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CASE REPORT

Delayed hemoadsorption in paraquat poisoning: A case report

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ABSTRACT. Paraquat (PQ) is a highly lethal bipyridyl herbicide. Its toxicity is driven by the generation of superoxide radicals, which lead to severe cellular damage. The ingested dose remains the factor with the greatest impact on mortality, and additional prognostic determinants include age, route of exposure, intentionality, and delays in initiating treatment. Although several interventions have been explored, including gastrointestinal decontamination, immunomodulation, and antioxidant therapy, extracorporeal support techniques continue to accumulate evidence for their use even when initiated late. We describe a successful case of delayed extracorporeal renal support therapy after ingestion of a lethal dose of PQ.

Keywords: *Paraquat; Pyridinium compounds; Hemoadsorption; Sorption detoxification; Renal replacement therapy.*

Paraquat (1,1′-dimethyl-4,4′-bipyridyl dichloride; PQ) is a nonselective, highly toxic quaternary ammonium herbicide with a molecular weight of 257 Da.[1,2] The oral route is the most common pathway of poisoning in intentional exposures, whereas cutaneous and inhalational routes predominate in accidental cases. After ingestion, PQ is distributed through the bloodstream to the kidneys, liver, heart, and lungs. Peak serum concentrations are reached within 2 to 4 hours and may remain stable for as long as 30 hours. The volume of distribution (VD) ranges from 1.2 to 1.6 L/kg, and elimination may extend up to 48 hours.[3] In the lungs, its half-life is prolonged because the compound is actively taken up by epithelial cells, initiating the generation of reactive oxygen species.[4,5]

PQ poisoning can rapidly lead to critical illness requiring multiorgan support because of the interdependence of pulmonary, cardiac, renal, and hepatic function.[6] Hemoadsorption (HA) can be used as an elimination technique for selected xenobiotics through solute adsorption and has emerged as a promising adjunct in the multidisciplinary management of paraquat-poisoned patients.[7] A xenobiotic is amenable to removal by HA when it meets specific physicochemical and pharmacokinetic criteria, including a VD of less than 1 L/kg, lipophilicity, high protein-binding capacity, and a molecular weight below 5,000 Da.[8,9] In this case, PQ was an appropriate candidate for extracorporeal removal owing to its low molecular weight and lipid solubility.

CLINICAL CASE

An 18-year-old male farmer with no known medical history was evaluated after intentionally ingesting 25 mL of a 20% w/v paraquat solution, corresponding to an estimated dose of 41 mg/kg body weight. One hour later, he consumed 200 mL of vegetable oil and 500 mL of milk, followed by multiple episodes of emesis. He presented to a primary care facility 2 hours after ingestion, already reporting progressive dysphagia. No gastrointestinal (GI) decontamination was performed. As his clinical condition deteriorated, he was airlifted to our hospital 48 hours after the exposure.

At the time of transfer, the patient reported odynophagia, epigastric pain, and nausea. Physical examination revealed gingival bleeding, an erythematous tongue, and patchy whitish exudates extending into the oropharynx (Fig. 1).

