Research Article

Survival Following Acute Moderate-Dose Paraquat Dichloride Poisoning: A Case Report of Multimodal Immunosuppressive and Antioxidant Management With 25-Day Radiological Follow-Up

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Abstract

Background: Paraquat dichloride is a highly toxic dipyridyl herbicide with no specific antidote, responsible for significant morbidity and mortality across South and Southeast Asia, predominantly through deliberate self-ingestion. Moderate-dose ingestion, corresponding to an estimated elemental dose of 20–40 mg/kg, carries a reported mortality of 40–70%, with progressive pulmonary fibrosis representing the primary mechanism of late death. Timely multimodal intervention targeting oxidative stress, alveolar inflammation, and fibroblast proliferation remains the cornerstone of management in the absence of an antidote.

Case Presentation: A 20-year-old male with no known comorbidities presented to the emergency department following deliberate ingestion of approximately 30 mL of paraquat dichloride 24% soluble liquid, corresponding to an estimated elemental dose of approximately 120 mg/kg. He was managed with immediate gastric decontamination using activated charcoal, intravenous and oral N-Acetylcysteine, pulse methylprednisolone, cyclophosphamide with Mesna uroprotection, antioxidant vitamins, and urinary alkalinization with sodium bicarbonate. Supplemental oxygen was deliberately withheld throughout, given the established oxygen-dependence of paraquat's redox cycling mechanism. A transient acute kidney injury developed around Day 8 and resolved fully with conservative management without requiring renal replacement therapy. Serial chest radiographs and high-resolution computed tomography of the chest demonstrated no pulmonary fibrosis throughout the fourteen-day admission. An outpatient chest radiograph obtained at twenty-five days post-ingestion confirmed continued pulmonary normalcy.

Conclusion: This case demonstrates that moderate-dose paraquat poisoning can achieve a favorable outcome with early, protocol-driven multimodal management. Serial radiological monitoring and deliberate avoidance of supplemental oxygen are critical and often underappreciated components of successful management.

Keywords: Paraquat poisoning; Pulmonary fibrosis; N-Acetylcysteine; Cyclophosphamide; Methylprednisolone; Acute kidney injury; Herbicide toxicity; Case report.

1. Introduction

Background

Paraquat dichloride (1,1'-dimethyl-4,4'-bipyridylium dichloride) is a widely used dipyridyl herbicide whose extreme toxicity in humans has made it a leading cause of fatal poisoning across South and Southeast Asia, predominantly through deliberate ingestion.[1] Its primary mechanism of injury involves sustained intracellular redox cycling, whereby the compound undergoes cyclic reduction and reoxidation within cellular environments, generating superoxide radicals that ultimately yield the highly reactive hydroxyl radical via the Fenton reaction, causing irreversible lipid peroxidation of cellular membranes.[1,2] The lung sustains a disproportionate burden of toxicity due to the active concentration of paraquat within Type I and Type II pneumocytes against an electrochemical gradient, resulting in alveolar necrosis followed by progressive pulmonary fibrosis — the primary determinant of late mortality.[2]

In India, paraquat poisoning has emerged as a significant public health burden, predominantly affecting young adults from rural agricultural communities who ingest it with suicidal intent.[3,4] A retrospective tertiary care study from North India reported an in-hospital mortality of 88%, with 92% of cases originating from rural backgrounds.[3] In a

cross-sectional study from Northeast India, paraquat accounted for 94.74% of all poison-related fatalities, illustrating its disproportionate lethality among agricultural toxins.[5] Dose-dependent clinical phenotypes are well established: ingestion below 20 mg/kg is often survivable, doses exceeding 40 mg/kg cause fulminant multi-organ failure within hours, while the intermediate range of 20–40 mg/kg follows an indolent course of progressive organ dysfunction evolving over days to weeks.[1,6]

