RESEARCH PAPER

Suspected Pesticide (Paraquat & Chlorantraniliprole) Poisoning Presenting as Chemical Pneumonitis & Severe Ards Requiring Mechanical Ventilation

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ABSTRACT

Paraquat is a highly toxic bipyridyl herbicide associated with severe multisystem toxicity and high mortality, particularly due to progressive pulmonary injury leading to chemical pneumonitis, acute respiratory distress syndrome (ARDS), and pulmonary fibrosis. Paraquat poisoning is a medical emergency due to its propensity to cause multisystem toxicity. Chlorantraniliprole toxicity in humans is rarely reported. Mixed pesticide exposure further complicates the clinical course. Delayed presentation significantly worsens prognosis because of progressive respiratory failure and multiorgan dysfunction.

We report the case of a 15-year-old female patient with suspected mixed pesticide poisoning involving paraquat and chlorantraniliprole who presented with progressive breathlessness following accidental ingestion of the toxic formulation. Imaging revealed bilateral pneumonitis with progressive worsening on serial chest radiographs and HRCT showing bilateral consolidations with diffuse ground-glass opacities. Serial arterial blood gas analysis demonstrated progression from hypoxemic Type 1 respiratory failure to hypercapnic Type 2 respiratory failure. Despite aggressive management with lung-protective ventilation, prone positioning cycles, corticosteroids, antioxidants, renal replacement therapy and planned extracorporeal membrane oxygenation (ECMO), the patient succumbed to refractory hypoxemia and multiorgan dysfunction. This case highlights the devastating pulmonary toxicity of paraquat poisoning, especially in delayed presentations, and emphasizes the importance of early recognition, prompt decontamination, and aggressive supportive care. Mixed pesticide poisoning involving paraquat carries a grave prognosis, and public awareness regarding early referral and emergency management remains crucial for improving outcomes.

Keywords: Paraquat, Chlorantraniliprole, Poisoning, Chemical pneumonitis, ARDS, Mechanical ventilation.

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INTRODUCTION

Paraquat (1,1′-dimethyl-4,4′-bipyridinium dichloride) is a highly toxic, non-selective bipyridyl herbicide widely used in agriculture because of its rapid action and effectiveness in weed control. Despite its agricultural benefits,

paraquat remains one of the most lethal poisons encountered in clinical toxicology due to its narrow therapeutic index and the absence of a specific antidote. Even small quantities of ingestion can result in severe systemic toxicity and high mortality. Toxicity primarily occurs through the generation of reactive oxygen species, leading to oxidative stress, lipid peroxidation, mitochondrial dysfunction, and progressive cellular injury affecting multiple organs, particularly the lungs, kidneys, liver, and gastrointestinal tract. The lungs are the principal target organ because paraquat selectively accumulates in type I and type II pneumocytes through the polyamine uptake system, resulting in alveolitis, chemical pneumonitis, acute respiratory distress syndrome (ARDS), and eventually irreversible pulmonary fibrosis.

Clinical manifestations vary depending on the dose and route of exposure but commonly include oral ulcerations, vomiting, abdominal pain, acute kidney injury, hepatic dysfunction, and progressive respiratory failure. Delayed presentation is associated with significantly worse outcomes because pulmonary injury continues to progress even after the initial exposure. Early decontamination, supportive care, antioxidant therapy, corticosteroids, renal replacement therapy, and advanced respiratory support remain the mainstay of management, although prognosis remains poor in severe poisoning cases.

Chlorantraniliprole is a relatively newer anthranilic diamide insecticide that acts by activating ryanodine receptors in insects, causing muscle paralysis and death. Human toxicity from chlorantraniliprole is rarely reported and is generally considered less severe compared to paraquat; however, mixed pesticide ingestion involving both compounds may complicate clinical presentation and worsen outcomes.

We report the case of a 15-year-old female with suspected mixed pesticide poisoning involving paraquat and chlorantraniliprole who presented late with progressive chemical pneumonitis, acute kidney injury, and severe ARDS requiring invasive mechanical ventilation. This case highlights the devastating pulmonary toxicity of paraquat, the impact of delayed referral, and the challenges in management despite maximal intensive care support.

