



Case Report

Multimodal Management of Moderate-to-Severe Paraquat Poisoning in a Patient with Uncontrolled Type 2 Diabetes: A Case Report.

	Abstract: <i>Background:</i> Paraquat poisoning carries a mortality rate of 60 to 90% and has no specific antidote, with outcomes driven largely by ingested dose and the speed of intervention. Management in comorbid patients, and in settings without access to plasma paraquat assays, remains poorly described. <i>Case Presentation:</i> We describe a 45-year-old man with uncontrolled Type 2 Diabetes Mellitus (HbA1c 11.7%) who ingested approximately 34 mg/kg of paraquat ion, placing him in the moderate-to-severe toxicity range by the Proudfoot classification. Plasma paraquat concentration was unavailable, so risk stratification relied on clinical and laboratory parameters, including metabolic acidosis, 3+ proteinuria, and hyperglycemia. He was treated with a four-pillar multimodal protocol: gastrointestinal decontamination with activated charcoal, two cycles of charcoal hemoperfusion at six and sixteen hours post-ingestion, immunosuppression with dose-reduced cyclophosphamide and dexamethasone, and antioxidant support with N-acetylcysteine. Supplemental oxygen was withheld given saturations above 97%, and steroid-induced hyperglycemia superimposed on pre-existing uncontrolled diabetes was managed with a continuous insulin infusion. The patient survived without progression to pulmonary fibrosis or significant renal impairment. <i>Conclusion:</i> This case demonstrates that early, individualized multimodal therapy can achieve survival in moderate-to-severe paraquat poisoning even when plasma paraquat measurement is unavailable and diabetes complicates immunosuppressive dosing, supporting the feasibility of protocol-driven management in resource-limited settings.
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INTRODUCTION

Paraquat is a widely used non selective contact herbicide that remains highly lethal to humans upon ingestion. The mortality rate for paraquat poisoning is estimated at 60 to 90%, and despite decades of research, no specific antidote has been identified^[1,3]. The clinical effects and prognosis are dose dependent: ingestion of less than 20 mg/kg of paraquat ion is associated with mild toxicity and likely recovery; 20 to 40 mg/kg causes moderate to severe poisoning with renal dysfunction, mucosal damage, and progressive pulmonary fibrosis; and doses exceeding 40 mg/kg result in fulminant multi-organ failure and death within hours to days^[1,2].

The pathophysiology of paraquat toxicity is driven by redox cycling, in which paraquat is reduced by cellular NADPH cytochrome P450 reductase to form a paraquat radical that reacts with molecular oxygen to generate

superoxide anions. This leads to lipid peroxidation, mitochondrial damage, NF-κB activation, and cellular death, particularly in the lungs, kidneys, and liver^[1,3]. Paraquat is actively concentrated in lung tissue via the polyamine uptake system, where it reaches concentrations 10-15 times higher than plasma, resulting in pneumonitis and irreversible pulmonary fibrosis in a condition known as “paraquat lung”^[3].

Current management strategies focus on four pillars: limiting gastrointestinal absorption, enhancing elimination through extracorporeal therapies, suppressing the inflammatory cascade with immunosuppressive agents, and providing antioxidant support.

We present the case of a 45 year old male with uncontrolled Type 2 Diabetes Mellitus who survived

moderate to severe paraquat poisoning through early multimodal intervention. This case is notable for the challenge of managing steroid induced hyperglycemia superimposed on pre existing uncontrolled diabetes, and for the use of a comprehensive treatment protocol in a resource limited setting where plasma paraquat concentration measurement was unavailable.

CASE PRESENTATION

A 45 year old male with a two year history of Type 2 Diabetes Mellitus, managed with oral hypoglycemic agents despite reportedly poor compliance, presented to the casualty department after intentionally consuming approximately 10 mL of 24% paraquat dichloride roughly three hours earlier. He had no prior psychiatric history and no history of hypertension, cardiac disease, renal disease, or prior hospitalizations.

