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Case Reports

Survival Following Severe Paraquat Poisoning a Case Study from the Andaman and Nicobar Islands

[1]Shiv Shankar Singh, [1]Colin Sanchez and [2]Prasan Kumar Panda

[1]Govind Ballav Pant Hospital, Andaman and Nicobar Islands Institute of Medical Sciences (ANIIMS), Port Blair, India
[2]Department of Medicine (Infectious Disease Division), All India Institute of Medical Sciences, Rishikesh, India

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Corresponding Author:
Shiv Shankar Singh
Govind Ballav Pant Hospital, Andaman and Nicobar Islands Institute of Medical Sciences (ANIIMS), Port Blair, India
Email: ssingh_gbp@yahoo.com

Abstract: Paraquat poisoning continues to pose a significant public health challenge in the Andaman and Nicobar Islands, largely due to its alarmingly high mortality rate. In this report, we present the case of a 39-year-old male farmer who ingested 30 mL of paraquat with the intent of suicide. Initially, the patient underwent prompt gastric lavage and exhibited relative stability. Upon admission, he complained of epigastric pain and vomiting but did not display classic symptoms such as oral ulceration or respiratory distress. Early laboratory findings were unremarkable, with no immediate evidence of organ dysfunction. However, as the days progressed, the patient developed severe non-oliguric renal impairment along with mild hepatic involvement. On the fifth day following ingestion, treatment was initiated with methylprednisolone (1 g intravenously, daily) and N-acetylcysteine (600 mg orally, three times a day), resulting in marked clinical improvement. Notably, his renal function improved rapidly and hepatic dysfunction resolved. Over the next several days, the patient's serum creatinine levels steadily decreased and liver enzymes normalized. He was discharged on the 12th day and continued to demonstrate complete recovery upon follow-up. This case highlights the potential benefits of early intervention with immunomodulatory and antioxidant therapies in counteracting the severe toxicity of paraquat, despite the historically poor prognosis associated with such poisonings in the region. The successful outcome in this patient emphasizes the critical role of timely diagnosis, aggressive decontamination, and individualized treatment strategies in resource-limited settings. Further research is essential to optimize treatment protocols and improve survival rates in paraquat poisoning cases, particularly in areas with limited access to advanced healthcare.

Keywords: Acute Kidney Injury, Herbicide Toxicity, Methylprednisolone, N-acetylcysteine, Paraquat Poisoning

Introduction

Paraquat, a widely used herbicide known for its cost-effectiveness, presents a significant public health challenge in developing regions like the Andaman & Nicobar Islands. Its accessibility and effectiveness in weed control have unfortunately made it a common choice for suicidal ingestion among agricultural workers and rural populations (Raghu *et al.*, 2013). This herbicide's toxicity is profound, with ingestion often resulting in severe multi-organ damage and a mortality rate that can exceed 70% (Asaduzzaman *et al*., 2023).

The Andaman and Nicobar Islands, with their agricultural dependency and remote healthcare infrastructure, face particular vulnerability to paraquat poisoning. In these settings, where timely medical intervention and advanced treatment options are often limited, the management of paraquat toxicity remains a daunting challenge (Asaduzzaman *et al*., 2023).

Paraquat and diquat, classified as bipyridyl compounds, undergo redox cycling in cells, producing reactive oxygen species that cause oxidative stress by depleting NADPH. This leads to cellular damage (via lipid peroxidation and apoptosis) and a secondary inflammatory response. Over hours to days, these

processes result in multi-organ failure, primarily affecting high-blood-flow organs like the lungs, heart, kidneys, and liver (Li *et al*., 2021; Buckley, 2001). Historically, local studies have reported high mortality rates, underscoring the urgent need for effective therapeutic strategies tailored to regional contexts.

Immunomodulatory therapies, including cyclophosphamide and methylprednisolone, have been explored for their potential to mitigate paraquat-induced organ damage, though outcomes have been variable (Buckley, 2001). The absence of a specific antidote complicates treatment, emphasizing the importance of early recognition, aggressive decontamination measures like gastric lavage, and supportive care to minimize systemic absorption and maximize survival chances (Marashi *et al*., 2015).

This case study aimed to report where after consumption of around 30 mL of Paraquat the patient developed severe non-oliguric acute kidney injury but recovered completely with the use of injection methylprednisolone and N-Acetylcysteine without the need for dialysis.

Method

It is an observational single case study. We followed CARE guidelines when reporting this case study.