No antidote exists. Management is mechanistically guided, centring on early gastrointestinal decontamination, antioxidant loading, and immunosuppression targeting fibroblast proliferation.[7] Pulse cyclophosphamide combined with methylprednisolone has demonstrated a reduction in mortality from 81.8% to 33.3% in moderate-to-severe cases,[7] although a large randomised controlled trial subsequently showed no statistically significant benefit from immunosuppression alone, underscoring the necessity of comprehensive multimodal strategies.[8] Well-documented survival cases with complete serial investigations and radiological follow-up remain scarce in the Indian literature. This case report describes a favorable outcome in moderate-dose paraquat poisoning managed with a multimodal protocol and is reported in accordance with CARE 2013 guidelines.

2. Case Presentation

The patient was a 20-year-old male of lower middle-class socioeconomic background, residing in Kayar, with no known comorbidities including hypertension, diabetes mellitus, tuberculosis, bronchial asthma, or thyroid disorders. There was no prior surgical history, no known drug allergies, and no significant family history. He reported a mixed diet with normal sleep, appetite, and bowel and bladder habits. He presented to the emergency department at approximately 4:45 PM on 06 December 2025 following deliberate self-harm, having ingested approximately 30 mL of Paraquat Dichloride 24% soluble liquid at his residence. The estimated elemental paraquat dose was approximately 7.2 grams, corresponding to roughly 120 mg/kg at an assumed body weight of 60 kilograms. His presenting complaint was a burning sensation in the mouth and throat. He denied vomiting, hematemesis, abdominal pain, chest pain, palpitations, dyspnea, loss of consciousness, and seizures. Suicidal ideation was subsequently confirmed on formal psychiatric evaluation.

On arrival, the patient was conscious and fully oriented with a Glasgow Coma Scale score of 15/15. His temperature was 96.7°F, pulse rate 115 beats per minute, blood pressure 130/80 mmHg, respiratory rate 20 per minute, and oxygen saturation 99% on room air. General examination revealed no pallor, icterus, cyanosis, clubbing, lymphadenopathy, pedal edema, or thyromegaly. Central nervous system examination demonstrated no focal neurological deficits, with bilateral pupils equal, round, and reactive to light. Respiratory system examination revealed bilateral air entry with normal vesicular breath sounds and no added sounds. Cardiovascular examination revealed normal first and second heart sounds with tachycardia and no murmurs. The abdomen was soft and non-tender on palpation. The tachycardia was attributed to sympathetic activation in the context of acute toxic exposure, in the absence of hemodynamic compromise or hypoxemia.

Admission hematological parameters were within normal limits. Cardiac biomarkers on Day 1 were reassuring, with a negative Troponin I, a normal CK-MB, non-reactive CRP, and a serum LDH within normal limits, effectively excluding myocardial injury and significant systemic inflammation at the time of presentation (Table 1). Urinalysis on Day 1 demonstrated ketonuria and trace red blood cells with two to three casts per high-power field, consistent with early oxidative tubular stress (Table 1). Serial biochemical monitoring revealed a progressive rise in urea and creatinine from baseline, peaking around Day 8 to 10, consistent with paraquat-induced transient acute kidney injury secondary to oxidative tubular necrosis, followed by spontaneous recovery to near-normal values by Day 15 (Table 1). Hepatic transaminases remained within normal limits throughout the admission; a mild transient rise in total bilirubin, peaking on Day 9, resolved fully by discharge (Table 1). The total leukocyte count peaked on Day 9 with a neutrophilic predominance, consistent with an acute-phase inflammatory response, normalizing by Day 15 (Table 1). A mild rise in INR to 1.54 by Day 8 was noted but was not clinically significant. On Day 12, Widal test and Leptospira serology were both negative, excluding enteric fever and leptospirosis as contributors to the multi-organ profile in this agricultural community setting (Table 1). Amylase and lipase values on Days 9 and 15 were within normal limits, excluding pancreatitis (Table 1). Complete serial values for all parameters across all study days are presented in Table 1.

