ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

Paraquat Poisoning: A Contemporary Review

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Publication Date: 2026/01/06

Abstract: Paraquat poisoning remains a major public health problem worldwide due to its high lethality, absence of a specific antidote, and rapid progression toward multiorgan failure. Paraquat is a widely used bipyridyl herbicide whose toxicity is primarily mediated by massive oxidative stress, preferential pulmonary accumulation, and subsequent development of acute lung injury followed by extensive pulmonary fibrosis. Small ingested volumes are sufficient to cause severe systemic damage, and mortality rates remain extremely high despite aggressive supportive care. Current management strategies focus on early decontamination, supportive therapy, and various extracorporeal or pharmacological interventions, although evidence supporting their effectiveness remains limited and inconsistent. This review provides an updated synthesis of the epidemiology, pathophysiological mechanisms, clinical manifestations, prognostic factors, and current therapeutic approaches in acute paraquat poisoning, highlighting recent advances and persistent controversies in its management.

Keywords: *Paraquat, Intoxication, Toxicology, Clinical Manifestations, Management.*

How to Cite: Jhoan David Maldonado; Rafael David Moreno; Andrés Felipe Ospina; Cristian Rodrigo Hernández (2026) Paraquat Poisoning: A Contemporary Review. *International Journal of Innovative Science and Research Technology*, 11(01), 1-7. https://doi.org/10.38124/ijisrt/26jan085

I. INTRODUCTION

Paraquat (1,1-dimethyl-4,4′-bipyridinium) is a bipyridylium herbicide widely used in agriculture due to its high efficacy, low cost, and rapid action (1). Since its commercialization in the 1960s, it has become one of the most extensively used herbicides worldwide, particularly in agricultural regions of Asia and Latin America, where availability remains high and regulatory control is limited (2,3). Moreover, paraquat is recognized as one of the most toxic substances to humans: ingestion of small volumes can induce multiorgan failure and death, with case fatality rates exceeding 60–80% in multiple clinical series (4–6).

Its clinical relevance is driven by several critical factors. First, its intrinsic toxicity is explained by rapid accumulation in highly metabolically active tissues, particularly the lungs and the proximal renal tubules, where redox cycling is promoted, leading to massive free radical generation and progression toward acute alveolitis followed by extensive pulmonary fibrosis (4,7,8).

This mechanism, extensively documented in animal models and clinical studies, ultimately results in severe hypoxemia and respiratory failure, which constitutes the leading cause of death (4,7). Second, no specific antidote exists, and available therapeutic strategies—including antioxidants, immunosuppressive agents, hemoperfusion, and extracorporeal therapies—have shown limited or inconsistent efficacy, even when administered early (2,6,9,10). The absence of effective interventions renders management a complex clinical challenge associated with high mortality.

This toxicological scenario is compounded by the persistent epidemiological burden of paraquat poisoning. Paraquat intoxication continues to represent a substantial cause of morbidity and mortality in multiple countries. Globally, between 250,000 and 370,000 deaths attributable to pesticide poisoning are reported annually, and more than 90% of acute paraquat cases result from intentional ingestion (7,11). The majority of deaths occur in Southeast Asia, Central America, and South America (11). Population-based studies demonstrate a considerable disease burden: in Antioquia, Colombia, paraquat poisoning accounted for 3,299 years of life lost due to premature mortality and disability between 2010 and 2016, corresponding to 53.4 years of life lost per 100,000 inhabitants—approximately four times higher than that estimated for all chemical poisonings combined (12). This burden predominantly affects young adult agricultural workers, resulting in a significant socioeconomic impact

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

(13,14). In addition, multiple hospital-based studies indicate that most patients present after intentional ingestion, with rapid systemic deterioration and high mortality rates (8,15,16).

Although several countries have implemented total or partial bans, the current regulatory landscape remains highly heterogeneous (11,17). Paraquat continues to be available in many rural regions due to its low cost and ease of access. Evidence suggests that regulatory bans can substantially reduce pesticide-related suicides: following its withdrawal in Taiwan, paraquat-associated deaths decreased by up to 58% (17). Nevertheless, its continued presence in agricultural markets across Latin America and Asia sustains significant population-level exposure (11,18,19).

Taken together, paraquat poisoning constitutes a major toxicological emergency, characterized by extreme toxicity, absence of a specific antidote, rapid progression of pulmonary injury, and high lethality, predominantly associated with intentional ingestion within an unequal regulatory framework that perpetuates its availability. The convergence of these factors underscores the need for updated reviews and critical analyses aimed at clinical practice, providing a rigorous narrative synthesis of the pathophysiological, clinical, therapeutic, and regulatory aspects of paraquat poisoning, with particular emphasis on recent advances in the management of acute paraquat intoxication.

