Paraquat Toxicity in Humans: Advances in Diagnosis, Management and Prevention

*Mondal RN,[1] Jahan SM,[2] Rani M,[3] Ali SS,[4] IslamMN[5]

Abstract

Background: Paraquat, a widely used herbicide, remains one of the most lethal pesticides for humans due to its high toxicity and lack of a definitive antidote. Ingestion, even in small amounts, often leads to multi-organ failure and death, particularly in resource-limited settings.

Objective: Objectives of this review was updated synthesis of the current understanding of paraquat toxicity, with a focus on recent advances in diagnosis, therapeutic management, and preventive strategies.

Methods: Relevant literature was retrieved through a comprehensive search of PubMed, Scopus, and Google Scholar databases using terms such as "paraquat poisoning," "diagnosis," "treatment," and "prevention." Articles published from 2000 to 2025 were considered, with emphasis on clinical trials, observational studies, and recent guidelines. As there were limited publications about paraquat poisoning, we took some articles published before 2000.

Results: Early diagnosis using plasma or urine paraquat levels, supported by prognostic tools such as the Proudfoot and Hart nomograms, improves clinical decision-making. Management strategies now include aggressive decontamination, immunosuppressive therapy (e.g., cyclophosphamide, corticosteroids), antioxidants (e.g., N-acetylcysteine), and early hemodialysis in selected cases. However, outcomes remain poor in severe cases. Recent public health efforts have focused on restricting paraquat use, improving labeling, and enhancing community awareness to prevent intentional and accidental exposure.

Conclusion: While advances in diagnostic and treatment modalities offer some hope in managing paraquat poisoning, prevention remains the most effective strategy. Continued research, policy regulation, and education are crucial to reducing the global burden of paraquat-related morbidity and mortality.

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1. **Dr. Ratindra Nath Mondal, Associate Professor, Department of Medicine, Prime Medical College. dr.ratinmondal@gmail.com.*
2. *Professor Dr. Shah Md. Sarwer Jahan, Department of Medicine, Dinajpur Medical College*
3. *Dr. Moni Rani , Medical Officer, Department of Radiotherapy, Rangpur Medical College*
4. *Professor Dr. Sk Sadek Ali, Department of Paediatrics, Dinajpur Medical College*
5. *Professor Dr. Md Noor Islam, Department of Medicine, Prime Medical College*

**For correspondence*

Introduction

Paraquat is a toxic bipyridyl compound that was discovered in 1950s and found its way

into agricultural use by 1962. Initially developed for controlling weeds and grasses, this liquid herbicide was later classified as a 'restricted-use' chemical due to its high toxicity.[1] Despite its toxicity, the low cost and effectiveness of this pesticide have led to its widespread and often unregulated use in developing countries such as ours. Its high lethality after ingestion, even in small amounts, makes it a public health concern. Paraquat poisoning carries a high mortality rate, with fatalities reported in up to 70% of cases.[2] Despite partial bans or restrictions in countries like South Korea and Sri Lanka, paraquat continues to account for thousands of deaths annually, particularly from intentional ingestion in agricultural communities across South and Southeast Asia.[3]

In our country clinical management strategies for paraquat toxicity if mainly symptomatic, hence morality and morbidity is very high. There are huge challenges in treatment approach for paraquat poisoning ranges from confirmation, decontamination, lack of facilities of removal of poison (hemoperfusion) and management of the complications. Therefore, we are going to review this topic to synthesizes recent insights into pathophysiology, clinical manifestations, diagnostics, and evolving therapeutic strategies, and highlights epidemiological trends, especially from high-burden regions.

Methods

This review was conducted to synthesize current knowledge on the diagnosis, clinical management, and prevention of paraquat toxicity in humans. A structured literature search was carried out using the following approach:

Search Strategy

A comprehensive search of electronic databases including PubMed, Scopus, Google Scholar, and Web of Science was performed. The search included literature published from January 2000 to June 2025. The following keywords and MeSH terms were used in various combinations: “Paraquat poisoning”, “herbicide toxicity”, “pulmonary fibrosis”, “antioxidants”, “cyclophosphamide”, “immunosuppressive therapy”, “diagnosis”, “management”, “treatment outcome”, and “prevention”.

