Case Report

Dual Organophosphate and Paraquat Poisoning: Successful Management with Early Hemoperfusion and Ventilation: A Case Report

Ramesh Yelanati[1], Dharanindra Moturu[2*], Ramesh Babu Potineni[3], Supriya Rayana[4], Mohammad Noor Shaik[5], Sravani Thommandru[4], Kondaveeti Devaki[4], Kavya Sruthi Grandhi[5], Jahnavi Naga Pavani Pucha[5]

1. *DM Consultant Nephrologist, Aster Ramesh Hospital, Vijayawada, Andhra Pradesh, India*
2. *DM Critical Care Medicine, Advanced Trainee, Westmead Hospital, Australia*
3. *DM Cardiology, HOD Cardiology and Managing Director, Aster Ramesh Hospital, Vijayawada, Andhra Pradesh, India.*
4. *Pharm D, Assistant Professor, Department of Pharmacy Practice, KL College of Pharmacy, Koneru Lakshmaiah Education Foundation, Vaddeswaram, Guntur, Andhra Pradesh, India.*
5. *Doctor of Pharmacy Program, KL College of Pharmacy, Koneru Lakshmaiah Education Foundation, Vaddeswaram, Guntur, Andhra Pradesh, India.*

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ABSTRACT

Background: Organophosphate poisoning is a major public health issue in many low and middle-income countries. Paraquat, a widely used herbicide, can often cause severe, usually fatal poisoning in humans. Dual poisoning involving both organophosphates and paraquat is rare.

Case Presentation: A 17-year-old male who ingested both substances was admitted to the emergency department intensive care unit. He was treated with four sessions of hemoperfusion, along with intravenous atropine and pralidoxime. After the fourth hemoperfusion, the patient developed respiratory distress due to acute cholinergic syndrome secondary to organophosphate poisoning, requiring endotracheal intubation. By day five, his clinical condition improved, and he was extubated. Six hours after extubation, he developed sudden respiratory distress and was re-intubated with a 7.5 mm endotracheal tube and connected to a mechanical ventilator. The patient was diagnosed with intermediate syndrome secondary to organophosphate poisoning, and atropine infusion was restarted. By day seven, his condition improved and he was successfully weaned off ventilation. He was discharged hemodynamically stable on day 10.

Conclusion: This mixed organophosphate-paraquat poisoning case was clinically complex and challenging. Management involved a combination of mechanical ventilation and hemoperfusion, which had to be carefully titrated. Further studies are needed to improve treatment protocols and outcomes for mixed poisoning cases.

*** Corresponding Author:**
Moturu Dharanindra, MD
DM Critical Care Medicine, Advanced Trainee, Westmead Hospital, Australia.
E-mail: *newgenmedico@outlook.com*

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Introduction

In India, organophosphates (OPs) are the most widely used pesticide for suicide by poisoning [1]. It can present acutely with a cholinergic crisis, respiratory distress, and intermediate syndrome, or it may cause delayed toxic effects [2].

OPs inhibit acetylcholinesterase, leading to the stimulation of cholinergic and nicotinic receptors [2]. Atropine is the primary treatment for OPs poisoning [3]. While atropine counteracts muscarinic cholinergic effects, oximes are required to treat the nicotinic neurological manifestations. Oximes should be administered promptly, as their effectiveness decreases significantly after 13 hours [3].

Paraquat is a commonly used herbicide that can lead to severe, often fatal poisoning in humans [4]. It causes acute lung injury and irreversible pulmonary fibrosis, with no specific antidote available. Respiratory failure is the leading cause of death in paraquat poisoning cases. Management is mainly supportive care [5, 6]. Gut decontamination with activated charcoal is recommended in patients who present within 1-2 hours of ingestion [7]. Other treatments include gastric lavage or whole gut irrigation with adsorbents like Fuller's earth or activated charcoal, intensive hemoperfusion, antioxidants like N-acetyl cysteine, immunosuppressive therapy with cyclophosphamide, and mechanical ventilation for respiratory support. Despite these interventions, mortality in severe paraquat poisoning remains high [5, 6].

Here, we report a case of mixed organophosphate and paraquat poisoning admitted to a tertiary care hospital. The aim of this case report is to present the clinical features and successful management of mixed organophosphate-paraquat poisoning, highlighting the role of early hemoperfusion and targeted supportive therapy.