Results

A 39-year-old male farmer was admitted to our tertiary care hospital on 13 August 2023 after ingesting 30 mL of paraquat herbicide (the bottle with a distant expiry date) with suicidal intent. He reported the ingestion occurred around 5:00 pm, following which he experienced five episodes of vomiting within an hour. Despite vomiting, he denied any oral ulceration or significant throat pain at the time of admission.

On initial examination at a nearby primary health center, the patient was conscious, oriented and hemodynamically stable. His vital signs included a pulse rate of 112 beats per minute, blood pressure of 112/84 mmHg and a respiratory rate of 18 breaths per minute. Physical examination revealed mild epigastric tenderness, but no signs of respiratory distress or oral cavity ulceration were noted. Following gastric lavage at the primary health center, he was promptly referred to our facility for further management.

Upon arrival at our hospital approximately 5 h post-ingestion, the patient continued to complain of mild epigastric pain, but remained hemodynamically stable without respiratory distress. His initial laboratory investigations, including complete blood count, liver function tests and renal function tests, were within normal limits (Table 1). However, given the history of paraquat ingestion and the potential for delayed toxicity, close monitoring was initiated.

Over the next 48 h, the patient developed new-onset burning throat pain and mild dysphagia, prompting a reassessment of his clinical status. Repeat laboratory investigations revealed a progressive rise in serum creatinine levels, peaking at 7 mg/dL on the fifth day of admission with metabolic acidosis (pH = 7.15). Concurrently, there was a mild elevation in serum bilirubin levels, indicating acute kidney injury and mild hepatic impairment. Serum Paraquat levels couldn't be assessed due to lack of facility.

The differential diagnosis at this stage included paraquat poisoning with associated multi-organ dysfunction, particularly acute kidney injury and liver injury. Given the rapid onset of renal impairment following ingestion and the absence of significant respiratory symptoms, alternative diagnoses such as other herbicide ingestions or infectious etiologies causing systemic toxicity were considered less likely based on clinical presentation and history.

Table 1: Basic investigations of the patient during hospital stay

Parameter	Day 1	Day 3	Day 5	Day 7	Day 9	Day 12
Hemoglobin (g/dL)	12.8			12.2		12.4
Total Leukocyte count (/μL)	5610			5450		9390
Total Platelets (×10^3/μL)	187			175		282
Random blood glucose (mg/dL)	84			96		
Urea (mg/dL)	25	40	58	97	78	68
Creatinine (mg/dL)	1.1	3.5	7.0	7.1	2.6	1.9
Sodium (mEq/L)	142	140		133	145	144
Potassium (mEq/L)	3.7	3.8		3.7	4.0	3.7
Total Bilirubin (mg/dL)	1.2	2.1	3.0	1.4	0.8	0.9
AST– Aspartate transaminases (U/L)	21	35	58	27	41	48
ALT – alanine transaminases (U/L)	17	38	50	51	45	58

Management strategies included supportive care with intravenous fluids, soda bicarbonate, and pantoprazole for gastric protection, alongside close monitoring of renal and hepatic functions. Despite these measures, the patient's clinical course continued to deteriorate with worsening renal parameters and rising bilirubin levels.

On the fifth day post-ingestion, in light of progressive renal dysfunction and evolving liver injury, a decision was made to initiate methylprednisolone pulse therapy (1 g IV daily) and oral N-acetylcysteine (NAC, 600 mg three times daily). This therapeutic approach aimed to attenuate the inflammatory response and oxidative stress induced by paraquat toxicity, potentially preventing further organ damage and improving clinical outcomes (Dinis-Oliveira *et al*., 2008).

Following the initiation of methylprednisolone and NAC therapy, there was a notable improvement in renal function within 48 h, with serum creatinine levels declining steadily. Liver function markers also began to normalize by the fifth day of therapy, indicating a positive response to treatment. Throughout the hospitalization, the patient maintained adequate urine output, obviating the need for renal replacement therapy such as hemodialysis.

After five days of therapy, methylprednisolone and NAC were discontinued and the patient was discharged in stable condition on the 12th day of admission. Follow-up examinations three and subsequent 30 days post-discharge revealed complete recovery of kidney and liver functions, underscoring the efficacy of early intervention and combined therapy in mitigating severe paraquat toxicity.