II. METHODOLOGY

This review aims to provide a contextualized and critical summary of current knowledge regarding the toxicology, mechanisms of action, clinical implications, and management of paraquat poisoning. To this end, a comprehensive literature search was conducted across the electronic databases PubMed/MEDLINE, Embase, ClinicalKey, and Google Scholar.

The search was restricted to articles published between January 1, 2010, and October 30, 2025, in order to ensure inclusion of the most recent evidence, with a focus on publications in English and Spanish.

Search terms were combined using Boolean operators and included: “Paraquat” AND “Intoxication,” “Paraquat” AND “Toxicology,” “Paraquat” AND “Management” OR “Treatment,” and “Paraquat” AND “Clinical Manifestations.” An additional manual search was performed by reviewing the reference lists of key articles identified to capture relevant studies not retrieved in the initial search.

Articles identified in the initial search were screened and filtered based on title and abstract, followed by full-text review of potentially eligible studies. Included publications comprised review articles, original studies, case reports with literature review, clinical guidelines, and book chapters specifically focused on paraquat as an herbicide, its toxicity in humans, cellular mechanisms of toxicity, or treatment strategies, published in English or Spanish. Editorials, letters to the editor without original data, conference abstracts, studies focused on herbicides or pesticides other than paraquat, publications in other languages, and purely agronomic or environmental studies without medical relevance were excluded.

Key information from the selected articles was extracted and qualitatively synthesized, focusing on the following domains: epidemiological characteristics of paraquat poisoning, lethal doses and prognostic factors, pathophysiological mechanisms of toxicity—particularly pulmonary, renal, and hepatic injury—acute and chronic clinical manifestations, and available medical treatment options.

III. PATHOPHYSIOLOGY OF PARAQUAT TOXICITY

Paraquat toxicity is characterized by a complex sequence of biochemical and cellular events that culminate in multiorgan damage. Its pathophysiology involves massive oxidative stress, lipid peroxidation, mitochondrial dysfunction, exaggerated inflammatory responses, and activation of apoptotic pathways, with a marked predilection for the lungs, kidneys, and liver (4,7,8,20). This section outlines the fundamental mechanisms underlying its high lethality.

A. Oxidative Stress and Reactive Oxygen Species.

The central mechanism of paraquat toxicity lies in its ability to undergo intracellular redox cycling. Paraquat accepts electrons from electron donors such as NADPH- and FAD-dependent enzymes, generating a paraquat free radical that subsequently transfers the electron to molecular oxygen, producing superoxide anion ($O_2^{\bullet-}$) (4,7,8,20).

This process, known as redox cycling, occurs repetitively, amplifying free radical generation while simultaneously depleting intracellular NADPH stores, leading to:

- Increased production of hydrogen peroxide (H_2O_2)
- Generation of the highly cytotoxic hydroxyl radical (•OH)
- Depletion of reduced glutathione (GSH)
- Failure of endogenous antioxidant defense mechanisms

These phenomena have been demonstrated in both experimental and clinical studies, where paraquat accumulation results in structural damage to pneumocytes, renal tubular cells, and hepatocytes (4,7,8). The oxidative overload provides the mechanistic rationale for the use of antioxidants such as *N*-acetylcysteine or vitamin E; however, clinical evidence supporting their efficacy remains inconsistent (2,6).

B. Genetic Susceptibility

0Several studies have identified genetic variants in antioxidant enzymes that modulate the severity of paraquat-induced injury. Reported polymorphisms include:

- Superoxide dismutase (SOD, V16A)
- Catalase (CAT, C262T)

These enzymes constitute the first line of defense against reactive oxygen species. Genetic dysfunction reduces the capacity to neutralize superoxide radicals and hydrogen peroxide, thereby increasing the risk of pulmonary fibrosis,

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

multiorgan failure, and mortality (17,21). These findings suggest that genetic variability contributes to prognosis and may partially explain the heterogeneity observed in clinical outcomes.

C. Target Organs and Mechanisms of Injury

Paraquat exhibits high affinity for tissues with active polyamine transport systems and high metabolic activity. The primary target organs are the lungs, kidneys, and liver.

