

A Rare Case of Paraquat Poisoning

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Abstract: Paraquat is a highly toxic herbicide known for its severe health impacts on humans. Ingesting paraquat can cause acute symptoms such as sore throat, difficulty swallowing, and oral ulcers, leading to multi-organ dysfunction and potentially death. A case involving a 23-year-old male with paraquat poisoning highlights the critical need for prompt medical intervention. The patient received initial treatment with gastric lavage and supportive care, including intravenous fluids and antioxidants. Despite severe initial symptoms, the patient recovered well after comprehensive management, including hemodialysis and anti-inflammatory medication. The case underscores the importance of early diagnosis and aggressive treatment in paraquat poisoning to improve survival rates. Regular follow-up is crucial due to the high mortality rate associated with paraquat exposure, despite the best medical efforts.

Keywords: Paraquat, Paraquat lung, acute respiratory distress syndrome (ARDS), reactive oxygen species, multi organ dysfunction, steroids, dialysis.

1. Introduction

Paraquat is a toxic bipyridyl herbicide, bright green corrosive liquid with a pungent smell. Its herbicidal properties were discovered in 1950s and first marketed in 1962. ⁽¹⁾ Paraquat is toxic to humans (Category II) by the oral route and moderately toxic (Category III) through the skin. Pure paraquat, when ingested is highly toxic to mammals, including humans, causing severe inflammation and potentially leading to severe lung damage (e. g., irreversible pulmonary fibrosis, also known as 'paraquat lung'), acute respiratory distress syndrome (ARDS), and death. ⁽²⁾

Lung injury is a main feature of poisoning. Multi organ dysfunction like Liver, heart, lung, and kidney failure can occur within several days to weeks that can lead to death up to 30 days after ingestion. Oral exposure can cause oral ulceration, neck swelling, esophagitis etc. Those who suffer large exposures are unlikely to survive. Chronic exposure can lead to lung damage, kidney failure, heart failure, and esophageal strictures. The mechanism underlying paraquat's toxic damage to humans is thought to be caused by the generation of highly reactive oxygen species and nitrite species that results in oxidative stress. The mortality rate is estimated between 60 - 90%. ⁽²⁾

2. Case Report

A 23 year old male presented with a complaint of sore throat, difficulty in swallowing, oral ulcer and swelling in neck for 2 days. Patient gave a history of ingestion of paraquat poison and was taken to a nearby hospital where he was given gastric

lavage with activated charcoal, and managed conservatively with intravenous fluids, antiemetics, and H2 blockers. Patient brought to our hospital after 3 days with the above complaints. There was no complaint of abdominal pain, vomiting, fever, cough and breathlessness. There was no significant past medical history or past psychiatric illness.

On general examination, the patient was conscious, oriented to time, place and person. Patient was well built and well nourished. Patients vitals were normal. On examination there was no pallor, icterus, clubbing, cyanosis, lymphadenopathy or edema. There was visible neck swelling and oral pigmentation with ulceration.

The identity of the poison was confirmed as the family showed a photograph of a bottle of "Gramoxone" and it was suspected to be a case of accidental poisoning according to history given by relatives.

Laboratory investigation

Haemoglobin	13.8mg/dl
Total WBC	15, 700/mm ³
Platelet	2.27 lakhs/mm ³
S. Creatinine	6.35
S. Urea	>128
SGPT	155

Urine routine microscopy showed presence of protein. HIV, HbsAg, antiHCV, coagulation profile was normal. USG Neck and APK was normal. Serum cholinesterase levels was normal. According to patients' history, it was suspected of due to paraquat poisoning.

