Case Series

Paraquat poisoning: A silent and deadly threat

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Abstract

Background: Paraquat poisoning is a life-threatening condition with a high mortality rate, especially in developing countries like India. This article presents four patients with paraquat ingestion with severe complications, including fatal outcomes despite early hospital admission and aggressive management such as hemoperfusion. It highlights epidemiology, clinical presentation, management strategies, and emphasizes the importance of prevention and awareness, particularly among young individuals.

Key words: Paraquat poisoning; Hemoperfusion

1. Introduction

Paraquat is a highly toxic herbicide widely used in agriculture. Even small quantities can lead to multi-organ failure and death. There is no specific antidote, making early recognition and supportive care crucial. In India, paraquat poisoning is increasingly reported, especially among young individuals due to easy availability and intentional ingestion.

1.1. Paraquat poisoning, Incidence in India

- Paraquat accounts for a significant proportion of pesticide poisoning cases.
- Mortality rate: 50–90%
- Increasing trend in rural and semi-urban areas
- Commonly associated with intentional self-harm
- Lack of strict regulation and easy accessibility contributes to rise in incidence

1.2. Young age affected

- Most affected group: 15–30 years
- Around 60–70% of cases occur in young individuals
- Higher incidence in females in some regions

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1.3. Common reasons

- Emotional stress
- Social pressure
- Lack of awareness about toxicity

2.1. Patient 1

A 23-year-old female presented to the hospital with a history of paraquat ingestion at home around 7:40 PM. She was brought to the hospital within approximately three hours, with initial complaints of vomiting.

On admission, she was conscious and oriented. Her vital signs were relatively stable, with a pulse rate of 62 per minute, blood pressure of 100/60 mmHg, and oxygen saturation of 99% on room air. A urine Dithionite test was performed, which turned out to be strongly positive, confirming paraquat poisoning.

Immediate treatment was initiated. Gastric decontamination was done using Ryle's tube wash. She received antioxidants, steroids, cyclophosphamide, proton pump inhibitors, and antiemetics. Hemoperfusion was also initiated, and one cycle was completed.

However, during the second cycle of hemoperfusion, the patient developed hypotension, which rapidly progressed to cardiac arrest. Cardiopulmonary resuscitation was started as per ACLS protocol, there was no return of spontaneous circulation.

She was declared dead at 11:00 PM on the following day. The immediate cause of death was cardiac arrest, with the underlying cause being paraquat poisoning.

2.2. Patient 2

A 26-year young male presented with a history of alleged paraquat ingestion (50–70 ml) at home and was initially treated at outside hospital, where six cycles of hemoperfusion and six units of FFP were administered before referral for further management.

On admission, the patient was conscious but critically ill, with icterus and tachypnea. Vitals showed PR 112/min, BP 110/70 mmHg, and SpO2 72% despite 15 L oxygen. Respiratory examination revealed basal crepitations, and the abdomen was soft. Investigations demonstrated deranged renal and liver function tests, consistent with acute kidney injury, acute liver injury, and acute lung injury secondary to paraquat poisoning.

The patient was managed in the ICU with N Acetyl Cysteine infusion, steroids, antibiotics, proton pump inhibitors, antiemetics, diuretics, and supportive care, along with pulmonology consultation. However, persistent hypoxia (SpO_2 70–72%) and one episode of hematemesis were noted during the hospital course.

Despite aggressive management, the patient's condition deteriorated to multiorgan dysfunction. In view of the poor prognosis, the family was counseled, and the patient was discharged against medical advice (DAMA).

2.3. Patient 3

A 58years female presented with a history of alleged consumption of paraquat dichloride 24% (approximately 100 ml) and was status post two cycles of hemoperfusion. The patient developed giddiness within 45 minutes, followed by three episodes of vomiting, with no history of chest pain, shortness of breath, or loss of consciousness, and had no known comorbidities.

On examination, the patient was conscious, oriented, afebrile, and tachypneic. Vitals revealed a heart rate of 88/min, blood pressure of 90/50 mmHg, respiratory rate of 28/min, and SpO2 of 98% on room air. Systemic examination showed normal heart sounds, bilateral normal breath sounds, and a soft, non-tender abdomen.

Investigations revealed a strongly positive urine Dithionite test. Echocardiography showed normal cardiac chambers with an ejection fraction of 60% and Grade I diastolic dysfunction, with no major abnormalities.

The patient underwent gastric decontamination with activated charcoal and was treated with N-acetylcysteine infusion, steroids, cyclophosphamide, and supportive care, along with two cycles of hemoperfusion.