Inclusion Criteria

- Articles published in English
- Peer-reviewed original research articles, systematic reviews, meta-analyses, and clinical guidelines
- Studies involving human subjects with confirmed or suspected paraquat poisoning
- Articles discussing mechanisms, diagnosis, treatment, or prevention strategies

Exclusion Criteria

- Non-English language articles without English translation
- Studies conducted exclusively on animals or in vitro models, unless they provided relevant mechanistic insights
- Case reports with insufficient data
- Articles not directly relevant to the objective of the review

Selection Process

Two independent reviewers screened the titles and abstracts of identified articles for relevance. Full texts of potentially eligible articles were retrieved and assessed according to the specified criteria for selection and elimination. Any discrepancies were resolved through discussion and consensus, or by consultation with a third reviewer.

Data Extraction and Synthesis

Relevant data were extracted into a standardized format, including study design, population, intervention/treatment modalities, outcomes, and key findings. The findings were then narratively synthesized, focusing on recent advancements in diagnosis

(biomarkers, imaging), management (supportive care, antioxidants, immunosuppressants), and prevention strategies.

Quality Assessment

Where applicable, the quality of included studies was assessed using appropriate tools (e.g., Cochrane Risk of Bias Tool for randomized trials, Newcastle-Ottawa Scale for observational studies). However, due to this review's focus on a narrative synthesis, no quantitative meta-analysis was performed.

Epidemiology

Global and regional insightsParaquat poisoning disproportionately affects low- and middle-income countries in Asia, Latin America, and parts of Africa. Recent multicenter data show:- In China, paraquat remains one of the top three agents involved in fatal self-poisoning.[4] In Bangladesh and Nepal, small-scale surveys report case fatality rates of ~60–70%.5- Sri Lanka, after banning paraquat in 2014, saw a >50% reduction in pesticide-related suicides.6

Lethal dose threshold (LD_1) associated with paraquat ingestion

Lethal dose (LD_{50}) for humans is estimated to be 20–40 mg/kg of body weight when ingested. This corresponds to approximately 10-15 mL of a 20% (w/v) paraquat solution**,** which is commonly used in agriculture.[7,8]

Ingestion of more than 30 mg/kg is generally associated with a poor prognosis and near 100% mortality, especially in the absence of prompt treatment. Even smaller amounts (as little as 5–10 mL**)** can be fatal if not managed aggressively and early.[9]

The toxicity is dose-dependent:

- <20 mg/kg**:** Mild poisoning; some patients may recover with supportive care.
- 20–40 mg/kg**:** Moderate to severe poisoning; prognosis is variable.
- >40 mg/kg: Fulminant poisoning with widespread organ dysfunction resulting in death, often within 24–72 hours.[10]

Pathophysiology of paraquat poisoning

Paraquat is a highly toxic herbicide. Its toxicity is mainly due to its ability to oxygen-derived free radicals, leading to oxidative stress and multi-organ damage, particularly in the lungs.

Stepwise mechanism:

1. Cellular uptake
   - Paraquat is actively taken up into cells, especially type I and type II alveolar epithelial cells in the lungs, via the polyamine uptake system.[9]
2. Redox cycling and ROS generation
   - Inside the cell, paraquat undergoes enzymatic one-electron reduction (mainly by NADPH-cytochrome P450 reductase) to form a paraquat radical.
   - The paraquat radical reacts rapidly with molecular oxygen to regenerate paraquat and produce superoxide anion ($O_2^{-}\cdot$).
   - This process repeats continuously ("redox cycling"), leading to excessive ROS production.[10]
3. Oxidative stress and cellular injury
   - Superoxide anion and derived ROS (like hydrogen peroxide and hydroxyl radicals) cause:
     - Lipid peroxidation → damages cell membranes.
     - Protein oxidation → alters enzyme and structural protein function.
     - DNA damage → impairs cell viability.
   - These lead to cell death by necrosis and apoptosis.[9]
4. Inflammation and fibrosis
   - In the lungs, the damage triggers an inflammatory response, recruitment of neutrophils, and release of pro-inflammatory cytokines.

