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CASE REPORT

Paraquat Poisoning: Clinical Insights from a 33-year-old Iranian Survivor with Lung Injury

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Abstract

Introduction: Paraquat is one of the most widely used herbicides worldwide. It is quick-acting and non-selective, killing green plant tissue on contact. Paraquat poisoning is a critical condition with a high mortality rate due to its extreme toxicity, particularly with regard to its impact on lung health.
Case Presentation: This case report delineates the clinical progression and therapeutic interventions for a 33-year-old Iranian male who survived a substantial ingestion of paraquat, an occurrence that is frequently associated with deleterious outcomes. The patient was admitted following a suicide attempt involving paraquat, manifesting symptoms such as nausea, vomiting, and erythematous rashes. Despite receiving immediate and intensive treatment, including hemodialysis, antioxidant therapy, and comprehensive supportive care, the patient experienced acute lung injury on the fifth day of hospitalization, as confirmed by computed tomography (CT) scans.
Discussion: This case underscores the critical importance of early intervention and ongoing monitoring in the management of paraquat poisoning, particularly in reducing the risk of severe complications like lung injury. While the patient was ultimately discharged in stable condition, the development of lung damage underscores the need for further research into more effective therapies for paraquat toxicity.
Conclusion: This report contributes to the limited clinical knowledge surrounding paraquat poisoning, offering valuable insights for healthcare providers dealing with similar cases.

Keywords: Paraquat poisoning, Lung injury, Toxic effect, Lung CT Scan

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INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-bipyridinium dichloride) has been extensively utilized as a contact herbicide in developing nations for several decades. While it can be safe when applied correctly, exposure to the substance can lead to severe poisoning, which has a high mortality rate. Paraquat poisoning typically occurs due to intentional, occupational, or accidental exposure. The majority of fatal cases globally, particularly in developing nations, are attributed to self-poisoning through oral ingestion [1]. This can result in serious health issues such as acute renal failure, liver inflammation, lung scarring, and even death, depending on the degree of exposure [2]. The amount that can be fatal for adults is quite small. Specifically, just 5 to 15 milliliters of a 20% PQ aqueous solution can be lethal. The primary organ affected is the lung, where the concentration of paraquat is found to be 10 to 90 times greater than that observed in plasma, and respiratory failure from lung injury is the most common cause of death [3]. The purpose of this case report is to highlight the clinical presentation, management, and outcomes associated with paraquat poisoning, with a particular focus on lung involvement and the therapeutic strategies employed to address this severe complication.

CASE REPORT

The patient was a 33-year-old man who ingested approximately a cup of paraquat, an agricultural poison, in a suicide attempt four hours prior to his arrival. He was referred to our emergency department from a small town located 50 kilometers from our poison control center. Upon admission, the patient reported experiencing nausea, vomiting, and a burning sensation in his eyes and mouth, along with burns on his body. He stated that he vomited immediately after ingesting the paraquat and had also poured the poison onto his chest and hands. He did not bring up any issues with swallowing, excessive drooling, or voice problems. Additionally, there was no record of abdominal pain, seizures, or fever. Notably, the patient had a history of suicide attempts involving agricultural pesticides from 12 years ago. During the physical examination, the patient measured 165 cm in height and weighed 55 kg, resulting in a BMI of 20.20.

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His vital signs were stable: a heart rate of 74 beats per minute, a temperature of 37 degrees Celsius, blood pressure of 118/70 mmHg, and a respiratory rate of 14 breaths per minute. The patient appeared plethoric and showed slight agitation. There were itchy, red rashes scattered across his chest and distal forearms on both sides, with some areas having small, ruptured blisters, particularly around the eyes (Figure 1). A thorough examination of the oral cavity revealed the presence of numerous small superficial ulcers predominantly located on the tongue, hard palate, and buccal mucosa. His pupils were mid-sized and responsive. Cardiac and pulmonary auscultation demonstrated no pathological findings. The abdominal examination indicated the absence of tenderness, and bowel sounds were present. Muscle strength and reflexes were assessed as normal and within the expected range.