On arrival he was conscious and oriented, with a Glasgow Coma Scale score of 15. He was hemodynamically stable but hypertensive, with a blood pressure of 180/90 mmHg, and tachycardic at 113 beats per minute; oxygen saturation was 98% on room air. Notably, he did not report the typical early symptoms of vomiting, abdominal pain, or breathlessness, and oral examination revealed no significant caustic mucosal injury at initial assessment; chest and abdominal examinations were unremarkable.

A Ryle's tube was inserted, 50 g of activated charcoal was administered, and dexamethasone 4 mg was given intravenously, while the patient was kept nil per oral and started on intravenous normal saline at 125 cc/hr. Within roughly fifteen minutes, a loading dose of N-acetylcysteine (150 mg/kg IV) was begun over one hour. At about 1.5 hours after arrival, reassessment showed a blood pressure of 170/80 mmHg and a pulse of 106 bpm, prompting admission to the Medical Intensive Care Unit and a nephrology consultation. On MICU arrival his vitals were a heart rate of 109 bpm, blood pressure of 180/100 mmHg, and oxygen saturation of 97% on room air, with chest and abdominal examinations again normal.

Roughly six hours after ingestion, the first cycle of charcoal hemoperfusion was performed over four hours, with a 2000 unit intravenous heparin bolus for anticoagulation. This was followed by a further 40 g dose of activated charcoal via Ryle's tube and another 4 mg dose of intravenous dexamethasone. Immediately after this first hemoperfusion cycle, the patient received cyclophosphamide 500 mg IV with mesna 200 mg IV given both before and after it, a continuous 24-hour infusion of N-acetylcysteine (900 mg), cefoperazone-sulbactam 1.5 g IV, pantoprazole IV, and ondansetron IV, while dexamethasone 4 mg IV was continued three times daily and an insulin infusion was initiated for glycemic control. A second cycle of charcoal hemoperfusion followed at approximately sixteen hours post-ingestion.

Initial laboratory studies showed a hemoglobin of 17.3 g/dL, sodium 138 mEq/L, potassium 3.73 mEq/L, and an INR of 1.1. Glycemic parameters were markedly deranged, with a random blood glucose of 362 mg/dL and an HbA1c of 11.7%. Arterial blood gas analysis showed a pH of 7.3, pCO₂ of 37.6 mmHg, bicarbonate of 19.8 mmol/L, and a lactate of 3.02 mmol/L, consistent with a primary metabolic acidosis with appropriate respiratory compensation. Renal function was preserved at presentation, with a creatinine of 0.89 mg/dL and blood urea of 15 mg/dL, while liver enzymes were mildly elevated (SGOT 78 U/L, SGPT 73 U/L). Urinalysis revealed 3+ proteinuria, 1+ glycosuria, and 2+ ketonuria, with a protein creatinine ratio of 1.65. Chest radiography was normal, and electrocardiography and two dimensional echocardiography showed concentric left ventricular hypertrophy with a preserved ejection fraction of 60%.

The ingested dose of 10 mL of 24% paraquat dichloride equates to approximately 2.4 g of active paraquat ion; given the patient's documented body weight of 70 kg, this corresponds to an estimated dose of roughly 34 mg/kg, placing him in the moderate-to-severe toxicity range by the Proudfoot classification (20-40 mg/kg), a category associated with renal and hepatic dysfunction, progressive pulmonary fibrosis, and death typically occurring two to three weeks post-ingestion^[1,2,4]. Plasma paraquat concentration was not available at our facility, precluding calculation of the Severity Index of Paraquat Poisoning, which is derived from the product of plasma paraquat concentration and time since ingestion, with values above 10 associated with high mortality^[4].

