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Case Series: Forensic Analysis of Paraquat Poisoning Fatalities

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Abstract

Paraquat, a highly toxic bipyridinium herbicide is also a leading cause of fatal poisoning in agricultural communities with more mortality rates exceeding around60%. This case series examines three fatal cases of paraquat poisoning in a tertiary care hospital in South India focusing on mainly the clinical presentations, biochemical profiles, toxicological confirmation, and also the histopathological findings. All cases presented with acute renal failure, hepatic dysfunction, and progressive pulmonary fibrosis, confirmed through autopsy and HPE. Biochemical markers included elevated serum creatinine (2.8–4.5 mg/dL), liver enzymes (AST 120–150 U/L, ALT 95–110 U/L), and metabolic acidosis (pH 7.1–7.2). Histopathology revealed characteristic alveolar fibrosis, hepatic centrilobular necrosis, and renal proximal tubular degeneration. Forensic analysis highlighted the most importance of toxicological screening and scene investigation to differentiate intentional from accidental ingestion. In spite of aggressive interventions like hemodialysis all the cases resulted in death within 3–5 days due to multi-organ failure. This study underscores some diagnostic challenges of paraquat poisoning and the absence of effective antidotes, and also the need for regulatory restrictions on paraquat access. Forensic medicine doctors play a critical role in documenting these cases to inform public health strategies. Enhanced toxicological facilities and rapid diagnostic tools are very much essential in improving the outcomes in resourcelimited settings.

1. Introduction

Paraquat (1,1'-dimethyl-4,4'-bipyridinium dichloride) is a non-selective herbicide widely used in india for agricultural purposes. Its high toxicityalong with lack of specific antidotes, and easy accessibility donate to its notoriety as a deadly poison, with ingestion leading to mortality nearly to 60% of cases. Paraquat poisoning occurs primarily through ingestion and causing very rapid oxidative stress, cellular damage, and also multi-organ failure. The lungs are very often disproportionately affected due to paraquat's selective uptake by alveolar cells the main cause which is leading to pulmonary fibrosis. In India, paraquat is a significant public health concern, mainly in the rural areas where it is used extensively and often stored in unsafe way.

Clinically, paraquat poisoning progresses in phases: an initial gastrointestinal phase where the symptoms mainly are vomiting, mucosal dmage etc followed by these there will be renal and hepatic failure, and finally a delayed pulmonary phase characterized by fibrosis. Forensic

medicine plays a essential role in identifying these deaths, as intentional ingestions (suicides) are very common, though in some accidental exposures occur. Toxicological confirmation using GC-MS is essential, also the histopathological examination to document organ damage. This case series describes three fatal cases of paraquat poisoning, highlighting clinical, biochemical, and forensic and histopathological findings. The objectives here are to characterize its typical presentations, validate diagnostic tools, and discuss implications in forensic medicine and public health policy in India.

2. Materials and Methods

This retrospective case series analyzed three fatal cases of paraquat poisoning at a tertiary care hospital between 2023 and 2024. Inclusion criteria were confirmed paraquat ingestion via history and subsequently death as the outcome. Exclusion criteria included survival beyond hospitalization. Data were collected on clinical symptoms, laboratory parameters, toxicological results, and autopsy findings.

Clinical data included vital signs, oxygen saturation, and symptoms like vomiting or dyspnea. Laboratory tests comprised serum creatinine, liver function tests (AST, ALT, bilirubin), arterial blood gases (pH, pO2), and complete blood counts. Toxicological screening used GC-MS to detect paraquat in gastric aspirate, blood, or urine. Autopsies were performed within 24 hours of death, with tissue samples (lungs, liver, kidneys, esophagus) fixed in 10% formalin for histopathological analysis. Hematoxylin and eosin staining was used to examine cellular changes.

Data were anonymized to protect patient confidentiality, and ethical approval was not required for retrospective analysis. Descriptive statistics summarized biochemical and histopathological findings. The study adhered to forensic research guidelines, ensuring reproducibility and relevance to the Journal of IAFM.

