

# Paraquat poisoning

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## Correspondence to

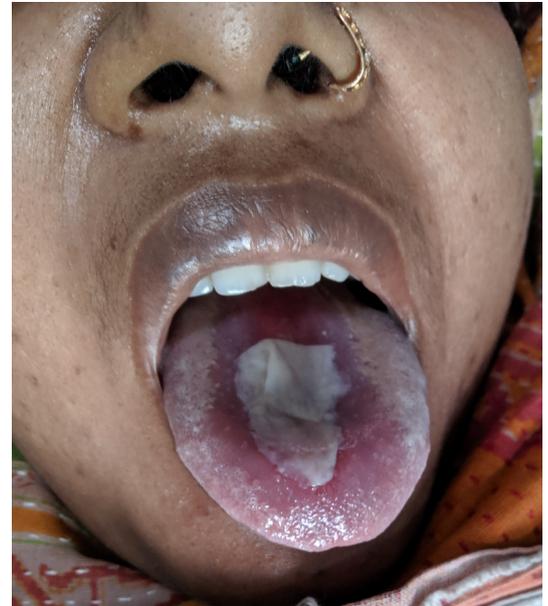
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## DESCRIPTION

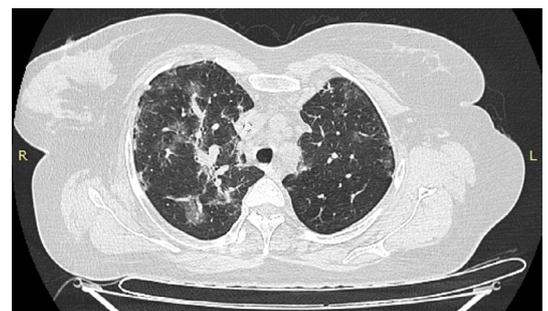
A 45-year-old woman without any known comorbidity was admitted to our facility with painful ulcerations in the mouth, dysphagia and decreased urine output for the last 2 days following suicidal consumption of 30 mL of 20% w/v of paraquat poison. On admission, she was afebrile (temperature: 98°F), her pulse rate was 104 beats/minute, blood pressure was 108/78 mm Hg and peripheral oxygen saturation was 98% on room air. Examination revealed ulcerated, coated tongue and oral mucosa along with the formation of slough (paraquat tongue) (figure 1). Basic blood parameters revealed raised urea (96 mg/dL; reference range, 5–20) and creatinine (5.4 mg/dL; reference range, 0.6–1.2). Initial radiography of the chest did not reveal anything unusual. She was put on injection N-acetyl-cysteine and was managed conservatively. Haemodialysis was started for the management of oliguric acute kidney injury. The patient responded well to this management and urine output gradually increased. However, on day 7 of admission, she reported cough and shortness of breath. At that point, the patient was tachypneic (respiratory rate 30 breaths/minute) and hypoxemic (peripheral oxygen saturation was 78% on room air) with associated bi-basal crackles. Arterial blood gas analysis showed features of type I respiratory failure. Radiography of the chest revealed bilateral opacities in the lower zone. A high-resolution CT scan of the thorax was performed, which showed areas of bilateral ground-glass opacities along with patchy fibrosis, predominantly in the lower lobes. (figure 2 and video 1) The patient was put on mechanical ventilation for progressive hypoxemia. However, she died within 2 days despite our best efforts.