The urine dithionite test, a bedside qualitative assay that offers prognostic information based on time to a negative result, was also not performed^[7]. In the absence of these tools, risk stratification relied on clinical and laboratory parameters; recent evidence identifies the amount ingested, absolute neutrophil count, urine protein concentration, serum bicarbonate, serum creatinine, and blood glucose as independent prognostic factors for mortality, with a composite index achieving an area under the curve of 0.921^[8]. In this patient, several adverse prognostic indicators were present, including metabolic acidosis, significant hyperglycemia, 3+ proteinuria, and elevated lactate, while relatively preserved renal function and a normal chest radiograph at admission were reassuring^[8,9]. The principal diagnostic challenge in this case was the inability to measure plasma paraquat concentration, the gold standard prognostic tool, a limitation common in resource limited settings in India that necessitated reliance on clinical and laboratory surrogates.

The overall management strategy rested on four pillars: gastrointestinal decontamination, extracorporeal toxin removal, immunosuppression, and antioxidant therapy.

Gastric lavage was deliberately avoided, consistent with current recommendations against lavage in paraquat poisoning given the risk of worsening caustic gastrointestinal injury^[3]. Activated charcoal, one of the recommended adsorbents alongside Fuller's earth, was instead given in the two doses described above to limit further systemic absorption^[3].

For extracorporeal elimination, two cycles of charcoal hemoperfusion were performed, the first within approximately six hours of ingestion and the second at roughly sixteen hours, reflecting paraquat's multi-compartmental pharmacokinetics: the first session was intended to clear the initial plasma peak, and the second to address the anticipated rebound in plasma concentration as the toxin redistributed from peripheral tissues back into the vascular compartment^[10]. A comprehensive review of extracorporeal therapies concluded that early and repeated hemoperfusion, particularly within four to six hours of ingestion, improves survival by directly adsorbing paraquat,^[10] and a large retrospective study of 621 patients identified hemoperfusion within four hours of exposure, with a second treatment within twenty hours, as an independent predictor of survival^[11].

To prevent progression from alveolitis to irreversible pulmonary fibrosis, an immunosuppressive regimen of cyclophosphamide 500 mg IV, given post hemoperfusion to suppress fibroblast and inflammatory cell proliferation, and dexamethasone 4 mg IV three times daily (12 mg/day), to stabilize lysosomal membranes and inhibit neutrophil and macrophage recruitment to lung tissue, was employed; mesna 200 mg IV was given before and after cyclophosphamide to neutralize its toxic metabolite acrolein and prevent hemorrhagic cystitis. The cyclophosphamide dose used here, approximately 7 mg/kg, was notably lower than standard published protocols, which typically use 15 mg/kg/day for two days combined with methylprednisolone 1 g/day for three days^[12]. This dose modification, and the omission of methylprednisolone, were pragmatic decisions made in light of the patient's uncontrolled diabetes (HbA1c 11.7%), given the significant risk of infection and further metabolic decompensation that more intensive immunosuppression could pose. A nationwide Taiwanese study of 1,811 patients found that the combination of methylprednisolone, cyclophosphamide, and daily dexamethasone was associated with the highest survival rate of the regimens studied (48%, $P < 0.001$)^[13].

Antioxidant support was provided with N-acetylcysteine, given as a 150 mg/kg intravenous loading dose over one hour beginning at 3:10 a.m., followed by a continuous infusion of 900 mg over 24 hours; NAC acts as a precursor to glutathione, augmenting the cell's natural antioxidant defenses and directly scavenging reactive oxygen species^[3].

Supplemental oxygen was deliberately withheld throughout the admission, as the patient's oxygen saturation remained above 97% on room air. This approach was informed by a multi center retrospective cohort study of 416 patients showing that liberal oxygen therapy, administered before saturation fell below 90%, was associated with significantly increased mortality (adjusted OR 5.97, 95% CI 1.692–21.049),^[14].