During the hospital course, despite multidisciplinary management, the patient developed sudden hypoxia and bradycardia. Cardiopulmonary resuscitation and ACLS protocol were initiated; however, the patient could not be revived.

The immediate cause of death was respiratory failure secondary to paraquat poisoning, with no other contributing conditions. This case highlights the high mortality associated with paraquat poisoning, where even early and aggressive treatment may not prevent a fatal outcome.

2.4. Patient 4

A 15-year-old girl was brought to the hospital after allegedly consuming paraquat solution at her home in the early hours of the morning.

It began around 1 AM, when she ingested the toxic substance. Soon after, she developed repeated episodes of vomiting. Recognizing the seriousness of the situation, her family rushed her to a nearby hospital, where immediate first aid measures were initiated. Gastric lavage was performed, and activated charcoal was administered through a Ryle's tube to reduce further absorption of the toxin.

As the morning progressed, she was referred to a higher center for advanced management. On arrival, she was conscious, oriented, and not in apparent distress. Her vital signs were stable, offering a brief sense of reassurance.

However, paraquat poisoning is often deceptive. Further investigations revealed a positive urine dithionite test, confirming paraquat exposure. Considering the severity, hemoperfusion was initiated on the same day to enhance toxin removal from the bloodstream.

Gradually, she began to develop new symptoms, including severe throat pain and difficulty swallowing even saliva—suggestive of corrosive injury caused by the toxin.

The medical team explained the guarded prognosis to her family. Despite aggressive management with intravenous fluids, antibiotics, proton pump inhibitors, antiemetics, antioxidants, steroids, and other supportive measures, her condition remained critical.

A multidisciplinary team—including specialists from gastroenterology, pulmonology, and ENT—was involved in her care. A second cycle of hemoperfusion was advised to improve outcomes.

At this critical juncture, the family, overwhelmed by the situation, chose not to continue further advanced treatment. She was discharged against medical advice.

Subsequently, she was taken to Government Hospital for further care. Despite continued management, her condition deteriorated, and she was later declared dead.

3. Management of Paraquat poisoning

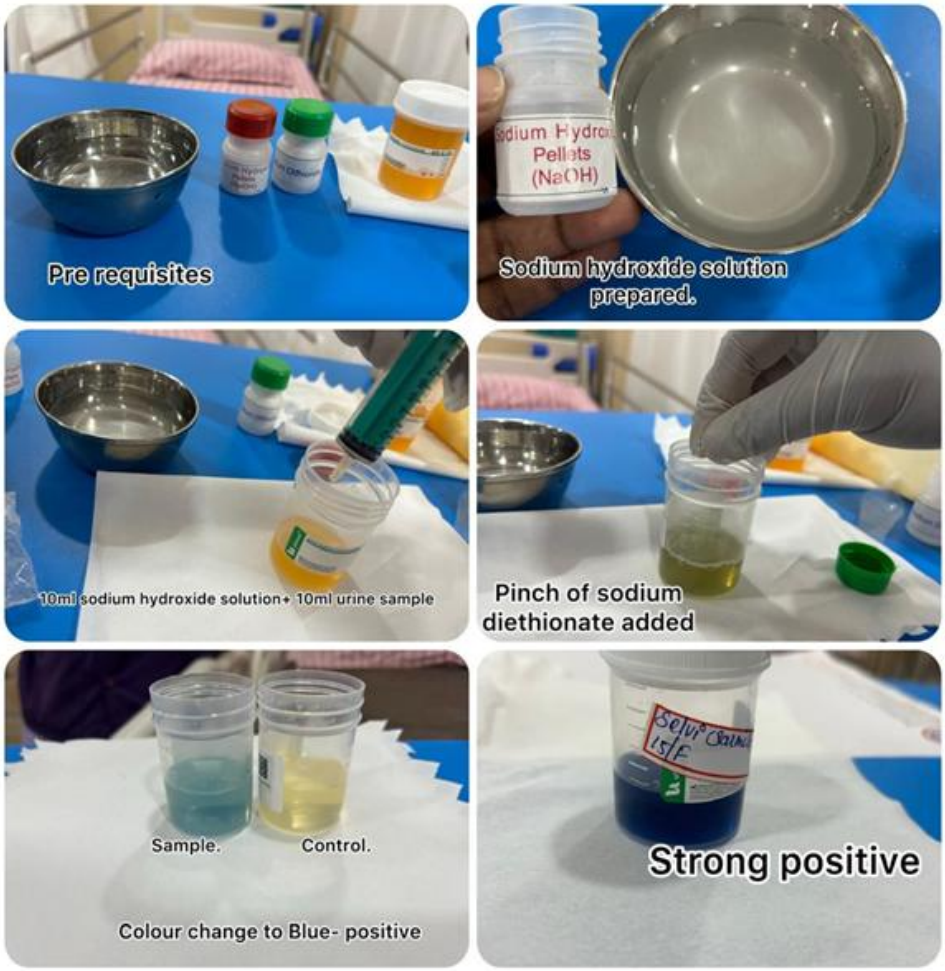
3.1. Early Management

- Immediate gastric decontamination (within 1–2 hours)
- Activated charcoal / Fuller's earth