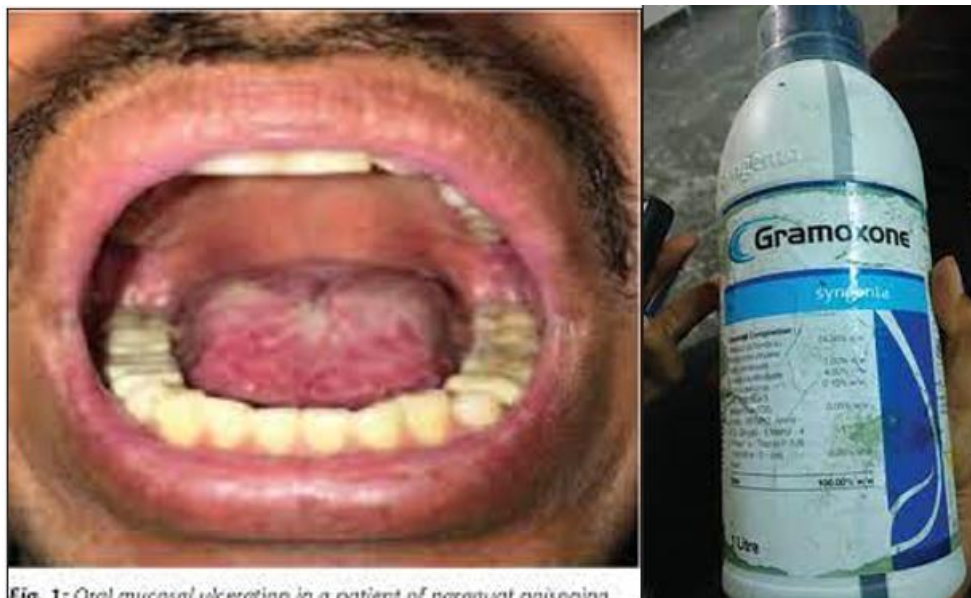


Fig. 1: Oral mucosal ulceration in a patient of paraquat poisoning

Management

Gastric lavage was given with activated charcoal. Injectable steroids (Inj. Solumedrol 1 gm IV once a day for 3 days) along with antioxidants like (N - acetyl cysteine, Vitamin C, Vitamin E) and other supportive analgesics, antacids and IV fluids were administered as anti-inflammatory agents to prevent free radical damage. Dexamethasone (8mg) iv 8hrly for 5 days followed by oral prednisolone in tapering dose over 2 weeks. One cycle of hemodialysis was done. Patient recovered well and was discharged later. Patient is under follow-up with no complaint and normal RFT and LFT values.

3. Discussion

Since bipyridyl salts are caustic, the gastrointestinal tract can be severely injured after ingestion of a concentrated solution. ⁽¹⁾

Symptoms of paraquat ingestion are usually dose-dependent, and intoxication can be categorized to mild, moderate, and fulminant. Mild intoxication can happen with doses ≤ 20 mg/kg, which usually produce minor gastrointestinal problems like transient vomiting, diarrhea, and oropharyngeal burns, but usually complete recovery is possible. Moderate intoxication can occur with doses between >20 mg/kg and <50 mg/kg of the poison. Patients may suffer lung injury, pulmonary fibrosis, acute renal failure, and in the majority of cases, death occurs within 2 - 3 weeks. Fulminant intoxication of ≥ 50 mg/kg of the poison, may lead to death within 3 days, because of multiple organ failure. ⁽¹⁾

Diagnosis of paraquat poisoning is usually made based on circumstantial evidences. ⁽¹⁾

Conventional treatment includes nasogastric tube fixation, gastric lavage with normal saline, charcoal-sorbitol lavage, forced alkaline diuresis and hemodialysis or hemoperfusion. ⁽¹⁾

Some antioxidants like vitamins C and E have been clinically used to protect against free-radical damage. N - acetyl

cysteine is also used as an antioxidant because of its free radical scavenging property, and it will increase intracellular glutathione levels. ⁽¹⁾

Glucocorticoids are potent anti-inflammatory agents. Moreover, pulse methylprednisolone has also been shown to suppress superoxide production by neutrophils and macrophages and the formation of superoxide in the arachidonic acid cascade. This action is further potentiated by cyclophosphamide therapy, a broad spectrum immunomodulator, which influences virtually all components of cellular and humoral immune response and reduces the severity of inflammation. ⁽⁴⁾

4. Conclusion

There is no specific antidote available for paraquat poisoning. It is important to diagnose early and to pursue aggressive decontamination to prevent further absorption. ⁽⁵⁾ Despite the presence of possible appropriate management approach, the case fatality is very high. Therefore, delayed death occurs in PQ poisoning and is regarded to continue as described management approach, and there should be a follow-up for these patients over a long period. ⁽⁶⁾

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