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Patients Survival After Paraquat Poisoning: A Report of Three Cases

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ABSTRACT

Background: Paraquat poisoning is a common and often fatal herbicide poisoning in society. This study presents a clinical case series of patients who survived after paraquat poisoning.

Cases Presentation: This study evaluated patients hospitalized between March 2016 and March 2021 with paraquat (PQ) poisoning who survived. Out of 115 patients with PQ poisoning, three cases of severe toxicity with an average age of 24.33 years are presented here. The urinary sodium dithionate test result was positive in all three surviving patients. All patients arrived at the poisoning emergency center within an hour of ingestion and received gastric lavage and charcoal therapy. They were also treated with corticosteroids, N-acetylcysteine (NAC), vitamins C and E, Curcumin, and Livergel. Hemodialysis was performed for the patients, with one undergoing hemodialysis and hemoperfusion after ingesting 250 mL of PQ 20%. After a six-month follow-up, all surviving patients were in good health.

Conclusion: Various factors, such as early admission after exposure, prompt gastrointestinal (GI) decontamination, corticosteroids with Curcumin and Livergel, antioxidants, hemodialysis, and hemoperfusion in one case may have contributed to the survival of patients with PQ poisoning in this study. However, individual vulnerability should also be considered a crucial factor requiring further investigation.

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Introduction

Paraquat (PQ) is a fast-acting, non-selective herbicide [1]. PQ poisoning is the leading cause of mortality in developing countries owing to the high toxicity of pesticides [2-4]. Mortality rates in acute poisoning range from 33% to 78% [5]. Ingestion and skin contact are primary causes of lethal poisoning [1]. Intentional ingestion of PQ is a standard method of suicide using herbicides in Central Iran [2].

Toxicity mechanisms are primarily related to oxidative damage, immune system activation, and inflammatory mediators [6]. Acute respiratory failure caused by oxidative damage and pulmonary fibrosis is the leading cause of death due to PQ poisoning [7]. PQ concentration in the lungs is more than 10 times higher than in the plasma.

No definitive treatment exists for PQ toxicity [8]. Conventional treatment protocols recommend reducing PQ absorption in the GI tract through gastric lavage and using adsorbents, such as activated charcoal (AC). Increasing PQ removal from plasma can be achieved through hemodialysis, hemoperfusion, and continuous veno-veno hemofiltration. Management of PQ poisoning is primarily supportive [7]. Here, we describe a case series of PQ poisoning survivors who were admitted to the referral poisoning emergency center of Khorshid Hospital from March 2016 to March 2021.

Cases Presentation

The current study was a retrospective analysis of all patients admitted to the Poisoning Emergency Center of Khorshid Hospital, affiliated with the Isfahan University of Medical Sciences, Isfahan, Iran. Patients with paraquat poisoning who presented within 24 hours of acute paraquat poisoning between March 2016 and March 2021 and survived were included in the study. Follow-up was conducted by contacting the patients in May 2022. PQ poisoning was confirmed by a positive urinary sodium dithionate test [7]. During the study period, 115 patients were hospitalized for paraquat poisoning. Three surviving patients with severe poisoning were included in the study. The following information was extracted from the patient's files, recorded on a data-gathering form, and entered into a computer.

Patients' basic demographic information, such as age, sex, marital status, occupation, place of residence, history of addiction and physical illness, the season of poisoning, route and type of exposure, amount of PQ ingested (as reported by the patient or their relative), interval time between PQ exposure and admission, and length of hospital stay were collected. The severity of intoxication was classified as follows: Mild toxicity, which included being asymptomatic or experiencing only gastrointestinal (GI) symptoms and ingesting <7.5 mL of PQ 20%; moderate toxicity, which included ingesting 7.5-15 mL of PQ 20%; and severe toxicity, which included rapidly progressive multiple organ damage and ingesting more than 15 mL of PQ 20% [9]. Clinical manifestations and vital signs at admission, laboratory tests, including blood glucose, complete blood count, venous blood gas analysis, liver enzymes (alanine transaminase [ALT], and aspartate transaminase [AST]), renal marker function (creatinine and blood urea nitrogen), coagulation tests (prothrombin time [PT]/partial thromboplastin time [PTT]/international normalized ratio [INR]), serum electrolytes (sodium, potassium, calcium, and magnesium), and repeated urinary sodium dithionate were recorded. The severity of poisoning, according to the sodium dithionate test, was also categorized as mild, moderate, or severe toxicity based on the intensity of the blue color from 1 to 3 [5, 9]. Polycythemia, anemia, leukocytosis, thrombocytopenia [10] and hypokalemia, hypocalcemia, hyponatremia were defined as mentioned previously [11].

As none of the patients experienced hypoxia, airway management, and ventilation were unnecessary. Intravenous crystalloid serum administration (15–20 mL/kg in 20-40 minutes) was repeated as necessary until adequate urine output was achieved [2]. All patients were prescribed antacids, intravenous proton pump inhibitors, and pantoprazole 40 mg twice daily, as needed. Endoscopy was performed in patients who required the procedure [2].