Case Presentation

A 17-year-old male was admitted to the hospital 4 hours after intentional ingestion of a poisonous mixture containing paraquat and organophosphate (Chlorpyrifos). He presented in a confused state, tachypnea (40 breaths/min), and a Glasgow Coma Scale (GCS) of 13 (E3+V4+M6). On admission, vital signs were as follows: pulse rate of 110 beats/min, blood pressure (BP) of 80/50 mmHg, and SpO_2 of 94% on room air. On examination, there was mild expiratory wheeze, a slightly distended abdomen, and sinus tachycardia on electrocardiogram (ECG). Immediate decontamination was performed with gastric lavage using a nasogastric tube. The Urine sodium dithionate test was positive, confirming paraquat ingestion (Figure 1). Serum cholinesterase was 1800 U/L (normal range 0-14000 U/L), indicating approximately 85% inhibition of enzyme activity and reflecting severe organophosphate poisoning. Laboratory investigations (Complete blood picture, Renal and Liver function tests, and arterial blood gas analysis) are summarized in Tables 1 and 2.

Figure 1. Urine dithionate result tested strongly positive (blue color at 4hours).

The patient was transferred to the emergency room intensive care unit (ER ICU). Airway, Breathing, and Circulation were stabilized. As the patient had secretions in the airway, oral suctioning was performed. He was provided oxygen via a venturi mask, an intravenous (IV) fluid bolus of 500 ml was given, and maintenance fluids were started. Hemoperfusion was initiated within 1 hour of presentation to the hospital to remove paraquat from the circulation as soon as possible. The urine dithionite test was done before each hemoperfusion session. A total of 4 sessions were performed at 2-hour intervals. After the 4th hemoperfusion session, the urine dithionite test became negative, leading to cessation of hemoperfusion. He also received intravenous fluids, steroids, antibiotics, and vitamin C, intravenous atropine (4mg/hour), and Pralidoxime (500mg bolus followed by 500 mg over 5 hours).

The patient developed respiratory distress after completion of the 4th hemoperfusion. On examination, bilateral crepitations were observed, and the clinical findings were consistent with acute cholinergic syndrome. Chest X-ray revealed pulmonary edema (Figure 2), therefore, Non-invasive ventilation (NIV) was initiated. Subsequently, he was endotracheally intubated. Initially, mucosal erythema, congestion, and edema were noted, which later progressed to erosion and ulceration. Leukocytosis was noted on day 2

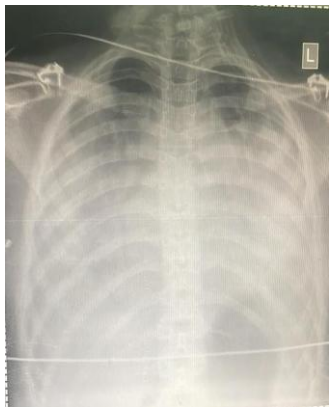

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Figure 2. Pulmonary edema showing bilateral pulmonary infiltrates.

(White Blood Cell (WBC) count: 12,600).

infusion was restarted.

On day seven, the patient's general condition improved with no residual organ dysfunction (Figure 3). The Sequential Organ Failure Assessment (SOFA) score was 1. He was successfully extubated and tolerated spontaneous breathing. He was transferred to the ward on day 8 and was discharged in a stable hemodynamic condition on day 10. He was referred to Psychiatry for follow-up counselling (Table 4).

Discussion

Mixed poisoning cases involving organophosphates (OPs) and paraquat are exceptionally rare, with only isolated case reports and small series available, most notably from South Asia and India, where both compounds remain widely accessible [1, 2, 14]. As per our search in databases like PubMed Central, Google Scholar, and Embase, this is the second case report on

Table 1. Complete Blood Picture, Renal Function Tests and Liver Function Tests.

Parameters	Day1	Day 2	Day 6	Day 7	Day 10	Reference Range
Complete Blood Picture						
Hemoglobin (g/dl)	12.8	12.2	11.6	12.4	12.9	13-17
Total RBC count (millions/cu.mm)	4.70	4.42	4.11	4.47	4.67	4.5–5.5
Total WBC count (cells/cu.mm)	10,700	12,600	10,700	10,400	10,200	4000-11,000
Neutrophils (%)	84	86	65	66	47	45–75
Lymphocytes (%)	12	08	25	23	41	15–45
Eosinophils (%)	02	02	01	01	02	1–6
Monocytes (%)	02	04	09	10	10	2-10
Basophils (%)	00	00	00	00	00	1–2
Renal Function Tests and Liver Function Tests						
Serum sodium (mEq/L)	143.5	133	--	134	132.1	135 – 145
Serum potassium (mEq/L)	3.48	3.4	--	--	4.65	3.5 – 5.0
Serum creatinine (mg/dl)	0.6	0.5	--	--	0.6	0.66–1.25
Total bilirubin (mg/dl)	0.3	0.3	0.6	--	--	0.2-1.3
Total protein (gm/dl)	6.0	5.1	5.4	--	--	6.3-8.2
Serum albumin (gm/dl)	3.4	2.9	3.0	--	--	3.5-5.0

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RBC= Red Blood Cell, WBC=White Blood Cell

By day 3, hemodynamics was stable with a GCS of E3VTM5. On day four, sedation was weaned off, and he was maintained on a spontaneous mode of ventilation with a fraction of inspired oxygen (FiO_2) of 0.3. Despite 70 hours of atropine and pralidoxime infusion, neuromuscular weakness persisted.