Supportive care included intravenous fluid resuscitation with normal saline at 125 cc/hr to maintain renal perfusion and promote urinary excretion of paraquat,^[7] a continuous insulin infusion to manage glycemia in the setting of uncontrolled Type 2 Diabetes exacerbated by high dose dexamethasone, cefoperazone 1000 mg with sulbactam 500 mg IV for infection prophylaxis, pantoprazole IV for stress ulcer prophylaxis, and ondansetron IV for antiemetic cover; the patient was kept nil per os for 48 hours.

DISCUSSION

This case illustrates the successful management of acute paraquat poisoning in a patient with comorbid uncontrolled Type 2 Diabetes Mellitus using a multimodal approach. The estimated ingested dose of approximately 34 mg/kg placed the patient in the moderate to severe toxicity category by the Proudfoot classification, a range associated with progressive pulmonary fibrosis and mortality typically occurring two to three weeks post-ingestion^[2,4].

Accurate prognostic assessment is essential in paraquat poisoning to guide treatment intensity and clinical decision making. The most validated prognostic tools include the Proudfoot survival curves, the Severity Index of Paraquat Poisoning, and the plasma or urine dithionite tests^[4,7]. The principal prognostic factor identified in clinical data is the time required to achieve a negative urine dithionite test, and survival is reported to be 100% when the area affected by ground-glass opacity is less than 20% of total lung volume on high-resolution CT imaging seven days post-ingestion^[7]. Neither plasma paraquat concentration nor the urine dithionite test was available in this case, a common limitation in resource-limited settings. However, recent evidence demonstrates that a composite index incorporating routine laboratory parameters, amount ingested, neutrophil count, urine protein, serum bicarbonate, creatinine, and blood glucose provides an area under the curve of 0.921 for predicting mortality, offering a practical alternative when plasma paraquat levels are unavailable^[8].

Gastric lavage was deliberately avoided in this case, consistent with the recommendation of Gawarammana and Buckley that lavage should not be performed in paraquat poisoning due to the risk of exacerbating caustic mucosal injury^[3]. Activated charcoal was given instead, in two doses totaling 90 g, to adsorb residual paraquat in the gastrointestinal tract; activated charcoal

and Fuller's earth remain the recommended adsorbents^[3].

The cornerstone of the elimination strategy was rapid initiation of charcoal hemoperfusion. Paraquat follows a multi-compartmental pharmacokinetic model, remaining in plasma only briefly before being actively transported into the lungs via the polyamine uptake system^[3]. A comprehensive review of extracorporeal therapies concluded that early and repeated hemoperfusion, particularly within four to six hours of ingestion, improves survival, and that combining hemoperfusion with continuous renal replacement therapy produces synergistic effects^[10]. A large retrospective study of 621 patients similarly identified hemoperfusion within four hours of exposure, with a second treatment within twenty hours, as an independent predictor of survival^[11]. The evidence is nonetheless not unequivocal: Gawarammana and Buckley concluded that elimination methods such as hemodialysis and hemoperfusion are unlikely to change the clinical course, a discrepancy that underscores the need for further randomized controlled trials to define the role of extracorporeal therapies in paraquat poisoning^[3].

The secondary phase of paraquat poisoning is characterized by an intense inflammatory response in the alveolar spaces that matures into irreversible pulmonary fibrosis,^[1] and an immunosuppressive regimen of cyclophosphamide and dexamethasone was used to try to prevent this progression. The evidence for immunosuppressive therapy remains of low certainty: the 2021 Cochrane systematic review concluded that glucocorticoid with cyclophosphamide may have a small effect on in-hospital mortality but little or no effect at three months post-discharge, and a randomized controlled trial of 299 patients in Sri Lanka found no significant difference in in-hospital mortality between immunosuppression and placebo groups (53% versus 62%, $p = 0.12$)^[1,12]. By contrast, a nationwide Taiwanese study of 1,811 patients found that adding immunosuppressive treatment to hemoperfusion significantly increased survival, from 24.3% to 29.3% ($P < 0.001$), with the combination of methylprednisolone, cyclophosphamide, and daily dexamethasone associated with the highest survival rate of 48% ($P < 0.001$)^[13]. The absence of methylprednisolone from the regimen used in this case is a notable deviation from the most extensively studied protocols; this decision reflected concern that the additional hyperglycemic burden of methylprednisolone (1 g/day for three days) posed an unacceptable risk of metabolic decompensation given the patient's uncontrolled diabetes.