3.2. Hospital Management:

Urine dithionite test (for rapid confirmation)

- Bedside Urine Dithionite Test (Image-based Steps)
- Collect urine sample
- Add sodium hydroxide solution
- Add sodium dithionite
- Blue color → Positive (Paraquat present)
- Dark blue = strong positivity (high toxicity)



Definitive Treatment: Hemoperfusion (early initiation improves survival)

4.1. Immunosuppressive therapy

- Steroids
- Cyclophosphamide
- Antioxidants:
- Vitamin C
- Vitamin E
- N-acetylcysteine

4.2. Supportive care

- Oxygen therapy (cautious use)
- Fluid management
- Organ support

4.3. Why Oxygen must be used carefully?

- Paraquat toxicity increases with high oxygen levels
- Enhances free radical formation
- Worsens lung injury

4.4. Accelerates fibrosis

Clinical rule: Use oxygen only if $SpO_2 < 90\%$, and give minimal required amount

4.5. How does Hemoperfusion help?

- Removes paraquat via adsorption
- More effective than hemodialysis
- Best if started within 4–6 hours

Limitation: Less effective after tissue distribution

4.6. Are there poison- specific filters?

- No paraquat-specific filters
- Charcoal cartridges preferred
- Adsorb paraquat effectively

4.7. How do Steroids + Cyclophosphamide help?

- Reduce inflammation
- Prevent alveolar damage
- Delay lung fibrosis
- Suppress immune-mediated injury

Limitation: Limited benefits in severe poisoning

4.8. How do antioxidants help?

Paraquat causes

- Free radical (ROS) formation
- Oxidative stress → cell death

Antioxidants

- Neutralize free radicals
- Reduce cellular damage

- Protect lungs

Causes of Cardiac arrest

Main causes

- Hypoxia (most common)
- Metabolic acidosis
- Multi-organ failure
- Shock
- Not primarily due to direct cardiotoxicity

5.1. Lung damage- Step by Step mechanism and outcome

Mechanism

- Selective accumulation in alveolar cells
- Generation of reactive oxygen species (ROS)
- Lipid membrane damage
- Inflammatory response
- Alveolitis (acute lung injury)
- Fibroblast activation
- Collagen deposition

Outcome

- Early: Acute lung injury → hypoxia
- Intermediate: ARDS-like picture
- Late: Irreversible pulmonary fibrosis
- Final cause of death in most cases: Respiratory failure

5.2. Is Serum Paraquat level measurement required?

Not mandatory in all settings, especially resource-limited hospitals

Useful for:

- Prognosis prediction
- Severity assessment (using nomograms)

Limitations

- Not widely available
- Time delay reduces usefulness
- Clinical condition is more important

Practical approach: Urine dithionite test + clinical assessment is sufficient in most cases

Prognosis

Depends on:

- Amount ingested
- Time to treatment
- Organ damage severity
- Overall prognosis remains poor

6. Nursing management: Nurses play a critical role in management:

Assessment

- Monitor vital signs continuously
- Assess respiratory status
- Observe for signs of organ failure

Interventions

- Assist in gastric lavage
- Administer medications as prescribed
- Maintain fluid balance
- Provide oxygen therapy carefully
- Supportive Care
- Psychological support to patients and family
- Infection prevention
- Documentation of clinical changes
- Emergency Care
- Immediate response during cardiac arrest
- Assist in CPR and advanced life support

Prevention of Paraquat Poisoning

- Strict regulation of sales
- Safe storage practices
- Avoid storing in drink bottles
- Public awareness programs
- Mental health counseling

7. Conclusion

Paraquat poisoning remains one of the most lethal toxicological emergencies with extremely high mortality despite aggressive treatment. Early gastric decontamination, prompt hemoperfusion, and supportive care are the cornerstones of management, but outcomes remain poor due to rapid lung injury and fibrosis. Prevention, awareness, and restriction of access are the most effective strategies to reduce morbidity and mortality.

8. Strong closing line!

In paraquat poisoning, survival is rare once toxicity sets in—prevention is not just better than cure, it is often the only cure."