CASE PRESENTATION

A 15-year-old female with no known prior comorbidities was brought to the emergency department with progressively worsening breathlessness following accidental ingestion of a pesticide formulation containing paraquat and chlorantraniliprole approximately 20 days earlier. Initial treatment was provided at a peripheral healthcare facility where she was managed conservatively. However, over the following two weeks, her respiratory symptoms progressively worsened, and she developed increasing oxygen requirement, oral ulcerations, reduced urine output, and generalized weakness. She was subsequently referred to tertiary care center for further management due to worsening respiratory distress and suspected acute kidney injury requiring renal replacement therapy.

On admission, the patient was already intubated and mechanically ventilated because of severe hypoxemia and respiratory failure. She appeared critically ill, with tachycardia and persistent hypoxemia despite ventilatory support. Bilateral coarse crepitations were heard on chest auscultation. Oral mucosal ulcerations were noted, suggestive of corrosive injury secondary to pesticide ingestion. No prior significant medical illness, surgical history, or known drug allergies were reported by the family. There was no history suggestive of chronic respiratory disease or renal dysfunction before the poisoning episode.

Laboratory investigations revealed leucocytosis (14500/mm3), elevated liver enzymes (SGOT/SGPT- 98/72), and significant renal dysfunction (urea 132 mg/dl, s. creatinine 2.9 mg/dl) consistent with acute kidney injury and urine paraquat was weakly positive. The patient required haemodialysis during the course of treatment. Serial arterial blood gas analyses demonstrated progression from severe hypoxemic respiratory failure to hypercapnic respiratory failure, eventually consistent with Type 2 respiratory failure. Chest radiograph on admission showed bilateral diffuse pneumonitis, with follow-up imaging demonstrating progressive worsening of bilateral pulmonary infiltrates. High-resolution computed tomography (HRCT) of the chest revealed extensive bilateral consolidations with diffuse ground-glass opacities, consistent with severe chemical pneumonitis and ARDS.

The patient was managed in the intensive care unit with lung-protective invasive mechanical ventilation, multiple prone ventilation cycles, corticosteroids, antioxidant therapy, broad supportive care, and renal replacement therapy. Despite aggressive treatment, respiratory status continued to deteriorate with refractory hypoxemia. Extracorporeal membrane oxygenation (ECMO) was planned for severe ARDS; however, before definitive escalation could be achieved, the patient developed progressive multiorgan dysfunction and succumbed to her illness on day 33 following ingestion. This case reflects the fulminant and often fatal course of delayed paraquat poisoning complicated by severe pulmonary toxicity and multiorgan failure.

SERIAL CHEST X-RAYS

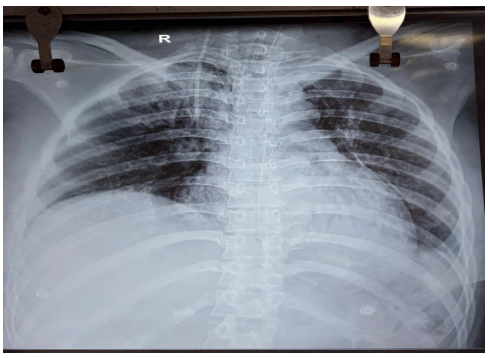


Figure 1: Chest X-ray on admission showing bilateral pneumonitis.

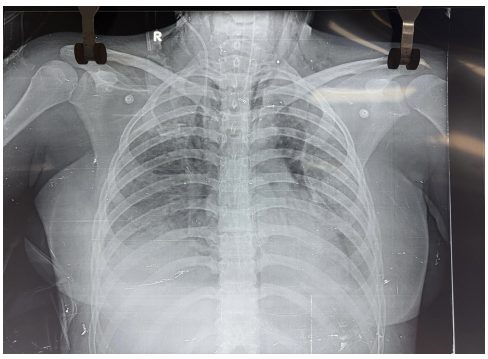


Figure 2: Follow-up chest X-ray showing progression of bilateral pulmonary infiltrates.

