Case Report

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Early and Aggressive Management of Severe Paraquat Poisoning in a 17-Year-Old Female: A Rare Case Report.

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Abstract

N, N′-dimethyl-4, 4′-bipyridinium dichloride (paraquat) is a commonly used synthetic, nonselective contact herbicide. Ingesting of paraquat in significant amount can cause severe effects on the lungs, gastrointestinal (GI) tract, kidneys, liver and heart and in many cases may prove to be fatal. Currently, there are no specific antidotes, and none of the existing treatments have been proven effective. The prognosis remains poor globally and even with aggressive treatment, mortality remains high. Despite its widespread use as an herbicide, there are very few reported cases from India. We hereby report a case of 17-year-old girl who presented to emergency department with an attempt of suicide by ingestion of paraquat. This case emphasizes the importance of early intervention and aggressive management to prevent fatal outcome.

Keywords:- Paraquat poisoning, N-acetylcysteine, haemodialysis, Outcome

INTRODUCTION

Paraquat, a widely used herbicide, is highly toxic upon ingestion, causing severe damage to multiple organs.[1] Paraquat poisoning causes extensive tissue damage through the production of superoxide radicals and reactive oxygen species (ROS). The severity depends on the dose ingested: less than 20 mg/dL causes local erosions; 20-50 mg/dL leads to renal and hepatic necrosis with pulmonary fibrosis; over 50 mg/dL results in multiorgan failure and rapid death.[2]

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Management focuses on reducing absorption and oxidative damage, with early gastric lavage and adsorbents like activated charcoal or Fuller's earth. Antioxidant therapy with N-acetylcysteine and vitamins C and E is critical, along with supportive care such as IV fluids and antibiotics. Advanced treatments like hemoperfusion and haemodialysis support renal function and manage complications. Immunosuppressive therapy with glucocorticoids and cyclophosphamide shows promise in reducing mortality. Early and aggressive management is vital for improving outcomes, with long-term follow-up necessary for managing complications like pulmonary fibrosis and renal failure.

CASE REPORT

A 17-year-old female presented to the emergency department with a history of attempted suicide by ingesting paraquat. She initially experienced symptoms of nausea, vomiting, and abdominal pain. On admission, her vital signs were recorded as follows: blood pressure 110/70 mmHg, heart rate 90 beats per minute, respiratory rate 20 breaths per minute, and temperature 37.2°C. Her renal and hepatic function tests were within normal limits.

Immediate management included gastric lavage to remove the ingested paraquat, followed by the administration of IV antibiotics to prevent secondary infections. IV antiemetics were given to control nausea and vomiting, and antacids were administered to manage gastrointestinal symptoms. Additionally, IV N-acetylcysteine was started for its antioxidant properties, and IV fluids were provided to ensure proper hydration and support renal function.

On the second day of hospitalization an HRCT chest was done which showed Multiple alveolar infiltrates with ground-glass opacities in both lung fields, these opacities were seen to be forming a patch of consolidation with positive air bronchogram suggestive of aspiration pneumonia. In addition to aspiration pneumonia patient also developed mouth ulcers and melena (black stools). In response, she was treated with multivitamins and vitamin K supplementation. Despite these interventions, her condition continued to deteriorate over the next few days, with worsening symptoms and clinical signs indicating progressive organ damage.

By the fifth day, the patient's clinical status had significantly declined, necessitating hemodialysis to manage the toxic effects of paraquat. Hemodialysis sessions were carried out over the following days, and the patient began to show signs of gradual improvement.

By the tenth day of admission, the patient's condition had stabilized. Her symptoms had improved, and her vital signs were within normal limits. She was subsequently discharged from the hospital with advice for psychiatric evaluation to address her suicidal tendencies and prevent future attempts. The multidisciplinary approach, including prompt decontamination, supportive care, and addressing complications, played a crucial role in her recovery.

DISCUSSION

Paraquat is a bipyridyl compound that causes direct cellular damage by generating superoxide radicals, reactive oxygen species, and nitrite radicals. The severity of poisoning depends on the dose. Ingesting less than 20 mg/dL can cause erosions in the tongue, oral mucosa, and gastrointestinal tract. Moderate toxicity, from consuming 20–50 mg/dL, can lead to renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis, with death typically occurring in 2–3 weeks. Severe toxicity, from ingesting more than 50 mg/dL, can cause multiorgan dysfunction and shock, resulting in death within 3 days. [3]

