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Case Report

Paraquat Poisoning in an Young Adolescent: A Case Report

Harsha Vardhan Reddy[1], Misbah Rangwala[2], Udgeeth Thaker[3], Bhupendra Nayak[4], Sefali Patel[5]* and Khushi Thakor[5]

[1]2nd Year Resident, Department of Critical Care Pharmacotherapy, Parul Institute of Pharmacy, India

[2]MD Anesthesia, IDCCM, EDIC, Critical Care Consultant, Parul Sevashram Hospital, India

[3]MD General Medicine, IDCCM, Critical Care Consultant, Parul Sevashram Hospital, India

[4]MD Emergency Medicine, DrNB Critical Care Medicine, Critical Care Consultant, Parul Sevashram Hospital, India

[5]Doctor of Pharmacy Intern, Department of Pharmacy Practice, Parul Institute of Pharmacy, India

***Corresponding Author:** Sefali Patel, Doctor of Pharmacy Intern, Department of Pharmacy Practice, Parul Institute of Pharmacy, India.

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Abstract

Paraquat poisoning is a major public health problem, owing to the widely application of the highly toxic herbicide paraquat in agrarian regions. Paraquat (N,N′-dimethyl–4, 4′-bipyridinium dichloride) is a highly toxic compound with reported LD50 of about 35 mg/kg for humans. We report a case of a 17-year-old female who developed gastrointestinal injuries, acute kidney injury (AKI), acute liver injury and acute respiratory distress syndrome (ARDS) after the ingestion of 8–10 mL of paraquat. The patient developed oral ulceration and vomiting after ingestion, subsequently requiring intensive care unit admission for marked renal failure and respiratory distress. Although she was placed on dialysis and treated aggressively—the effort to save her was heroic—she quickly deteriorated and died as multiple organ system failure ensued. This case illustrates an important paradigm of urgent medical intervention for paraquat ingestion and the subsequent difficulty in treatment due to a lack of focused antidote. Moreover, it highlights the need for preventive measures as well as better awareness of paraquat exposure (particularly in agriculture) among vulnerable subgroups. The findings from this case reinforce the necessity for ongoing research into effective treatment modalities for paraquat poisoning and advocate for enhanced educational efforts on safe handling practices to mitigate the risk of future poisonings.

Keywords: Paraquat Poisoning; Acute Respiratory Distress Syndrome (ARDS); Multi-organ Failure; Acute Kidney Injury (AKI); Poison Management

Introduction

Paraquat poisoning is an important public health dilemma, especially in areas with high agricultural application of this potent herbicide. AbstractParaquat (N,N′-dimethyl-4,4′-bipyridinium dichloride) is a widely used herbicide but also well known for its high toxicity and an estimated human LD50 of 35 mg/kg. This means that only 10-15 mL of a 20% solution can be lethal, and acts primarily through inducing oxidative stress that leads to multi-organ failure [1,2]. Paraquat has been available since the 1960s; suicidal ingestion is much more common with this substance than accidental exposure or homicides. It seems particularly prevalent in developing countries, where there is little regulation on the availability of these chemicals and where self-harm among those less able to manage their mental health during the crisis is rising. The

clinical effects are so lethal that ingestion of paraquat is commonly followed by gastrointestinal injuries, acute respiratory distress syndrome (ARDS), renal failure, and hepatic necrosis. After ingestion, paraquat is quickly absorbed and mainly bio-accumulates in the lung, where it produces reactive oxygen species leading to cellular destruction and inflammation [1,5].

Although paraquat poisoning is serious, there is still no specific antidote available; and treatment strategies concentrate on supportive care and alleviating the toxin effects. Within this time window early steps such as gastric decontamination and antioxidant therapy can be considered, although the prognosis is dismal in patients with advanced symptoms or high paraquat plasma concentrations. Introduction Paraquat has unique clinical presentation and management, apart from other poisons.

Case Report

A 17-year-old female patient presented to the emergency department with chief complaints of oral ulcers for 2 days following the ingestion of less than 5 ml of paraquat which is less than the lethal dose, followed by emesis on June 10, 2024, at 9:00 AM. After the ingestion, she experienced three episodes of vomiting and was initially referred to a private hospital from where she was told to refer to our centre. Upon arrival, her blood pressure was 120/68 mmHg, pulse rate was 100 beats per minute, and a respiratory rate of 28 breaths per minute. Systemic examination revealed secretory sounds in the respiratory system, and her peripheral pulses were palpable. She was hemodynamically stable at the time of admission, although she had experienced one episode of vomiting. Diagnostic tests revealed acute kidney injury (AKI) and acute liver injury. On the second day of hospitalization, she continued to vomit and experienced hemoptysis, with her serum creatinine level rising to 6.6 mg/dl. However, by June 17, her serum creatinine had decreased to 3 mg/dl after undergoing two days of dialysis and her HRCT revealed soft tissue density basal dependent consolidation with ground-glass opacifications with interlobular septal thickening involving basal segment of bilateral lower lobes which was suggestive of possibility of aspiration pneumonitis and subpleural fibrotic linear parenchymal opacities and interstitial septal thickening involving bilateral lungs suggest possibility of old infective etiology (Figure 1, Figure 2). Initially kept nil per oral (NPO) and on a non-rebreather mask (NRBM), she began tolerating sips of water on June 17 and progressed to oral intake by June 21, with improvement in the oral lesions noted by June 24. On June 25 the Patient was shifted to Pediatric ward from ICU.