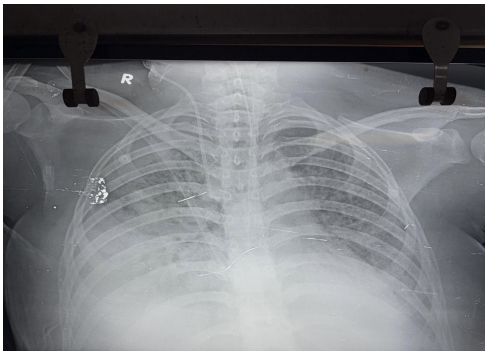
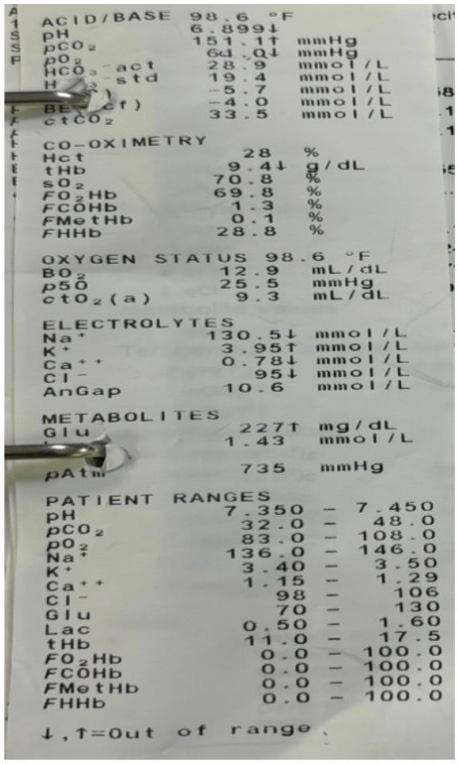


Figure 3: Further worsening with diffuse bilateral opacities consistent with severe chemical pneumonitis and ARDS.

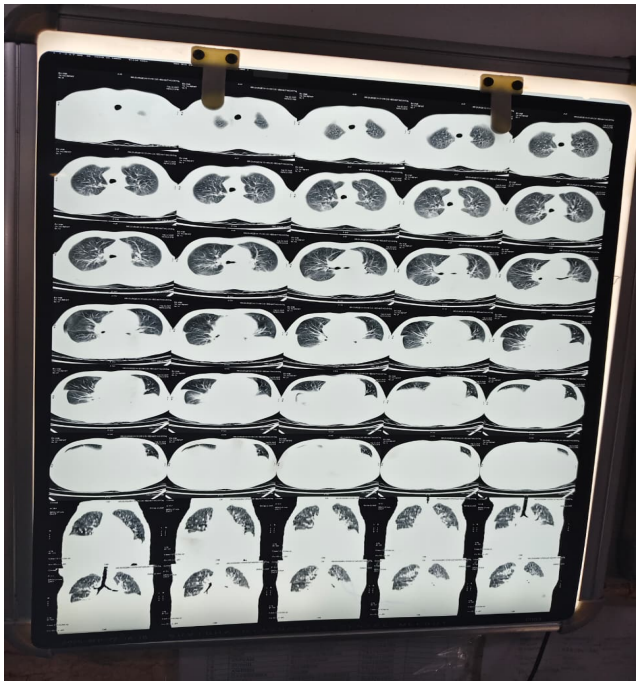
ARTERIAL BLOOD GAS ANALYSIS

ACID/BASE 98.6 °F		
pH	7.400	
pCO_2	49.0↑	mmHg
pO_2	51.1↓	mmHg
HCO_3^- act	29.7	mmol/L
HCO_3^- std	28.0	mmol/L
BE(B)	4.3	mmol/L
BE(ecf)	4.9	mmol/L
$ctCO_2$	31.2	mmol/L
CO-OXIMETRY		
Hct	26	%
tHb	9.0↓	g/dL
sO_2	79.4	%
FO_2Hb	79.0	%
FCOHb	0.2	%
FMetHb	0.3	%
FHHb	20.5	%
OXYGEN STATUS 98.6 °F		
BO_2	12.4	mL/dL
p50	30.6	mmHg
$ctO_2(a)$	10.0	mL/dL
ELECTROLYTES		
Na^+	139.0	mmol/L
K^+	3.13↓	mmol/L
Ca^{++}	0.86↓	mmol/L
Cl^-	102	mmol/L
AnGap	10.5	mmol/L
METABOLITES		
Glu	92	mg/dL
Lac	0.69	mmol/L
pAtm	729	mmHg
PATIENT RANGES		
pH	7.350 – 7.450	
pCO_2	32.0 – 48.0	
pO_2	83.0 – 108.0	
Na^+	136.0 – 146.0	
K^+	3.40 – 3.50	
Ca^{++}	1.15 – 1.29	
Cl^-	98 – 106	
Glu	70 – 130	
Lac	0.500 – 1.60	
tHb	11.0 – 17.5	
FO_2Hb	0.0 – 100.0	
FCOHb	0.0 – 100.0	
FMetHb	0.0 – 100.0	
FHHb	0.0 – 100.0	
↓,↑=Out of range		