The patient underwent pulse oximetry and cardiac monitoring, and subsequently, the patient's dietary regimen was modified to NPO (nil per os). Blood glucose monitoring was conducted, alongside a routine workup for potential poisoning, which also included serum and red blood cell cholinesterase assessments. Laboratory data of the patient is illustrated in Table 1. The patient received fluid therapy and was administered 40 mg of injectable pantoprazole daily. Additionally, 100 mg of vitamin E was administered via injection twice daily, along with N-acetylcysteine (NAC) at a dosage of 10 mg twice daily, and dexamethasone at a dosage of 8 mg twice daily. A psychiatric consultation was also requested. A Shaldon catheter was successfully inserted into the right internal jugular vein for dialysis using the Seldinger technique. There was no sign of a hematoma or active bleeding at the site of the Shaldon catheter. Additionally, a controlled chest X-ray was performed, which confirmed that the Shaldon catheter placement was uncomplicated, with no evidence of pneumothorax. Following a negative HBs-antigen test, the patient underwent hemodialysis sessions lasting 6 hours, 4 hours, and 4 hours on the second, fourth, and fifth days, respectively. Given that the patient's serum potassium level was recorded at 2.8 mEq/L on the fifth day of hospitalization, a prescription for 10 cc of potassium chloride syrup was initiated to be administered every 8 hours. The patient was referred for a skin consultation due to skin lesions. The consultation revealed that the lesions were caused by the toxic effects of herbicide exposure. As a result, the patient was prescribed Rivanol solution and zinc oxide cream. Moreover, the patient was monitored for the healing of the lesions and any potential secondary infections that could arise.

The patient underwent a lung computed tomography (CT) scan on the fifth day of hospitalization due to patient's complaint of a mild dyspnea and abnormal findings noted during pulmonary auscultation. The imaging revealed the presence of a mild pneumothorax on the right side, accompanied by a small volume of pleural effusion and passive collapse of the lower lung in the right hemithorax. Additionally, there were signs of focal bronchiectasis in the right middle lobe, consolidation in the right lower lobe, and an atelectatic band in the right lower lobe. The scan also identified several reactive lymph nodes in the axillary region

