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CASE REPORT

Fatal Paraquat Poisoning in a Young Female: A case report

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ABSTRACT

Paraquat (PQ) is a highly toxic and lethal herbicide. In countries like Nepal, PQ poisoning is not uncommon. PQ affects the lungs, liver, and kidneys, causing acute failures of these organs due to oxidative stress. In this case report, we describe a 24-year-old female who presented with multiple episodes of vomiting and abdominal pain following suicidal ingestion of 10-15ml of PQ. Initially, the patient was normal clinically, and blood investigations also yielded normal results. However, condition of the patient deteriorated over time, and she passed away due to respiratory distress, acute kidney injury, and acute liver failure while being managed in the intensive care unit.

Keywords: *Acute kidney injury, acute respiratory failure, paraquat.*

INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-bipyridium dichloride; PQ) is a non-selective herbicide commonly used for its fast-acting properties in agriculture.[1] However, it is highly toxic, leading to a ban in numerous regions worldwide, including Nepal, where it was banned in 2024 AD.[2] PQ usually involves hepatic, renal, or pulmonary systems and is associated with a very high mortality (case fatality rate of 50-70%) even when consumed in minimal amounts (15-20 ml of 20% w/v).[3] High fatality is also due to lack of specific antidote, and thus only supportive management is possible.[3] In this case report, we discuss a 24-year-old female who ingested PQ with suicidal intent and highlight the clinical and radiological features, as well as the management challenges of PQ poisoning.

CASE REPORT

A 24-year-old previously healthy female presented to the emergency department 12 hours after suicidal ingestion of approximately 10–15 ml of PQ. She had multiple episodes of non-projectile, non-bilious vomiting, abdominal pain, and loose stools. There was no history of loss of consciousness, abnormal body movements, or frothing. No significant past medical, surgical, or drug history was reported.

On arrival, her vitals were stable: blood pressure 110/70 mmHg, pulse 86/min, respiratory rate (RR) 18/min, SpO_2 98% on room air, and temperature 36.7°C. Glasgow Coma Scale (GCS)was 15/15. Examination revealed whitish patches over the tongue ("paraquat tongue") but was otherwise unremarkable. Baseline chest X-ray and abdominal ultrasonography were normal. Initial blood investigations were within the reference ranges.

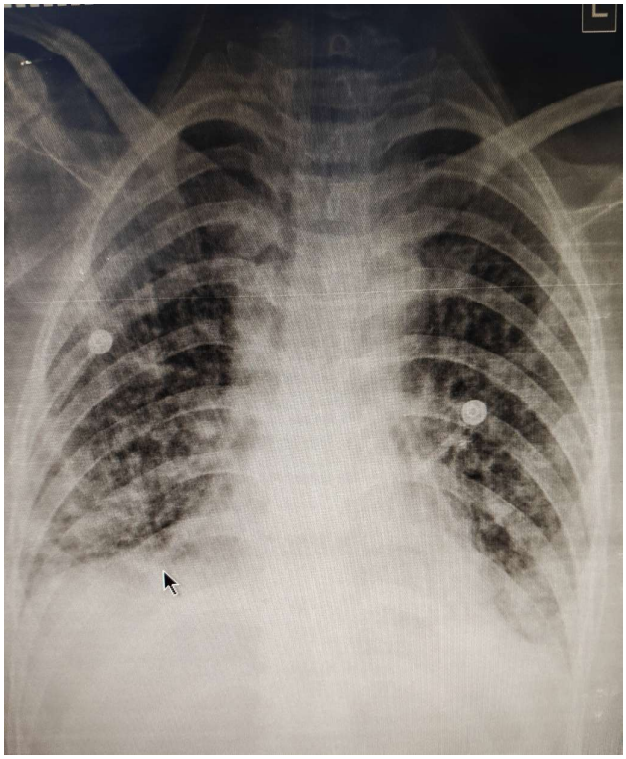

Figure 1: Chest X-ray anteroposterior view showing bilateral heterogeneous opacities predominantly in the lower zones with blunting of the left costophrenic angle.

She was admitted to the observation ward. Within 6 hours, she developed progressive respiratory distress with tachypnea (RR 32/min), SpO_2 dropped to 82% on room air, and GCS deteriorated to 13/15 (E3V4M6). She was placed on high-flow nasal cannula (HFNC) oxygen at 40 L/min with FiO_2 60%, after which SpO_2 improved to 92%. Arterial blood gas (ABG) at that time revealed pH 7.32, PaO_2 56 mmHg, $PaCO_2$ 30 mmHg, HCO_3^- 16 mmol/L, and lactate 3.5 mmol/L, consistent with type I respiratory failure with metabolic acidosis. Repeat chest X-ray showed new bilateral patchy opacities (Figure 1).