On day five, the patient's general condition improved, neuromuscular power improved, and no secretions were observed. The GCS was E4VTM6. He was weaned off the ventilator and extubated. Post-extubation, there was no respiratory distress. Six hours after extubation, he developed sudden respiratory distress and required re-intubation.

The patient was diagnosed with intermediate syndrome based on clinical examination, peripheral nerve conduction study, and electromyogram. Atropine

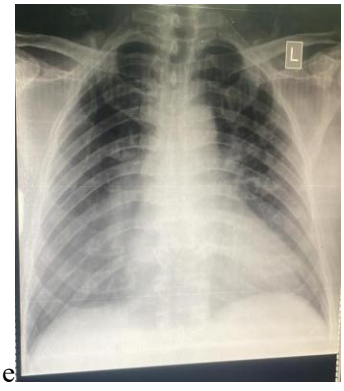

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Figure 3. Post extubation X-ray showing resolution of pulmonary edema & well-aerated lung.

organophosphate and Paraquat mixed poisoning. Epidemiological data consistently highlight the significant global burden of pesticide poisoning, with paraquat and OPs leading to high mortality, especially in developing regions [6, 14, 15].

It is important to recognize that each compound affects different biological pathways and produces distinct toxic manifestations. In this case of paraquat and Organophosphate Poisoning (OPP), the OPs caused cholinergic toxidrome and neuromuscular weakness and required mechanical ventilation. Oxygen delivery at higher concentrations is known to cause higher mortality in paraquat poisoning, which is harmful and worsens the lung damage secondary to paraquat poisoning [12]. In this scenario, we aimed for low oxygen targets (FiO_2 0.3–0.4, SpO_2 88–92%) to minimize paraquat-induced lung damage while supporting ventilation for cholinergic toxidrome.

Early hemoperfusion prevented multiorgan dysfunction from paraquat while potentially delaying acute cholinergic syndrome [9]. In the present case, hemoperfusion was started within 1 hour, which may have contributed to the limited tissue accumulation of paraquat and the patient's favorable outcomes.

Once ingested, paraquat is rapidly distributed to organs, with about 90% excreted unchanged in urine within 12–24 hours. The sodium dithionite test detects paraquat in urine by producing a blue color. The test can detect paraquat concentrations as low as 1 µg/mL in the urine [10].

Organophosphates cause irreversible inhibition of acetylcholinesterase in the blood and nervous system. Without acetylcholinesterase, the body cannot break down acetylcholine, leading to its accumulation and resulting in a cholinergic toxidrome [8].

Ajay Mishra et al reported the case of a 27-year-old man who developed multi-organ dysfunction syndrome following the intentional ingestion of a 50:50 mixture of two OP compounds, dichlorvos and profenofos.

Table 2. Arterial Blood Gas (ABG) Analysis.

Parameters	Day 1	Day 2	Day 6	Day 7	Day 8
pH	7.273	7.380	7.506	7.524	7.482
PCO2 (mmHg)	46.0	37.1	32.3	26.5	25.7
PO2 (mmHg)	61.3	268	143	65.8	70.0
Sodium *(ISE Direct) (*meq/L)	132	133	131	126	131
Potassium *(ISE Direct) (*meq/L)	2.5	3.4	3.3	3.3	3.8
SO2 %	87.8	99.1	98.8	98.5	95.4
HCO3 ACT (mmol/L)	19.3	22.2	27.3	23.6	21.8
BE(ECF) (mmol/L)	6.1	2.7	3.2	1.0	3.1
Random blood glucose (mg/dl)	206	325	125	111	99

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pH =Hydrogen Ion Concentration, PCO_2= Partial Pressure of Carbon Dioxide, PO_2=Partial Pressure of Oxygen, SO_2=Oxygen Saturation, HCO_3=Bicarbonate, BE (ECF)= Base Excess (Extracellular Fluid)

Table 3. Comparison of Similar Cases with the Present Case.