3. Results

- Case 1: A 35-year-old male presented 6 hours after ingesting 50 mL of 20% w/v paraquat solution. He reported vomiting, abdominal pain, and oral ulcerations. On admission, blood pressure was 110/70 mmHg, pulse 92 bpm, and oxygen saturation 88%. Laboratory findings showed serum creatinine 3.2 mg/dL, AST 120 U/L, ALT 95 U/L, total bilirubin 1.8 mg/dL, and arterial pO2 60 mmHg with pH 7.2. GC-MS confirmed paraquat in gastric aspirate (3.8 μg/mL). Treatment included gastric lavage, activated charcoal, and supplemental oxygen. By day 2, he developed acute respiratory distress syndrome (ARDS). Hemodialysis was initiated but ineffective. Death occurred on day 3. Autopsy revealed corroded esophageal mucosa, hemorrhagic pulmonary edema, and enlarged kidneys. Histopathology showed alveolar fibrosis, hyaline membrane formation, and proximal tubular necrosis. Kidney tissue examinations [Figure 1 and Figure 2] highlighted significant pathological changes, including necrotic tubular cells, inflammatory infiltrates, and preserved glomeruli, consistent with paraquat-induced renal damage.

- Case 2: A 28-year-old female presented 8 hours after intentional ingestion of 30 mL paraquat. Symptoms included nausea, epigastric pain, and jaundice. Initial vitals were stable (BP 120/80 mmHg, pulse 88 bpm), but oxygen saturation dropped to 85% by day 2. Laboratory results showed creatinine 4.5 mg/dL, AST 130 U/L, ALT 100 U/L, total bilirubin

5.8 mg/dL, and pH 7.1. GC-MS detected paraquat in blood (2.5 µg/mL). She received hemodialysis and N-acetylcysteine, but renal and hepatic failure progressed. Death occurred on day 5 from multi-organ failure. Autopsy showed jaundiced tissues, centrilobular hepatic necrosis, and fibrotic lungs. Histopathology confirmed diffuse alveolar damage, interstitial pneumonitis, and extensive tubular degeneration in kidneys.

- Case 3: A 42-year-old male was admitted 4 hours post-ingestion of 40 mL paraquat, presenting with vomiting, dyspnea, and metabolic acidosis (pH 7.2, pO2 55 mmHg). Laboratory findings included creatinine 2.8 mg/dL, AST 150 U/L, ALT 110 U/L, and total bilirubin 2.5 mg/dL. Paraquat was confirmed in urine (4.2 µg/mL) via GC-MS. Despite mechanical ventilation and supportive care, he developed pulmonary fibrosis and died on day 4. Autopsy revealed esophageal erosions, fibrotic lungs, and swollen kidneys. Histopathology showed interstitial pneumonitis, alveolar hemorrhage, and tubular necrosis, with mild hepatic steatosis.

4. **Discussion**

Paraquat poisoning follows a well-documented toxicological pathway. Initial gastrointestinal damage results from direct mucosal corrosion, as seen in all three cases with esophageal erosions and vomiting. Systemic absorption leads to oxidative stress, mediated by reactive oxygen species, causing renal and hepatic injury within 24–48 hours. Elevated creatinine (2.8–4.5 mg/dL) and liver enzymes (AST 120–150 U/L, ALT 95–110 U/L) in these cases align with prior studies, reflecting acute tubular necrosis and centrilobular necrosis. The lungs, however, bear the brunt of paraquat's toxicity due to its accumulation in type II pneumocytes, triggering fibroblast proliferation and irreversible fibrosis. Histopathological findings of alveolar fibrosis and diffuse alveolar damage are pathognomonic, observed consistently across all cases.

Toxicological confirmation via GC-MS is critical in forensic settings, as clinical history alone is unreliable, especially in suspected suicides. Paraquat concentrations (2.5–4.2 µg/mL) in blood and urine correlated with fatal outcomes, consistent with literature thresholds (>0.2 µg/mL). However, rapid diagnostic tests are lacking, delaying intervention in resource-limited hospitals. Treatment remains supportive, with hemodialysis and antioxidants like N-acetylcysteine showing limited efficacy, as evidenced by the rapid progression to death in all cases.

Forensic implications are significant. Intentional ingestion, likely in Cases 2 and 3, requires scene investigation to rule out foul play or accidental exposure. Autopsy findings, such as corroded mucosa and fibrotic lungs, are distinctive but non-specific without toxicology. Histopathology bridges this gap, providing evidence of paraquat-specific damage. In Case 1, kidney histopathology [Figures 1 and 2] demonstrated proximal tubular necrosis, inflammation, and interstitial edema, reinforcing the multi-organ impact. These cases highlight the need for standardized forensic protocols, including mandatory GC-MS screening in suspected herbicide poisonings.

Public health challenges include paraquat's unregulated availability in India, despite bans in over 50 countries. Rural farmers, as likely represented in these cases, face occupational risks due to inadequate storage and labeling. Educational campaigns and stricter regulations could reduce incidence. Limitations of this study include its small sample size and retrospective

design, which preclude survival analysis or treatment efficacy evaluation. Future research should explore early biomarkers and novel therapies to improve outcomes.