If the patient was referred within four hours of ingestion, gastric lavage and charcoal administration at a rate of 1 g/kg were ordered during hospitalization. The treatment protocol also included N-acetylcysteine (NAC) 150-300 mg/kg 24-hour infusion daily (Aurum Pharmaceutical Limited, Romford Essex, England), vitamin E 300 units intramuscularly every 12 h (Osvah Pharmaceutical Company, Tehran, Iran), and vitamin C 150 mg/h intravenous infusion daily (Daroupakhsh Pharmaceutical Company, Tehran, Iran). This treatment protocol was administered daily during hospitalization. Curcumin 1 g every 8 hours (500 mg capsule of Dineh Iran Pharmaceutical Company, Tehran, Iran), silymarin 140 mg tablets daily (Livergol, Goldaru Herbal Pharmaceutical Company, Isfahan, Iran), and methylprednisolone (Jaber Pharmaceutical Company, Tehran, Iran) at the rate of 1

g daily intravenous infusion for up to 3 days gradually reducing dosage every three days were prescribed.

Hemodialysis was performed for 6-8 hours (between 1 and 3 times) (Fresenius 4008, Fresenius Medical Care, Homburg, Germany) with a membrane surface of at least 1.6-1.8 m^2 and a blood flow rate greater than 250-300 mL/min. Repeated dialysis was performed based on the positivity of the dithionate test or the presence of acute renal failure. Hemoperfusion with polystyrene resin for 3-4 hours once daily (through femoral venous catheters at a blood flow rate of 250-300 mL/min) was also performed for one patient. An upper GI endoscopy was performed on one patient. Statistical analysis of data is expressed as Mean±SD, frequency, and percentage.

Results

Table 1 presents the demographic characteristics of the three surviving patients with severe PQ poisoning. All patients were men, with a mean age of 24.33 years (min-max: 16-36). None of the patients had any medical history or a history of addiction to various substances such as heroin, cigarettes, opium, methamphetamine, and methadone.

Table 1. Demographic details of paraquat poisoning patients

Variables		No. (%)
Gender	Male	3(100)
Age (y)	Min-max	16-33
	Mean±SD	24.33±9.71
Marital status	Single	2(66.7)
	Married	1(33.3)
Occupation	Self-employment	2(66.7)
	Student	1(33.3)
Residence	City	1(33.3)
	Suburb	2(66.7)
Season of poisoning	Summer	2(66.7)
	Winter	1(33.3)
Intention of poisoning	Suicide	3(100)
Route of exposure	Oral	3(100)
Time from PQ poisoning to admission (h)	Min-max	0.5-1
Amount of paraquat ingested (mL)	Min-max	45-250
	Mean±SD	165±106.88
Severity of poisoning	Severe	3(100)
Hospitalization (d)	Mean±SD	7.33±0.57
	Min-max	7-8
Time admitted to ICU (d)	2nd day	2(66.7)
	3rd up	1(33.3)

ICU: Intensive care unit; PQ: Paraquat.

All three patients were orally poisoned with PQ and experienced suicidal ideation. The mean amount of PQ consumed was 165±106.88 mL (min-max: 45-250 mL) of 20% PQ. The Mean±SD length of hospital stay was 7.33±0.57 days. All patients were admitted to the intensive care unit (ICU) for 1-4 days. Upon admission, vital signs were typical, except for one patient with tachypnea.

Based on exposure to PQ, all patients experienced severe poisoning. All three patients arrived at the poisoning emergency within an hour of ingestion. Upon admission, all the patients exhibited GI symptoms with vomiting. At admission, the Mean±SD values for blood urea nitrogen (BUN), creatinine (Cr), AST, ALT, creatine phosphokinase (CPK), lactate dehydrogenase (LDH), PT, and PTT were 18.33±8.38 (mg/dL), 0.90±0.1 (mg/dL), 22.33±1.52 (U/L), 23.66±20.40 (U/L), 207±84.7 (U/L), 226±1.25 (U/L), 16.4±3.60 (s) and 60.6±51.7 (s), respectively. Liver enzyme levels were elevated in one patient, while elevated blood urea levels were observed in one patient. Lung involvement was not observed in any of the patients.

Normal electrolyte levels (potassium and sodium) have been reported, with only one patient exhibiting hyperglycemia. Upon admission, one patient developed respiratory alkalosis. Within 24 hours of admission, one patient required intubation.

The urinary sodium dithionate test resulted in a deep blue (3+) color in two patients (66.7%), while another patient showed a light blue (2+) color. All patients underwent gastric lavage and charcoal therapy and received corticosteroids, NAC, and vitamins C and E at the indicated doses. In addition to these treatments, all patients were administered curcumin and livergel.

Hemodialysis was administered to all the patients during hospitalization. One patient who had ingested 250 mL of PQ 20% received hemodialysis for 8 h and hemoperfusion for 4 h due to severe toxicity. Endoscopy revealed an ulcer in the distal esophagus. One patient did not undergo hemodialysis or hemoperfusion. Six months after discharge, all patients were in good health.