- Over time, this results in progressive pulmonary fibrosis, which is the major cause of death in survivors.[10]

Organ involvement in paraquat poisoning

1. Lungs

Lungs are primary target of paraquat. In the lungs paraquat is accumulated in the epithelial cells, redox cycling generates oxygen-derived free radicals, causing lipid peroxidation and cellular injury. This will lead to

- Acute alveolitis and edema
- Followed by extensive pulmonary fibrosis**,** leading to respiratory failure, the major cause of death in survivors.

2. Kidneys

In the kidneys, paraquat causes direct tubular toxicity and oxidative stress, which resulting in

- Acute tubular necrosis (ATN)
- Rapid onset of kidney dysfunction (AKI) often occurs early and may contribute to paraquat accumulation due to reduced clearance.

3. Liver

Mild to moderate hepatocellular injury with elevation of liver enzymes occurs due to oxidative damage to hepatocytes.

4. Other organs

Less commonly, paraquat can damage heart (myocarditis, arrhythmia) and adrenal glands and nervous system (rare and typically subclinical).[10]

Clinical manifestations associated with paraquat toxicity

Clinical features depend upon time course and amount of ingestion. Symptoms are classified as early (occurs due to caustic effect of concentrated paraquat), intermediate (reflects systemic absorption and multi-organ toxicity) and late (due to alveolitis leading to pulmonary fibrosis i.e. fibrosing alveolitis")

Time course	Clinical features
Early symptoms (within hours)	- Gastrointestinal irritation: Nausea, vomiting (possibly with blood), diarrhea, abdominal pain, and oral ulcers (notably "paraquat tongue").[11] - Oropharyngeal damage: Painful swallowing, sore throat, and mucosal ulcerations.[12] - Systemic effects: Headache, dizziness, fever, and lethargy.[13]
Intermediate phase (1–5 days post-ingestion)	- Renal impairment: Elevated serum creatinine and urea levels indicating acute kidney injury.[8] - Hepatic dysfunction: Elevated liver enzymes and jaundice.[12] - Pulmonary symptoms: Cough, shortness of breath, and hypoxia due to developing pneumonitis.[14]
Late phase (5–14 days and beyond)	- Pulmonary fibrosis: Progressive scarring of lung parenchyma leading to respiratory failure.[13] - Multi-organ failure: In severe cases, failure of the heart, liver, kidneys, and lungs.[8]

Investigations in paraquat poisoning

Paraquat poisoning requires prompt confirmation and assessment of severity to guide prognosis and management. Below is a structured summary of the key investigations used in clinical practice.

Purpose	Investigation	Details / interpretation
Confirm exposure & assess severity	Plasma paraquat concentration[7]	Best single test; is recommended to be done within the first 4–6 hours post-ingestion. Repeat at 12–24 hours if initial level not available or ingestion time unclear. Measurement should be plotted on Proudfoot or Hart nomogram to predict prognosis (higher levels = poorer outcome)
	Urine dithionite test (sodium dithionite test)[15]	Screening test: urine turns blue if >1–2 µg/mL paraquat; qualitative, rapid, bedside test.
Assess target organ damage[8,9]	Chest X-ray	May be normal initially; later shows diffuse infiltrates or fibrosis
	High-resolution CT (HRCT) of chest	Detects early alveolitis or fibrosis before X-ray changes
	Arterial blood gas (ABG)	Detects hypoxemia; may show metabolic acidosis
	Renal function tests (creatinine, urea)	Detect renal dysfunction from tubular necrosis
	Liver function tests	May show transaminase elevation
Baseline & supportive	Full blood count, electrolytes	Evaluate for complications, hemoconcentration, or associated findings

Urine dithionite test

Quick and useful in resource-limited settings; negative test after several hours suggests lower risk.

Purpose: Rapid bedside test to detect paraquat exposure.

Method: Add sodium dithionite to an alkaline urine sample. A blue coloration indicates paraquat presence; darker shades suggest higher concentrations.