She was shifted to ICU for further management. A right internal jugular vein dialysis catheter was inserted, and hemodialysis was initiated on day 2 for worsening acute kidney injury (urea 18.2 mmol/L, creatinine 579 µmol/L). She was started on intravenous methylprednisolone 1 g/day for 3 days followed by tapering doses, and ceftriaxone 2 g once daily as prophylaxis, continued for 7 days. Intravenous fluids (crystalloids) were given for hydration, and vasopressor support with norepinephrine was required transiently on day 4 due to hypotension (BP 80/50 mmHg).

On day 5, the patient's condition worsened with increasing oxygen requirement; she was intubated and placed on mechanical ventilation with a lung-protective strategy (low tidal volume, high PEEP). Proning was performed intermittently as per the guidelines. Despite this, oxygenation remained poor (PaO_2/FiO_2 ratio of 75). Serial chest X-rays showed bilateral diffuse opacities. CT thorax (day 7) demonstrated extensive consolidation, ground-glass opacities, and septal thickening involving bilateral lower lobes with minimal pleural effusion (Figure 2). Inflammatory markers (C-reactive protein (CRP)186 mg/L, procalcitonin 3.1 ng/mL) were elevated on day 7, raising suspicion of ventilator-associated pneumonia. Endotracheal aspirate culture was done, which showed growth of Pseudomonas, after which ceftriaxone was upgraded to piperacillin-tazobactam. Coagulation profile revealed INR 1.6 with thrombocytopenia (platelets 90,000/µL), suggesting evolving coagulopathy but there was no obvious disseminated intravascular coagulation. Upper gastrointestinal endoscopy performed on day 7 (due to odynophagia and suspicion of caustic injury) showed diffuse mucosal congestion without ulcers in the esophagus or stomach.

Despite two sessions of hemodialysis, steroid therapy (Methylprednisolone 1g IV for 3 days followed by prednisolone 1mg/kg/day in tapering doses), broad-spectrum antibiotics, and ventilatory support, the patient developed progressive multi-organ failure. A summary of blood investigations is described in Table 1. On day 10, she had worsening hepatic dysfunction (bilirubin 133 µmol/L, AST 404 U/L, ALT 421 U/L), refractory hypoxemia, and succumbed to respiratory failure and shock.

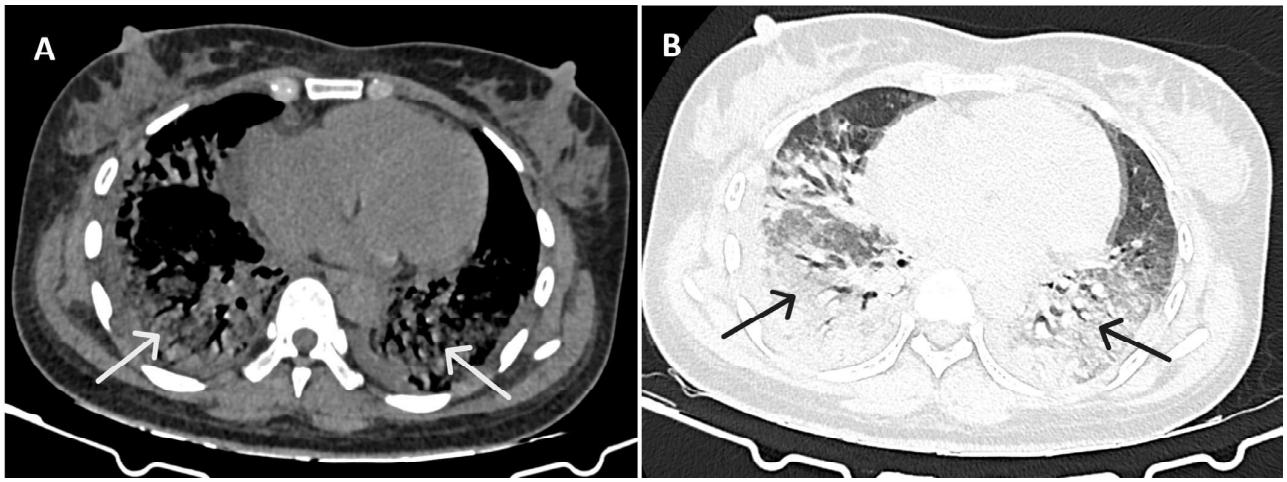

Figure 2: Computerized tomography (CT scan) **A:** Soft tissue window; **B:** Lung Window showing extensive consolidation and ground glass opacities and septal thickening predominantly involving lower lobes and posterior segments.

Table 1. Serial blood investigation results of the patient.

