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(Case Report)

Fatal Paraquat-Induced Pulmonary Fibrosis in a Young Adult: A Case Report

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Abstract

Paraquat is a highly toxic herbicide that can cause fatal pulmonary fibrosis even with small ingestions. We report a 35-year-old male with a history of depression and previous suicide attempt who presented with gastrointestinal symptoms after ingesting 20 mL of concentrated Paraquat. Despite intensive care measures including hemodialysis, corticosteroids, and supportive therapy, he developed progressive respiratory failure due to worsening pulmonary fibrosis and died on hospital day 51. This case underscores the severity of Paraquat poisoning and the limited effectiveness of available treatments, highlighting the importance of early recognition, the need for novel biomarkers denoting severity, and the combination of supportive care with extracorporeal detoxification modalities in managing such cases.

Keywords: Paraquat Poisoning; Pulmonary Fibrosis; Acute Kidney Injury; Respiratory Failure

1. Introduction

Paraquat is a bipyridyl herbicide widely used for weed control in many countries, despite its well-known human toxicity. Ingestion of even small amounts can cause multiorgan failure, with pulmonary fibrosis being the principal cause of delayed mortality. The compound generates reactive oxygen species, leading to lipid peroxidation, NF-κB activation, mitochondrial dysfunction, and apoptosis in multiple organs [1]. Management of paraquat poisoning remains largely supportive and focuses on modifying the toxicokinetics of the poison [2].

Although rare, fatal paraquat poisoning provides important clinical and educational insights. Reporting this case highlights the mechanisms of paraquat-induced lung injury, the clinical course of poisoning, and the challenges in management, particularly regarding oxygen therapy and antioxidant strategies. Sharing such cases helps clinicians recognize early warning signs, anticipate complications, and understand the limitations of current interventions, contributing to improved awareness and potentially guiding future therapeutic strategies.

Here, we present a fatal case of paraquat poisoning in a young adult, emphasizing the pathophysiological challenges and limited therapeutic options.

2. Case presentation

A 35-year-old male, with a history of depression, and a previous suicidal attempt (2019 by organophosphate ingestion) presented to our Emergency Department (ED) complaining of intractable vomiting, watery diarrhea after ingestion of a non-specific amount of a presumed pesticide few days ago. His home medications included a combination of Valproic acid, Quetiapine, and Mirtazapine which he stopped taking one month ago.

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In addition to his chief complaint, the patient also reported bronchorrhea, sialorrhea, and decreased per os (PO) intake due to odynophagia after his intoxication.

On physical exam, the patient was lethargic but oriented in time, place, and person, he was afebrile with a pulse rate of 84 bpm, and a blood pressure of 100/70 mmHg. He had red oropharyngeal mucosa, excessive secretions and dorsal glossal lesions (Figure 1), and slow skin recoil. His chest auscultation revealed good bilateral air entry with bilateral scattered rhonchi and a respiratory rate of 20 Breaths per min. His oxygen saturation was 98% on room air. The remainder of his physical exam was unremarkable.

In ED, our patient was started on vigorous IV hydration and received three doses of 3 mg of atropine every 5 min as management for presumed organophosphate poisoning in agreement with toxicology center decision. His bronchorrhea and sialorrhea decreased and then ceased.

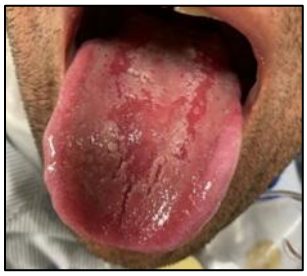

Figure 1 Multiple erosions and superficial ulcers on the tongue of the patient

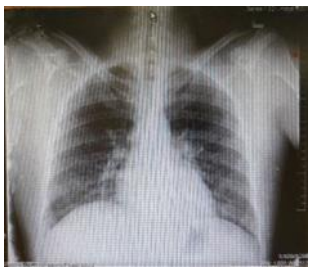

Figure 2 Patient Chest X-Ray on the day of presentation

Laboratory results including hemoglobin level, platelets count, electrolytes, liver function test, creatinine phosphokinase, where all within normal range except for high white blood cell count (WBC)14,400cells/μL, creatinine 10.15mg/dL and C-reactive protein(CRP) 108mg/L.

