

## Paraquat poisoning: A case report and review of literature

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### ABSTRACT

Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride), a brown syrupy liquid is an effective herbicide that has low chronic toxicity because of its rapid deactivation on contact with soil. A high dose of paraquat or severe poisoning has a poor prognosis. At present there is no specific antidote to paraquat poisoning, hence the need to focus on prevention and in case of exposure or ingestion, aggressive decontamination to prevent further absorption. Although uncommon, paraquat ingestion can lead to severe and often fatal toxicity. However, despite its widespread availability, reports of this herbicide poisoning in India are uncommon.

**Key words:** Mortality, outcome, paraquat positioning

### INTRODUCTION

Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride), a brown syrupy liquid is an effective herbicide that has low chronic toxicity because of its rapid deactivation on contact with soil.<sup>[1-5]</sup> Although it is uncommon, paraquat ingestion can lead to severe and often fatal toxicity.<sup>[6-8]</sup> However, although it is widely available, reports of this herbicide poisoning are not common in India.<sup>[9-13]</sup> We discuss a fatal case of suicide in which paraquat was consumed and review the literature.

### CASE REPORT

An 18-year-old female was admitted to the emergency room with an alleged history of attempted suicide in which an unknown quantity of paraquat (liquid form) was consumed at her residence. The patient was managed at local hospital with (intravenous (IV) fluids, antiemetic, and H2 blockers) and brought to our hospital for further treatment after 24 h. She had difficulty in opening her mouth and a decreased urine output. There was no history

of vomiting, loose stools, abdominal pain, seizures, or fever. At the time of examination, the patient was conscious and oriented. There was neck edema. Examination of the oral cavity showed mucosal erosion of tongue, palate, and lips with oral bleeding [Figure 1]. On clinical examination, the pulse rate was 98 beats per min, regular, blood pressure (BP) was 130/80 mmHg, with respiratory rate of 22 per min. Cardiovascular system was normal. There was difficulty in breathing, but there were no added sounds on examination of the respiratory system. Pupils were bilaterally equal and reactive to light. The patient was intubated as there was associated laryngeal edema and difficulty in breathing. Gastric lavage was performed and charcoal was given in the emergency department. She was kept on elective ventilation. In the intensive care unit (ICU), she received IV fluids and an antiemetic as a supportive measure. Her initial chest X-ray showed a left lower zone infiltrate [Figure 2]. She had high serum urea (221 mg/dL) and creatinine (8.78 mg/dL) on the day of admission that gradually reduced to normal after two sessions of dialysis. Blood and urine cultures were sterile. Other blood investigations including thyroid and liver functions were normal. Urine examination was normal. Ultrasound of the abdomen showed bilateral grade I changes in the kidneys. Two-dimensional (2D) echo and electrocardiogram (ECG) were normal. upper gastrointestinal (GI) endoscopy showed corrosive injury to the esophagus and proximal stomach. Endotracheal culture showed *Klebsiella pneumoniae*. She received intravenous methylprednisolone 1 g in 200 ml normal saline every 2 h daily for 3 days, IV

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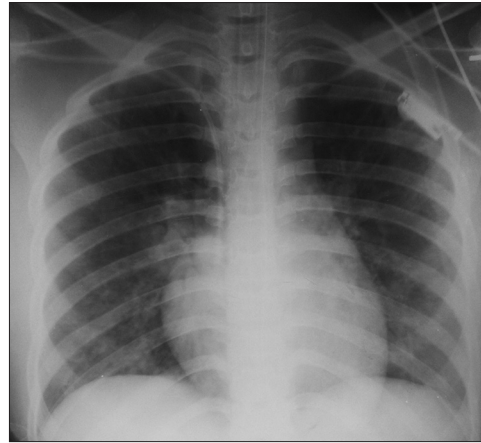
**Figure 1:** Clinical photograph showing extensive oral ulcerations

cyclophosphamide 750 mg (15 mg/kg/day) in three divided doses (in 200 ml of N-saline over 2 h) for 2 days followed by IV dexamethasone 5 mg 6 hourly, injection N-acetylcysteine 2 g stat followed 1 gm TID for 5 days, vitamin C (500 mg/amp) 6 g/day IV, vitamin E (400 i.u./tab) 2 tabs QID. The patient did not respond to treatment and expired on the 12<sup>th</sup> day post injury as a result of septicemia and respiratory failure.

## DISCUSSION

When consumed orally, Paraquat is sequestered in the lungs 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.<sup>[14-18]</sup> In fatal cases of paraquat poisoning, histopathological findings range from pulmonary congestion, edema, and hemorrhage to extensive pulmonary fibrosis.<sup>[19]</sup> Paraquat toxicity produces local as well as systemic effects.<sup>[12,20]</sup> As seen in the present case, paraquat ingestion results in an inflammation of the tongue, oral mucosa and throat, corrosive injury to the gastrointestinal tract, renal tubular necrosis, hepatic necrosis, and pulmonary fibrosis.<sup>[14]</sup> The patient complains of burning and ulceration of the throat, tongue, and esophagus.<sup>[8,14,20,21]</sup> The pulmonary manifestations of paraquat poisoning begin with diffuse consolidation, which evolves several days later into cystic lesions followed by focal fibrotic lesions with very high mortality.<sup>[22]</sup> Ingestion of large amounts is considered to be uniformly fatal from multiorgan failure and cardiogenic shock.<sup>[14,18]</sup> Identification of paraquat in urine has not only been used to confirm the diagnosis,<sup>[14]</sup> but also investigated for the prognostication.<sup>[23]</sup> It has been found that plasma concentration of >1.6 pg/ml 12 h after ingestion is universally fatal.<sup>[8]</sup>

As there is no specific clinically proven antidote for paraquat poisoning, supportive treatment is given to avoid free radical



**Figure 2:** X-ray chest posteroanterior (PA) view showing bilateral patchy consolidation involving lower lobes

injury to lungs (vitamins C and E),<sup>[9,24-27]</sup> with pulse therapy using steroids (methylprednisolone or dexamethasone) and cyclophosphamide to prevent pulmonary fibrosis,<sup>[13,28,29]</sup> elimination of paraquat from circulation (hemodialysis), and gastric decontamination.<sup>[11,27,30-32]</sup> In contrast, the use of oxygen can enhance the toxicity of Paraquat by providing more electron acceptors and should be given in lower concentrations to the hypoxic patients.<sup>[30,33,34]</sup> In spite of advances in medical care, prompt treatment, and supportive care, mortality is high (mainly due to multiorgan system and respiratory failure) in patients with paraquat poisoning.<sup>[8,12,25,27,35-39]</sup> Although there have been isolated case reports of survivors (mainly due to the smallness of the dose or effective and early treatment),<sup>[12]</sup> an ingestion of a high dose or severe paraquat poisoning has a poor prognosis. At present, there is no specific antidote to paraquat poisoning. Therefore, it is recommended that the crucial focus should be on preventive measures and in case of exposure, when it has been ingested, the institution of aggressive decontamination to prevent further absorption.

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