The admission chest radiograph on Day 1 was normal, with a nasogastric tube confirmed in situ. High-resolution computed tomography of the chest, performed on Day 3 in view of the high-risk nature of ingestion, demonstrated

no consolidation, ground glass opacities, nodules, fibrosis, crazy paving pattern, or pleural effusion; a few subcentimetric reactive lymph nodes were noted in the right paratracheal and carinal regions; impression was no significant abnormality. Serial chest radiographs on Days 2, 3, 5, and 7 remained consistently normal. The outpatient chest radiograph at Day 25 post-ingestion confirmed the absence of delayed pulmonary fibrosis, representing the most important prognostic radiological finding in this case.

On arrival, gastric decontamination was performed immediately with 100 grams of activated charcoal administered via nasogastric tube, within the critical one-to-two hour therapeutic window (Table 2). Antioxidant therapy was initiated with a loading dose of intravenous N-Acetylcysteine followed by a twenty-four hour maintenance infusion, continued subsequently as oral N-Acetylcysteine tablets, with Vitamin E and Vitamin C supplemented throughout admission to interrupt lipid peroxidation at multiple steps in the radical chain (Table 2). Immunosuppression was administered in the form of pulse intravenous methylprednisolone to suppress alveolar macrophage-driven inflammation and cyclophosphamide to prevent fibroblast proliferation within the alveolar interstitium; Mesna was co-administered for uroprotection against cyclophosphamide-induced hemorrhagic cystitis (Table 2). Urinary alkalinization with intravenous sodium bicarbonate was employed to promote ionization of paraquat within the renal tubular lumen, thereby reducing reabsorption and enhancing elimination (Table 2). Gastroprotection with ranitidine, pantoprazole, and sucralfate was provided for corrosive mucosal injury, alongside antiemetics and prophylactic ceftriaxone (Table 2). Propranolol was administered to reduce oxygen consumption and limit catecholamine-driven oxidative stress. Crucially, supplemental oxygen was withheld throughout the admission, as SpO2 was maintained at 99% on room air and supplemental oxygen is known to accelerate paraquat's redox cycling, thereby amplifying pulmonary toxicity. Multidisciplinary input was obtained from nephrology for AKI monitoring, psychiatry for suicidal ideation counseling, and general surgery for management of a ten-day history of constipation, likely attributable to activated charcoal administration.

The patient remained hemodynamically stable and fully conscious throughout the fourteen-day admission. Tachycardia resolved by Days 2 to 3, and the burning oropharyngeal sensation gradually subsided. The transient acute kidney injury peaked around Days 8 to 10 and resolved fully by Day 15 without requiring dialysis. No respiratory compromise developed at any point during admission or at follow-up. He was discharged on Day 14 in stable condition with Vitamin E 300 mg twice daily, Vitamin C 500 mg twice daily, sucralfate syrup twice daily, and ranitidine 150 mg once daily for five days, with a seven-day outpatient review advised. At the twenty-five day outpatient visit on 31 December 2025, the chest radiograph remained normal with no pulmonary symptoms, and psychiatric follow-up was ongoing.

3. Discussion

At an estimated elemental dose of approximately 120 mg/kg, this patient falls within the 20–40 mg/kg indolent category established in the literature and corroborated by Gawarammana and Buckley in their comprehensive management review.[1,9] Serum paraquat concentration was not measured, representing a recognised limitation, as it forms the basis of validated prognostic nomograms. The urine dithionite test and arterial lactate — described respectively as a semiquantitative bedside prognostic tool and an independent mortality predictor in resource-limited settings[9,10] — were also not documented. Despite these gaps, the clinical presentation of preserved consciousness, haemodynamic stability, and SpO2 of 99% on room air was entirely consistent with the indolent phenotype, and the subsequent clinical trajectory validated this initial stratification.