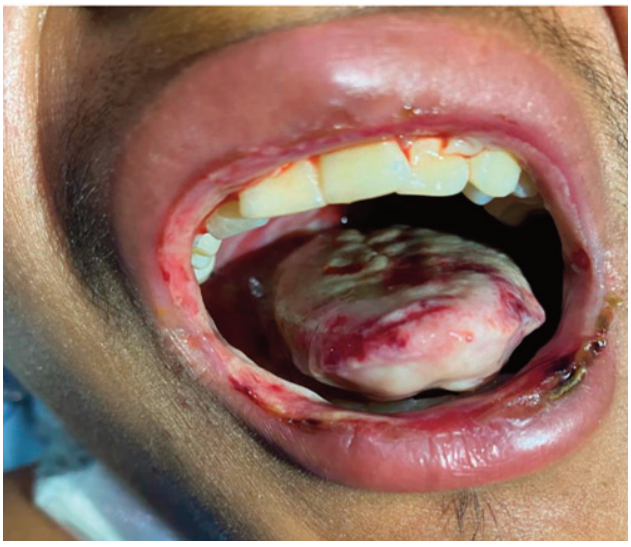

Figure 1. Paraquat tongue characterized by marked lingual edema, extensive ulceronecrotic lesions, and a thick yellow-white fibrinous pseudomembrane with multifocal hemorrhagic areas on the dorsal and lateral surfaces. Associated swelling, erosive lesions, and hemorrhagic crusts are also visible on the labial mucosa (Credits: courtesy of the authors).

Laboratory studies showed elevated creatinine and blood urea nitrogen levels, leukocytosis, and metabolic acidosis. The peak creatinine level was 3.65 mg/dL at 72 hours of illness, and lactate levels remained within normal limits throughout. Serum PQ concentrations could not be measured. Details of the laboratory findings over time can be found in Table 1.

Treatment was initiated with 3 g of methylprednisolone administered in three divided doses, along with acetaminophen, tramadol, ondansetron, and omeprazole. After evaluation by a nephrologist, extracorporeal treatment (ECTR) was initiated for xenobiotic removal despite a 62-hour delay after ingestion. A 6-hour HA session performed in series with intermittent hemodialysis (HD) was carried out using a conventional machine (4008S Classic, Fresenius Medical Care). HD was delivered with a high-flux Optiflux dialyzer, and HA was performed using a HA230 ion-exchange resin cartridge (Jafron Biomedical, Zhuhai, Guangdong, China).

Chest computed tomography showed no apparent abnormalities. Nonetheless, spirometry revealed moderate restrictive impairment, the most characteristic finding, reflecting loss of lung volume due to fibrosis. Peripheral airway obstruction was also present, likely due to ventilatory dysfunction. The patient was subsequently discharged 10 days after admission to our hospital. A summary of the therapeutic interventions is shown in Fig. 2.

During clinical follow-up, 1 month after PQ exposure, esophagogastroduodenoscopy revealed chronic follicular gastropathy. No pulmonary injury was documented at that time.

DISCUSSION

Epidemiology

A comprehensive analysis of mortality trends from acute pesticide poisoning in Mexico (2000–2021) identified self-poisoning as the leading cause of death. The highest mortality rates were observed among individuals aged 15 to 19 years, likely due to their heightened susceptibility to suicidal behavior combined with easy access to highly lethal pesticides.[10]

Survival and prognosis

Several factors influence survival in patients poisoned with PQ: the patient's intent, which correlates with the amount of xenobiotic ingested; concomitant food intake, which may delay GI absorption; the time to presentation at

TABLE 1. Patient biochemical and blood gas parameters.

	Creatinine (mg/dL)	BUN (mg/dL)	WBC (x10^9/L)	HGB (g/dL)	HCT (%)	pH	PCO_2 (mmHg)	PO_2 (mmHg)	HCO_3- (mmol/L)	Lactate (mmol/L)
Admission	3.6	29	11,400	15	44	7.38	29.6	66.4	17.6	0.9
After HA	3.65	74	6,700	14.6	41	7.37	30.8	79.5	18.3	1.1
Final	0.87	22	5,600	12.9	37.2	7.48	30.2	68.6	22.6	0.7

HA: hemoadsorption; BUN: blood urea nitrogen; WBC: white blood cells; HGB: hemoglobin; HCT: hematocrit; PCO_2: partial pressure of carbon dioxide; PO_2: partial pressure of oxygen; HCO_3-: bicarbonate.

