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PARAQUAT POISONING: A CASE REPORT AND REVIEW OF PATHOPHYSIOLOGY, CLINICAL SPECTRUM, AND CURRENT MANAGEMENT APPROACHES

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Abstract
Paraquat poisoning remains a life-threatening emergency, often leading to respiratory failure due to pulmonary fibrosis. We present a case of an 18-year-old pregnant woman who ingested paraquat and developed progressive respiratory distress despite intensive therapy. Paraquat induces oxidative stress and fibrosis, with management centered on early decontamination, antioxidant therapy, and immunosuppression. Novel treatments such as edaravone and extracorporeal membrane oxygenation (ECMO) may improve organ protection and allow oxygen-sparing ventilation, though survival remains poor. Prevention through strict regulation and awareness continues to be the most effective strategy against paraquat-related mortality.

Keywords Paraquat poisoning; Pulmonary fibrosis; Oxidative stress; Hemoperfusion; ECMO

INTRODUCTION

Paraquat ingestion results in multi-organ failure, with the lungs being the primary target due to active uptake of the compound by alveolar cells [3]. The absence of a specific antidote and poor outcomes despite aggressive treatment underline the need for continued case-based understanding [4]. Although supportive care, decontamination, hemoperfusion, immunosuppression, and antioxidant therapy are commonly employed, a definitive antidote is lacking and clinical outcomes remain poor [5]. Because paraquat is inexpensive and highly effective, its unregulated use in developing countries like India has contributed to its continued prevalence [6]. Here, we present a fatal case of paraquat poisoning in early pregnancy and briefly review current pathophysiology and management considerations.

CASE PRESENTATION

An 18-year-old, four-month pregnant female presented with an alleged history of ingestion of approximately 5 mL of 24% paraquat dichloride. She initially received gastric lavage at a private

hospital but was discharged after 24 hours.

Five days later, she presented to our emergency department with progressively worsening breathlessness, dry cough, and bilateral flank pain. The patient reported no vomiting or diarrhea but complained of generalized weakness and myalgia.

On admission, her vital signs showed blood pressure 108/66 mmHg, pulse rate 104/min, respiratory rate 30/min, and SpO_2 88% on room air. Chest auscultation revealed bilateral coarse crackles, predominantly in the lower zones.

Laboratory investigations showed a rising trend in serum urea (58 → 136 mg/dL) and creatinine (1.2 → 3.4 mg/dL) over three days, with adequate urine output. Haemoglobin was 9.6 g/dL, with microcytic hypochromic anaemia. HRCT thorax revealed bilateral ground-glass opacities (Figure 1), patchy peripheral consolidations, and reticulonodular opacities in the right upper lobe, consistent with aspiration pneumonitis and evolving pulmonary fibrosis (Figure 2 & 3). Ultrasound of the abdomen was normal, and obstetric ultrasonography confirmed a single viable intrauterine pregnancy of 14 weeks with a low-lying placenta.

The patient underwent three sessions of hemodialysis due to rising azotemia, resulting in partial biochemical improvement. Despite meticulous oxygen titration and a lung-protective ventilation strategy, her respiratory function worsened, with escalating oxygen requirements and deteriorating compliance. During the ICU course, intrauterine fetal demise occurred. The patient subsequently developed refractory hypoxemia and multi-organ failure and expired despite maximal supportive measures.

Representative HRCT chest images are shown below:

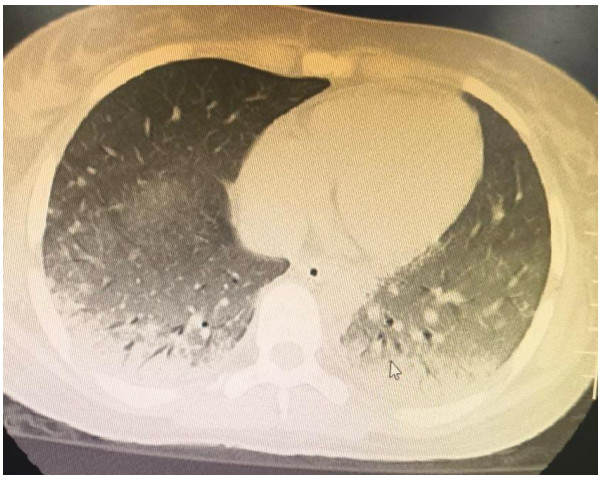

Figure 1: HRCT thorax showing bilateral ground-glass opacities

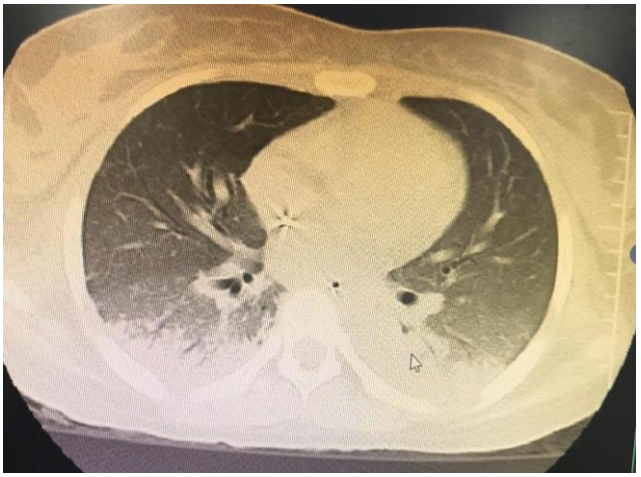

Figure 2: HRCT thorax showing patchy peripheral consolidations

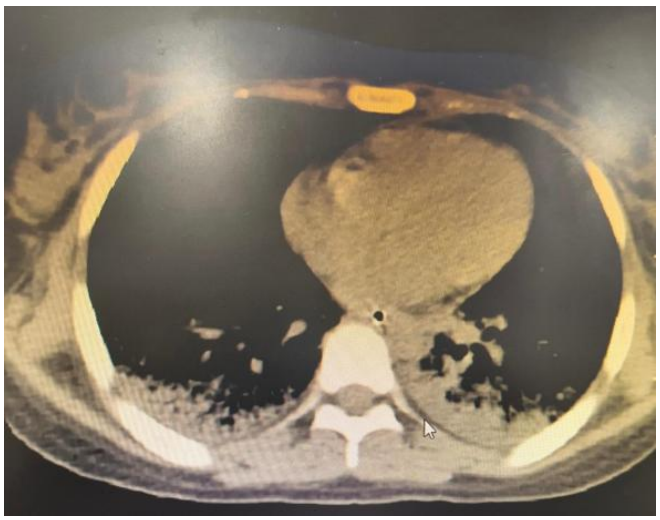

Figure 3: HRCT thorax showing reticulonodular changes and fibrosis

DISCUSSION

Paraquat is an effective, broad-spectrum, and highly poisonous water-soluble quaternary ammonium herbicide. The pathophysiology of paraquat toxicity primarily involves the generation of reactive oxygen species (ROS), leading to lipid peroxidation, mitochondrial damage, and extensive cellular necrosis [7]. The lungs are particularly susceptible due to energy-dependent paraquat uptake, resulting in alveolitis and subsequent pulmonary fibrosis. Renal and hepatic injuries frequently accompany respiratory compromise. High-dose corticosteroids and cyclophosphamide are widely used, though evidence of efficacy remains weak [7]. Hemoperfusion and hemofiltration may be beneficial when initiated within four hours of ingestion. Novel agents such as edaravone have shown renal and hepatic protective effects in retrospective analyses. Management focuses on airway support, limiting oxygen exposure, and antioxidant therapy with N-acetylcysteine and vitamin C. Extracorporeal membrane oxygenation (ECMO) and lung transplantation have been reported as rescue measures in isolated cases [8].

In the present case, the patient initially received gastric lavage at a private hospital. Gastric lavage should ideally be performed within two hours of ingestion to minimize further systemic absorption of paraquat. Because paraquat is rapidly absorbed and distributed to most tissues, particularly the lungs and kidneys, decontamination becomes less effective once absorption has occurred, underscoring the importance of early intervention [9].

Clinically, paraquat ingestion commonly produces inflammation and ulceration of the oral mucosa, corrosive gastrointestinal injury, renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis, as previously reported by Kondle Raghu et al. [6]. Trakulsrichai S. et al. also noted that most patients present with predominant gastrointestinal symptoms [10]. In the current case, breathlessness, dry cough, and bilateral flank pain were the early presenting symptoms, later progressing to respiratory failure. Despite hemodialysis sessions providing partial biochemical improvement, the patient's pulmonary status worsened, ultimately leading to death.