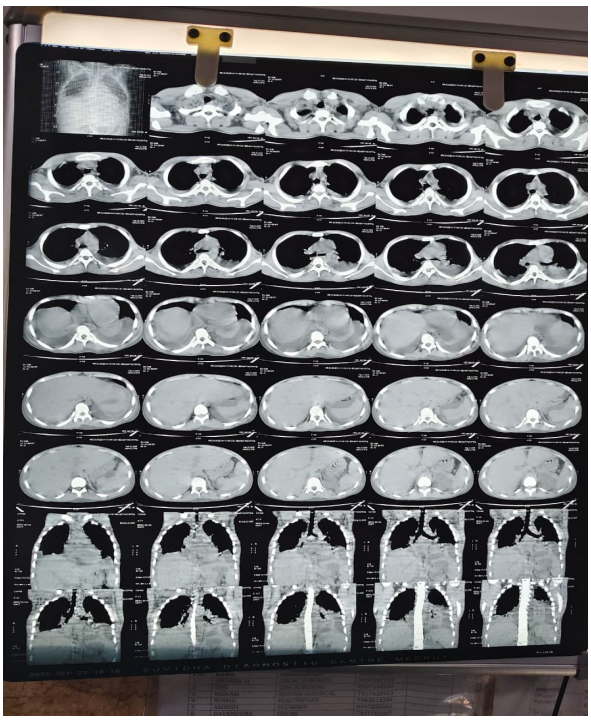
ACID/BASE	98.6 °F	
pH	6.899↓	
pCO_2	151.1↑	mmHg
pO_2	64.0↓	mmHg
HCO_3^- act	28.9	mmol/L
HCO_3^- std	19.4	mmol/L
BE(ecf)	-5.7	mmol/L
BE(b)	-4.0	mmol/L
$ctCO_2$	33.5	mmol/L
CO-OXIMETRY		
Hct	28	%
tHb	9.4↓	g/dL
sO_2	70.8	%
FO_2Hb	69.8	%
FCOHb	1.3	%
FMetHb	0.1	%
FHHb	28.8	%
OXYGEN STATUS	98.6 °F	
BO_2	12.9	mL/dL
$p50$	25.5	mmHg
$ctO_2(a)$	9.3	mL/dL
ELECTROLYTES		
Na^+	130.5↓	mmol/L
K^+	3.95↑	mmol/L
Ca^{++}	0.78↓	mmol/L
Cl^-	95↓	mmol/L
AnGap	10.6	mmol/L
METABOLITES		
Glu	227↑	mg/dL
	1.43	mmol/L
pAtm	735	mmHg
PATIENT RANGES		
pH	7.350 - 7.450	
pCO_2	32.0 - 48.0	
pO_2	83.0 - 108.0	
Na^+	136.0 - 146.0	
K^+	3.40 - 3.50	
Ca^{++}	1.15 - 1.29	
Cl^-	98 - 106	
Glu	70 - 130	
Lac	0.50 - 1.60	
tHb	11.0 - 17.5	
FO_2Hb	0.0 - 100.0	
FCOHb	0.0 - 100.0	
FMetHb	0.0 - 100.0	
FHHb	0.0 - 100.0	

↓,↑=Out of range



Day 0	Accidental ingestion at farm
Days 1–10	Managed in local hospital; developed acute kidney injury → dialysis initiated
Day 20	Referred for worsening respiratory distress
Day 21–24	Intubated, placed on AC/VC mode; diagnosed with severe ARDS
Day 24–30	Received 5 cycles of prone ventilation
Day 31	Planned for ECMO due to refractory hypoxemia
Day 33	Developed multiorgan dysfunction; succumbed despite maximal supportive measures

