## **Case Report**

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# **Paraquat poisoning: a case report**

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## ABSTRACT

Paraquat (1, r-dimethyl-4, 4'-bipyridium dichloride) is a commonly used herbicide in Asia. In spite of its wide availability, poisoning due to this herbicide is uncommon. An ingested dose of more than 10 ml is known to cause irreversible lung injury. Due to lack of any specific diagnostic test or specific clinical signs, diagnosis is often difficult without identifying the compound from history. Poisoning with high doses is associated with poor prognosis. As there is no specific antidote, treatment is aggressive decontamination and supportive management. However, in high doses, cases are often fatal.

Keywords: Paraquat, Poisoning, Pulmonary fibrosis, Prognosis

#### **INTRODUCTION**

N, N'-dimethyl-4, 4'-bipyridinium dichloride, commonly known as paraquat is a herbicide commonly used in India and rest of the world. It is the second highest sold herbicide worldwide. It is a corrosive liquid usually in brown to bluish in colour. Cases of poisoning with paraquat produce both local and systemic toxicity and can often be fatal. Here we discuss a case of suicidal intake of paraquat, which turned fatal.

#### **CASE REPORT**

A 17 Year old girl was brought to emergency department with an alleged history of intake of about a mouthful of liquid herbicide from her home. She was brought to the hospital about 1 hour after the intake of the weedicide. She had pain on opening the mouth and had developed erosions in the oral cavity. There was no history of vomiting, loose stools, seizures, abdominal pain and loss of consciousness or increased secretions from the mouth. On examination, she was conscious and oriented, heart rate 96/min, blood pressure 128/76 mmHg, oxygen saturation 99% on room air, respiratory rate of 20/min. Axillary temperature was 98.8°F. Examination of the oral cavity revealed mucosal erosions and bleeding. Examination of the cardiovascular system was normal and chest was clear with bilaterally equal air entry. Pupils were equal and reacting to light bilaterally. Gastric lavage and activated charcoal was given in the emergency department along with Intravenous fluids. Her blood and urine investigations, liver and renal function test on admission were within normal limits (Table 1).



Figure 1: Chest X-ray of the patient showing bilateral dense opacities.

ECG showed sinus tachycardia. On being stable, she was kept under observation in the medical intensive care unit, with supportive measures of anti-emetics and intravenous fluids. On the next day morning her family members produced the empty packet of paraquat dichloride 24%. Upon diagnosis of acute paraquat poisoning, she was started on dialysis as per the opinion of the consulting nephrologist. Her serum SGPT and SGOT and serum urea and creatinine gradually began to rise from day 2 and she developed breathing difficulty and decreased urine output. She was electively incubated and was kept on mechanical ventilation. She was on treatment with intravenous methylprednisolone 1 gram in 200 ml normal saline 2 hourly, injection N-acetyl cysteine 2 grams stat

followed by 1 gram thrice daily, vitamin C, 6 grams per day and lidocaine mouth wash. Her blood and urine cultures were sterile. She initially responded to treatment, with serum creatine and liver function tests normalizing and she was gradually extubated after weaning off (Table 1). She was under close observation in the intensive care unit for the next 20 days with regular chest X-ray and blood tests. On twenty second day, she developed severe breathing difficulty and was re-incubated. Chest X-ray showed bilateral coarse coalescent opacities with diffuse alveolitis, suggesting acute respiratory distress syndrome. However, despite our best efforts, the patient expired on twenty forth day due to respiratory failure.

#### **Table 1: Investigations.**

Investigation	Day 1	Day 3	Day 7	Day 15	Day 23
S. creatinine (mg/dl)	0.7	5	5.7	2.1	4.5
Blood urea (mg/dl)	15	81	107	35	99
SGOT (U/l)	26	36	333	132	156
SGPT (U/l)	16	29	243	51	207

### DISCUSSION

After oral intake, paraquat gets sequestered in the lungs, releasing superoxide anions, which damage the cell membranes by producing oxygen free radicals, which can lead to pulmonary fibrosis, nephro and hepatotoxicity.<sup>1</sup> Histopathological examination of fatal cases demonstrate pulmonary congestion, hemorrhage and extensive pulmonary fibrosis. Paraquat is known to cause both local as well as systemic toxicity. Local effects include ulcerations and mucosal erosions of the lips, oral cavity and esophagus with patients complaining of burning sensation in the mouth and throat, and systemic effects include pulmonary fibrosis, renal tubular necrosis and hepatic necrosis.<sup>2</sup> Pulmonary toxicity starts with diffuse consolidation, which may progress to pulmonary fibrosis, which is associated with high mortality.<sup>3</sup> Poisoning with high doses may be associated with multi organ dysfunction and cardiogenic shock, which may be rapidly fatal.<sup>2</sup> Detection of paraguat in urine maybe a method to diagnose poisoning as well as grading prognosis.<sup>2,4</sup> A urine paraquat level above 1.6 pg/ml after 12 hours of poisoning is almost always fatal.5

Due to lack of any specific antidote, the mainstay of treatment of cases is usually supportive to avoid free radical mediated injury.<sup>6</sup> Studies have shown that Steroid pulse therapy and cyclophosphamide may be used prevent pulmonary fibrosis.<sup>7</sup> Hemodialysis and gastric decontamination are found to be helpful in the management. Due to increased generation of oxygen free radicals, oxygen is usually avoided in patients, though hypoxic patients may be given low dose oxygen.<sup>8</sup> In spite of aggressive treatment and decontamination, mortality remains high in cases due to development of multi organ dysfunction and irreversible lung damage thus making

the prognosis poor.<sup>9</sup> Poisoning with very low doses and prompt decontamination may favor better outcomes and survival in some cases.

## CONCLUSION

Poisoning with paraquat is uncommon but highly fatal. At present, there is no antidote for paraquat and hence management of cases is limited to supportive care, decontamination and hemodialysis. Unknown poisoning with renal and lung injury should arouse the suspicion of paraquat.

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