Parameters	At presentation	Day 3	Day 7	Day 10	Reference range
Total Leukocyte Count (TLC)	4800	8100	11300	10100	4000-11000 (/mm^3)
Hemoglobin (Hb)	11.6	11.3	11.4	9.9	12.5-15 (gm%)
Urea	3.3	20	23.5	11.2	2.8-7.2 (mmol/L)
Creatinine	65	656	650	107	58-96 (µmol/L)
Total Bilirubin (TB)	5	68	63	133	5-21 (µmol/L)
Direct Bilirubin (DB)	1	45	43	88	<4 (µmol/L)
Alanine aminotransferase (ALT)	34	214	167	421	0-35 (U/L)
Aspartate aminotransferase (AST)	30	122	109	404	0-35 (U/L)
Alkaline phosphatase (ALP)	42	168	205	625	30-120 (U/L)
Serology	Non-reactive	-	-	-	

DISCUSSION

Poisoning is the second most common method of suicide in Nepal after hanging.[2] Although Nepal has banned the sale of paraquat commercially, it is available in the market due to a lack of proper regulation, leading to multiple poisoning cases. However, the exact impact of PQ as a public health problem is yet to be evaluated.[2] After completely banning paraquat sales in Korea in 2012, overall pesticide-related suicide mortality decreased from 5.26 to 2.67 per 100,000 population between 2011 and 2013.[1] We present a case of a young female following suicidal ingestion of PQ with typical clinical features of poisoning.

The mode of poisoning in PQ includes both suicidal and accidental, and the most common route of intake is oral, with less common routes being intramuscular, intravenous, and subcutaneous.[2] Whatever the mode of poisoning and route of intake, PQ is highly toxic and quickly spreads to body tissues, with lungs and kidneys having the highest concentration.[4] The toxicity arises from the generation of intracellular reactive oxygen species, causing cellular damage through lipid peroxidation, activation of nuclear factor kappa B, mitochondrial dysfunction, and apoptosis. These processes are responsible for pulmonary fibrosis, nephrotoxicity, and hepatotoxicity.[2,5]

Table 2. Clinical features of Paraquat ingestion according to doses ingested.

Grading of poisoning	Amount ingested (mg per kg body weight)	Clinical features
Mild	<20	Gastrointestinal symptoms
Severe	20-40	Acute renal failure, Acute hepatic involvement in the form of jaundice, elevated transaminases, Acute lung injury, Progressive pulmonary fibrosis, Respiratory failure
Fulminant	>40	Multiorgan failure (renal failure, cardiac arrhythmias, convulsions, oesophageal perforations) within hours to a few days of ingestion

The clinical features of PQ poisoning are classified based on the amount consumed as elaborated in Table 2.[6,7] In addition to these features, patients may present with a mucosal ulceration in the oral cavity and tongue, characteristically known as "Paraquat tongue" as was present in our patient at the time of presentation.[3] In a case series by Mishra et. al., diffuse inter- and intra-lobar septal thickening with superimposed areas of ground-glass opacities was the predominant finding within 7 days. Other early findings included pneumomediastinum, pneumothorax, and pleural effusions.[4] Later consolidation occurs in association with bronchiectasis, and fibrotic change may be seen on a CT scan as explained by Huh et. al. in their study.[4,8] Pulmonary function tests (PFT), if performed, show a mild restrictive pattern initially, but the lung function may deteriorate rapidly within one month of ingestion.[8] In our case, the initial X-ray was normal, but the patient subsequently developed heterogeneous opacities along with septal thickening. CT also revealed ground-glass opacities, making the findings in line with the current literature.

Blood investigations such as plasma PQ concentration, leukocyte count, blood urea nitrogen, serum creatinine, alanine transaminase, and aspartate transaminase, along with urinary PQ concentration, serve as important predictors of outcome.[7] Management of PQ poisoning is challenging owing to a lack of a specific antidote or chelating agent; thus, only supportive management is possible.[7] General measures include gastric lavage with fuller's earth or activated charcoal to prevent absorption if the patient presents within 2-4 hours of ingestion. This was not performed in our case due to delayed presentation.[9] This can be followed by extracorporeal methods such as hemoperfusion/hemodialysis to enhance elimination. Other methods include the use of immunosuppressants (cyclophosphamide, steroids) and antioxidants (Vitamin E, Vitamin C, N-acetyl-cysteine, deferoxamine, and salicylic acid). Another important aspect of management is clinical monitoring of patients and looking for signs of acute renal failure, liver toxicity, and acute respiratory failure.[9,10] Anthrahydroquinone - 2 - 6 - disulfonate (AH2QDS) has been proposed as a potential antidote by some studies, as it provides mitochondrial protection, reduces oxidative stress, and improves survival in rat models, but more studies are required to demonstrate its effectiveness in humans.[2]

However, none of these methods is the definitive therapy and does not guarantee favorable outcomes. We performed supportive management with oxygen supplementation, intravenous steroids and hemodialysis, but the outcome was unfavorable.

CONCLUSION

Paraquat is a herbicide with serious toxicity concerns and can be fatal even when consumed in small amounts. Even if patients are stable at the time of presentation, their condition can worsen dramatically within a short period of time, thus demanding high level of vigilance. Supportive care and close monitoring are the cornerstones of management.

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