Conservative measures were continued while thorough investigations were conducted to identify the ingested substance. Patient first showed stability in overall clinical and hemodynamic state but then progressively complained of dyspnea with an increase in oxygen requirement, becoming dependent on a Non-rebreather face mask (NRBFM) at 15 L/min within 48 hours of his admission and having an oxygen saturation of 90%.

He also developed oliguria with a urine output (UOP) of 15 cc/hr. A new chest X-ray (Figure 3) revealed hilar congestion with mild bilateral pleural effusion. A right internal jugular dialysis catheter was inserted and urgent hemodialysis (HD) in the intensive care unit (ICU) was initiated with the removal of 2 Liters on his first session.

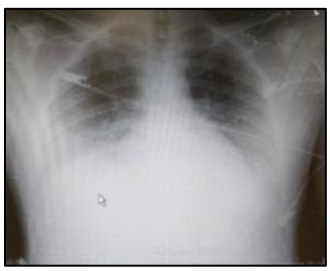

Figure 3 Chest X-ray revealing bilateral hilar prominence and blunting of costophrenic angles

Following this deterioration, patient's family provided us with a picture of the ingested liquid: Paraquat 276 g/l (27.6%) of which he drank approximately 20 ml, with no dilution, followed by forced vomiting due to its thick consistency.

As soon as the substance was recognized Dexamethasone 8 mg every 8 hours was initiated along with vitamin C 300mg po bid for 7 days.

A total of five HD sessions were done with the patient acute kidney injury (AKI) resolving with good UOP and a creatinine level returning to normal (0.8 mg/dl) within 25 days. However, his oxygen requirement continued to increase needing a NRBFM of 15 L/min and a high-flow nasal cannula on a flow rate of 60 L/min and an FiO_2 of 100%.

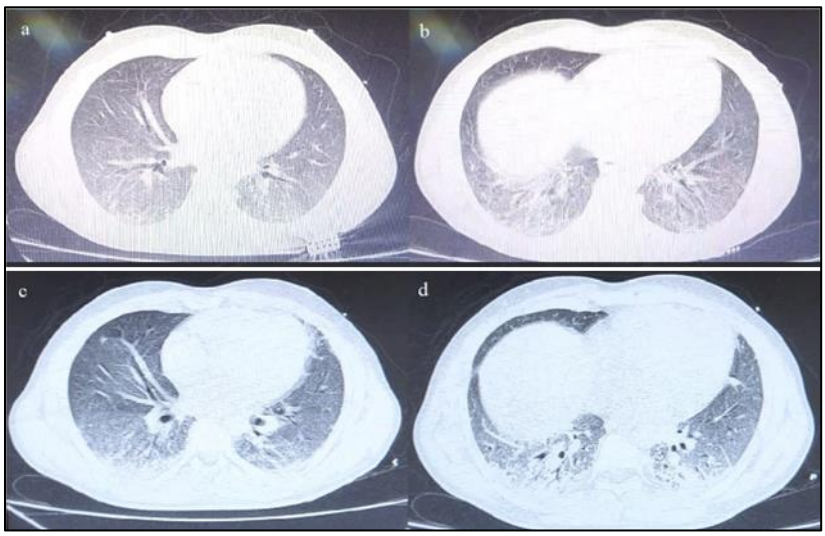

Figure 4 CT scan of the chest on day 4 (a & b) showing bilateral lower lobes reticulo-nodular infiltrates with ground glass opacities (GGOs) and 13 days after his admission (c & d) showing more extensive and diffuse GGOs with bilateral lower lobes consolidations and reticular infiltrates

Table 2 Laboratory workup during hospital stay. WBC: white blood cells, Hgb: Hemoglobin, BUN: Blood urea nitrogen, Crea: creatinine, K^+: Potassium, CO_2 : Bicarbonate , CRP: C-reactive protein, T. Bil.: Total bilirubin, GGT: gamma-glutamyl transferase, SGPT: serum glutamate pyruvate transaminase – ALT, Ca: calcium, Ph: phosphorus.