N-acetylcysteine was chosen for its established antioxidant mechanisms: it acts as a glutathione precursor and a direct scavenger of reactive oxygen species, and may also inhibit NF- κ B activation^[3]. A prior case report of survival after massive paraquat

ingestion used a higher maintenance dose of 300 mg/kg/day by continuous infusion for three weeks;^[15] while no published human trials have established an optimal NAC dosing protocol in paraquat poisoning, the rationale for its use here is well supported mechanistically.

A distinctive and clinically significant feature of this case was the patient's uncontrolled Type 2 Diabetes, with an HbA1c of 11.7% at presentation. High-dose dexamethasone is known to cause substantial glycemic excursions, which were managed with a continuous insulin infusion; this dual challenge, treating paraquat toxicity while simultaneously managing steroid-induced hyperglycemia superimposed on pre-existing uncontrolled diabetes, required careful balancing of therapeutic priorities throughout the admission. Hyperglycemia itself can exacerbate oxidative stress and impair tissue repair, potentially worsening paraquat induced organ damage, and blood glucose has independently been identified as a prognostic factor in paraquat poisoning, further underscoring the importance of strict glycemic control in this setting^[8]. Paraquat itself may contribute to hyperglycemia through stress-mediated cortisol and catecholamine release and suppression of insulin secretion, as demonstrated in animal models,^[16] and the glycosuria and ketonuria present on admission indicated a degree of metabolic decompensation requiring simultaneous stabilization alongside toxin-specific treatment.

Paraquat is primarily excreted unchanged in the urine, and while the kidneys are the principal route of elimination, they are also a target of toxicity, often resulting in acute tubular necrosis^[3]. Acute kidney injury complicates approximately 50% of paraquat poisonings, with serum creatinine typically peaking around five days post-ingestion and usually normalizing within three weeks^[7]. The 3+ proteinuria observed in this patient was an early marker of renal strain, and maintaining high volume intravenous fluid resuscitation was considered vital to ensure adequate renal perfusion and reduce the contact time of paraquat with the tubular epithelium.

Finally, supplemental oxygen was deliberately withheld throughout management. A multi-center retrospective cohort study of 416 patients demonstrated that liberal oxygen therapy was associated with significantly increased overall mortality (adjusted OR 5.97, 95% CI 1.692–21.049). Current recommendations advise withholding supplemental oxygen unless oxygen saturation falls below 90%^[14].

CONCLUSIONS

This case demonstrates the successful management of acute paraquat poisoning in a patient with comorbid uncontrolled Type 2 Diabetes Mellitus. The estimated ingested dose of approximately 34 mg/kg placed the patient in the moderate to severe toxicity category with

a high risk of mortality. A multimodal approach incorporating early activated charcoal administration, two cycles of charcoal hemoperfusion at six and sixteen hours post ingestion, immunosuppressive therapy with cyclophosphamide and dexamethasone, and antioxidant support with N acetylcysteine was employed. The deliberate avoidance of supplemental oxygen and the careful management of steroid-induced hyperglycemia with continuous insulin infusion were critical components of care. This case underscores the importance of early prognostic stratification, rapid initiation of multimodal therapy, and individualized management when comorbidities such as uncontrolled diabetes complicate the clinical picture. It also highlights the feasibility of effective management in resource-limited settings where plasma paraquat concentration measurement is unavailable, using clinical and routine laboratory parameters for prognostic assessment.

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