Case Report

Paraquat poisoning: Case report of a survivor

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Abstract

N, N′-dimethyl-4, 4′-bipyridinium dichloride (paraquat) is a widely used synthetic, nonselective contact herbicide. Ingestion of toxic doses of paraquat can be fatal with life-threatening effects on the lungs, gastrointestinal (GI) tract, kidney, liver, heart, and other organs. Till date, there are no specific antidotes and none of the current treatments have proven efficacious. The prognosis is uniformly poor worldwide, including those who treat aggressively with multimodal therapies. Long-term survivors are few, and have GI and pulmonary complications. Hence, prevention needs to be the utmost priority, and on exposure, aggressive decontamination should be initiated. Although it is a very common herbicide, there are very few cases reported from India and awareness among people needs to be widened.

Keywords: Acute renal failure, herbicide, mortality, paraquat, pulmonary fibrosis

Introduction

N, N'-dimethyl-4, 4'-bipyridinium dichloride (paraquat), a pungent smelling corrosive liquid is currently the second highest selling herbicide worldwide.[1] Its accidental or deliberate ingestion is associated with a high mortality rate, and it produces both local and systemic toxicity.[2] Here, we report a case of deliberate self-harm with paraquat which was complicated with renal failure and esophageal erosions with no features of pulmonary toxicity. The patient was managed supportively and he recovered completely.

Case Report

A 28-year-old gentleman presented to our hospital with an alleged history of consumption of 10 ml of 24% paraquat, 12 days before at 10 pm, following a familial dispute. Eight hours after consumption of the poison, he was found by his relatives unconscious and was taken to a local hospital where he was given gastric lavage, activated charcoal, and managed conservatively with intravenous fluids, antiemetics, and H2 blockers. During his stay in that hospital, he became oliguric with a progressive increase in serum creatinine values. He also complained of multiple episodes of vomiting, burning sensation of the tongue, ulcers in his oral cavity, difficulty in opening the mouth, and swallowing solid foods. At this point, he was referred to our hospital for further management.

At the time of arrival at our center, his main complaints were a progressive difficulty in swallowing, associated with multiple painful oral ulcers. On examination, his vitals were stable and he was conscious and oriented. There were erosions over his lips and oral cavity. His chest was clear with no crepitations or wheeze and other systemic examination was normal.

On evaluation, his white blood cell counts were normal, creatinine was 2.12 mg/dl, urea was elevated (97 mg/dl), and he had a hypokalemia with a potassium of 3.1 mmol/l. He was managed conservatively with adequate hydration and H2 blocker, with which his renal failure resolved and creatinine normalized. He was initiated on a liquid diet and a gastroenterology opinion was sought for the management of possible esophageal strictures. An upper gastrointestinal (GI) scopy was deferred since he was considered a high-risk candidate in view of the risk of esophageal

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rupture. A barium swallow was advised 4–6 weeks later if the dysphagia continued to progress. His chest X-ray was normal. He was discharged in a stable condition and was advised to be on regular follow-up with serial chest x-rays and spirometry to rule out pulmonary fibrosis.

Discussion

Paraquat is a bipyridyl compound which causes direct cellular damage by production of superoxide radicals or other reactive oxygen species and nitrite radicals.[3] The clinical course is usually dose dependent and it causes both local and systemic toxicity. Oral ingestion can lead to erosions of the tongue, oral mucosa, and corrosive injury to the GI tract with poison dose <20 mg/dl. Renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis can be seen with moderate toxicity with consumption of 20–50 mg/dl where death usually occurs in 2–3 weeks. In fulminant toxicity (consumption of more than 50 mg/dl), death is due to multiorgan dysfunction and shock which usually occurs in 3 days. The elimination of paraquat is mainly by kidneys usually within 24 h in minor poisonings. However, the terminal elimination half-life could exceed 100 h. Our patient presented to us 12 days after consumption of the poison, so the serum and urine paraquat levels could not be done and the diagnosis was based on history, clinical findings, and documentation of consumption of the poison.

The management of paraquat poisoning is mainly supportive as there is no known antidote. Gastric lavage, adsorbents such as activated charcoal (1–2 g/kg) and Fuller's earth (1–2 g/kg) should be initiated as early as possible to prevent the absorption of the poison. Antioxidants such as Vitamins C and E and N-acetyl cysteine, a free radical scavenger has also been used.[3] Hemoperfusion has shown to be effective in decreasing the paraquat level if given within 4 h of ingestion. Hemodialysis is used only as a supportive treatment for patients who develop acute tubular necrosis. Role of immunosuppression is still being studied. A recent cochrane meta-analysis concluded that patients who received glucocorticoid with cyclophosphamide in addition to standard care had a lower risk of death at final follow-up than those receiving standard care only (risk ratio 0.72; 95% confidence interval 0.59–0.89).[4]

The outcome depends on the severity of the poisoning and the time taken to avail medical help. The high mortality rates are due to the toxicity of the compound itself and the lack of a specific antidote. Young age, percutaneous or inhalational route, exposure to less paraquat, and lesser degrees of leukocytosis, acidosis, renal, hepatic, and pancreatic failures on admission were good prognostic factors.[5] The common late complications among survivors were renal failure, esophageal erosions, esophagitis, and strictures. Progressive pulmonary fibrosis is another important late complication of paraquat toxicity which can cause death 2–3 weeks later due to hypoxia and respiratory failure. The reasons why our patient survived could be because of the quantity of poison taken, the lack of any pancreatic or hepatic failure, acidosis, and the good supportive treatment that was given.

Conclusion

Paraquat is a common herbicide worldwide with a high mortality on ingestion. Early diagnosis with prompt history taking and aggressive decontamination with gastric adsorbents is the cornerstone of treatment. The late complications of paraquat ingestion include oropharyngeal ulcerations, esophageal erosions, renal failure, and pulmonary fibrosis.

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Conflicts of interest

There are no conflicts of interest.

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