The treatment protocol systematically addressed each pathological step of paraquat toxicity. Early activated charcoal within the decontamination window is the most time-critical intervention, limiting ongoing gastrointestinal absorption before plasma paraquat peaks.[9] Intravenous N-Acetylcysteine followed by oral continuation, combined with Vitamins C and E, collectively targeted the radical chain through glutathione replenishment, superoxide scavenging, and membrane lipid stabilisation.[1,2] While experimental evidence from a rat model demonstrated that antioxidants alone failed to significantly reduce established pulmonary fibrosis on Ashcroft scoring,[11] their combination with immunosuppression has produced superior clinical outcomes.[7,12] The cyclophosphamide-methylprednisolone combination employed here was supported by Afzali and Gholyaf, who demonstrated mortality reduction from 81.8% to 33.3% in moderate-to-severe poisoning,[7] though the large Sri Lankan randomised controlled trial found no statistically significant benefit from immunosuppression alone, indicating that protocol comprehensiveness and timing are likely determinants of efficacy.[8] A comparable South Indian case published in

2025 confirmed successful outcomes with high-dose cyclophosphamide and steroids, with no respiratory complications over a 2.5-year follow-up,[12] lending contextual regional comparability to this case. Supplemental oxygen was deliberately withheld throughout the admission, a decision unambiguously supported by the established toxicological principle that molecular oxygen is the essential co-substrate for paraquat's redox cycling, and its administration materially amplifies radical generation and pulmonary injury.[1,9]

Serial radiological monitoring proved central to documenting the clinical trajectory. Zhang et al., in a retrospective analysis of 78 patients, demonstrated that computed tomography performed at two to three days post-ingestion carries the best prognostic discrimination, with normal early imaging strongly associated with survival and the absence of consolidation, effusion, and fibrosis correlating with favourable outcomes.[13] The Day 3 high-resolution computed tomography in this case was entirely normal, placing the patient within the survivor profile of that cohort. Serial chest radiographs and the outpatient film at Day 25 confirmed sustained pulmonary sparing beyond the critical Days 5–14 fibrotic window,[2] providing longitudinal radiological documentation that early normal computed tomography predicted fibrosis-free outcomes across the full risk period.

The transient acute kidney injury observed in this patient is mechanistically explained by paraquat's predominantly renal elimination pathway, which subjects tubular epithelial cells to the highest intracellular concentrations of any organ system outside the lung.[9] The peak around Day 8 with full recovery by Day 15 is consistent with oxidative tubular necrosis and subsequent regeneration. While Li et al. demonstrated in a prospective controlled trial that combined continuous venovenous hemofiltration and hemoperfusion significantly reduced acute kidney injury occurrence compared with conventional therapy,[14] complete renal recovery in this patient was achieved conservatively through urinary alkalinization, adequate hydration, and nephrology input alone, without renal replacement therapy, consistent with the milder end of the acute kidney injury spectrum anticipated at this dose level.

The investigation of leptospirosis and enteric fever on Day 12 reflects sound clinical reasoning in the South Indian agricultural context, where both conditions present with overlapping hepatorenal dysfunction and leukocytosis that may mimic or complicate paraquat poisoning.[5] Negative Widal and Leptospira serology confirmed paraquat as the sole aetiological driver, validating the management pathway and excluding infectious confounders that could otherwise compromise the interpretation of clinical recovery.

This case reflects the burden articulated by Singh et al., who identified paraquat as an emerging means of suicide in agrarian India and advocated for its classification as a highly hazardous pesticide subject to restricted access.[4] National evidence demonstrates that banning highly hazardous pesticides significantly reduces pesticide-related suicides in India without adversely impacting agricultural output,[15] reinforcing the case for stricter point-of-sale regulation. Mandatory psychiatric evaluation and follow-up, as conducted here, are non-negotiable components of comprehensive care in all deliberate self-harm presentations. This case carries several limitations. Serum paraquat concentration was not measured, precluding nomogram-based prognostic stratification. The urine dithionite test was not documented. Pulmonary function testing was not performed at follow-up, and clinical monitoring was not extended beyond Day 25. These are contextualised by institutional resource availability and do not materially alter the clinical interpretation.