HRCT CHEST



DISCUSSION

Paraquat poisoning is one of the most severe and life-threatening forms of pesticide toxicity encountered in clinical practice, particularly in developing countries where agricultural exposure is common and access to the compound remains relatively easy. Even small quantities of ingestion are associated with significant morbidity and mortality because paraquat has no specific antidote and causes progressive multisystem damage. The primary mechanism of toxicity involves the generation of reactive oxygen species, leading to oxidative stress, mitochondrial dysfunction, lipid peroxidation, and widespread cellular injury. Although multiple organs may be affected, the lungs are the principal target because paraquat selectively accumulates in type I and type II pneumocytes through active uptake mechanisms, resulting in alveolar epithelial destruction, inflammatory pneumonitis, acute respiratory distress syndrome (ARDS), and ultimately irreversible pulmonary fibrosis.

Clinical presentation depends on the amount ingested, concentration of exposure, and timing of

medical intervention. Patients commonly present with oral ulcerations, vomiting, abdominal pain, acute kidney injury, hepatic dysfunction, and progressive respiratory failure. Oral mucosal ulceration, often described as "paraquat tongue," is an important early clinical clue suggestive of corrosive ingestion. Renal dysfunction is also a significant prognostic marker because paraquat is primarily excreted through the kidneys, and acute kidney injury leads to impaired clearance and worsening systemic toxicity. Delayed referral significantly worsens outcomes because pulmonary injury continues to progress even after the initial toxic exposure, often leading to severe hypoxemia and ventilator-dependent respiratory failure.

In the present case, the patient developed progressive chemical pneumonitis with worsening bilateral pulmonary infiltrates, severe ARDS, and acute kidney injury requiring intermittent hemodialysis. HRCT findings of bilateral consolidations with diffuse ground-glass opacities correlated with extensive alveolar injury and inflammatory lung damage. Serial arterial blood gas analysis demonstrated progression from hypoxemic respiratory failure to hypercapnic Type 2 respiratory failure, indicating advanced pulmonary involvement and poor prognosis. The associated ingestion of chlorantraniliprole, although less commonly associated with severe human toxicity, may have contributed to the complexity of presentation and systemic deterioration.

Management of paraquat poisoning remains largely supportive and includes early gastrointestinal decontamination when feasible, oxygen therapy with caution, corticosteroids, antioxidants, immunosuppressive therapy in selected cases, renal replacement therapy, and advanced ventilatory support. Lung-protective ventilation and prone positioning are essential in severe ARDS, while extracorporeal membrane oxygenation (ECMO) may be considered as a rescue therapy in refractory hypoxemia. However, survival remains poor in patients presenting late with established pulmonary fibrosis or severe multiorgan dysfunction. In our case, despite invasive mechanical ventilation, multiple prone cycles, corticosteroids, antioxidants, dialysis, and planned ECMO, the patient eventually succumbed to refractory hypoxemia and multiorgan failure.

This case highlights the devastating pulmonary toxicity of paraquat and emphasizes the importance of early diagnosis, prompt referral, and aggressive supportive care. Public awareness regarding the lethal nature of paraquat poisoning and the need for immediate medical intervention is essential to improve outcomes and reduce mortality in such cases.

CONCLUSION

Mixed pesticide poisoning involving paraquat carries a poor prognosis, particularly when presentation is delayed and significant pulmonary injury has already developed. Because clinical manifestations are often progressive, early recognition and prompt referral are essential to improve survival. Severe complications such as chemical pneumonitis, acute respiratory distress syndrome
(ARDS), acute kidney injury, and multiorgan dysfunction require aggressive supportive management, including intensive respiratory and renal support.

This case highlights the devastating pulmonary toxicity of paraquat and the challenges associated with managing advanced poisoning despite maximal intensive care measures. Increased public awareness regarding the dangers of paraquat exposure, along with early hospital presentation and timely multidisciplinary intervention, remains crucial for reducing mortality and improving clinical outcomes.