5. Conclusions

This case series underscores the lethal nature of paraquat poisoning, characterized by rapid multi-organ failure and characteristic histopathological changes. Biochemical markers (elevated creatinine, liver enzymes) and GC-MS confirmation are essential for diagnosis, while autopsy and histopathology provide forensic clarity. The absence of effective treatments and high mortality necessitate urgent public health interventions, including restricted paraquat access and farmer education. Forensic pathologists are pivotal in documenting these cases, aiding legal and preventive efforts. Enhanced toxicological infrastructure and rapid diagnostics are critical to address this crisis in India.

References

1. Gawarammana Indika B, Buckley Nicholas A. Medical management of paraquat ingestion. British Journal of Clinical Pharmacology. 2011;72(5):745-757.

2. Dinis-Oliveira Ricardo Jorge, Duarte José Alberto, Sánchez-Navarro Angel, Remião Fernando, Bastos Maria de Lourdes, Carvalho Félix. Paraquat poisonings: mechanisms of lung toxicity, clinical features, and treatment. Critical Reviews in Toxicology. 2008;38(1):13-71.

3. Senarathna Lalith, Eddleston Michael, Wilks Martin F, Woollen Brian H, Tomenson John A, Roberts Darren M, Buckley Nicholas A. Prediction of outcome after paraquat poisoning by measurement of the plasma paraquat concentration. QJM: An International Journal of Medicine. 2009;102(4):251-259.

4. Houzé Philippe, Baud Frédéric J, Mouy Rémy, Bismuth Chantal, Bourdon René, Scherrmann Jean-Michel. Toxicokinetics of paraquat in humans. Human & Experimental Toxicology. 1990;9(1):5-12.

5. Agarwal Ritesh, Srinivas Rajesh, Aggarwal Ashutosh N, Saxena Ritu. Experience with paraquat poisoning in a respiratory intensive care unit in North India. Singapore Medical Journal. 2006;47(12):1033-1037.

6. Lee Eun Young, Hwang Kyung Yeon, Yang Jae-Oh, Hong Sung-Yong. Predictors of survival after acute paraquat poisoning. Toxicology and Industrial Health. 2002;18(4):201-206.

7. Sabzghabaee Ali Mohammad, Eizadi-Mood Nastaran, Montazeri Kamran, Yaraghi Ali, Golabi Masoumeh. Fatality in paraquat poisoning. Singapore Medical Journal. 2010;51(6):496-500.

8. Wilks Martin F, Fernando Rezvi, Ariyananda Pradeepa L, Eddleston Michael, Berry David J, Tomenson John A, Buckley Nicholas A, Jayamanne Shaluka, Gunnell David, Dawson Andrew. Improvement in survival after paraquat ingestion following introduction of a new formulation in Sri Lanka. PLoS Medicine. 2008;5(2):e49.

9. Tan John T, Letchuman Ramanathan G, Choy Min P, Leela R, Balasubramaniam P. Paraquat poisoning: experience in a Malaysian hospital. Journal of Environmental Health Research. 2013;13(1):65-71.

10. Kavousi-Gharbi Saeed, Jalli Reza, Rasekhi Alireza, Marjani Majid, Tabatabaee Hamid Reza. Acute paraquat poisoning: a case report and review of the literature. Journal of Acute Disease. 2016;5(2):171-174.

11. Eddleston Michael, Wilks Martin F, Buckley Nicholas A. Prospects for treatment of paraquat-induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome. QJM: An International Journal of Medicine. 2003;96(11):809-824.

12. Kim Young Tae, Joung Hyung Keun, Kim Young Hoon, Hong Yeong S, Shin Young Kee, Park Jong Seok, Yang Jae Min. Pathologic findings in acute paraquat poisoning. Korean Journal of Pathology. 2004;38(2):75-82.

13. Raghu K, Mahesh V, Sasidhar P, Reddy P R, Venkataramaniah V, Agrawal Amit. Paraquat poisoning: a case report and review of literature. Journal of Family Medicine and Primary Care. 2013;2(4):360-362.

14. Bismuth Chantal, Garnier Robert, Baud Frédéric J, Muszynski Jacques, Keyes Casey. Paraquat poisoning: an overview of the current status. Drug Safety. 1990;5(4):243-251.

15. Suntres Zacharias E. Role of antioxidants in paraquat toxicity. Toxicology. 2002;180(1):65-77.

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