Case Report

# Paraquat poisoning with spontaneous pneumothorax in the era of online shopping

**Manish Jha[1], Nayanika Gaur[2,3]**

*[1]Department of Anesthesia and Critical Care, Dr. S. N. Medical College, Jodhpur, Rajasthan, [2]Department of Obstetrics and Gynecology, All India Institute of Medical Sciences, Jodhpur, Rajasthan, [3]Consultant, Kirti IVF Clinic and Gynae Endoscopy Center, Jodhpur, Rajasthan, India*

## Abstract

This is a case of a 22-year-old male student who took paraquat as a substance abuse. He presented with flu-like symptoms and chest X-ray depicting changes of acute respiratory distress syndrome (ARDS). After looking at his purchase history in his cell phone, he was found to have purchased paraquat dichloride online and its use was later confirmed by his roommate. The patient developed respiratory distress with renal failure and succumbed to his condition after 11 days of hospital admission. This report highlights an important social issue of probable substance abuse with paraquat which is a commonly used contact herbicide in rural settings but not-so-common in urban population. Also, the report highlights the unusual complication sequalae of pneumothorax. The high mortality with this substance is because of the unavailability of an antidote and of its unscrutinized availability.

**Keywords:** Acute respiratory distress syndrome, paraquat poisoning, pneumothorax

## Introduction

Paraquat dichloride (1,1-dimethyl-4, 4'-bipyridium dichloride) is a widely used and highly toxic, broad-spectrum (non-selective) contact herbicide and a powerful desiccant. Self-poisoning with pesticides is a major public health concern, especially in developing countries with an estimated 300,000 deaths occurring annually in the Asia-Pacific region alone.[1,2] While the organophosphate class accounts for the majority of hospital admissions, the highest case fatality (>50%) is from paraquat poisoning.[3,4]

## Case Presentation

A previously healthy, 22-year-old male student from urban dwelling, presented to the out-patient department (OPD) at a tertiary care hospital in India, with complaints of sore throat and oral ulcers and multiple episodes of nausea and vomiting over 7 days.

Patient was treated symptomatically in OPD until he complained of passage of fresh blood with the vomitus for which he was admitted to the medical ward (8th day from first symptom). Investigations revealed normal liver function test, severely deranged kidney function test (urea/creatinine 210 mg/dL/10.8 mg/dL) but no metabolic acidosis. Chest X-ray was normal.

On the second day of admission, the patient developed a low-grade fever but was conscious and oriented. His pulse was 110/min, blood pressure (BP) 110/80 mmHg, respiratory rate 20/min and oxygen saturation ($SPO_2$) 97%. The patient was stable during the following week until on the 7th day, he developed severe respiratory distress, $SPO_2$ 85% on high-flow oxygen mask, and respiratory rate 45/min. The patient was intubated and shifted to intensive care unit (ICU). Chest auscultation was unremarkable at that time with chest X-ray showing acute respiratory distress

**Address for correspondence:** Dr. Manish Jha, B-4, Shastri Nagar, Jodhpur - 342 003, Rajasthan, India.
E-mail: manishjha18@gmail.com

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syndrome (ARDS) like in the picture as shown in Figure 1. The patient was empirically started on anti-flu treatment (oseltamivir 75 mg twice a day, broad-spectrum antibiotics, protein pump inhibitors, enteral nutrition and intravenous (IV) fluids). Prone ventilation session was initiated concerning severe ARDS.

On 8th day of admission (15 days after the primary symptom), the patient's relatives reported online purchase of paraquat by the patient by looking at the purchase history in his cell phone. The fact of this substance's abuse by this 22-year-old male was confessed by his roommate. With this information, the patient was given injectable methylprednisolone 1 g for 3 days (followed by dexamethasone 8 mg IV 8 hourly), cyclophosphamide 500 mg IV 12 hourly, acetylcysteine 12 g once a day, Vit C 1.5 g 6 hourly, and vitamin E (alpha-tocopherol) 400 units once a day. In the following days, the patient developed spontaneous right-sided pneumothorax with subcutaneous emphysema followed by left-sided pneumothorax in spite of lung protective ventilation. Pneumothorax was then relieved after bilateral intercostal drainage (ICD) tube insertion as shown in Figure 2. The patient had now stopped responding to vasopressors. No improvement in saturation was found. Later patient went into cardiac arrest but could not be revived despite best efforts.

