Case Report

A rare case of Paraquat poisoning developing pneumothorax

Ram S Kaulgud*, Arun B S, Nagaraj A R and Vijayalakshmi P B

Department of Internal Medicine, Karnataka Institute of Medical Sciences, Hubli, India.

*Correspondence Info:
Dr. Ram S Kaulgud
Assistant Professor,
Department of Internal Medicine,
Karnataka Institute of Medical Sciences, Hubli, India.
E-mail: ramk72@yahoo.com

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 many countries across the world. In South-East Asian countries, it constitutes it is 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 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 form renal, pulmonary complications or from multi-organ failure. Onset of renal failure with increase in creatinine >3 μmol/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. 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. The treatment modalities are likely to be useful only if the patient survives beyond 48 hours after poisoning. 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.

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.

References

1. Pond SM. Manifestations and management of paraquat poisoning. Med J Aust 1990; 152: 256-9.
2. Rusell, LA. Paraquat poisoning: toxicologic and pathologic findings in three fatal cases. Clin Toxicol 1981; 18: 915-928.
3. Ruiz-Bailen M, Serrano-Corcoles MC, Ramos-Cuadra JA. Tracheal Injury Caused by Ingested Paraquat. Chest 2011; 119(6): 1956-1957.
4. Chen, KW, Wu, MH, Huang, JJ, et al Bilateral spontaneous pneumothoraces, pneumopericardium, pneumomediastinum, and subcutaneous emphysema: a rare presentation of paraquat intoxication. Ann Emerg Med1994; 23: 1132-1134.
5. Bismuth C, Garnier R, Dally S, et al. Prognosis and treatment of paraquat poisoning: A review of 28 cases. J Toxicol Clin Toxicol 1982;19:461-74.
6. Ragoucy-Sengler and Pileire, 1996 . C. Ragoucy-Sengler, B. Pileire A biological index to predict patient outcome in paraquat poisoning. Hum. Exp. Toxicol 1996; 15: 265–26.
7. Changes in the concentrations of creatinine, cystatin C and NGAL in patients with acute paraquat self-poisoning. Darren M. Roberts, Martin F. Wilks, Michael S. Roberts, Ramasamyiyer Swaminathan. Toxicology Letters 2011; 202(1): 69–74.
8. Agrwal R, Sirinivas R, Agrawal AN, Gupta D. Immunosuppressive therapy in lung injury due to paraquat poisoning: A metaanalysis. Singapore Med J.2007; 48(11):1000-5.
9. Eddleston M, Wilks MF, Buckley NA. Prospects for treatment of paraquat-induced lung fibrosis with immunosuppressive drugs and the need for better prediction of outcome: a systematic review. Q. J. Med 2003; 96: 809–824.
10. Vale JA, Meredith TJ, and Buckley BM. Paraquat poisoning: Clinical features and immediate general management. Hum Toxicol 1987; 6: 41-7.