➢ *Lung*

The lung is the principal site of toxicity due to active uptake of paraquat by type I and type II pneumocytes via the polyamine transport system (4,7). Pulmonary concentrations may reach levels 10–20 times higher than those in plasma (4). Pulmonary injury occurs in two distinct phases:

- Acute destructive phase: Characterized by necrosis of type I and type II pneumocytes, alveolar edema, diffuse alveolar collapse, infiltration of neutrophils, macrophages, and eosinophils, hemorrhage, and endothelial damage. Destruction of type II pneumocytes impairs surfactant production, increasing surface tension and perpetuating alveolar edema (4,7,8).
- Proliferative phase and pulmonary fibrosis: Characterized by fibroblast proliferation, accelerated collagen deposition, complete loss of alveolar architecture, progression to massive pulmonary fibrosis, and lethal anoxia. Histopathological analyses reveal extensive and progressive fibrosis, resulting in profound structural and functional alteration of the alveolar–capillary unit (2,7,8,22).

➢ *Kidney*

The kidney plays a dual role as both a target organ and a key route of paraquat elimination. Paraquat accumulates in the proximal renal tubules, where it induces:

- Cytoplasmic vacuolization
- Proximal tubular necrosis
- Reduced glomerular filtration rate
- Marked reduction in toxicant excretion

More than 90% of paraquat may be excreted within the first 24 hours if renal function is preserved; however, early tubular injury markedly reduces clearance, creating a vicious cycle of increased accumulation and systemic toxicity (12,23,24).

D. Lipid Peroxidation and Mitochondrial Dysfunction

Lipid peroxidation represents one of the most critical mechanisms of paraquat toxicity. Paraquat interacts with cellular membrane lipids, particularly in pulmonary tissue, where it reduces phospholipid levels such as lecithin and generates reactive aldehydes that disrupt cellular integrity (4,8).

Experimental studies have demonstrated:

- Significant increases in lipid peroxidation products in paraquat-exposed models
- Increased vulnerability in animals deficient in vitamin E or selenium
- Partial protective effects of reducing antioxidants such as vitamin C

Membrane lipid disruption not only damages alveolar structures but also compromises mitochondrial function (7,25,26).

Paraquat interferes with mitochondrial respiratory chain complexes, leading to mitochondrial dysfunction, reduced ATP production, and rapid progression toward apoptosis—particularly in pulmonary and renal cells—through activation of caspases, release of cytochrome *c*, and generation of mitochondrial reactive oxygen species (6,8,25,27).

IV. TOXIC AND LETHAL DOSES

Paraquat poisoning exhibits one of the most aggressive dose-response relationships described in clinical toxicology. Small amounts can induce severe multi-organ damage, and moderate volumes are often fatal even with advanced life support. Rapid gastrointestinal absorption, preferential accumulation in the lungs and kidneys, and massive generation of reactive oxygen species explain the compound's high lethality (4,7,8).

➢ *Toxic Doses*

A toxic dose is defined as one capable of causing clinically relevant damage, even without necessarily compromising survival. Clinical studies and hospital series agree that volumes as low as 5–10 mL of concentrated paraquat solution (20–24%) can produce significant clinical manifestations, especially if the patient seeks medical attention late (7,15).

➢ *Effects Associated with Toxic Doses Include:*

Gastrointestinal irritation, oral ulcers and corrosive esophagitis, early renal tubular damage with rapid elevation of creatinine, increased inflammatory and oxidative markers, and the onset of lung injury due to selective accumulation in pneumocytes.

Prognostic models based on plasma concentrations have shown that even lower doses can exceed mortality thresholds when presentation is late or individual susceptibility factors are present (28–31).

➢ *Lethal Doses*

Several narrative reviews, case series, and multicenter studies indicate that paraquat has one of the lowest lethal doses among agricultural herbicides (4, 6, 8, 15).

Reported clinical ranges: 10–20 mL of concentrated solution is associated with very high mortality in most patients. Ingestions of ≥30 mL are associated with near 100% mortality, even with intensive management and extracorporeal therapies.

Volumes greater than 40–50 mL usually cause fulminant multiorgan failure within 24–48 hours (4,6,8,15).

These values, although approximate, are consistently reproduced in studies from Asia, the Middle East, and Latin America, regardless of the healthcare context (8,13,14). The high mortality rate is explained by the combination of acute destructive alveolitis, accelerated pulmonary fibrosis, proximal tubular necrosis, and oxidative liver damage (7,8,12,32).

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

➢ *Dose-Time Relationship*

The amount ingested alone does not fully explain the outcome. The relationship between the ingested dose and the time to intervention is a fundamental prognostic determinant.

➢ *Key Studies have Demonstrated:*

Late presentation increases mortality even with moderate doses (20,33).Delayed negativity of the urinary dithionite test is associated with a greater systemic burden and mortality (34).