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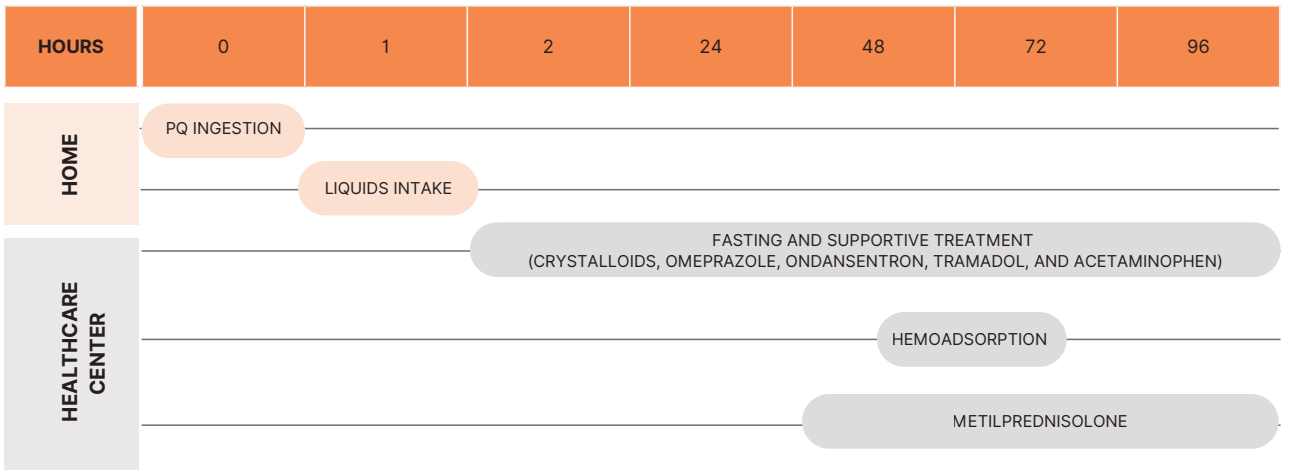

PQ: paraquat.

Figure 2. Timeline of interventions during the first 96 hours of hospitalization.

a medical facility; the use or omission of GI decontamination; and the application of elimination-enhancing techniques.[5-11] The lethality of PQ derives from both its intrinsic toxicity and the absence of an effective treatment.[12] Ingestion of 10 to 20 mL of the 20% w/v formulation is equivalent to approximately 55 mg/kg of body weight in a 70-kg adult and can be fatal.[13]

To assess prognosis, the Severity Index of Paraquat Poisoning (SIPP) should be used, which is calculated by multiplying the PQ concentration (mg/L) by the time elapsed since ingestion (hours). Patients with a score below 10 have a substantially higher likelihood of survival.[14] Similarly, the use of a qualitative colorimetric test with sodium dithionite (paraquat dithionite solution, PDS) allows correlation of urine discoloration with serum PQ concentrations and is particularly valuable in hospitals lacking the capacity for quantitative PQ assays.[15]

Other prognostic indicators have been proposed in PQ poisoning. In a cohort of 143 patients treated in a Chinese emergency department, a serum lactate level ≥2.95 mmol/L was associated with poor prognosis.[16] The APACHE score has also been evaluated as a predictor of mortality, with a proposed cutoff of >9 points.[17] In addition, clinical severity correlates with the amount ingested and is categorized as mild (<10 mL), moderate (10–40 mL), or severe (>40 mL).[18] Finally, patients with PQ poisoning who have a base excess greater than –5.5 are at increased risk of mortality.[19]

Extracorporeal therapies

The removal o PQ using different extracorporeal techniques, including HA, conventional HD, and continuous renal replacement therapies (CRRT) such as continuous venovenous hemofiltration (CVVH), has been used to treat intoxicated patients, with variable survival outcomes.[20]