Discussion

PQ poisoning is the leading cause of herbicide poisoning [7]. Despite advancements in medical treatment, it remains a significant cause of mortality in emergency departments [12], accounting for approximately 55% of in-hospital fatalities [13].

This study examined three patients with severe toxicity who survived PQ poisoning. The toxicity of acute PQ poisoning depends on various factors, including the amount of exposure, absorption, excretion, and individual vulnerability [5]. The survival of our patients may be attributed to a combination of the following factors.

The results revealed that all patients with paraquat toxicity arrived at the hospital within the first hour. Additionally, initial treatments, such as gastric lavage and AC administration, were performed for most patients who reached the emergency department within one hour of exposure. Early GI decontamination can reduce the amount of absorbed PQ and its toxicity.

Antioxidants, including NAC, vitamin C, and E, were prescribed to all the patients with poisoning. Several other studies have reported the use of various antioxidants. NAC has been used as a free radical scavenger, limiting the glutathione synthesis rate in pulmonary alveolar pneumocytes and reducing pulmonary inflammation [2]. Vitamin C and E, possessing antioxidant properties, normalize biochemical and histological parameters [9] and prevent the progression of pulmonary fibrosis [13]. Additionally, a previous report showed that silymarin and curcumin improved patient outcomes [6]. However, some studies have indicated that these treatments offer little benefit in term of survival and are unable to reduce mortality [7, 13]. Therefore, subsequent studies have explored using different antioxidant doses [7].

Our results showed that all patients received corticosteroids. Although there is limited evidence regarding the therapeutic dose and duration of treatment for immunosuppression caused by glucocorticoid therapy [2, 14], initiating treatment with corticosteroids as soon as possible after high-dose exposure and gradually reducing the dose may have the best effect on PQ toxicity, as previously reported [14]. However, some studies have shown that immunosuppressive therapies do not exert beneficial effects [6, 15, 16].

Our surviving patients underwent hemodialysis, and one patient who ingested a high amount of PQ received a combination of hemodialysis and hemoperfusion in the early hours. Several studies have also suggested using extracorporeal removal techniques, such as hemodialysis and hemoperfusion, to increase toxin excretion and maintain kidney function [17]. Hemodialysis can be used in cases where hemoperfusion is unavailable [2, 4]. However, owing to the unavailability of hemoperfusion in our department, all patients underwent hemodialysis.

Patient survival can be influenced by several factors, including young age, low levels of PQ exposure, short time from exposure to decontamination in patients with a high amount of ingested PQ, and typical vital signs [5]. In our study, patients who survived were young. The time between exposure and admission was approximately one hour. In addition, all three patients' liver and kidney functions were normal upon admission. Previous studies have indicated that low levels of leukocytosis, acidosis [5], hypoxia, and hypotension [8] are good prognostic factors. Among survivors, common late complications included renal and hepatic insufficiency upon admission [5], esophageal erosions, esophagitis, and strictures. Progressive pulmonary fibrosis is another significant late complication of paraquat toxicity, which can lead to death 2–3 weeks later owing to hypoxia and respiratory failure [18].

However, early management to prevent toxin absorption, increase the rapid excretion of toxins, and use hemodialysis [19] can improve patient survival. The reasons why our patient survived could also be the quantity of poison taken, the absence of any history of kidney or hepatic failure, and the early supportive treatment provided. Curcumin and livergel were also administered to the patients. In vitro, curcumin treatment prevented paraquat-induced reactive oxygen species and apoptotic cell death [20]. Additionally, curcumin treatment abolished the PQ-induced reduction in lung angiotensin-converting enzyme (ACE) and bronchoalveolar lavage (BAL) cells and lung glutathione levels [21]. These results indicate that curcumin has significant therapeutic implications in facilitating early suppression of PQ lung injury.

Conclusion

Several factors, including the time from exposure to admission, early decontamination, implementation of paraquat elimination techniques, and administration of antioxidants and anti-inflammatory medications, may contribute to the survival of patients with paraquat poisoning. In addition to these factors, individual vulnerability should also be considered a critical risk factor that needs further investigation. Randomized, large-sample trials are necessary to determine the effectiveness of various variables and treatment methods for paraquat poisoning [13].

Limitations

This study has some limitations. We did not categorize the severity of toxicity based on serum PQ level at admission, which is the best criterion for defining toxicity severity. However, it should also be mentioned that serum PQ measurement is not available in many centers.

Ethical Considerations

Compliance with ethical guidelines

This study was conducted by the ethical standards of the 1964 Declaration of Helsinki and its later amendments or comparable standards. A sentence confirming that written informed consent was obtained from all participants or, if participants were under 16 years of age, from a parent or legal guardian. This article presents registry design data approved by Isfahan University of Medical Sciences, Isfahan, Iran (Code: IR.ARI.MUI.REC.1400.031).

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Authors' contributions

All authors contributed equally to the conception and design of the study, data collection and analysis, interception of the results, and manuscript drafting. Each author approved the submission of the final version of the manuscript.

Conflict of interest

The authors declared no conflict of interest.

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