Similar poor outcomes have been described by Havaldar A.A. [2] and Pathak V. et al. [11], who documented fatal cases despite aggressive therapy. Although a few successful outcomes have been reported with high-dose cyclophosphamide and corticosteroid therapy, consistent benefit remains uncertain. The timing of hemodialysis or hemoperfusion appears critical, with the greatest benefit observed when initiated within four to six hours of ingestion; delayed therapy, as in this case, may have limited impact once tissue distribution is complete.

Pregnancy adds an additional layer of complexity because of potential fetoplacental transfer of paraquat and the physiological constraints of gestation on hemodynamics and ventilation [12]. Literature on paraquat poisoning during pregnancy is limited, but fetal loss has been documented in severe maternal poisoning, as reported by Chen J. et al. [13] and Trakulsrichai S. et al. [10]. Our case

similarly demonstrated intrauterine fetal demise in the setting of refractory maternal hypoxemia.
Prognostication tools such as the Acute Physiology and Chronic Health Evaluation II (APACHE II) score have shown utility in predicting mortality among paraquat poisoning cases and may aid clinicians in guiding goals-of-care discussions in advanced disease [12]. Overall, the present case aligns with previous reports of poor outcomes following delayed presentation, underscoring the critical importance of primary prevention, rapid diagnosis, early risk stratification, and prompt initiation of evidence-based interventions. Early recognition, timely decontamination, and aggressive multidisciplinary management remain key to improving outcomes, but prevention through regulation, restriction, and public awareness is paramount.

MANAGEMENT AND OUTCOME

Initial stabilization involves airway protection, oxygen titration, and early gastrointestinal decontamination with activated charcoal or Fuller's earth when ingestion is recent. Hemoperfusion (HP) [14] is most effective within four hours of ingestion and can be combined with continuous kidney replacement therapy (CVVH/CVVHD) for solute clearance and acid–base management [15]. Immunosuppressive therapy with corticosteroids and cyclophosphamide aims to attenuate inflammation and fibroproliferation. Antioxidant therapy with N-acetylcysteine helps replenish glutathione reserves, while vitamins C and E act as additional free radical scavengers [7].

RECENT ADVANCES

Lung-protective ventilation (tidal volume 4–6 mL/kg predicted body weight, plateau pressure ≤30 cm H_2O) and oxygen restriction remain central to avoid oxygen-exacerbated lung injury. In cases of refractory hypoxemia, adjunctive measures such as prone positioning, conservative fluid management, and neuromuscular blockade may be employed. In the index case, despite aggressive management, progressive pulmonary fibrosis led to respiratory failure requiring mechanical ventilation, ultimately proving fatal [16].Edaravone is a small-molecule free radical scavenger that donates electrons to neutralize hydroxyl radicals and other reactive oxygen species (ROS). It interrupts lipid peroxidation chains, inhibits NF-κB activation and downstream cytokine release [17], and stabilizes endothelial integrity, thereby potentially reducing interstitial edema.
Retrospective data in paraquat poisoning suggest improvements in renal and hepatic biomarkers and delayed fibrosis; however, a consistent survival benefit has not yet been demonstrated. Extracorporeal membrane oxygenation (ECMO), particularly the veno-venous mode for respiratory failure, provides extracorporeal gas exchange that allows ultra-protective ventilation with very low FiO_2, reducing oxygen-augmented ROS injury [17]. ECMO can serve as a bridge to recovery or lung transplantation in select cases, though its risks and resource demands are considerable. Other investigational approaches include hemadsorption cartridges, targeted antifibrotic therapies such as pirfenidone and nintedanib, and the development of novel prognostic biomarkers [17].

CONCLUSION

Paraquat poisoning remains a formidable clinical challenge with limited therapeutic options and high mortality. Its pathophysiology involves oxidative injury, alveolar destruction, and progressive pulmonary fibrosis. Early decontamination, cautious ventilatory strategies, and timely initiation of hemoperfusion or immunosuppression may offer modest survival benefits. The introduction of novel agents such as edaravone and advanced supportive modalities like ECMO provides emerging hope for organ preservation and oxygen-sparing support. However, prevention through strict regulation, public education, and safe herbicide practices remains the most effective strategy. Future research should focus on antifibrotic therapies and the development of specific antidotes to alter the natural course of this lethal poisoning.

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