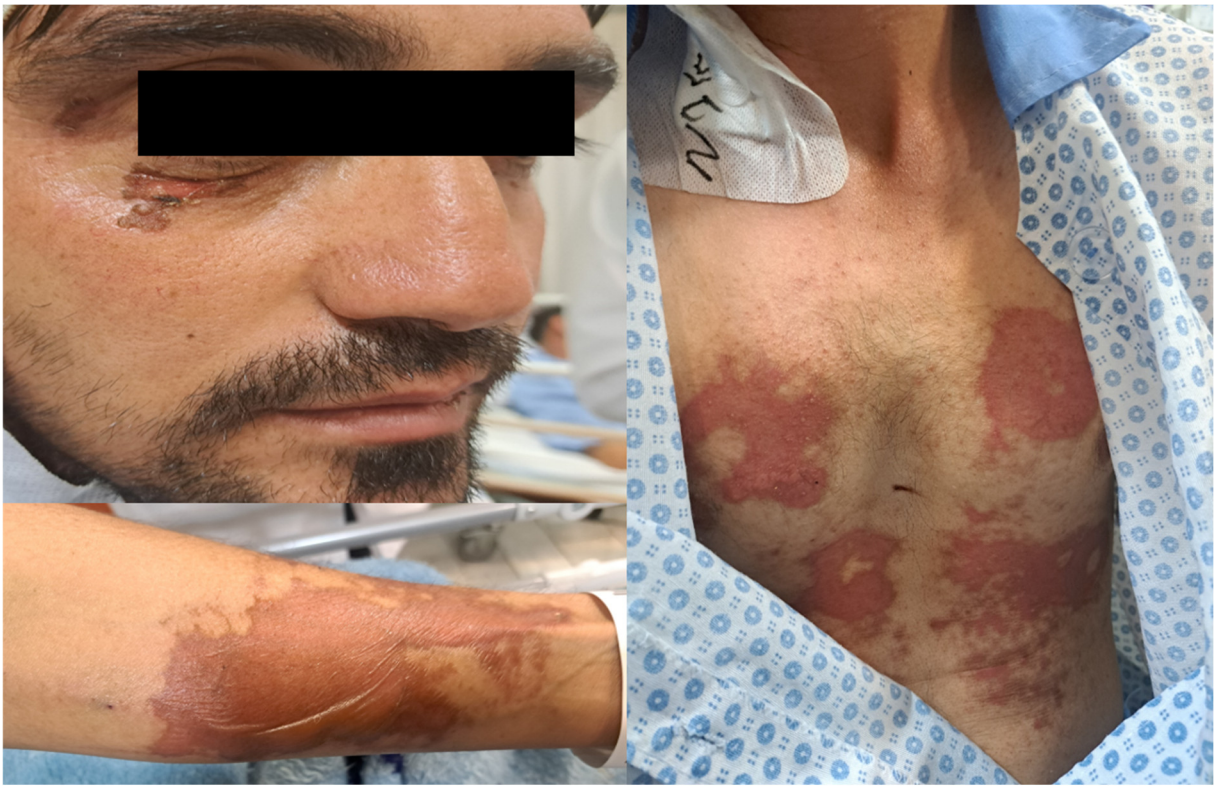

Figure 1. Skin lesions following contact with paraquat. Multiple erythematous and itchy rashes of varying sizes are scattered on the distal forearms, periorbital area, and anterior chest. Some ruptured blisters are also observed on the distal forearm

bilaterally, with the largest measuring 8.5 mm in short axis diameter on the right side. The patient presenting with suspected pneumonia was administered ampicillin-sulbactam at a dosage of 3 grams every six hours, in conjunction with ciprofloxacin at a dosage of 400 milligrams twice daily, for a duration of two days. The patient underwent an evaluation for daily respiratory distress and was prescribed simvastatin at a dosage of 40 mg orally once daily to mitigate inflammation.

On the third day of hospitalization, the patient demonstrated the ability to tolerate oral intake; consequently, the dietary regimen was adjusted to a liquid diet. After the hemodialysis sessions and starting potassium chloride syrup on the 7th day of hospitalization, the serum potassium level increased and stabilized at 3.6 mg/dL. Moreover, on the sixth day of admission, the dithionate test showed no presence of paraquat. The test was repeated two more times, and both results were also negative. On the seventh day of treatment, the patient transitioned from injectable antibiotics to an oral regimen of clindamycin, prescribed at a dosage of 600 milligrams every eight hours. A controlled lung CT scan was scheduled for the ninth day of hospitalization. The lung CT results revealed no pneumothorax recurrence, a reduction in pleural effusion volume, and no evidence of adenopathy. Furthermore, the rashes caused by the herbicide's toxic properties gradually faded and became less severe, and the

Table 1. Patient laboratory results at various time intervals after hospital admission

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 10
Parameters									
WBC (*10^3/μL)	8.6	-	8.9	-	-	-	-	-	12.3
RBC (*10^6/μL)	5.07	-	4.49	-	-	-	-	-	4.69
Hb (g/dL)	14.7	-	12.7	-	-	-	-	-	13.5
Platelet (*10^3/μL)	183	-	145	-	-	-	-	-	220
PT (sec)	14.3	-	15.3	-	-	-	-	-	-
INR (sec)	1.14	-	1.23	-	-	-	-	-	-
PTT (sec)	30.6	-	32	-	-	-	-	-	-
BS (mg/dl)	92	-	75	-	-	-	-	-	-
Urea (mg/dl)	27	13	-	-	25	26	23	34	33
Cr (mg/dl)	1.0	0.8	1.0	-	1.0	1.0	1.1	1.0	0.9
AST (U/L)	24	27	24	-	21	17	32	19	-
ALT (U/L)	18	32	29	-	26	21	25	21	-
ALP (U/L)	-	-	84	-	-	-	-	-	-
CPK (U/L)	-	167	98	-	-	-	-	-	-
Na	151	137	144	-	143	140	142	139	-
K	3.6	3.5	3.3	-	2.8	2.8	3.6	3.6	3.6
Bilirubin (mg/dL)									
Total	-	1.7	1.2	-	1.0	1.1	1.0	0.8	-
Direct	-	0.4	0.4	-	0.3	0.3	0.3	0.3	-
VBG									
PH	7.4	7.4	7.4	7.4	7.5	-	7.4	-	7.48
HCO3	23.2	27.4	25.0	28.8	33.0	-	30.4	-	26.5
PCO2	37.0	42.0	38.8	48.1	42.0	-	47.1	-	35.3
BE	-1.1	2.8	0.7	3.4	9.1	-	+5.5	-	+4.2
Serum-PChE (U/L)	3055	-	-	-	-	-	-	-	-
RBC-AChE (IU/mL)	3.4	-	-	-	-	-	-	-	-
Urine toxicology	-	-	-	-	-	-	-	-	-
Dithionite test									
Paraquat	+	+	+	+	+	-	-	-	-
Diquat	-	-	-	-	-	-	-	-	-

Abbreviations: WBC, white blood cell, RBC, red blood cell, BS, blood sugar, AST, aspartate aminotransferase, ALT, alanine aminotransferase, ALP, alkaline phosphatase, CPK, creatine phosphokinase, VBG, venous blood gas, PChE, plasma cholinesterase, AChE, acetylcholinesterase.