	D2	**D3**	**D5**	**D6**	**D7**	**D10**	**D13**	**D17**	**D22**	**D32**	**D44**	**D48**	**D50**
WBC	13.98	19.63	9.14	13.7	10.42	20.4	32.8	31.3	19.06	16.04	15.6	15.6	-
Hgb	16.8	16.4	16.5	15.8	15.3	15.8	15.4	15.6	16.3	14.9	16.2	15.4	-
BUN	77	102	114	112	84	57	45	32	33	-	-	-	31
Crea	12.49	15.2	14.13	10.5	6.08	2.36	1.54	1.24	0.95	0.78	1.28	0.88	1.87
K+	4.08	4.08	4.89	4.36	4.48	4.3	4.6	4.5	-	4.2	3.54	4.4	4.9
CO2	22.2	23	17.2	23.6	19.5	18.8	23	22.2	24.5	21.9	21.9	21.9	23.2
CRP	-	435.6	423.2	292.4	129.5	-	13.3	-	3	1.4	2	13.6	-
T. Bil.	0.54	-	-	-	0.32	-	-	-	-	0.38	-	-	-
GGT	-	48	-	-	102	-	-	-	-	59	-	-	-
SGPT	-	12	-	-	72	-	-	-	-	34	-	-	-
Ph	-	7.67	9.17	-	6.14	4.4	3.6	4.16	-	4.5	-	-	-

The patient had severe rapidly progressing respiratory failure due to paraquat-induced pulmonary fibrosis, he was intubated on day 40. He developed low grade fever (38°.2) without clear focus of infection. Pan cultures were taken and he was started on Meropenem plus Linezolid for presumed nosocomial infection. All cultures turned to be negative but patient had sustained severe hypoxemia and passed away on Day 51.

3. Discussion

Paraquat poisoning remains a major toxicological emergency due to its rapid systemic absorption and preferential accumulation in pulmonary tissue. The herbicide exerts its toxicity primarily through redox cycling in type I and II alveolar epithelial cells, generating reactive oxygen species that cause cellular apoptosis, necrosis, and ultimately irreversible pulmonary fibrosis [2]. Even with early therapeutic intervention, the onset of lung fibrosis typically heralds a poor prognosis.

Prognosis often correlates with plasma Paraquat concentrations. Levels exceeding 2 mg/L within the first 24 hours post-ingestion are associated with near-certain mortality [3]. Unfortunately, this diagnostic tool is unavailable in many clinical settings, including ours, limiting the ability to stratify risk and guide treatment based on quantitative exposure.

Recent studies have identified novel biomarkers that may enhance prognostic accuracy in paraquat poisoning. Proteomic analyses have highlighted upregulation of S100A8 and S100A9 proteins in patient plasma, suggesting their potential use as diagnostic and prognostic markers [4]. These neutrophil-derived alarmins are linked to inflammatory signaling as well as fibroblast activation in lung disease. Elevated S100A8/A9 has been associated with idiopathic pulmonary fibrosis, and experimental studies have shown antifibrotic responses following S100A8/A9 blockade [5]. Together, these findings indicate that S100A8/A9 may reflect the severity of lung injury in paraquat poisoning and could serve as targets for therapeutic intervention.

Additionally, elevated serum creatinine levels have been reaffirmed as significant predictor of mortality, with a meta-analysis demonstrating high sensitivity and specificity for this marker [6].

Furthermore, Tang et al. developed and validated a prognostic nomogram for in-hospital mortality in acute paraquat poisoning patients, incorporating clinical indicators such as decreased level of consciousness (Glasgow Coma Scale score < 15), neutrophil-to-lymphocyte ratio, alanine aminotransferase, creatinine, carbon dioxide combining power, and paraquat plasma concentrations at admission. The nomogram demonstrated outstanding discriminative ability, facilitating early-stage evaluation and prognosis prediction [7].

