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Case Report

Catastrophic Outcomes: Rapid Multi-Organ Failure from Paraquat Poisoning- A Case Report

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Abstract

Paraquat dichloride, a potent herbicide widely used in agriculture, poses a severe health risk due to its high toxicity. Ingesting even small amounts can cause fatal multi-organ failure. We report a case of a 19-year-old male who ingested approximately 5 ml of 24% paraquat dichloride. He presented with stable vital signs, but initial laboratory results showed elevated urea (26mg/dL), creatinine (0.97 mg/dL), and hypokalemia (3.2 mEq/L). Despite interventions including gastric lavage, N-Acetyl cysteine, methylprednisolone, and supportive care, he developed severe metabolic acidosis (HCO^{3-} 22.8 to 16.3 mEq/L), acute renal failure (creatinine 0.97 to 4.62 mg/dL, urea 26 to 99 mg/dL, serum potassium 3.2 to 2.62 mEq/L), and multi-organ dysfunction. The unavailability of hemoperfusion has impacted the outcome. The patient's rapid deterioration highlights paraquat's aggressive nature and underscores the necessity for better therapeutic strategies and regulatory measures to prevent such poisoning.

Keywords: Paraquat Poisoning; Multi-Organ Failure; Case Report; Oxidative Stress; Hemoperfusion; Renal Injury.

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Introduction

Paraquat dichloride, a potent herbicide extensively used in agriculture, particularly in developing countries like India, poses a significant health hazard due to its high toxicity [1]. Known for its rapid action and effectiveness in controlling weeds, paraquat's lethal nature is compounded by its accessibility and low cost [1]. However, its toxicity is formidable, with a mortality rate that can exceed 70% among those exposed, making it one of the most dangerous poisons commonly encountered in agricultural settings[1].According to the literature, in India, paraquat exhibits a median case-fatality ratio of 54.2%, markedly higher than other widely used pesticides such as organophosphates (11.1%), endosulfan (22.9%), and glyphosate (6.1%)[2]. In contrast, substances like pyrethroids (0.7%) and imidacloprid (0.0%) demonstrate significantly lower toxicity, highlighting the exceptional human health risk posed by paraquat [2].

The severity of paraquat poisoning is dose-dependent; minor exposures can cause severe and irreversible health effects, while ingestions above certain thresholds almost invariably result in death, primarily due to multi-organ failure [3,4]. Clinical manifestations of paraquat poisoning include gastrointestinal damage, acute kidney injury, liver failure, and particularly catastrophic effects on the lungs, such as progressive pulmonary fibrosis [3,4].

Statistically, paraquat accounts for a high number of poisoning cases in agricultural regions, with a substantial portion resulting from either accidental or intentional ingestion [5]. Despite its known risks, the lack of effective regulatory policies in some areas allows its continued widespread use [5].

Current therapeutic strategies for paraquat poisoning remain largely supportive due to the absence of a specific and effective antidote [6,7]. Management primarily involves symptomatic treatment and attempts to limit systemic damage. Pharmacological interventions such as antioxidants to mitigate oxidative stress and corticosteroids to control the inflammatory response have been employed, although their clinical efficacy remains variable and often inconclusive [7,8].

Among advanced therapeutic options, hemoperfusion has shown potential benefits, particularly when initiated within a narrow time window post-ingestion—commonly referred to as the “golden hours”—but its application is often limited by resource availability and inconsistent outcomes across clinical settings [6]. Furthermore, paraquat-induced multi-organ dysfunction complicates treatment efforts and underscores the need for early and aggressive intervention [8,9].

This case highlights paraquat’s deceptive clinical course—initial stability followed by sudden multi-organ failure—despite early intervention. It underscores the urgent need for accessible prognostic tools and therapeutic strategies in resource-limited settings.

Case Detail

A 19-year-old male patient with no significant family, personal, or psychiatric history was admitted to our hospital after consuming approximately 5 ml of 24% paraquat dichloride six hours prior to being discovered by a relative. He was initially taken to a local hospital where he received gastric lavage and intravenous fluids before he was referred to our facility for further management. Before admission, the patient reported experiencing 2 to 3 episodes of vomiting at home. Examination revealed that he was conscious, cooperative, and well-oriented, with stable vital signs: afebrile, heart rate of 84 bpm, respiratory rate of 20 breaths per minute, blood pressure of 124/80 mm Hg, and SpO2 of 98% on room air. The systemic examination was otherwise normal. He was admitted to the Critical Care Unit (CCU).