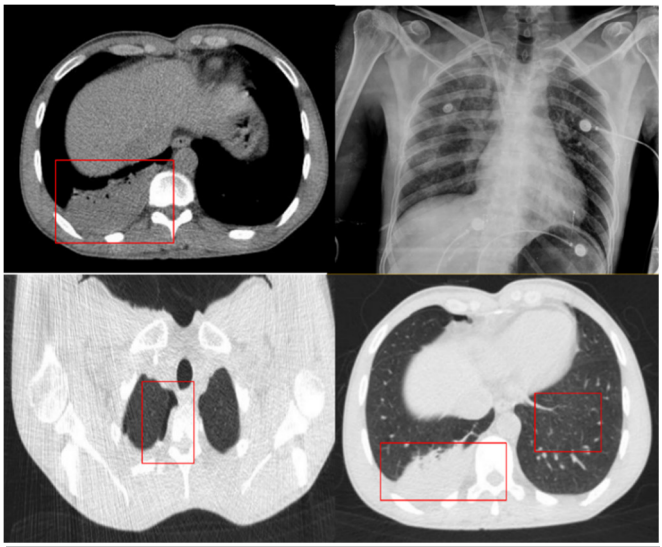

Figure 2. Lung CT scan of a patient after paraquat poisoning. The image displays various signs of lung injury that may result from paraquat exposure

patient reported no more burning or itching. Consequently, the patient was discharged from the toxicology unit in a favorable overall condition, exhibiting stable vital signs. The discharge included caution regarding potential danger signs and a recommendation for outpatient follow-up at a psychiatric clinic.

DISCUSSION

Widely used as a herbicide, paraquat poses lethal risks even with minimal exposure. The case fatality rate for paraquat poisoning is notably high, and there is a significant lack of proven treatment protocols for its treatment. Our case pertains to a young male patient with a documented history of paraquat ingestion. He maintained that the quantity ingested was negligible and reported subsequent emesis of the substance; however, he nonetheless experienced subacute lung injury during his hospitalization. The amount of paraquat ingested directly influences the clinical effects observed. When large quantities of liquid concentrate are consumed—specifically more than 50–100 ml of 20% ion—patients may experience rapid organ failure. Those affected typically present with symptoms such as hypoxia, shock, and metabolic acidosis, often leading to fatalities from multiple organ failure within hours to days. In contrast, smaller ingestions generally result in toxicity that primarily impacts the kidneys and lungs, with symptoms developing over a span of 2 to 6 days. This scenario is commonly referred to in clinical literature as ‘moderate to severe’ poisoning, and the mortality rate for this group remains above 50% [4]. In our case, the patient initially presented with a burning sensation in the oral cavity, body, and facial regions that had come into direct contact with paraquat. Aside from this symptomatology, he remained asymptomatic. Nevertheless, dialysis was initiated due to the potentially life-threatening consequences associated with paraquat exposure. Approximately 90% of the paraquat absorbed is rapidly excreted unchanged in the urine within 12 to 24 hours after ingestion. In instances of severe poisoning, however, there is a significant decline in renal clearance within just a few hours. As a result, the kidneys eliminate the small amount of paraquat that enters the deeper compartments at a much slower pace, which can take several days to weeks. While the initial elimination half-life is about 6 hours, this duration increases to approximately 4 days after the first day [5]. Following five consecutive positive dithionate tests, paraquat serum levels were observed to be negative on the sixth-day post-ingestion and continued to remain negative in the subsequent two tests. The evaluation of plasma paraquat concentration plays a crucial role in confirming cases of poisoning and predicting patient prognosis. Five established nomograms and formulas are available that leverage plasma paraquat levels to forecast outcomes in instances of self-poisoning, offering predictions ranging from 4 to 200 hours after ingestion. These predictive tools have shown consistent reliability in assessing mortality risk within their designated time frames. Patients who display evident systemic toxicity within the first 24 hours are faced with a poor prognosis, as their chances of survival are minimal. Furthermore, the emergence of renal failure, changes noted on chest radiographs, and the occurrence of gastrointestinal lesions are all regarded as unfavorable prognostic signs [5,6]. Despite the prompt commencement of dialysis, the patient developed a lung injury on the fifth day of hospitalization. He subsequently reported mild dyspnea at rest, and auscultation of the lungs revealed abnormal findings. A CT scan of the lungs later confirmed the diagnosis of lung injury. Paraquat exhibits its most severe toxicity in the lungs, leading to the development of acute alveolitis. Several other effects, including extensive alveolar collapse, and vascular congestion [7]. In a similar manner, the patient developed a hydropneumothorax in the right lung. Furthermore, consolidation and focal bronchiectasis were noted, which may be attributable to an infection as a sequela of the lung injury. The pulmonary lesion progresses through two distinct phases. The alveolar epithelium serves as the primary site of toxicity within the lungs. In the acute 'destructive phase,' both type I and type II pneumocytes exhibit signs of swelling, vacuolation, and damage to their mitochondria and endoplasmic reticulum. This phase is characterized by the sloughing of alveoli, which is accompanied by pulmonary edema. Following this initial phase, a proliferative phase ensues, during which the alveolar space becomes populated with mononuclear profibroblasts that differentiate into fibroblasts over the course of days to weeks. Ultimately, this process leads to the development of lung fibrosis [8]. Initially, our patient exhibited no symptoms apart from a burning sensation in the face and body. However, the lung injury that emerged several days post-admission greatly deteriorated the patient's prognosis. Furthermore, it has been observed that patients experiencing a burning sensation on their skin often face a similarly poor outlook. Zhao et al. investigated the relationship between lung CT findings and the prognosis of patients who had been poisoned by paraquat [9]. They noted that consolidation appeared as an early indicator, while bronchiectasis observed in later CT scans was associated with a relatively favorable prognosis.