As for management, a therapeutic paradox exists with oxygen administration. While essential for hypoxemia, excessive supply worsens oxidative injury and accelerates damage. In a multi-center retrospective cohort study, Lin et al, reported that early liberal oxygen therapy increased the risk of mortality in paraquat-poisoned patients, suggesting that oxygen should be reserved for those with clear hypoxia (SpO2<90%) [8]. These findings highlight the importance of careful oxygen titration.

Moreover, as oxidative stress plays a key mechanism in paraquat-induced injury, adjunctive antioxidants, including N-acetylcysteine, vitamins C and E, may help limit cellular damage. Although not sufficient as standalone therapy, early administration could enhance clinical outcomes. However, owing to a lack of pharmacokinetic data, the optimal dose is unknown [9].

In parallel with supportive measures, extracorporeal removal techniques have been explored, with hemoperfusion offering greater theoretical efficacy than conventional hemodialysis. Hemoperfusion has been shown to significantly reduce plasma paraquat concentrations both in vitro and in vivo, emphasizing its essential role in the management of acute paraquat poisoning [10]. A prospective controlled study demonstrated that combining hemoperfusion with continuous venovenous hemofiltration (CVVH) significantly improved 90-day survival compared with conventional therapy [11]. Separately, an observational cohort found that initiating hemoperfusion within the first 4-5 hours of ingestion was associated with reduced mortality [12]. In our case, only hemodialysis was performed, and it was initiated after renal dysfunction had developed, likely beyond the window of maximal efficacy.

The use of immunosuppressive agents most notably corticosteroids and cyclophosphamide has been investigated for their potential to modulate the inflammatory cascade and prevent fibrosis. Both cyclophosphamide and corticosteroid were used based on the hypothesis that paraquat-induced lung injury was caused by inflammatory processes and that suppression of inflammation might improve outcome [9]. Some observational studies suggest improved survival in moderate poisoning cases, though evidence remains inconclusive, particularly in severe exposures.

Our patient's course was complicated by an initial diagnostic uncertainty due to the lack of exposure history and delayed confirmation of the ingested substance in addition to his late presentation. Supportive therapy, including fluid resuscitation and corticosteroids, was initiated, but more definitive measures such as hemoperfusion and immunosuppressive combination therapy were not executed.

This case underscores several key challenges in managing Paraquat poisoning: the rapid progression of toxicity, the absence of readily available prognostic biomarkers, and the crucial importance of timing.

Ultimately, this case exemplifies the limitations of current treatment modalities, the urgent need for preventive strategies and emerging new therapies. Restriction of access to highly toxic herbicides and bolstering mental health support systems are essential public health measures.

4. Conclusion

This case illustrates the severe and often fatal nature of Paraquat poisoning, especially in settings with limited diagnostic and therapeutic resources. Despite prompt supportive care and corticosteroid therapy, the delayed identification of the ingested substance and unavailability of hemoperfusion limited the effectiveness of treatment. Plasma Paraquat concentration and biomarkers of lung injury, play a crucial role in guiding treatment decisions, yet are often inaccessible in real-world practice. The case underscores the importance of early intervention and access to specialized therapies like hemoperfusion, showing potential benefits when applied within the first few hours of exposure and the potential - but still uncertain - benefit of immunosuppressive agents. Eventually, this case highlights both the clinical challenges and the critical necessity of implementing mitigation measures, including stricter regulation of hazardous herbicides and enhanced mental health services.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflicts of interest related to this case report.

Statement of ethical approval

Ethical approval was not required for this single-patient case report.

Statement of informed consent

Written informed consent for publication was obtained from the patient at the time of hospitalization.

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Author Contributions

All authors contributed to the management of the patient, the writing of the manuscript, and approved the final version. Mariam R. Issawi led the clinical documentation and manuscript drafting. Zaynab Zaylaa contributed to literature review and data analysis. Layal Olaywan supervised the clinical management and critically revised the manuscript.

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