Case Report

Paraquat Poisoning Presenting as the "Daisley Barton Syndrome"

Souvik Chaudhuri, Maddani Shanmukhappa Sagar, Sunil Ravindranath, Vinod Reddy[1]

Departments of Critical Care Medicine and [1]Anaesthesia, Kasturba Medical College, Manipal University, Manipal, Karnataka, India

Abstract

Paraquat is a bipyridilium herbicide which is highly toxic. Due to its corrosive effects, it can lead to esophagitis, esophageal rupture, and gastritis. It leads to the formation of reactive oxygen species which causes multiorgan damage, prominent among those being pulmonary, renal, and liver dysfunction. One of the rare causes of respiratory distress after paraquat poisoning is spontaneous pneumothorax due to pneumocyte injury, also known as the "Daisley Barton Syndrome." We report a case of paraquat poisoning who developed alveolitis initially and later had bilateral spontaneous pneumothorax. Bilateral chest tubes were inserted, and the patient recovered initially but later deterorated due to other complications. We should be aware of this phenomenon, and sudden worsening of the respiratory parameters should alert the clinician for prompt treatment.

Keywords: Daisley Barton syndrome, paraquat poisoning, reactive oxygen species, spontaneous pneumothorax

Introduction

Paraquat, a quaternary ammonium herbicide, gets concentrated in the lungs post ingestion.[1] It leads to the formation of hydrogen and superoxide anions and reactive oxygen species (ROS), causing free-radical damage in the lungs, liver, kidney as well as pancreas.[1,2] High mortality associated is due to the absence of any specific antidote.[2] Respiratory dysfunction is the most common cause of death in paraquat poisoning.[2] Spontaneous bilateral pneumothorax after ingestion is rare and has been reported up to 7 years after ingestion.[3] We must be aware of this underdiagnosed manifestation of paraquat poisoning both in the acute and chronic phases of treatment.[4]

Case Report

A male patient aged 18 years was admitted to the intensive care unit (ICU) with a history of ingestion of about 50 mL of paraquat (24% v/w). He was initially treated at another hospital where gastric lavage with activated charcoal was done. He had gradual worsening of clinical condition along with multiorgan dysfunction for which he was referred to our hospital. At presentation, he was tachypneic with respiratory rate of about 38–40/min, was hemodynamically stable, and had SpO_2 of 84% on room air. There was extensive corrosive damage to the lips and tongue. Chest roentgenogram (CXR) revealed diffuse bilateral infiltrates suggestive of alveolitis. He had nonoliguric acute kidney injury (AKI) with serum urea of 144 mg/dL and creatinine 6.88 mg/dL. He also had liver dysfunction with raised bilirubin and liver enzymes. He was started on measures to ameliorate free-radical injury and inflammation. He was administered injection dexamethasone, injection *N*-acetyl cysteine (NAC), and Vitamin C as supportive therapy. However, his respiratory distress increased on the 2nd day of admission with SpO_2 decreasing to 85% on 40% venturi by face mask. He also had subcutaneous emphysema over the neck and anterior chest. Lung ultrasonography (USG) revealed the absence of lung sliding on the right side, and CXR was done immediately, which revealed bilateral pneumothorax [Figure 1]. Immediate intercostal tube drain was secured, and CXR confirmed

Address for correspondence: Dr. Maddani Shanmukhappa Sagar, Department of Critical Care Medicine, Kasturba Medical College, Manipal University, Manipal, Karnataka, India.
E-mail: sagarmaddani04@gmail.com

Access this article online

Quick Response Code:

Website: www.ijrconline.org

DOI: 10.4103/ijrc.ijrc_47_19

How to cite this article: Chaudhuri S, Sagar MS, Ravindranath S, Reddy V. Paraquat poisoning presenting as the "Daisley Barton syndrome". Indian J Respir Care 2020;9:110-2.