Conversely, they identified ground-glass opacity (OR: 2.013), interstitial pulmonary fibrosis (OR: 3.779), and mediastinal emphysema (OR: 33.118) as significant risk factors contributing to mortality from lung injury due to paraquat exposure. In recent years, it has been recognized that high levels of oxidative stress play a key role in the underlying mechanisms of paraquat poisoning [10]. Considering the suggested mechanism behind paraquat toxicity, there have been multiple opportunities to disrupt the toxic process. The management approach has mainly focused on eliminating paraquat from the gastrointestinal tract to prevent absorption, enhancing its elimination from the bloodstream, and implementing strategies to mitigate lung damage using anti-inflammatory medications. At present, there are no pharmacological agents available that can counteract paraquat, nor are there any chelating agents that can effectively bind the toxin in the bloodstream or other tissues [11]. We administered Vitamin E and dexamethasone to our patient due to their antioxidant and anti-inflammatory properties. Additionally, we included NAC because research has demonstrated its ability to protect alveolar type II cells, which are particularly vulnerable to paraquat toxicity [2]. Extracorporeal treatments like intermittent hemodialysis are expected to be almost equally effective in improving the removal of paraquat, given that paraquat has low protein binding [12]. Therefore, we started promptly in the first 24 h after exposure dialysis to prevent poison uptake by pneumocytes and other tissues even with normal renal function tests. This approach did not prevent lung injury but could reduce the extent of lesion caused by paraquat intoxication. Consequently, we initiated dialysis within the first 24 hours following exposure to prevent the absorption of toxins by pneumocytes and other tissues, even when renal function tests were normal. While this strategy did not completely avert lung injury, it may have mitigated the severity of the damage resulting from paraquat poisoning.

CONCLUSION

This case report details an unusual instance of survival after significant paraquat poisoning in a young male from Iran, highlighting the essential role of prompt intervention and thorough management in enhancing patient outcomes. Although paraquat is known for its severe toxic effects, especially on the respiratory system, the patient's recovery was aided by the timely use of hemodialysis, antioxidant therapy, and intensive supportive care. Nevertheless, the emergence of lung injury despite these interventions points to the necessity for ongoing research into more effective treatments for paraquat toxicity. This case underscores the significance of early detection, diligent monitoring, and a collaborative approach in addressing such critical poisonings, providing important insights for healthcare professionals encountering similar situations in the future.

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