However, on June 26, she developed a cough and then patient was again shifted to ICU and the CT Scan was suggestive of Pneumomediastinum and pneumopericardium . She was again placed on NRBM and kept NPO. By July 1, she was stable enough to resume oral intake, but on July 2, she required a face mask at 6 L/min for oxygen support. On July 3, she experienced significant respiratory distress, with a respiratory rate of 50 breaths per minute and an SpO2 of 74%. The following day, she was intubated and placed on volume-controlled ventilation, with chest X-ray findings suggestive of acute respiratory distress syndrome (ARDS). Her heart rate escalated to 130-140 beats per minute, and by July 7, her Glasgow Coma Scale (GCS) score had deteriorated to E0V1M0. Despite sedation on July 11, her GCS remained poor, but it improved to E1V1M1 by July 12. Tragically, on July 13 at 9:00 AM, she suffered a sudden bradyarrest. Immediate cardiopulmonary resuscitation (CPR) was initiated, and intravenous adrenaline was administered per ACLS protocol, but there was no improvement in her blood pressure or pulse rate. Despite multiple attempts at resuscitation, her condition did not stabilize, and she was declared dead at 9:39 AM. This case underscores the severe complications associated with paraquat poisoning and highlights the challenges in managing such critical cases, where the prognosis remains poor despite aggressive medical intervention.

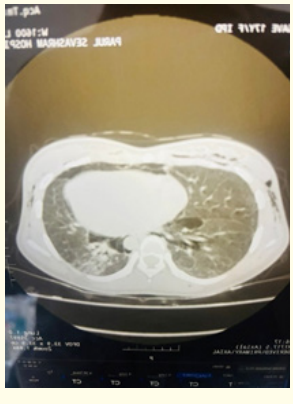

Figure 1

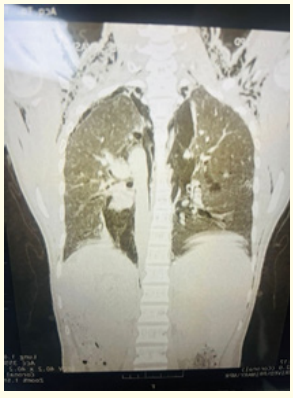

Figure 2

Discussion

Paraquat is a highly toxic herbicide, and paraquat poisoning poses great threat to life and health with lack of an effective antidote. This report presents a 17-year-old girl who had poor outcome following paraquat ingestion less than 5 ml typically less than the lethal dose and emphasizes the necessity of quick action as during this period, she could have gone deteriorated within minutes. Paraquat poisoning is characterized by corrosive injury to the gastrointestinal tract and subsequent multiple organ system failure, and our patient presented with both clinical signs consistent with paraquat toxicity: at an early point in his presentation he was noted to have oral ulcers which led to vomiting [1,4].

Her course with acute kidney injury (AKI) and acute liver injury demonstrates the multi-organ effect of paraquat. Serum creatinine increased to 6.6 mg/dl highlighting extensive renal dysfunction, a common effect of paraquat ingestion due to its direct nephrotoxicity [1,3]. Her clinical status deteriorated further in spite of vigorous therapy including dialysis and she needed to be intubated for respiratory failure due to ARDS. Acute respiratory distress syndrome (ARDS) is an important complication and a major cause of high mortality associated with paraquat poisoning [3,4].

This report also emphasizes the importance of supportive therapy and monitoring in patients with paraquat poisoning. Oxygen therapy and later mechanical ventilation were necessary as her respiratory function deteriorated. However, despite attempts to resuscitate the patisent , bradyarrest occured and she died unfortunately highlighting the poor prognosis of severe paraquat toxicity. The overall mortality due to paraquat poison in the case of multisystem involvement can be as high as 60% to 80% [2,3,5].

It also highlights the importance of knowing how to limit your exposure if you live or work near farms using paraquat. Awareness-raising about the risks of paraquat and safe handling practices could be beneficial to prevent such life-threatening poisonings.

Conclusion

None the less, this case report depicts again the potentially devastating effects of PQ poisoning and the difficulties we encounter as regard its management. The speed at which symptoms develop to life-threatening multi-organ failure underscores the need for immediate medical treatment after ingestion. Patients that develop severe complications such as ARDS and multiple organ dysfunction syndrome have a dismal prognosis despite aggressive treatment strategies including dialysis, supportive care.

The information obtained from this case highlights the need for further investigation of new treatment modalities in paraquat poisoning, as there is no current effective therapy available. Moreover, there should be prevention of exposure among those susceptible (especially farm workers where paraquat is widely used). Improved knowledge and awareness on paraquat poisoning mechanisms may hold important preventive potential in future cases, thereby helping patients recover faster.

Conflict of Interest Statement

All the authors - Dr. Harsha Vardhan Reddy (1), Dr. Misbah Rangwala (2), Dr. Udgeeth Thaker (3), Dr. Bhupendra Nayak (4), Ms. Sefali Patel (5), Ms. Khushi Thakor (6) have no conflict of interest.

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