Case	Age/Sex	Poison Ingested	Key Symptoms	Organ Failure	Interventions	Outcome
Ajay Mishra et al [11]	27/M	Dimethoate + Chlorpyrifos	Cholinergic crisis, Acute Renal Failure (ARF), multi-organ dysfunction syndrome (MODS)	MODS	Atropine, ventilation	Cardiac arrest, death at 48 hours
Thunga G et al [6]	21/M	OP + Paraquat	Cholinergic crisis, ARF	Renal failure	Initial supportive care	Discharged left against medical advice (LAMA), outcome not available
Present case	17/M	OP + Paraquat	Cholinergic crisis, lung injury, intermediate syndrome	Lungs	Atropine, oximes, ventilation, and early hemoperfusion	Survived

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OP=Organophosphate, ARF=Acute Renal Failure, MODS= Multi-organ dysfunction syndrome, LAMA=Left against medical advice

Table 4. Clinical Progression and Treatment Timeline of the Patient.

Day	Clinical Status	Key Investigations/Findings	Interventions/Treatments
Day 1	Confused, tachypneic, BP 80/50 mmHg, SpO_2 94% room air. GCS E3V4M6.	Urine soidum dithionate test: positive (paraquat). Serum cholinesterase: 1800 U/L.	Gastric lavage, airway suctioning, O_2 via venturi mask, IV fluids, antibiotics, steroids, Vit. C, hemoperfusion (1st session, total 4), Inj. Atropine (4 mg/hr), Inj. Pralidoxime.
Day 2	Mild leukocytosis, pulmonary edema were noted.	WBC: 12,600. Chest X-ray: pulmonary edema.	2nd hemoperfusion session, NIV started, later intubated due to respiratory distress.
Day 3–4	Sedation weaned; spontaneous mode ventilation. Neuromuscular weakness persisted.	-----	Continued atropine + pralidoxime, 3rd and 4th hemoperfusion sessions.
Day 5	Clinical improvement, power regained, no secretions. Extubated.	GCS: E4VTM6.	Atropine tapered. 6 hrs post-extubation: respiratory distress → re-intubated, diagnosed intermediate syndrome.
Day 6–7	clinically stable, SOFA ≈ 1.	ABG: improving.	Gradual weaning off mechanical ventilation, successful extubation on Day 7.
Day 8	Stable, no organ dysfunction.	—	Shifted out of the ICU.
Day 10	Hemodynamically normal.	—	Discharged from the hospital. Psychiatry follow-up arranged.

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BP=Blood Pressure, SpO_2=Peripheral Oxygen Saturation, GCS= Glasgow Coma Scale, WBC=White Blood Cell, NIV=Non-Invasive Ventilation, ABG=Arterial Blood Gas, SOFA=Sequential Organ Failure Assessment, ICU=Intensive Care Unit, O_2=Oxygen, IV=Intravenous

Despite intensive resuscitation efforts, the patient suffered cardiac arrest and passed away 48 hours after hospitalization [11].

Thunga G et al. reported the case of a 21-year-old man admitted to the emergency department after allegedly ingesting 30-40 ml of a mixture containing Monocrotophos (an organophosphate compound) and Gramoxane (paraquat). The patient showed a steady rise in urea and creatinine levels, indicating acute renal failure likely caused by the poisoning. Due to the ongoing increase in these levels, he was advised to undergo hemodialysis on the 3rd, 5th, and 6th days. On the 6th day, while still on ventilator support, the patient's urea and creatinine levels improved, but his arterial blood gas continued to deteriorate. This was explained to his family, but due to financial difficulties, they opted to discharge him against medical advice [6]. Similar cases are summarized in Table 3. In middle- and low-income countries, financial difficulties and socio-economic problems are one of the main reasons for intentional self-harm with agricultural and industrial poisons. Ramesh Yelanati et al found that in paraquat poisoning cases, early targeted hemoperfusion reduced morbidity, mortality, and intensive care unit (ICU) length of stay, thereby reducing the socio-economic impact on the patient and the family [13]. These cases, alongside our own, highlight the overlapping syndromes, complexity in management, and often poor prognosis associated with mixed pesticide poisoning.

In the present case, the patient was discharged in stable condition with no residual organ dysfunctions. Both 30-day and 90-day follow-ups showed normal general condition with no residual organ dysfunction. The Department of Psychiatry further followed the patient for counselling and was treated for impulse control disorder and depression.

Conclusion

This mixed organophosphate and paraquat poisoning case presented unique and complex challenges in clinical management. Organophosphate poisoning led to a cholinergic toxidrome, causing neuromuscular weakness and requiring careful mechanical ventilation, while paraquat poisoning caused acute lung injury, necessitating strategies to minimize oxygen toxicity. Hemoperfusion played a pivotal role in preventing multiorgan dysfunction from paraquat toxicity. This case highlights the importance of early, aggressive treatment in managing mixed poisoning cases, though further research is needed to improve treatment protocols and outcomes for such cases.

Conflicts of Interest

The authors report there are no competing interests to declare.

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