The authors certify that they have obtained all appropriate patient consent forms in writing from the immediate family of the deceased patient. In the form, the patient's relative (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patient's relative (s) understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

## Discussion

Paraquat is one of the registered pesticides most frequently abused for committing suicide.[5] However, its use as a substance abuse in urban population is uncommon. In India, paraquat is easily available routinely used online portals like Amazon, Flipkart etc., in the name of Milquat, All Clear etc., The most likely route of exposure to paraquat that would lead to poisoning is ingestion. Our patient visited the OPD himself with vague complaints, thereby ruling out the possibility of attempted suicide. Also, the history of buying such a substance by an urban dweller student was a suspicious activity. But, it was only after 15 days after the primary symptom, that the possibility of paraquat poisoning due to probable abuse overdose was suspected. Paraquat is rapidly but incompletely absorbed and then largely eliminated unchanged in urine within 12–24 h. Therefore, the presence of paraquat in one's body can be confirmed using the urine dithionate test. Data suggest that cut-off duration of 30.4 h and 34.5 h has a sensitivity of 81% and 71.4%, respectively.[5]

Paraquat primarily affects the lungs by causing acute alveolitis in the alveolar epithelium leading to diffuse alveolar collapse, fibrosis and vascular congestion along with inflammatory cascade in the vascular endothelium, finally leading to apoptosis of cell in the lungs.[6] These events can be clinically correlated to the clinical picture found in our patient. Since our patient developed respiratory symptoms around the 7th–10th day, the mortality rate in paraquat suicide attempts is comparatively high (42–80%).[7] This may be due to delay in presenting to hospital and unavailability of specific antidote along with its inherent toxicity. Once signs of systemic toxicity in paraquat poisoning appear, aggressive and invasive therapy even in an ICU is unlikely to improve outcome.

Management in such a case aims at preventing further absorption of the poison, gastrointestinal decontamination, avoidance of free radical injury to the lungs, and prevention of pulmonary fibrosis. Use of activated charcoal (1 g/kg in water; maximum dose 50 g) or Fuller's earth (2 g/kg in water; maximum dose 150 g in water) is recommended as absorbents for paraquat poisoning as no specific antidote is available. In our case, we did not use the absorbents due to the delayed presentation. The role of haemodialysis and or haemoperfusion is limited with paraquat poisoning due to two reasons. First, most of the paraquat is spontaneously eliminated rapidly in the initial 6–12 h,

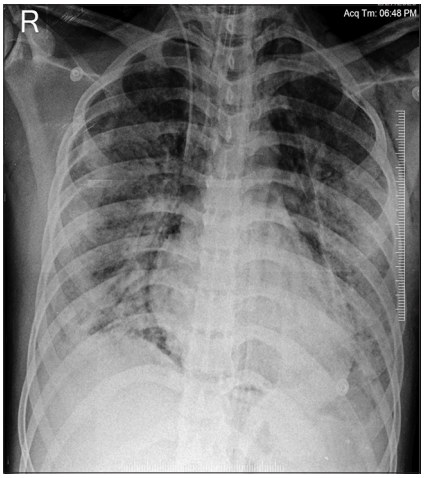


**Figure 1:** Day 1 of ICU admission – diffuse bilateral patches – ARDS

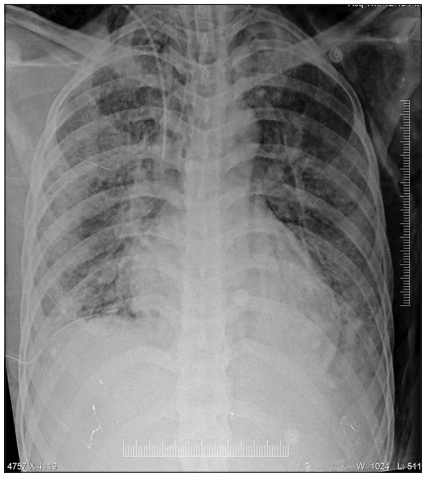

**Figure 2:** Day 4 of ICU admission – pneumothorax with ICD tube in right lung

and second, the time required for its impact on the lungs is very short. The use of immunosuppressive drugs is widely accepted based on the theory that paraquat leads to acute inflammatory reaction and these agents have anti-inflammatory properties, although clinical evidence to support their use is limited. Similarly, the supportive role of anti-oxidants, superoxide dismutase, *N*-acetylcysteine and nitrous oxide, to reduce the oxygen-free radical-related lung injury, is not supported by strong evidence, although animal models have shown some positive laboratory findings favouring their use.[8]

Since such presentations occur first to the primary care physicians, it is imperative for them to understand the importance of early diagnosis in paraquat poisoning. Urine dithionate test, which is a simple and cheap test, must be added in the initial diagnostic test checklist in every case of suspected poisoning as its sensitivity is highest when performed at the earliest. Lastly, primary care physicians must have a low threshold for spontaneous pneumothorax even if patient is on lung-protective ventilation in cases of paraquat poisoning.

## Conclusion

Early detection is the key point in managing paraquat poisoning as no antidote is available. Primary care professionals must be aware of the symptomatology of paraquat poisoning and timely use of urine dithionate test for early diagnosis and management. Deadly poisons such as paraquat must not be made readily available for our younger generations to experiment with. With this, we request our policymakers to mark the availability of such substances under licensed use.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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## Conflicts of interest

There are no conflicts of interest.

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