Baseline plasma concentrations accurately predict the risk of death in multiple cohorts (28–31).Early renal dysfunction limits the elimination of the toxin, amplifying its systemic toxicity (23,35).These findings reinforce the need for ultra-early interventions and explain why patients receiving seemingly lower doses can have fatal outcomes when treatment is delayed. Factors that modify toxicity

Interindividual variability can substantially alter the outcome for the same dose. The SOD V16A and CAT C262T polymorphisms have been associated with a worse clinical course, given the decrease in cellular antioxidant capacity (17). Associated clinical conditions such as exposure time, presence of corrosive skin or gastrointestinal lesions, comorbidities, delay in gastrointestinal or skin decontamination (20,36,37). These factors should be considered when evaluating the dose and prognostic risk.

V. CONTROVERSIES IN MANAGEMENT AND THERAPEUTIC ADVANCES

The clinical management of paraquat poisoning remains one of the most controversial areas in medical toxicology. The lack of a specific antidote and the heterogeneity of therapeutic studies have generated numerous debates about the actual efficacy of each intervention. The main areas of controversy and recent therapeutic advances are critically reviewed below.

➢ *Decontamination*

Gastrointestinal decontamination is one of the cornerstones of initial management, and its effectiveness depends strictly on the time elapsed since ingestion. Several studies indicate that the reduction in systemic absorption can be significant when treatment is initiated within the first hour, and its benefit diminishes rapidly as time progresses (33, 38).

➢ *Activated Charcoal*

Activated charcoal remains the most widely used method due to its broad availability and its ability to adsorb paraquat in the intestine (20). Although evidence is limited by the lack of controlled trials, its early use is considered reasonable and low-risk (39).

➢ *Fuller's Earth*

Fuller's earth has demonstrated an even greater adsorption capacity than activated charcoal, particularly at high concentrations of paraquat. Experimental and clinical studies suggest that it may be more effective in reducing gastrointestinal absorption, especially when administered repeatedly within the first few hours (20). However, its availability is limited in many centers, and clinical evidence remains heterogeneous.

Overall, gastrointestinal decontamination represents a low-risk intervention with potential benefit, the impact of which depends strictly on early administration (33, 39).

➢ *Elimination Therapies*

Extracorporeal elimination has been the subject of more studies than any other therapeutic intervention in paraquat poisoning, although substantial debate remains regarding its efficacy.

➢ *Hemoperfusion*

Hemoperfusion with activated charcoal cartridges has shown the ability to remove plasma paraquat in the first few hours after exposure (10,40). Several analyses, including a systematic review, indicate that its clinical benefit is greatest when administered very early, ideally within the first 4 to 6 hours (40). However, its effectiveness decreases rapidly once paraquat is taken up by tissues, especially the lungs (4,8). Meta-analyses agree that hemoperfusion can improve survival only in cases with moderate plasma concentrations and early treatment (10,40), while severe cases show inconsistent results (40).

➢ *Hemodialysis*

Conventional hemodialysis is less effective than hemoperfusion for paraquat removal due to the small molecular size and rapid tissue redistribution of the toxicant (8,10). Despite this, it is used for the management of metabolic and renal complications, rather than for herbicide clearance (10). A recent meta-analysis reported that hemodialysis does not significantly reduce mortality when used alone (41), although some studies describe a possible benefit when combined with early hemoperfusion (40).

➢ *Cyclophosphamide and Methylprednisolone*

The classic immunosuppression regimen with cyclophosphamide + methylprednisolone has been extensively studied, but the results are contradictory. Several studies have not demonstrated a clear reduction in mortality, even when combined with antioxidants or hemoperfusion (2, 27, 42). A recent meta-analysis on pulse immunosuppressive therapy concluded that the evidence is of low quality, with high methodological heterogeneity and no conclusive benefit in mortality (42). Furthermore, some retrospective studies suggest that the benefit may be limited to patients with moderate early respiratory failure, but not to severe or very advanced cases (42).

➢ *Antioxidants: N-acetylcysteine, Vitamin C, and Vitamin E*

N-acetylcysteine has been used to replenish glutathione and mitigate oxidative stress, while vitamins C and E have been proposed to reduce lipid peroxidation (6, 43). Although experimental studies show tissue protection, clinical evidence is limited and contradictory. For example, megadoses of vitamin C were recently re-evaluated without demonstrating conclusive benefits (44).

➢ *Biological and Emerging Therapies*

Strategies using mesenchymal stem cells, both alone and in combination with methylprednisolone, have been explored, showing a reduction in fibrosis and alveolar damage in animal models (45, 46). However, clinical evidence is preliminary,

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

and there are no controlled trials to support its routine implementation.