Initial blood tests showed raised urea at 26 mg/dL (reference range: 6-12 mg/dL), creatinine at 0.67 mg/dL (reference range: 0.4-1.4 mg/dL), sodium at 140 mEq/L (reference range: 135-145 mEq/L), low potassium at 3.2 mEq/L (reference range: 3.5-5.1 mEq/L), hemoglobin at 14.7 g/dL (reference range: 13-

17 g/dL), total leukocyte count (TLC) at 9970/mm³ (reference range: 4000-11000/mm³), and platelet count at 2.29 lacs/mm³ (reference range: 1.5-4 lacs/mm³). Arterial blood gas analysis (ABGA) indicated a pH of 7.4, pCO2 of 32.5, and HCO3 of 22.8. He was treated with intravenous N-Acetyl cysteine 8 gm in 200 ml 5% dextrose over 1 hour, followed by 3 gm in 500 ml 5% dextrose over 4 hours, followed by 6 gm in 1000 ml 5% dextrose over 16 hours, Injection Vitamin E 500 mg IV, Intravenous Methylprednisolone 500 mg in 250 ml Normal saline over 30 minutes for 3 days as per regimens suggested in severe paraquat-induced lung injury protocols, and symptomatic management in the CCU.

Throughout his hospitalization, he received prophylactic treatments including lavage with normal saline, antioxidants such as Vitamin C and E, N-acetyl cysteine, immunosuppressants like methylprednisolone and syrup Digene gel, syrup Mucaine gel, Xylocaine viscous gargles for ulcer prevention. These antioxidants and corticosteroid regimens were selected based on their ability to scavenge reactive oxygen species (ROS) and reduce inflammatory-mediated pulmonary fibrosis, a hallmark of paraquat toxicity. Early hemoperfusion was not possible because of the unavailability of the particular adsorbent [4].

Over the following days, his creatinine and urea levels increased, indicating intrinsic renal injury. Creatinine levels rose from 0.97 to 2.45, 3.03, 3.29, 4.04, 4.41, 3.15 and 4.62mg/dL, and urea levels increased from 267 – 597 – – 100 – 109 – 118 – 74 – 99mg/dL. Potassium levels fluctuated 3.2 – 3.1 – 2.4 – 2.8 – 2.6 – 2.62 – 4.8mEq/L. The patient was administered injectable potassium chloride to manage these fluctuations, and his random blood sugar (RBS) levels were also unstable throughout his hospital stay.

On the fifth day, the patient had three episodes of hematemesis and a sudden episode of brief jerky body movements followed by unconsciousness. He had experienced a fever the previous night and was treated within Vitamin K, Inj. Tranexamic acid, Inj. Diazepam, and Inj. Phenytoin. Blood tests on this day indicated a decreased platelet count (1,36,000/mm^3), low serum potassium, raised PT/INR, and elevated urea (109 mg/dL), creatinine (4.04 mg/dL), and bilirubin levels (Total Bilirubin 1.61 mg/dL). ABGA showed metabolic acidosis, and a USG thorax/abdomen revealed mild bilateral pleural effusion and minimal intra-abdominal and perinephric free fluid.

By the sixth day, the patient was unable to maintain oxygen saturation on room air and was put on BPAP with 60% O2 to prevent oxygen-related free radical injury. He underwent haemodialysis for acute kidney injury. Even after that on the same night, he became agitated and disoriented, necessitating invasive mechanical ventilation for 24 hours.

On the seventh day, the patient went into cardiogenic shock and required inotropic support. Despite all efforts, his condition continued to deteriorate, and he succumbed to death on the seventh day of admission.

Table 1: Profile of patients daily Investigations

Parameters	08-Jun	09-Jun	10-Jun	11-Jun	12-Jun	13-Jun	14-Jun morning	14-Jun	15-June
pH (7.35-7.45)	7.4	7.474↑	7.381	7.472↑	7.434	7.3↓	7.4	-	-
pCO2 (35-45 mm-Hg)	32.5	35	32↓	27.2↓	36.6	2.5↓	27↓	-	-
HCO3- (21-28 mEq/L)	22.8	20.8↓	18.5↓	19.4↓	24	15.9↓	16.3↓	-	-
S.LAC	-	-	-	-	0.92	3.5		-	-
Sodium (136-145 mEq/L)	-	140	136	137	139	144	152↑	148	151↑

Potassium (3.5-5.1 mEq/L)	-	3.2↓	3.1↓	2.8↓	2.4↓	2.8↓	2.6↓	2.62↓	4.8↓
Creatinine (0.72-1.25 mg/dL)	-	0.97	2.45↑	3.03↑	3.29↑	4.04↑	4.41↑	3.15↑	4.62↑
Urea (6-12 mg/dL)	-	26↑	59↑	75↑	100↑	109↑	118↑	74↑	99↑
Total Billirubin (0.2-1.5 mg/dl)	-	-	1.59↑	1.49	-	1.61↑	1.18	-	-
SGPT (0-45 IU/L)	-	-	46↑	32	-	41	39	-	-
SGOT (0-35 IU/L)	-	-	36↑	-	-	19	23	-	-
Hb (13-17 gm/dl)	-	14.7	15.08	14.11	13.9	14.55	13.14	13.28	-
RDW (11.6-14%)	-	11.3↓	11.18↓	11.33↓	11.1↓	10.84↓	11.42↓	11.44↓	-
WBC (4000-11000/cmm)	-	9970	13100↑	13800↑	9510	9200	11000	16700↑	-
Neutrophils (40-80%)	-	78	80	80	80	74	79	82↑	-
Lymphocytes (20-40%)	-	18↓	17↓	15↓	15↓	23	17↓	15↓	-
Platelet (150000-400000/cmm)	-	229800	199000	165000	161800	136000↓	151000	160000	-
PT-test	-	-	-	-	15.1	-	-	-	-
Pt-control	-	-	-	-	13.5↑	-	-	-	-
INR	-	-	-	-	1.12↑	-	-	-	-