Discussion

Paraquat, a toxic Bipiridyl compound discovered in 1950 that was initially used to kill marijuana weeds in the United States and Mexico, soon became popular worldwide as a cheap and effective herbicide. Paraquat poisoning poses a significant clinical challenge worldwide, particularly in regions like the Andaman and Nicobar Islands, where agricultural dependence and limited healthcare resources converge with the herbicide's ready availability. The toxic mechanism of paraquat involves its rapid absorption from the gastrointestinal tract, distribution to various organs, and subsequent generation of reactive oxygen species, leading to oxidative stress, cellular damage, and organ dysfunction (Marashi *et al*., 2015; Suntres, 2002).

Management strategies for paraquat poisoning have evolved, focusing on limiting absorption, enhancing elimination, and mitigating oxidative damage. Early decontamination through gastric lavage and administration of activated charcoal remains fundamental in reducing paraquat bioavailability and potentially improving clinical outcomes (Hong *et al*., 2024). However, the effectiveness of these measures diminishes rapidly after ingestion due to paraquat's quick absorption kinetics.

In our presented case, the patient ingested a significant quantity of paraquat but promptly received gastric lavage within hours of ingestion. Despite showing initial stability, he developed severe non-oliguric acute kidney injury and mild liver dysfunction within days. This clinical course underscores the unpredictable nature of paraquat toxicity, where patients may initially appear stable before deteriorating rapidly due to delayed organ-specific manifestations.

The role of immunosuppressive therapies in paraquat poisoning, including corticosteroids and cyclophosphamide, remains controversial. While some studies suggest potential benefits in mitigating inflammatory responses and reducing mortality rates, others have shown inconclusive results or minimal impact on overall survival, even in the same region with 12 years of study (Panda *et al*., 2021). In our case, methylprednisolone pulse therapy was initiated on the fifth-day post-ingestion, coinciding with the onset of acute kidney injury and worsening liver function. The decision to commence therapy was based on the rapid deterioration of renal function and liver injury markers, necessitating intervention to potentially modulate the inflammatory cascade and oxidative stress pathways implicated in paraquat toxicity.

The addition of NAC, a precursor of glutathione and a known antioxidant, aimed to counteract paraquat-induced oxidative stress and mitigate further cellular damage (Buckley, 2001). The combined regimen of methylprednisolone and NAC was associated with a rapid improvement in renal function, evidenced by declining serum creatinine levels and sustained urine output. Liver function markers also normalized within days of initiating therapy, reflecting the therapeutic efficacy of early immunomodulation and antioxidant supplementation in our patient.

The absence of a specific antidote for paraquat poisoning underscores the importance of supportive care and symptomatic management in optimizing patient outcomes. Hemodialysis, while indicated in severe cases of renal failure, was not required in our patient due to the preserved urine output and favorable response to methylprednisolone and NAC therapy (Adhikari *et al*., 2023). This highlights the potential for conservative management approaches in selected cases, emphasizing the individualized nature of treatment decisions based on clinical presentation and response to initial therapies.

The geographical context of paraquat poisoning in the Andaman and Nicobar Islands further complicates treatment paradigms, necessitating tailored approaches that consider local epidemiology, healthcare infrastructure, and resource availability. Our case report

contributes to the growing body of evidence supporting the role of early intervention and combined therapeutic strategies in improving outcomes for severe paraquat poisoning cases, particularly in settings with limited access to advanced medical care (Banday *et al*., 2024; Eizadi-Mood *et al*., 2022).

Being a single case study, this is always a limitation. However, everything about the case is available which can be analyzed in future cases and practiced if required.

Conclusion

Despite paraquat poisoning in the islands with reported 100% mortality, this case ascertains survival with early vomiting, gastric lavage, and steroid-NAC therapy. Continued research efforts are essential to refine treatment protocols, enhance therapeutic efficacy, and ultimately reduce the significant morbidity and mortality associated with paraquat ingestion in vulnerable populations.

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Author's Contributions

Shiv Shankar Singh: Gave concept and design, Did literature search, collected data, drafted the manuscript and approved.

Colin Sanchez and Prasan Kumar Panda: Did literature search, analyzed data, reviewed the manuscript and approved.

Ethics

The study was conducted according to accepted ethical guidelines for the conduct of research on HUMANS. No Animals were used in this research. All human research procedures followed were in accordance with the ethical standards of the committee responsible for human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008. Considering the retrospective observational single case study and de-identified nature of the case, ethical approval by ANIIMS (Port Blair) ethics committee/IRB was not required, however, individual Written informed consent was obtained.

Availability of Data and Materials

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials. However, any further requirements, it is available with corresponding author, and on request can be provided.

Conflict of Interest

The author or authors declare that they have no conflict of interest with respect to the author or publication of this article.

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