

## Pneumomediastinum: An Early Predictor of Mortality in Paraquat Poisoning: A Rare Case Presentation

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### Abstract

**Background:** Paraquat is a broad spectrum liquid herbicide (1,1'- dimethyl-4,4'- dipyridylum) associated with both accidental and intentional ingestion. It has a very high case fatality rate assuming to be due to inherent toxicity and lack of effective treatments. Poisoning by Paraquat herbicide has been a major medical problem in parts of Asia. Despite widespread availability, reports of Paraquat poisoning is not common due to very high case fatality rate might be due to not reaching to tertiary care centres and non specific clinical features with absence of proper history.

**Case Presentation:** We report a case of previously healthy 19 years old male who was brought to the emergency department 3 days after intentional ingestion of approximately 100ml of 24% Paraquat herbicide with complaints of retrosternal chest pain while breathing & dysphagia followed by several episodes of vomiting followed by the development of pneumomediastinum and death from respiratory failure. Paraquat poisoning should always be considered in the differential diagnosis in patients presenting with spontaneous pneumothorax or pneumomediastinum in places with high paraquat poisoning prevalence.

**Conclusion:** Though Pneumomediastinum being an early predictor of 100% mortality in Paraquat poisoning but an emergency physician should establish the diagnosis early and to pursue aggressive decontamination and prevention of further absorption. Increased awareness of the clinician and availability of the laboratory diagnostic methods will definitely help in successful management of paraquat poisoning and to decrease the case fatality rate.

What we already know? There are quite a few case reports that already mentioned about possibilities of occurrence of spontaneous pneumomediastinum, pneumothorax or lung fibrosis along with acute renal failure, liver toxicity and mucosal injury.

What this paper adds? This is rare case report of spontaneous pneumomediastinum in Paraquat poisoning presenting to our tertiary care centre. A chest radiograph or a CT chest should be performed which may be useful in early detection of pneumomediastinum, pneumothorax or lung fibrosis for assessing long term damage in survivors. We specifically point towards the fact that no obvious clinical guideline can decrease the case fatality rates in these kind of cases.

**Keywords:** Paraquat; Pneumomediastinum; Pneumothorax; Liquid herbicide; Case fatality.

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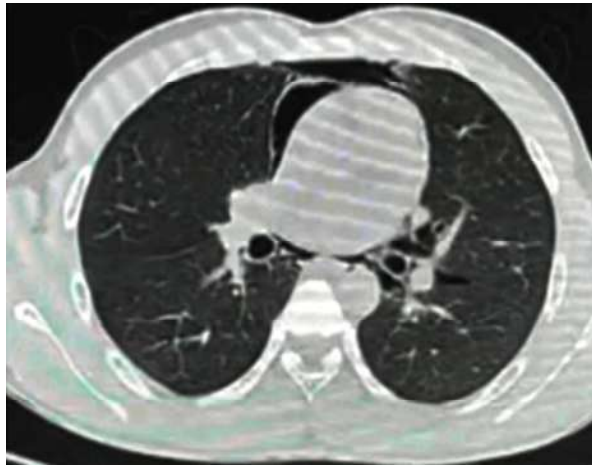
### Introduction

Paraquat is a quaternary nitrogen herbicide that is highly poisonous. About 20% of the patients with Paraquat poisoning develop pneumomediastinum as a complication with a mortality rate of almost 100%.<sup>1</sup> Pneumomediastinum occurring within 8 days after the ingestion is a specific predictor of mortality in Paraquat poisoning.<sup>1</sup> Globally, there are about 20,000 annual fatalities and more

than 2 million hospitalisations due to poisoning. Knowledge about Paraquat poisoning and its rare complication can make it a lot easier for emergency physician and early diagnosis and treatment.<sup>2</sup>

### Case Presentation

We report a case of previously healthy 19 years old male who was brought to the emergency department 3 days after intentional ingestion of approximately 100ml of 24% Paraquat herbicide with complaints of retrosternal chest pain while breathing & dysphagia followed by several episodes of vomiting. Relatives denied any episode of seizure. On examination, he was conscious, alert, afebrile with heart rate 102/min, regular, BP 100/70 mmHg, respiratory rate 22/min, and oxygen saturation (while breathing room air) 97%, Capillary blood glucose was 98mg/dl. His oral mucosa was congested and edematous with ulcerations. Pupils were bilateral 2 mm and reacting to light. Both lung fields were clear on auscultation. The remainder of the systemic examination was unremarkable.