In a study from Taiwan involving 207 patients with severe PQ poisoning, early HA (within <4 hours) using activated charcoal (AC) as the adsorbent was associated with a significant reduction in mortality risk.[21] In a retrospective study of 101 patients, Rao et al. found that mortality was significantly higher in those receiving only GI decontamination with supportive care (92.1%) compared to patients treated with HA within six hours of exposure (42.9%), with early HA associated with improved survival.[22] In addition, a multicenter retrospective study in China compared HA with HA plus CVVH, finding that serum PQ concentrations declined more rapidly and reached significantly lower levels in the HA + CVVH group.[23] These studies suggest that early initiation of HA (within 4 to 6 hours after ingestion) may improve survival. Futhermore, a separate multicenter study of 213 patients conducted in Taiwan found that early, multiple HA sessions were independently associated with increased survival after adjustment for age and renal function.[24]

In 2023, Ballesteros et al. reported a case of severe paraquat poisoning during pregnancy. Treatment was initiated 34 hours after herbicide ingestion and included HA and CVVH, along with cyclophosphamide and methylprednisolone. No adverse events attributable to extracorporeal therapy were reported, and the pregnancy resulted in a successful outcome.[25]

Finally, other extracorporeal therapies, such as intermittent HD or hemofiltration (HF), are likely to be nearly as effective as HA in enhancing elimination at comparable

TABLE 2. Clinical management of PQ poisoning.

	Intervention	Key points
GI decontamination	AC	1 g/kg (ideally <4 h)
	Fuller's Earth	2 g/kg; same as above
	Nasogastric tube	Early presentation; aspirate stomach before AC
	Gastric lavage/Forced emesis	Contraindicated
Supportive care	Resuscitation	Follow standard guidelines; avoid oxygen unless hypoxic; crystalloids (usually 2 or 3 L); correct electrolytes
	Advanced procedures	Intubation; ventilation; vasopressors only if active treatment pursued; endoscopy
	Palliative care	Analgesia (fentanyl/morphine); oxygen for dyspnea; focus on comfort
Extracorporeal removal	HA/HD/HF	Early initiation (<4 h); may combine therapies; follow with continuous technique to reduce rebound
Adjunctive therapy	Antioxidants	Acetylcysteine; controversial: deferoxamine, vitamin C/E and melatonin (low toxicity, may reduce oxidative damage)
	Anti-inflammatories/Anti-fibrotics	Dexamethasone (8 mg IV q8h, up to 5 wks if severe); cyclophosphamide + glucocorticoid not recommended; limited evidence for others

GI: gastrointestinal; AC: activated charcoal; HA: hemoadsorption; HD: hemodialysis; HF: hemofiltration.

blood flow rates. Moreover, when acute kidney injury is present and renal replacement therapy is indicated, HD can be coupled with HA,[26] as in our case. A comprehensive approach to the management of PQ poisoning is presented in Table 2.

Following ingestion of a lethal dose, rapid intervention is crucial; therefore, elimination therapies should not be delayed. The use of prognostic scoring systems supports clinical decision-making and should be regarded as a key component in the management of these patients. Early identification of target-organ damage should prompt consideration of interventions aimed at xenobiotic removal to prevent further injury. In this case, factors that likely contributed to the patient's survival included young age, absence of comorbidities, lack of pulmonary involvement, reduced xenobiotic absorption due to emesis, and ingestion with food, which may have delayed peak serum concentrations and facilitated elimination from the vascular compartment.

LIMITATIONS

This report describes a single PQ-poisoned patient treated with HA. Because serum PQ concentrations were not available at our institution, we were unable to calculate SIPP or quantify the amount of PQ removed through HA. These limitations should be taken into account when interpreting the clinical course and treatment response in this case.

CONCLUSIONS

When serum PQ concentration is unavailable, the ingested dose is an important predictor of mortality and immediate initiation of therapy is essential. AC is a first-line intervention in acute PQ poisoning, as it significantly reduces systemic absorption when administered promptly after ingestion. Early HA, especially within 4–6 hours of ingestion, is the most effective extracorporeal technique for PQ elimination and improves survival. Delayed HA may be considered in selected cases, but its efficacy is reduced and further evidence is needed to define its role.

Conflicts of interest

The authors declare no conflicts of interest.

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