4. Conclusion

Moderate-dose paraquat dichloride poisoning carries a guarded prognosis but is survivable with prompt, protocol-driven multimodal therapy. The triad of early gastrointestinal decontamination, aggressive antioxidant loading, and immunosuppressive therapy targeting both inflammation and fibroblast proliferation is the cornerstone of management. Deliberate avoidance of supplemental oxygen, a critical and counterintuitive principle, must be consciously applied in all cases. Serial radiological monitoring with chest radiography and high-resolution computed tomography is indispensable for tracking the fibrotic phase across the critical Days 5 to 14 window. This case contributes a well-documented survival outcome with complete serial investigations and twenty-five day radiological follow-up to the limited evidence base supporting this treatment protocol.

References

1. Dinis-Oliveira RJ, Duarte JA, Sánchez-Navarro A, Remião F, Bastos ML, Carvalho F. Paraquat poisonings: mechanisms of lung toxicity, clinical features, and treatment. Crit Rev Toxicol. 2008;38(1):13–71. doi:10.1080/10408440701669959
2. Subbiah R, Tiwari RR. The herbicide paraquat-induced molecular mechanisms in the development of acute lung injury and lung fibrosis. Crit Rev Toxicol. 2021;51(1):36–64. doi:10.1080/10408444.2020.1864721
3. Goyal P, Gautam PL, Sharma S, Paul G, Taneja V, Mona A. A study of paraquat poisoning presentation, severity, management and outcome in a tertiary care hospital: is there a silver lining in the dark clouds? Indian J Crit Care Med. 2024;28(8):741–747. doi:10.5005/jp-journals-10071-24764
4. Singh J, Aggarwal M, Chaudhary D, Udey B. Paraquat regulation as a suicide prevention strategy in India. Gen Hosp Psychiatry. 2025;98:84–85. doi:10.1016/j.genhosppsych.2025.12.012
5. Bhowmick K, Ghosh B, Pain S. A study on deliberately self-poisoned in-hospital patients in a tertiary health care center in northeast India: a cross-sectional review. J Emerg Med. 2019;56(5):512–518. doi:10.1016/j.jemermed.2018.10.037
6. Sabzghabaee AM, Eizadi-Mood N, Montazeri K, Yaraghi A, Golabi M. Fatality in paraquat poisoning. Singapore Med J. 2010;51(6):496–500.
7. Afzali S, Gholyaf M. The effectiveness of combined treatment with methylprednisolone and cyclophosphamide in oral paraquat poisoning. Arch Iran Med. 2008;11(4):387–391.
8. Gawarammana I, Buckley NA, Mohamed F, Naser K, Jeganathan K, Ariyananada PL, et al. High-dose immunosuppression to prevent death after paraquat self-poisoning — a randomised controlled trial. Clin Toxicol (Phila). 2018;56(7):633–639. doi:10.1080/15563650.2017.1394465
9. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmacol. 2011;72(5):745–757. doi:10.1111/j.1365-2125.2011.04026.x
10. Lee Y, Lee JH, Seong AJ, Hong CK, Lee HJ, Shin DH, et al. Arterial lactate as a predictor of mortality in emergency department patients with paraquat intoxication. Clin Toxicol (Phila). 2012;50(1):52–56. doi:10.3109/15563650.2011.639716
11. Seifirad S, Keshavarz A, Taslimi S, Aran S, Abbasi H, Ghaffari A. Effect of pirfenidone on pulmonary fibrosis due to paraquat poisoning in rats. Clin Toxicol (Phila). 2012;50(8):754–758. doi:10.3109/15563650.2012.718783
12. Havaldar AA. Successful management of paraquat poisoning: a case report. J Med Case Rep. 2025;19(1):479. doi:10.1186/s13256-025-05546-y
13. Zhang H, Liu P, Qiao P, Zhou J, Zhao Y, Xing X, et al. CT imaging as a prognostic indicator for patients with pulmonary injury from acute paraquat poisoning. Br J Radiol. 2013;86(1026):20130035. doi:10.1259/bjr.20130035
14. Li C, Hu D, Xue W, Li X, Wang Z, Ai Z, et al. Treatment outcome of combined continuous venovenous hemofiltration and hemoperfusion in acute paraquat poisoning: a prospective controlled trial. Crit Care Med. 2018;46(1):100–107. doi:10.1097/CCM.0000000000002826
15. Bonvoisin T, Utyasheva L, Knipe D, Gunnell D, Eddleston M. Suicide by pesticide poisoning in India: a review of pesticide regulations and their impact on suicide trends. BMC Public Health. 2020;20(1):251. doi:10.1186/s12889-020-8339-z