Received: 19-09-2019 **Revised:** 07-10-2019
Accepted: 21-10-2019 **Published:** 08-01-2020

the resolution of pneumothorax [Figure 2]. Patient's condition gradually stabilized with resolving AKI and liver dysfunction. The patient was shifted to a step-down unit but again developed respiratory distress 2 days later. He was readmitted to ICU due to hypoxic respiratory failure and required invasive mechanical ventilation. Two-dimensional echocardiography revealed moderate pulmonary artery hypertension. He had clinical deterioration with development of pneumomediastinum [Figure 3]. Finally, he succumbed during the course of the disease. Postmortem examination revealed increase in weight and firmness of both the lungs. The right lung weighed 1280 g and the left lung weighed 703 g, with histopathology showing pulmonary hemorrhage and organizing pneumonia.

Discussion

The mechanism of injury by paraquat is the generation of ROS and hydroxyl radical which destroy the integrity of cell membranes.[4] Although the toxic effects can be seen in the liver, kidney, and pancreas, the selective toxicity of the lung is due to energy-dependent uptake of paraquat by type 1 and type 2 alveolar epithelium.[2,4] The type 1 and type 2 pneumocytes are destroyed by the ROS, leading to the prevention of gas exchange and loss of surfactant. The loss of surfactant leads to increase in surface tension inside the alveoli, resulting in their rupture and eventually pneumothorax and pneumomediastinum.[4] The other mechanisms of pneumothorax have been explained due to secondary pulmonary hypertension, collagen deposition in alveoli, leading to increased alveolar pressures and eventually pneumothorax.[4,5] This phenomenon of pneumothorax and pneumomediastinum has been referred to as the "Daisley Barton syndrome."[3,4] This production and deposition of collagen in the alveoli has been reported to occur as early as the 3rd day after paraquat poisoning.[4] Since the postmortem findings showed pulmonary hemorrhage, alveolar rupture due to the loss of surfactant could have contributed to the pneumothorax in our patient. The initial CXR findings suggested diffuse inflammatory changes. However, due to the rapidly progressing respiratory dysfunction, USG was done which revealed absent lung sliding. CXR eventually revealed bilateral pneumothorax. Thus, in this patient, the cause of sudden increase in respiratory distress was the rare bilateral spontaneous pneumothorax, rather than the common alveolitis and consolidation.

The most common findings of lung injury include diffuse consolidation within 7 days, cystic linear pattern at 15 days, and interstitial pulmonary fibrosis after the acute event.[6,7] Cholestasis, acute tubular necrosis, pancreatitis, and cardiogenic shock are the other common features in the multiorgan dysfunction.[1,2]

Management in the absence of a specific antidote is directed at supportive treatment to avoid free-radical damage.[1] Initial gastric decontamination using adsorbents such as activated charcoal and hemoperfusion within 4 h of ingestion have been recommended.[2] Other modalities of treatment include

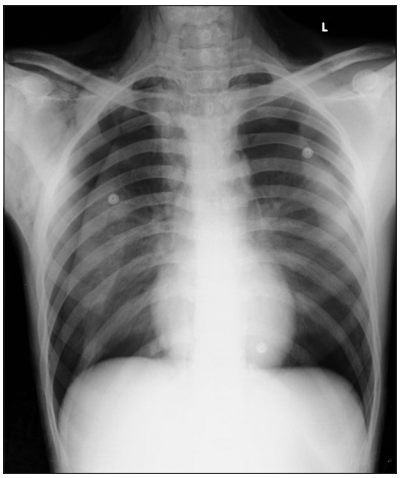

Figure 1: Chest radiography revealing the bilateral pneumothorax

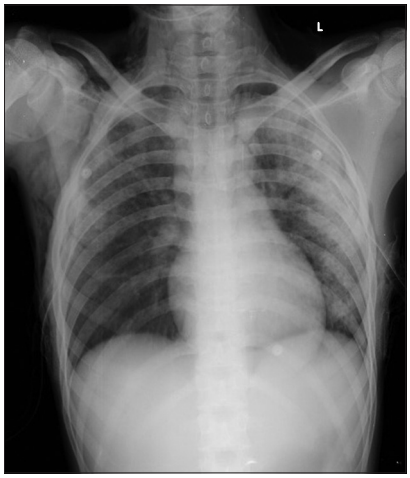

Figure 2: Resolution of the bilateral pneumothorax and lung expansion after the intercostal drain insertion on both sides

