-identified to protect confidentiality.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

De-identified data supporting this case series are derived from institutional clinical records and may be available from the corresponding author subject to institutional, ethical, and privacy restrictions.

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