

Case Report

A rare case of Paraquat poisoning developing pneumothorax

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Abstract

Paraquat poisoning can result in lung, kidney, heart, suprarenal gland damage but development of pneumothorax following paraquat consumption is rare. We report one such case.

Keywords: paraquat, pneumothorax, immunosuppression

1. Case study

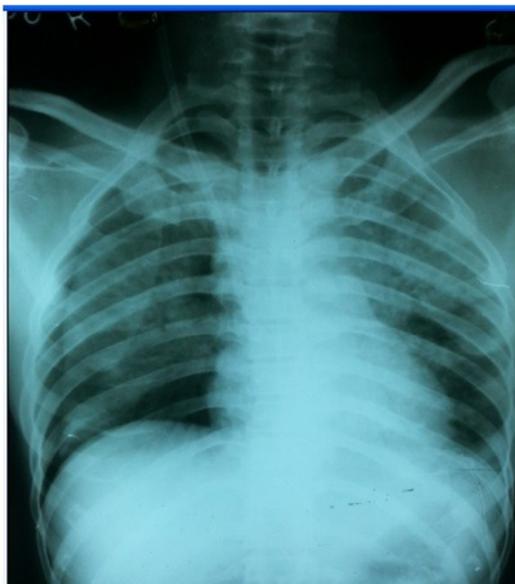
A gentleman aged 46 years presented to the casualty of our institute with alleged history of consuming paraquat 3 days back at his residence. The amount of poison consumed was about 10-12 ml of 20% concentrate. After consuming poison, stomach wash had been given 3 days back at a nearby primary health centre. On admission at our institute, the only presenting complaint was oliguria. His general condition was stable. Blood pressure was slightly higher-140/100mmHg. Systemic examination was within normal limits. Chest X ray done showed no significant abnormality

Fig1: Chest X Ray at presentation without any significant abnormality



Investigations revealed acute renal failure with blood urea-98.9 mg/dl and serum creatinine-4.29 mg/dl. Provisional diagnosis of paraquat poisoning with acute renal failure was made. Opinion by the nephrologist was sought; he favoured initiation of immunosuppressive therapy. He was administered immunosuppressive therapy with methylprednisolone. But, on day 4 of admission, renal functions worsened further with blood urea-169 mg/dl and serum creatinine-7 mg/dl. He was initiated on hemodialysis. He improved slightly after hemodialysis. But he started becoming breathless on day 6. Air entry was equal on both sides. There were crepitations in bilateral infraclavicular, mammary and axillary areas, more on left side. On X ray, he was found to have heterogenous opacities in bilateral upper and mid zone suggestive of air space consolidation. He was started on appropriate antibiotic therapy for the same. On next day, dyspnoea worsened suddenly. Repeat chest X ray done showed Pneumothorax on right side.

Fig 2: Pneumothorax on right side with air space consolidation in bilateral upper& mid zones.



Intercostal drainage tube was placed immediately, after which he improved slightly. But he developed acute respiratory distress syndrome early morning next day. Though he was intubated and initiated on mechanical ventilation, respiratory distress progressively worsened and he expired late evening on the same day.

2. Discussion

Paraquat (1,1'-dimethyl-4,4'-bipyridinium) dichloride is a non-selective contact herbicide widely used in many countries across the world. In South-East Asian countries, it constitutes an important cause of poisoning with suicidal intention. Poisoning is usually for attempting suicide or it is ingested accidentally. Though poisoning due to percutaneous absorption is theoretically possible, it is quite rare. The LD50 in humans is approximately 3-5 mg/kg, which translates into as little as 10-15 mL of a 20% solution.¹ It can cause damage to lungs, kidneys, heart, suprarenal glands or muscles.² The lung is the primary target organ of paraquat, and its effects on lungs represent the most lethal but unfortunately least treatable manifestation of toxicity. Pulmonary involvement can result in consolidation, lung fibrosis, acute respiratory distress syndrome. Acute exposure to high doses of paraquat can result in acute pulmonary edema. It is corrosive and can directly cause injuries to trachea, main bronchi.³ Rarely, it can cause pneumothorax, pneumomediastinum, pneumopericardium, subcutaneous emphysema.⁴ Unfortunately, our patient developed this rare complication. The most common pulmonary condition responsible for mortality following paraquat poisoning is pulmonary fibrosis, which usually develops 7-14 days after ingestion.⁵ Though the outcome of individual cases of paraquat poisoning is variable, it is a poison with quite high mortality. Large studies carried out earlier have shown the mortality figures after consumption of this poison to be varying between 40 and 60%. Most of the deaths due to this poison occur within 24-72 hours either from renal, pulmonary complications or from multi-organ failure. Onset of renal failure with increase in creatinine $>3 \mu\text{mol/L/h}$ has been proposed to be a predictor of mortality in patients with paraquat poisoning. This has been supported by a small study on 18 patients.⁶ Not only creatinine, but blood levels of some other markers have also been evaluated for their association with

mortality after paraquat poisoning. Among them, the rise in cystatin C has been found to be a useful marker of adverse outcomes following paraquat poisoning.⁷ But, another biomarker neutrophil gelatinase-associated lipocalin (NGAL) has not been found to be useful in predicting mortality. Treatment of poisoning consists of administration of adsorbents as soon after exposure as possible to prevent further absorption. Intravenous fluids may be administered to maintain adequate urine output. However if renal impairment develops, hemodialysis is indicated. Immunosuppression using cyclophosphamide and methylprednisolone has been found to be useful to improve prognosis in some clinical trials.⁸ The treatment modalities are likely to be useful only if the patient survives beyond 48 hours after poisoning.⁹ Cyclophosphamide could not be administered in our patient because of renal failure. So, only methylprednisolone was administered. Unfortunately, immunosuppression, though started early was not helpful in improving the prognosis.

Our case supports some of the theories proposed earlier.

1. High creatinine is poor prognostic indicator.
2. In consumption of paraquat in moderate doses (20-40 mg paraquat ion per kg body weight), death results because of pulmonary toxicity.¹⁰

From our case report, we can get a message that any patient with paraquat poisoning develops sudden breathlessness, one has to keep development of pneumothorax in mind, which is a treatable and curable as well.

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