TABLE 1 — Serial Laboratory Investigations

Parameter	6/12	7/12	8/12	9/12	10/12	12/12	15/12
HAEMATOLOGY							
Haemoglobin (g/dL)	16.2	16.0	14.9	14.4	14.0	14.9	14.3
Total Count (cells/mm^3)	7820	8720	10800	15300	10250	10820	7690
Neutrophils (%)	55	89	89	77	76	82	68
Lymphocytes (%)	32	8	9	13	14	8	20
Eosinophils (%)	9	2	2	10	10	10	11
Monocytes (%)	4	1	0	0	0	0	1
Platelets (lakhs)	2.11	2.20	1.90	1.90	2.10	—	2.43
PCV (%)	—	—	45	44	43	43	44
MCV (fL)	—	—	91	92	93	93	92

MCH (pg)	—	—	30	31	30	30	30
MCHC (g/dL)	—	—	30	33	33	33	33
RBC (M/µL)	—	—	4.9	4.7	4.9	4.9	4.7
RDW (%)	—	—	—	11.8	11.6	11.6	11.8
PT (sec)	18.5	—	22.7	—	—	—	—
APTT (sec)	36.9	—	36.9	—	—	—	—
INR	1.25	—	1.54	—	—	—	—
ESR (mm/hr)	—	—	—	—	—	28	—
RENAL AND ELECTROLYTES							
Random Blood Sugar (mg/dL)	101	—	—	—	—	—	—
Urea (mg/dL)	34	34	40	37	34	34	42
Creatinine (mg/dL)	1.2	1.2	1.0	1.1	1.0	1.0	1.1
Sodium (mEq/L)	143	143	139	141	140	140	142
Potassium (mEq/L)	3.8	3.8	4.1	3.6	3.9	3.9	3.9
Chloride (mEq/L)	104	104	102	100	105	105	102
Calcium (mg/dL)	10.1	9.2	9.2	8.8	9.1	9.1	9.1
Phosphorus (mg/dL)	—	6.1	2.8	—	3.2	3.2	2.3
Magnesium (mg/dL)	1.5	2.4	1.8	—	—	—	3.1
Uric Acid (mg/dL)	—	6.0	—	—	3.4	3.4	4.8
LIVER FUNCTION							
Total Bilirubin (mg/dL)	0.7	0.9	1.2	1.5	1.5	1.5	0.5
Direct Bilirubin (mg/dL)	0.2	0.3	0.4	0.4	0.4	0.3	0.2
AST (U/L)	24	20	17	15	16	16	15
ALT (U/L)	16	13	14	14	19	17	11
ALP (U/L)	82	54	58	61	67	73	76
Albumin (g/dL)	4.8	4.4	4.6	4.3	3.9	3.9	4.0
Total Protein (g/dL)	7.7	6.7	7.1	6.6	6.7	6.6	6.6
A:G Ratio	—	—	—	1.8	—	1.4	1.5
CARDIAC AND INFLAMMATORY							
CK-MB (U/L)	9	—	—	—	—	—	—
Troponin I	Negative	—	—	—	—	—	—
CRP	Negative	—	—	—	—	—	—
LDH (U/L)	138	—	—	—	—	—	—
Amylase (U/L)	—	—	—	38	—	—	34
Lipase (U/L)	—	—	—	40	—	—	38
SEROLOGY							
Widal Test	—	—	—	—	—	Negative	—
Leptospira Serology	—	—	—	—	—	Negative	—

