CASE REPORT

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A Case Report of Paraquat Poisoning

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Abstract

N, N2-dimethyl-4, 42-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 is no specific antidote. Although it is a very common herbicide, there are very few cases reported from India and awareness among people needs to be widened.

Introduction

Paraquat dichloride is a widely used and highly toxic herbicide. It is a broad-spectrum (non-selective) contact herbicide and a powerful desiccant. Paraquat is the third most widely used herbicide in the world. The World Health Organisation (WHO) categorises paraquat as a class II-moderately hazardous pesticide. Although the WHO has listed it as moderately hazardous, it is among the most hazardous pesticides in the world today. Paraquat is one of the pesticides most frequently used to commit suicide. It is a highly toxic compound and the fatality rate ranges between 60 and 80%[1] due to the lack of a specific antidote. A dose of 30 mg/kg may be fatal, which is equivalent to 8 - 10 ml of the 20% solution sold commercially[2]. It has been shown to cause significant damage to organs, including the lungs, liver, kidneys and myocardium, with the highest concentration found in the lungs[3]. The prognosis of patients with multiple organ dysfunction syndrome (MODS) caused by fulminant poisoning (> 40 mg PQ ion per kg of body weight) is extremely dangerous and patients may succumb within hours to a few days following ingestion[4,5]. Here, we report a case of paraquat poisoning which was complicated by renal failure and oesophageal erosions with no features of pulmonary toxicity. The patient was managed supportively and he recovered completely.

Case report

A 34-year-old male resident of Himachal Pradesh, with no previous comorbidity had alleged history of ingestion of ½ to 1 tea spoon of an unknown poison 5 days back for which he was taken to hospital nearby where gastric lavage was done and symptomatic treatment was given. He then presented in our emergency with complaints of multiple oral ulcers and difficulty in swallowing solid foods. There was no vomiting, loose stools, abdominal pain, hematemesis, malena, decreased urine output, shortness of breath, cough, chest pain, seizures or altered sensorium. On examination, patient was conscious and oriented to time, place and person. Vitals were stable. He was maintaining normal oxygen saturation on room air. Oral examination revealed mucosal erosions of tongue, palate, and lips. His chest was clear and other systemic examination was normal.

On evaluation, his kidney function tests were deranged , creatinine was 4.7 mg/dl and urea was 121 mg/dl. Rest of the blood investigations were normal. His chest X-ray was normal. ECG showed T-wave inversion in leads II , III , aVF, V3 to V6 but there were no dynamic changes in subsequent ECGs and Trop-T was negative. On day 1, a provisional diagnosis of corrosive poisoning with acute kidney injury with oral ulcers was kept. He was managed conservatively

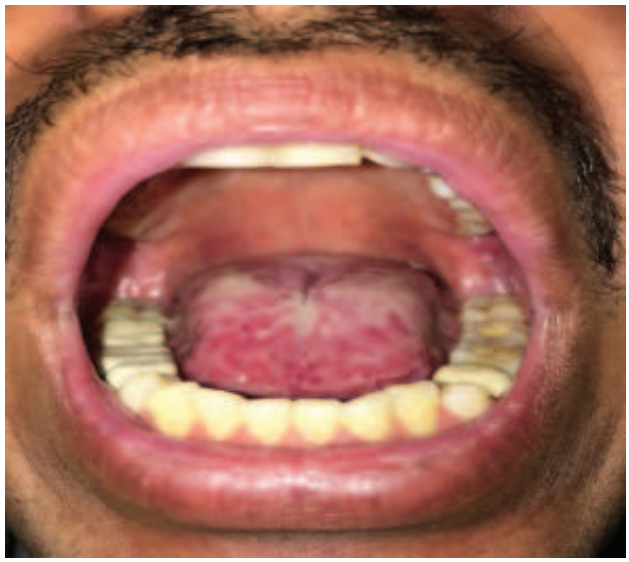

Fig. 1: *Oral mucosal ulceration in a patient of paraquat poisoning.*

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with adequate hydration and proton pump inhibitor. Gastroenterology opinion was sought. An upper gastrointestinal endoscopy date was booked. On 2nd day of admission, the relatives brought the poison bottle named "MILIQUAT" which contained "PARAQUAT". Interavenous Steroids and N-acetylcysteine were added to the treatment. Interavenous fluids, proton pump inhibitors and sucralfate were continued. Input/output charting and vitals monitoring was done. Patients general condition improved over period of time. He started accepting orally. Urine output was adequate. KFT was in improving trend and patient was able to maintain normal oxygen saturation on room air. He was then discharged with stable vitals and normal KFT and was asked to follow-up in the OPD.

Discussion

Paraquat is a bipyridyl compound which causes direct cellular damage by production of superoxide radicals or other reactive oxygen species and nitrite radicals[6]. Paraquat mainly affects the lungs, where it accumulates at up to 6 - 10 times the plasma concentration, sequestered in pulmonary type I, type II and Clara cells. When consumed orally, paraquat is sequestered in the lungs against gradient by active transport and causes a release of hydrogen and superoxide anions which cause lipid damage in the cell membranes, causing oxidant free radical damage that results in hepato/nephrotoxicity and pulmonary fibrosis. In fatal cases, histopathological findings range from pulmonary congestion, oedema, and haemorrhage to extensive pulmonary fibrosis.

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 less than 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. Identification of paraquat by urine dithionite test is used to confirm the diagnosis. Urinary paraquat concentrations of less than 1 mg/l within 24 hours of toxicity have a high probability of survival. It has been found that plasma concentration of more than 1.6 pg/ml 12 hour after ingestion is universally fatal.

The management of paraquat poisoning is mainly supportive as there is no known antidote. Use of 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 N acetylcysteine, vit E, vit C and salicylates might be beneficial through free radical scavenging, anti-inflammatory and NF-κB inhibitory actions. However, there are no published human trials. Hemoperfusion has shown to be effective in decreasing the paraquat level if given within 4 hour of ingestion. Haemodialysis 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 glucocorticoids 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)[7].

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.

We are publishing this case because despite paraquat's widespread availability, reports of this herbicide poisoning in India are uncommon. Its very important to know the exact causative agent of poisoning so that the right management can be done.

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