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"NAVIGATING THE CHALLENGES: A COMPREHENSIVE CASE REPORT ON SEVERE PARAQUAT POISONING"

General Medicine

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ABSTRACT

N,N′-Dimethyl-4,4′-bipyridinium dichloride, commonly known as paraquat, is a widely utilized non selective contact herbicide. Ingesting toxic amounts of paraquat can result in fatal outcomes, affecting vital organs such as the lungs, gastrointestinal tract, kidneys, liver, and heart. Unfortunately, there are no specific antidotes, and existing treatments lack proven efficacy, leading to a uniformly poor prognosis globally. Aggressive multimodal therapies have shown limited success, and long-term survivors often grapple with enduring gastrointestinal and pulmonary complications. Prevention stands as the paramount focus, necessitating swift and vigorous decontamination upon exposure. Despite being a prevalent herbicide, reported cases in India are sparse, underscoring the need for heightened awareness among the populace. This highlights the imperative nature of disseminating information and implementing stringent preventive measures to curb paraquat-related incidents and promote public safety.

KEYWORDS

INTRODUCTION

Paraquat, (N,N′-Dimethyl-4,4′-bipyridinium dichloride), a potent herbicide, is generally considered low in chronic toxicity due to rapid soil deactivation. Despite its widespread availability, cases of poisoning in India are rare. In a tragic incident, a fatal outcome resulted from accidental ingestion of paraquat, an infrequent occurrence in the country.[1] The victim's inadvertent consumption led to severe and often lethal toxicity, emphasizing the potential dangers associated with this herbicide.[2] This isolated case underscores the importance of heightened awareness and caution regarding the handling and accessibility of paraquat to prevent such tragic incidents.

Case Report

A 41-year-old male presented to the ER after accidentally ingesting approximately 350-400ml of paraquat mixed with alcohol. He complained of nausea and vomiting, displaying greenish discoloration in the vomitus.Admitted to Local hospital and later brought to our hospital for further management, the patient, initially stable, underwent gastric lavage, activated charcoal treatment, and was transferred to the ICU. Blood investigations revealed elevated total counts, normal Hb, and inflammatory markers within limits. Metabolic acidosis was observed on ABG, while electrolytes and renal function were initially normal. He was started on IV antibiotics (PIPTAZ), IV steroids(Dexamethasone), gastro-protectives and other symptomatic measures. Screening ECHO showed IVC fully collapsing and fair LV and no B lines. Next day Emergency hemodialysis and hemoperfusion were performed due to worsening creatinine levels.He had multiple episodes of vomiting following the dialysis, vomitus containing charcoal content non-blood stained or foul smelling and bowel opened once (greenish stools) which was managed symptomatically.

On subsequent days, the patient developed complications, including cough, sore throat, mucositis, and elevated liver enzymes.The patient underwent a 500mg Cyclophosphamide infusion over 3 hours, experiencing subsequent fever spikes and bilateral crackles. Despite interventions, creatinine remained high at 6.94, accompanied by a declining urine output of 50ml in 4 hours. The Nephrologist advised limiting fluid intake to 1L/day, Respiratory distress emerged, necessitating NIV CPAP, initiating NIV CPAP (SPO2-96%, PEEP-6, FiO2-25%), and administering IV antioxidants (NAC, EDARAVONE). Saturation dropped to 80%, prompting peripheral TPN for blood-tinged sputum and tachypnea (34/min). With elevated creatinine (9.86) and urea (163.4) Nephrology recommended Hemodialysis (D4, UF-1.5L), ongoing fluid restriction, resumed NIV, and introduced Pirfenidone. The treatment regimen included intravenous vitamin C at 6 g/day and vitamin E orally at 800 i.u./day (2 tabs QID). Despite these interventions, the patient did not show improvement and expired due to respiratory failure. The case highlights the challenges in managing cases with advanced complications, necessitating further research and alternative therapeutic approaches for better outcomes in such critical scenarios.

DISCUSSION

Paraquat, when orally consumed, primarily targets the lungs, inducing the release of hydrogen and superoxide anions, leading to lipid damage in cell membranes. This cascade results in oxidant free radical-induced hepato/nephrotoxicity and pulmonary fibrosis.[3] Fatal paraquat poisoning manifests histopathologically with pulmonary congestion, edema, and hemorrhage, progressing to extensive fibrosis. The toxicity extends beyond the lungs, causing inflammation of the tongue, oral mucosa, corrosive injury to the gastrointestinal tract, renal tubular necrosis, and hepatic necrosis.[4]The absence of a clinically proven antidote necessitates supportive treatments to mitigate free radical lung injury, employing vitamins C and E, along with pulse therapy using steroids and Pirfenidone to prevent respiratory failure. Hemodialysis aids in eliminating paraquat from circulation, while gastric decontamination is crucial.[5] Paradoxically, oxygen use, while commonly supportive, can exacerbate paraquat toxicity, emphasizing the complexity of its management. Despite advances, mortality remains high, primarily due to multiorgan failure and respiratory collapse. Survival is occasionally reported in cases of low-dose ingestion or with prompt, effective treatment. However, high-dose or severe poisoning presents a bleak prognosis.[6]

With no specific antidote available, prevention and aggressive decontamination post-ingestion become paramount. Identifying paraquat in urine not only confirms the diagnosis but also aids in prognostication, with a plasma concentration exceeding 1.6 pg/ml 12 hours post-ingestion universally indicating fatality.[3] Facing the challenge of paraquat intoxication, diverse supportive strategies were explored due to the absence of a definitive evidence-based therapy. Regrettably, our therapeutic endeavors failed to ameliorate the patient's clinical state, underscoring the pressing need for further research and innovative interventions in such critical medical scenarios

CONCLUSION

In conclusion, this case of paraquat poisoning illustrates the formidable clinical challenges and high mortality associated with this toxic agent. Despite aggressive interventions, including hemodialysis, cyclophosphamide, and antioxidants, the patient succumbed to respiratory failure and multi-organ dysfunction. The absence of a specific antidote highlights the critical need for preventive measures, rapid decontamination, and early detection in mitigating paraquat's devastating effects. The complexity of paraquat toxicity calls for continuous research and innovative therapeutic strategies to improve

outcomes. This case underscores the urgent necessity for increased awareness, stringent safety measures, and comprehensive educational efforts to minimize accidental exposures. The limitations in current treatment modalities emphasize the pressing need for collaborative efforts in the scientific community to enhance our understanding and management of paraquat poisoning.

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