Paraquat poisoning presents significant challenges due to its high toxicity and the lack of a specific antidote. The pharmacological management of paraquat poisoning is largely supportive and focuses on minimizing absorption, reducing free radical damage, and addressing complications. Paraquat induces toxicity primarily through the production of superoxide radicals and other reactive oxygen species (ROS), leading to cellular damage. The compound undergoes redox cycling, generating superoxide anions that cause lipid peroxidation, protein denaturation, and DNA damage. This process results in extensive tissue injury, particularly affecting the lungs, kidneys, liver, and gastrointestinal tract. [4]

Early intervention is crucial in the management of paraquat poisoning. Gastric lavage can help remove ingested paraquat from the stomach, reducing systemic absorption. This procedure is most effective if performed within the first hour of

ingestion. Adsorbents like activated charcoal (1–2 g/kg) or Fuller's earth (1–2 g/kg) can be administered to bind paraquat in the gastrointestinal tract, preventing further absorption. These adsorbents should be given as soon as possible after ingestion to be most effective. [5]

Antioxidant therapy plays a significant role in mitigating oxidative stress and free radical damage caused by paraquat. N-acetylcysteine (NAC) is a free radical scavenger that replenishes intracellular glutathione, enhancing the body's ability to neutralize ROS. It is administered intravenously to provide systemic antioxidant effects. Additionally, vitamins C and E act as antioxidants, helping to reduce oxidative damage. They are often used in combination with other treatments to provide additional protective effects against ROS.[6]

Supportive care is essential in the management of paraquat poisoning. Maintaining adequate hydration through intravenous fluids is crucial to support renal function and enhance the elimination of paraquat through the kidneys. Prophylactic antibiotics are administered to prevent secondary bacterial infections, which can occur due to mucosal erosions and other tissue damage. Antiemetics and antacids help manage nausea, vomiting, and gastrointestinal symptoms, improving patient comfort and preventing further complications. [7]

Advanced therapeutic interventions like hemoperfusion can effectively reduce paraquat levels in the blood if initiated within 4 hours of ingestion. Hemodialysis, while not directly removing paraquat, is used to manage acute kidney injury and other complications resulting from paraquat poisoning. It supports renal function and helps in the management of fluid and electrolyte imbalances. Recent studies have explored the role of immunosuppressive therapy in managing paraquat poisoning. A Cochrane meta-analysis suggested that patients receiving a combination of glucocorticoids and cyclophosphamide, along with standard care, had a lower risk of death compared to those receiving only standard care. This combination is thought to reduce inflammatory responses and tissue damage mediated by immune mechanisms.[8]

The prognosis of paraquat poisoning is influenced by the amount ingested, the timing of medical intervention, and the presence of organ failures. Early and aggressive supportive care, including the use of adsorbents, antioxidants, and hemoperfusion, can improve outcomes. Late complications such as pulmonary fibrosis, renal failure, and gastrointestinal strictures are common among survivors, necessitating long-term follow-up and management. [9]

Pharmacological management of paraquat poisoning is primarily supportive, focusing on early decontamination, antioxidant therapy, and supportive care to manage complications. Advanced treatments like hemoperfusion and immunosuppressive therapy show promise in reducing mortality. Prompt medical intervention and comprehensive care are essential for improving patient outcomes in paraquat poisoning cases.[10]

Management of such cases requires a multidisciplinary approach. Initial stabilization with blood transfusions to address severe anemia was critical in this case. This step was essential in improving the oxygen-carrying capacity and overall physiological stability of the child, allowing for better management of the respiratory infection. The use of broad-spectrum antibiotics such as ceftriaxone and vancomycin due to persistent respiratory distress and radiological evidence of pneumonitis and pleural effusion, underscores the importance of aggressive infection control in these patients.[8]

LIMITATION OF STUDY

This case report has several limitations. Firstly, the patient was not followed up for the long-term, which restricts our understanding of the delayed complications of paraquat poisoning. Long-term follow-up is crucial as survivors of paraquat poisoning are at significant risk for late complications such as pulmonary fibrosis, renal failure, and gastrointestinal strictures. These complications can develop weeks to months after the acute phase and greatly impact the patient’s quality of life and overall prognosis. Additionally, the absence of long-term data limits the ability to evaluate the effectiveness and outcomes of the treatments provided during the acute phase. Further research with extended follow-up is necessary to understand the full spectrum of paraquat poisoning and to develop comprehensive management strategies to address both immediate and delayed complications.

CONCLUSION

Paraquat poisoning carries a high risk of complications and mortality due to the lack of specific antidotes. Early and aggressive intervention is vital. Supportive management including maintaining hemodynamic stability and administration of N-acetylcysteine as a free radical scavenger can be effective. Advanced treatments such as hemodialysis, hemoperfusion and immunosuppressive therapy show potential in reducing mortality. Prognosis depends on timely intervention and comprehensive care.

Conflict of interest

None

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