**Fig.1:** High resolution CT chest showed pneumomediastinum on Day 1

by pesticides and agricultural chemicals. Paraquat poisoning accounted for only 0.34% of those cases, but paraquat poisoning had the highest mortality rate, accounting for 13% of all fatal cases.<sup>3</sup> Severe paraquat poisoning is characterised by multiple-organ failure, involving mainly the lungs, kidneys, and liver. The lung is a major target organ in Paraquat poisoning, and respiratory failure from lung injury is the most common cause of death. Spontaneous pneumomediastinum is usually defined as the presence of free air around the mediastinal structures, without any apparent precipitating cause or any underlying pulmonary disease.

After consumption of the herbicide, he was taken to the local hospital where Gastric lavage was performed, and repeated doses of charcoal were given in the emergency department, and he was admitted to the intensive care unit (ICU) for observation and further evaluation. In the ICU, he received IV fluid and antiemetic (Ondansetron) as supportive measure. Initial complete blood count, electrolytes, liver and renal function tests, arterial blood gas, serum amylase, and choline esterase levels were within normal limit. ECG revealed sinus tachycardia, and 2D echocardiography showed normal cardiac chambers with LVEF of 60%.

He was then brought to our tertiary care center for further evaluation and management. After admission, we started administering intravenous fluid, and the patient was admitted to the medical intensive care unit for close observation and further evaluation. Other supportive treatments included proton pump inhibitors, antiemetics and analgesics to control his epigastric pain. The patient was shifted to the CT room for CT thorax and blood drawn were sent for blood workup. High resolution CT chest showed pneumomediastinum. His blood panel showed renal function deteriorating, with markedly increased blood urea (143.5mg/dl) and creatinine levels (9.31mg/dl), and his urine output decreased, serum electrolytes were normal

TLC was raised (19000/ul), deranged Liver function test (Total bi 5.4mg/dl, Direct bil 3.6mg/dl, Indirect bil 1.86mg/dl, SGOT 186U/l, SGPT 175U/l) deranged. Hemodialysis was started. He became increasingly hypoxemic and required intubation and mechanical ventilation. Progressive multiple-organ failure ensued, and he died on day 3 after admission.

### Discussion and Conclusion

This case represents a typical presentation of fulminant paraquat poisoning. The patient had developed pneumomediastinum and multiple-organ failure within 24 hours. Patients with severe paraquat poisoning may be asymptomatic soon after ingestion, but deteriorate quickly within a few hours to days. Newer pesticides such as the synthetic derivatives of pyrethrin, which were believed to be relatively safe to humans, now appear to be implicated in some serious cases of intoxication.<sup>4</sup> This patient also developed pneumomediastinum, probably secondary to air leak from necrotising lung parenchyma or ruptured esophagus. This may also have been a contribution to death from factors other than paraquat toxicity such as complications

of treatment. For example, this patient underwent gastric lavage in other hospital which might have led to aspiration, asphyxia or to mediastinal perforation.<sup>5</sup>

Pneumomediastinum in paraquat poisoning indicates a very poor prognosis, with a mortality rate of almost 100%. Zhou et al. showed that early pneumomediastinum within eight days is a specific predictor of mortality in paraquat poisoning.<sup>3</sup> In humans, paraquat is highly toxic, with an estimated lethal dose in adults of about 3-6 g of paraquat ion. The most common route of poisoning is ingestion (either intentional or accidental) of the concentrated solution. After it is ingested, the gastrointestinal tract absorbs 20% of paraquat. The presence of ulcerated mucosa or an empty stomach increases the fraction of paraquat absorbed. After absorption, paraquat is distributed to highly perfused organs such as the lungs, kidneys, liver, and muscles, and remains partly in the intravascular space. Paraquat concentration in the lung parenchyma is very high (10-20 times greater than in plasma) because of active, energy dependent uptake of paraquat by type 1 and type 2 alveolar epithelium, via the polyamine uptake pathway. This explains why the lungs are the target organs in paraquat poisoning: high tissue concentrations from active uptake and abundant oxygen react with paraquat and form reactive oxygen radicals.

The clinical manifestations of paraquat poisoning range from local irritation to multiple organ failure and death.

In conclusion, the patient with severe paraquat poisoning has a poor prognosis. Because there is no specific antidote, the important approaches are to prevent accidental exposure and to pursue

aggressive decontamination and prevention of further absorption after ingestion. Pneumothorax and pneumomediastinum in a patient with paraquat poisoning is a less uncommon but under diagnosed finding. It has a high index of early mortality, hence reasonable clinical suspicion is always warranted. In places where paraquat poisoning is quite prevalent, it should still be considered in the differential diagnosis of patients presenting with unexplained pneumothorax, pneumomediastinum, or ARDS.

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