➢ *Melatonin and PINK1–BNIP3 Mitochondrial Regulation*

Experimental models have shown that melatonin can improve acute lung injury by promoting the expression of PINK1 and BNIP3, modulating the mitophage response, and reducing oxidative stress (26). This pathway is particularly relevant given the intense mitochondrial dysfunction described in paraquat. Despite its favorable pulmonary effects, there is no conclusive clinical evidence of improved mortality (26).

➢ *Modulation of Ferroptosis by NCOA4*

The role of ferroptosis, a form of iron-dependent cell death and lipid peroxidation, has gained attention. Inhibition of iron recycling mediated by NCOA4 has been shown to attenuate paraquat-induced lung injury and slow fibrosing progression (25). However, the data come from experimental studies and have not yet been successfully translated into clinical practice.

➢ *Mesenchymal Stem Cells (MSCs)*

Several preclinical studies report that mesenchymal stem cells, either alone or combined with methylprednisolone, decrease inflammation, alveolar apoptosis, and pulmonary fibrosis (46,47). Although the histological response is promising, the therapy remains experimental and without validation in controlled clinical trials.

➢ *Advanced Antioxidants and Metabolic Compounds*

Drugs such as edaravone, a free radical scavenger, have shown improvement in inflammatory and renal parameters in retrospective studies, without demonstrating a definitive impact on survival (48).

The compound 5-hydroxy-1-methylhydantoin has shown protective effects on lung injury through metabolomic mechanisms, decreasing oxidative markers and lipid peroxidation (21).

➢ *High-Dose ambroxol*

A recent meta-analysis on high-dose ambroxol described initial physiological improvements, but without solid evidence of a mortality benefit (49).

VI. SEQUELAE AND LINK TO CHRONIC DISEASES

Paraquat poisoning in those who survive the acute phase leaves a significant spectrum of systemic sequelae that can persist for months or years.

These complications reflect the pattern of oxidative damage, progressive fibrosis, and multi-organ dysfunction characteristic of this toxin (4,7,8). Available evidence also suggests a possible link between chronic or prolonged exposure and the development of neurodegenerative diseases such as Parkinson's disease (50,51). The main organic sequelae and their clinical relevance are reviewed below.

➢ *Neurological Sequelae*

Several studies have described neurological alterations in survivors of acute paraquat poisoning, especially in cognitive, motor, and sensory domains. A cohort of adults exposed to pesticides, including paraquat, showed manifestations such as attention deficits, impaired processing speed, and affective disorders, suggesting persistent neurotoxicity (52). The pathophysiological basis includes neuronal oxidative stress, mitochondrial dysfunction, and alterations in dopaminergic signaling, mechanisms described in studies exploring the neurotoxicity of paraquat and its relationship to demyelination processes (53,54).

These sequelae can be subtle and progressive, and in some cases related to structural and functional alterations of neuronal pathways specifically vulnerable to oxidative stress (52).

➢ *Pulmonary Sequelae*

The most frequent chronic complication is pulmonary fibrosis, resulting from the proliferative and cicatricial phase that follows acute alveolar damage (7,8). This process, characterized by fibroblast activation and massive collagen deposition, leads to:

Severe pulmonary restriction, progressive decrease in vital capacity, chronic hypoxemia, and exercise intolerance. Some studies have evaluated therapies aimed at slowing fibrosis, such as melatonin, mesenchymal stem cells, or ferroptosis modulators, but although they achieve antifibrotic effects in experimental models, there is no clear evidence of sustained benefit in human survival (25, 26, 47). Residual pulmonary fibrosis can continue to progress for weeks, even in people with apparent initial clinical recovery.

➢ *Renal Sequelae*

The kidney is a major target organ during the acute phase, and proximal tubular injury can leave permanent renal sequelae. Persistent deterioration of glomerular filtration rate, subclinical proteinuria, susceptibility to nephrotoxins, and an increased risk of progression to chronic kidney disease have been documented in various clinical studies (12, 14, 55).

In cases of severe exposure, tubular necrosis can lead to incomplete renal recovery, which represents a risk factor for long-term mortality (35).

VII. CONCLUSIONS

Paraquat poisoning remains one of the most lethal toxicological emergencies, characterized by a pathophysiological pattern dominated by massive oxidative stress, accelerated lung damage, and multiple organ failure. Despite experimental advances in antioxidant and immunosuppressive therapies, as well as strategies aimed at modulating fibrosis or ferroptosis, no intervention has conclusively demonstrated an improvement in human survival. Variability in management, the lack of an antidote, and rapid clinical progression reinforce the need for early interventions and standardized protocols. The body of evidence underscores the urgency of translational research and robust clinical trials to define truly effective interventions.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jan085

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