Timing: Most reliable within 12 hours post-ingestion.

Interpretation: A negative result after 6 hours suggests minimal exposure.[16,17]

Management

As there is no specific antidote, paraquat poisoning cases should be managed according to the following steps

1. Initial stabilization

- Airway, breathing, circulation (ABCs) should be assessed immediately.
- Oxygen therapy should be avoided unless SpO_2 is < 90%, as it may exacerbate lung damage through oxidative stress.[9]

2. Gastrointestinal Decontamination

- Activated charcoal (1-2 gm/kg) or Fuller's earth is effective if administered within 1-2

hour of ingestion[7] to bind paraquat in the gastrointestinal tract and reduce systemic absorption.

- Gastric lavage is controversial and generally not recommended unless patient present very early and under expert guidance.

3. Enhanced elimination

- Hemoperfusion may reduce plasma paraquat levels, although benefit is time-dependent.[18] Should be done ideally within 4 hours (may be done up to 12 hours) of ingestion to remove paraquat from the bloodstream before it distributes into tissues.[9]
- Hemodialysis may be used for renal support but has limited efficacy in toxin removal.

4. Immunosuppression

The use of glucocorticoids and cyclophosphamide is primarily indicated for moderate to severe paraquat poisoning cases**.** The goal is to suppress the inflammatory cascade and reduce lung fibrosis**,** which is the primary cause of death in paraquat toxicity.[19] Early initiation of therapy (preferably within 24 hours of ingestion) is crucial for potential efficacy.[20]

Indication of glucocorticoids and cyclophosphamide

1. Signs of pulmonary involvement, such as hypoxia, or imaging suggestive of lung injury.

Dose and duration of glucocorticoids and cyclophosphamide

Methylprednisolone:

- Dose: 1 gram/day IV for 3 consecutive days (pulse therapy)
- Followed by: Oral prednisolone 1 mg/kg/day for 2-3 weeks, then gradually tapered
- Duration: Up to 4-6 weeks, depending on clinical response

Cyclophosphamide:

- Dose: 15 mg/kg intravenously per day for 2 consecutive days.[21]
- Oral option: 2–3 mg/kg/day in some regimens for 2-3 weeks.

Adjunctive Therapy

Dexamethasone: 20 mg/day intravenously, continued until PaO_2 exceeds 11.5 kPa (approximately 86 mm Hg or SpO2 96-98%**).**[21]

Combination Therapy:

Combination of cyclophosphamide with high-dose corticosteroids (methylprednisolone or dexamethasone) has been shown to improve survival rates in some observational studies and case series, though RCT evidence remains limited.[7, 9, 10, 22, 23, 24, 25]

5. Antioxidant Therapy

Antioxidants are indicated in the early phase of paraquat poisoning to combat oxidative stress**.** Antioxidants aim to neutralize ROS and reduce organ damage. They shows greatest efficacy if given within the first few hours of ingestion, as part of combination therapy with immunosuppressive agents (e.g. steroids and cyclophosphamide).[7,9]

Among the antioxidants N-acetylcysteine (NAC), Vitamin C, and Vitamin E are administered commonly**.**[10, 23,24]

N-Acetylcysteine (NAC):

- Loading dose: 150 mg/kg IV over 1 hour,
- Followed by: 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours.
- Total duration: 21 hours; may be extended to 2–3 days depending on severity.
- Rationale: Replenishes glutathione and neutralizes free radicals.

Vitamin C (ascorbic Acid):

- Dos**e:** 1–3 g IV daily in divided into 2-3 doses
- Duration**:** 5–7 days or longer in severe poisoning

Vitamin E (tocopherol):

- Dose: 400–800 IU/day orally or IV
- Duration: 3–7 days or longer

6. Supportive Care

- Monitoring of renal, hepatic, and respiratory function is essential.
- Mechanical ventilation may be needed for respiratory failure, but is a poor prognostic sign.

Nutrition and fluid-electrolyte stability should be ensured

Renal support/Dialysis[7, 9, 22, 23,25]

1. Early removal of paraquat (if within 4-6 hours):

- Hemodialysis is less effective for paraquat removal because of its high tissue affinity but may be used if hemoperfusion is unavailable. For toxin removal hemoperfusion is better than hemodialysis.