Poisoning by pesticides is a major problem in low-income and middle-income countries. A systematic review of data extracted from 2006 to 2015 concluded that pesticides were responsible for almost 20% of global suicides leading to approximately 150 000 fatalities per year over the afore-mentioned period.<sup>1</sup> Unfortunately, this is a less commonly researched topic. Although organophosphate accounts for the majority of the hospital admissions, poisoning by paraquat compounds is a major medical problem as it is associated with a very high mortality (case fatality rate of 50%–70%) and morbidity.<sup>2</sup> Paraquat is a toxic bipyridyl compound, and it is lethal even in very small amounts (15–20 mL of 20% w/v). The mode of poisoning is usually suicidal or accidental; and it should always be suspected in



**Figure 1** Ulcerated and coated tongue along with the formation of slough (paraquat tongue).

cases of corrosive oral and oesophageal ulcerations along with features of hepatic, renal or pulmonary involvement.<sup>3</sup> Paraquat is incompletely absorbed from the gastrointestinal tract and mainly exerts its toxicity through production of reactive oxygen species (ROS) which causes cell damage by several mechanisms, such as mitochondrial toxicity, lipid peroxidation, oxidation of nicotinamide adenine dinucleotide phosphate (NADPH), activation of nuclear factor kappa B and apoptosis.<sup>4</sup> It targets both type I and II pneumocytes in the lung, leading to acute alveolitis. The initial 'destructive alveolitis' is followed by a 'proliferative' or 'cellular' phase, which ultimately leads to lung fibrosis.<sup>5,6</sup> Hepatorenal

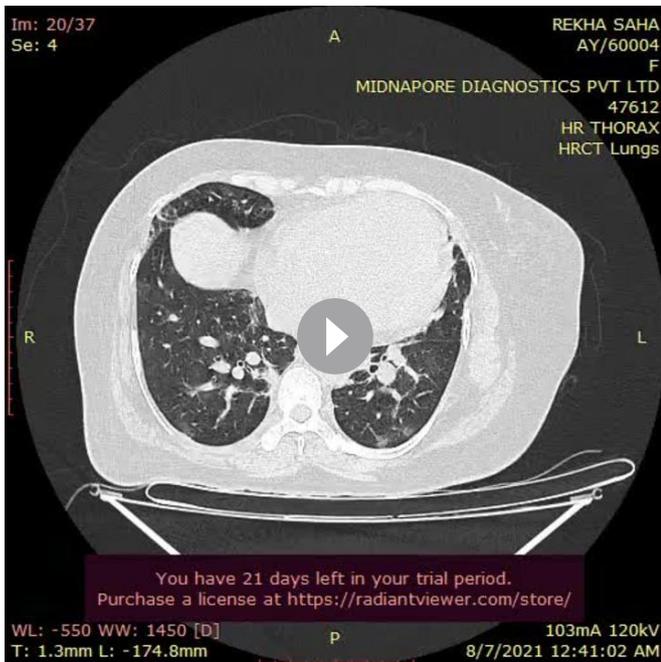


**Figure 2** High-resolution CT scan of the thorax showing areas of bilateral ground-glass opacities along with patchy fibrosis, predominantly in the lower lobes.



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**Video 1** High-resolution CT scan of the thorax of the patient

involvement in the form of acute tubular injury and hepatocellular damage is also common. Mucosal ulceration in the oral cavity and tongue, also known as ‘paraquat tongue’ is seen within the first few days of poisoning. No specific antidote is available for this poisoning, and there is also dearth of evidence-based recommendation for the treatment.<sup>7</sup> Management is mainly supportive. Gastric decontamination with activated charcoal is recommended in those presenting within 1–2 hours of ingestion; however, gastric lavage should not be done due to the corrosive nature of paraquat. Routine use of supplemental oxygen is not recommended in patients with mild-to-moderate hypoxia, as the hypoxemic strategy will reduce the production of ROS. Immunosuppressive agents such as cyclophosphamide, methylprednisolone and dexamethasone, along with antioxidants such as N-acetyl-cysteine

and ascorbic acid, have been investigated in its management without significant success.

### Learning points

- ▶ Paraquat is a toxic bipyridylium herbicide and its ingestion in very small amount can be fatal with toxic effects on kidney, liver, lungs, gastrointestinal tract and other organs.
- ▶ There is no specific antidote. Antioxidants and immunosuppressive agents have been investigated without much success. The prognosis is uniformly poor.

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