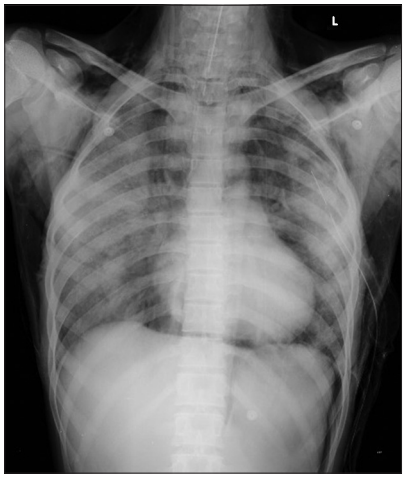

Figure 3: Nonhomogeneous infiltrates with pneumomediastinum appearing as the "continuous diaphragm sign"

Vitamin C and E administration to prevent free-radical induced pulmonary damage,[8,9] methylprednisolone, or dexamethasone to prevent pulmonary fibrosis.[10,11]

NAC to ameliorate hepatotoxicity[1] and continuous venovenous hemofiltration to avoid circulatory collapse.[12] Oxygen supplementation should be avoided, as it enhances ROS-induced damage and should only be given in cases of hypoxia.[1,2]

Conclusion

Utmost vigilance must be maintained while managing a case of paraquat poisoning in the critical care unit, and apart from alveolitis and diffuse consolidation, the possibility of spontaneous pneumothorax must be kept in mind. Prompt bedside lung USG and CXR should be ordered if there is sudden clinical deterioration to rule out pneumothorax.

Declaration of patient consent

The authors certify that they have obtained appropriate patient consent forms. In the form the legally authorized representative has given his consent for his images and other clinical information to be reported in the journal. The LAR understands that his name and initials will not be published and due efforts will be made to conceal his identity.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Raghu K, Mahesh V, Sasidhar P, Reddy PR, Venkataramaniah V, Agrawal A. Paraquat poisoning: A case report and review of literature. J Family Community Med 2013;20:198-200.
2. Shashibhushan J, Venugopal K, Lingaraja M, Patanjali CP, Suresh C, Huggi V. Paraquat: A fatal poison. Med J DY Patil Univ 2015;8:370-4.
3. Daisley H, Barton EN. Spontaneous pneumothorax in acute paraquat toxicity. West Indian Med J 1990;39:180-5.
4. Meyers D, Rampersad A, Christopher M, Pegus S, Daisley H. Underdiagnosed paraquat induced pneumothorax and pneumomediatinum, the Daisley Barton syndrome: A clinical feature of paraquat –Induced acute lung injury. Int J Clin Pharmacol Toxicol 2018;7:296-9.
5. Zhou CY, Kang X, Li CB, Li XH, Liu Y, Wang Z, *et al.* Pneumomediastinum predicts early mortality in acute paraquat poisoning. Clin Toxicol (Phila) 2015;53:551-6.
6. Ntshalintshali SD, Manzini TC. Paraquat poisoning: Acute lung injury – A missed diagnosis. S Afr Med J 2017;107:399-401.
7. Im JG, Lee KS, Han MC, Kim SJ, Kim IO. Paraquat poisoning: Findings on chest radiography and CT in 42 patients. AJR Am J Roentgenol 1991;157:697-701.
8. Khosya S, Gothwal S. Two cases of paraquat poisoning from Kota, Rajasthan, India. Case Rep Crit Care 2012;2012: Article 652146. p. 3.
9. Bismuth C, Garnier R, Baud FJ, Muszynski J, Keyes C. Paraquat poisoning. An overview of the current status. Drug Saf 1990;5:243-51.
10. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Experience with paraquat poisoning in a respiratory intensive care unit in North India. Singapore Med J 2006;47:1033-7.
11. Buckley NA. Pulse corticosteroids and cyclophosphamide in paraquat poisoning. Am J Respir Crit Care Med 2001;163:585.
12. Koo JR, Kim JC, Yoon JW, Kim GH, Jeon RW, Kim HJ, *et al.* Failure of continuous venovenous hemofiltration to prevent death in paraquat poisoning. Am J Kidney Dis 2002;39:55-9.