Discussion

This case exemplifies the severe and rapid progression of paraquat poisoning, a reflection of the broader challenges in managing such toxic exposures. Paraquat's toxicity, compounded by its rapid absorption and systemic distribution, often leads to catastrophic health outcomes, even with minor exposures [3,4,10]. In this instance, despite immediate medical intervention including gastric lavage and aggressive supportive care due to the unavailability of a required adsorbent for hemodialysis, the patient exhibited a progression of symptoms associated with paraquat poisoning, including gastrointestinal distress, acute renal failure, lung involvement and ultimately, multi-organ dysfunction.

The pathophysiology of paraquat involves the generation of superoxide radicals that cause extensive lipid peroxidation and cellular damage [11]. This oxidative stress primarily affects the lungs but also has severe implications for renal and hepatic function. This mechanism explains the initial stability observed in some patients, followed by sudden and severe deterioration as organ damage progresses [12].

The survival of patients with paraquat poisoning therefore depends on the availability of critical intervention strategies that influence outcomes. These interventions include.

Hemoperfusion and Hemodialysis:

Early Hemoperfusion: Initiating hemoperfusion within 4 hours of ingestion has been associated with improved survival rates. A recent study emphasized that the timing of hemoperfusion is critical, with earlier initiation leading to better outcomes [13].

Combined Therapies: Integrating continuous veno-venous hemofiltration (CVVH) with hemoperfusion has shown promise in enhancing patient survival. A study reported that this combination significantly improved outcomes in paraquat poisoning cases [14].

Immunosuppressive and Antioxidant Therapies:
The efficacy of immunosuppressive agents like cyclophosphamide and corticosteroids, along with antioxidants such as N-acetylcysteine, remain a topic of debate. Some studies suggest potential benefits, while others report minimal impact on survival rates. This inconsistency highlights the need for further research to establish standardized treatment protocols [15].

Supportive Care and Monitoring:
Comprehensive supportive care, including meticulous monitoring for organ dysfunction, is vital. Plasma paraquat concentration and SOFA (Sequential Organ Failure Assessment) score has been used to estimate toxicity severity and predict outcomes, though their reliability and accessibility remain variable [7]. These tools are not universally adopted and may be limited by timing and resource constraints [7]. Cases have demonstrated that despite aggressive supportive measures, the prognosis remains poor, especially when interventions are delayed or when patients present with severe poisoning [15].

Comparative Analysis of Recent Cases
Recent case studies reinforce the critical importance of early and aggressive intervention. For instance, a case report highlighted that delayed presentation and treatment were associated with high mortality, despite the availability of advanced supportive therapies [16]. Conversely, cases where early hemoperfusion was implemented, showed relatively better outcomes, underscoring the time-sensitive nature of paraquat toxicity management [16].

Alternative safer options
A wide range of effective and less hazardous alternatives to paraquat are available, including both chemical substitutes and non-herbicidal methods such as integrated weed management (IWM), cover cropping, mechanical weeding, and botanical herbicides, all of which have shown success without compromising agricultural productivity [17].

The high mortality rate associated with paraquat, coupled with the rapid clinical decline often seen in such poisonings, calls for an urgent re-evaluation of the availability of paraquat, especially in regions where regulatory oversight is limited [5]. This case, while tragic, provides crucial clinical insights into the severe impact of paraquat poisoning and the pressing need for enhanced clinical management and preventive strategies in agricultural practices [5].

Future studies and case reports should focus on validating accessible prognostic markers and evaluating scalable therapeutic interventions for rural and resource-constrained healthcare settings.

Conclusion

This case highlights the urgent need for immediate and specialized interventions in paraquat poisoning. Key lessons include the critical importance of rapid access to treatments and early interventions like hemoperfusion, which was not possible due to the unavailability of a particular adsorbent but could have potentially altered the outcome. This case underscores the deadly risk of even minimal paraquat exposure.

Ethical Considerations
For this case report detailing the unfortunate outcome of paraquat poisoning in a 19-year-old male, no ethical approval was required from the Institutional Review Board (IRB) of Government Medical College, Bhavnagar, Gujarat. This decision is based on the IRB's guidelines, which do not mandate

ethical approval for the publication of anonymized case reports. Further, all personal identifiers have been removed or altered to protect the privacy and confidentiality of the patient, in accordance with ethical standards for case reporting.

Consent for the publication of this case has been obtained from the deceased patient's next of kin. This consent was taken with a complete understanding of the intent to publish the case details to enhance medical knowledge and contribute to better therapeutic strategies for managing paraquat poisoning. The family was supportive of the publication, acknowledging the potential for this tragic case to provide valuable insights that could save future lives.

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