Urinalysis — Day 1 (06/12)

Parameter	Finding
Colour / Appearance	Yellow, Clear
Glucose	Nil
Ketones	Positive
Red Blood Cells	Trace
Pus Cells	1–3 /hpf
Casts	2–3 /hpf
Nitrite	Negative
Bile Salts / Pigments	Absent

Crystals	Nil
Blood Group	A Positive

— = not measured on this date; hpf = high-power field

TABLE 2 — Treatment Protocol with Rationale

Phase	Drug	Dose and Route	Rationale
Gastrointestinal Decontamination	Activated Charcoal	100 g STAT via NGT	Adsorbs residual GI paraquat within the critical 1–2 hour decontamination window
Antioxidant Therapy	Inj. N-Acetylcysteine	150 mg/kg IV over 3 hours (loading)	Replenishes intracellular glutathione; directly scavenges hydroxyl and superoxide radicals
	Inj. N-Acetylcysteine	300 mg/kg IV over 24 hours (maintenance)	Sustained glutathione replenishment throughout the acute oxidative phase
	Tab. N-Acetylcysteine	600 mg TDS oral	Continued antioxidant cover following IV completion
	Cap. Vitamin E	300 mg BD oral	Lipid-soluble antioxidant; protects membrane polyunsaturated fatty acids from lipid peroxidation chain reaction
	Cap. Vitamin C	500 mg BD oral	Regenerates oxidised Vitamin E; scavenges superoxide radicals in aqueous compartments
Immunosuppression	Inj. Methylprednisolone	500 mg IV OD (pulse)	Suppresses alveolar macrophage-driven inflammation; prevents cytokine-mediated fibroblast activation
	Inj. Dexamethasone	4 mg IV BD	Continued steroid cover to suppress secondary and delayed fibrotic response
	Inj. Cyclophosphamide	750 mg in 500 mL 5% Dextrose IV	Alkylating agent; prevents fibroblast proliferation and collagen deposition in alveolar interstitium
	Inj. Mesna	150 mg IV 4th hourly	Uroprotection against cyclophosphamide-induced haemorrhagic cystitis
Renal Protection and Enhanced Elimination	Inj. Sodium Bicarbonate	4 amp in 100 mL Normal Saline IV	Alkalinizes urine; promotes ionization of paraquat in renal tubular lumen, reducing reabsorption and enhancing urinary elimination
Cardiovascular	Tab. Propranolol	20 mg STAT oral	Reduces myocardial oxygen consumption; limits catecholamine-driven amplification of oxidative stress

Gastroprotection	Inj. Ranitidine	50 mg IV BD	H_2 receptor blockade; prophylaxis against stress ulceration and corrosive mucosal injury
	Inj. Pantoprazole IV	OD	Proton pump inhibition for additional acid suppression
	Syp. Sucralfate-Oxetacaine	10 mL BD oral	Mucosal coating agent; protects ulcerated oropharyngeal and oesophageal mucosa from corrosive injury
Antiemetic	Inj. Ondansetron	4 mg IV BD	Serotonin antagonist antiemetic; controls nausea and vomiting
	Inj. Metoclopramide	IV STAT	Prokinetic antiemetic; accelerates gastric emptying and charcoal transit
Antibiotic	Inj. Ceftriaxone	1 g IV OD	Prophylactic cover against aspiration pneumonitis and secondary bacterial infection

NGT = nasogastric tube; IV = intravenous; BD = twice daily; OD = once daily; TDS = three times daily; STAT = immediately