2. Renal failure: Initiate hemodialysis if there is:

- Oliguria/anuria
- Rising serum creatinine and urea
- Severe metabolic acidosis or electrolyte disturbances (e.g. hyperkalemia)

3. Severe metabolic acidosis:

- Dialysis helps correct refractory acidosis caused by multi-organ dysfunction.

4. Supportive care in multi-organ failure:

- Dialysis may be part of intensive supportive treatment when organ support is needed, even if toxin clearance is minimal.

Regional management practices

In China and Taiwan, hemoperfusion combined with steroids and cyclophosphamide is common.[26]

In Sri Lanka and Bangladesh, treatment often limited to decontamination and supportive care.

Experimental and Emerging Therapies in Paraquat Poisoning

Despite significant advances in understanding the pathophysiology of paraquat toxicity, effective therapies remain limited. Standard treatment modalities offer only modest benefit, particularly in cases of moderate to severe poisoning. In recent years, several experimental and emerging therapies[27,28] (still in the preclinical and clinical trials) have been investigated with the intention of improving survival and mitigating organ damage.

1. Mesenchymal Stem Cell (MSC) Therapy
2. Gene Therapy and RNA Interference
3. Nrf2 Activators **(**Nuclear factor erythroid 2–related factor 2)
4. Novel Antioxidants and Mitochondrial Protectants
5. Inhibitors of Fibrosis Pathways
6. Immunomodulators Beyond Steroids
7. Extracorporeal Therapies and Nanotechnology

Prevention of Paraquat Poisoning

Paraquat is a highly toxic herbicide, and prevention strategies are critical to reduce accidental or intentional exposure, especially in developing countries where its use is widespread. Effective prevention strategies can be categorized into regulatory, environmental, educational, and individual-level measures:

1. Regulatory Measures

- Restriction and ban: Due to its high toxicity, paraquat has been banned or heavily restricted in many countries and lack of an effective antidote.[29,30]
- Legislation on packaging: Regulations mandating paraquat to be sold in non-leak, clearly labeled, and uniquely shaped containers help reduce accidental ingestion.[31]

2. Safe Storage and Handling

- Secure storage: Paraquat should be stored in locked cabinets, away from children and food items, and never transferred to drinking containers.[32]
- Personal protective equipment (PPE): Farmers and handlers must use gloves, masks, and goggles to avoid dermal and inhalational exposure.[33]

3. Public Education and Awareness

- Community-based educational programs regarding the risks and proper handling of paraquat have shown efficacy in reducing poisoning cases.[34]
- Awareness campaigns also reduce suicidal ingestion by improving mental health literacy and de-stigmatizing help-seeking behavior.[35]

4. Product Formulation Changes

- Addition of emetics, stanching agents, and gelling agents to paraquat formulations has been implemented to reduce absorption and discourage ingestion.[36]

5. Suicide Prevention Strategies

- Integration of mental health services and pesticide regulation policies has demonstrated a reduction in suicide rates related to paraquat poisoning.[37]

Conclusion

Paraquat poisoning continues to pose a major public health concern because of its extreme toxicity and the absence of a specific antidote. Recent advancements in understanding its pathophysiology, improvements in diagnostic techniques such as plasma paraquat concentration monitoring, and emerging therapeutic strategies- including immunosuppressants, antioxidants, and extracorporeal removal methods- have contributed to better clinical outcomes in select cases. However, the prognosis remains poor in cases of moderate to severe exposures.

Early diagnosis and prompt initiation of aggressive treatment are critical to improving survival. Timely identification of at-risk patients using predictive biomarkers and scoring systems can guide clinical decision-making and resource allocation in emergency settings.

Equally vital is the emphasis on prevention. Public health efforts must focus on regulatory control, safe handling practices, public education, and mental health support to reduce both accidental and intentional paraquat exposures. Comprehensive prevention strategies, along with continued research and clinical innovation, are essential to mitigating the burden of paraquat poisoning globally.

Conflict of interest – none.

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