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A CASE REPORT

Daisley Barton Syndrome- A Rare Presentation of Paraquat Toxicity -A Case Report

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Abstract: ***Background***: Paraquat poisoning is a severe and often fatal condition resulting from exposure to the herbicide paraquat. Ingestion or significant inhalation can lead to rapid progression of symptoms, including respiratory failure, gastrointestinal distress, and multi-organ dysfunction. The toxicity primarily involves the generation of reactive oxygen species, resulting in oxidative stress and extensive cellular damage, particularly in the lungs. ***Case Presentation:*** Here, we report the case of a 27-year-old male from Chennai, India, who deliberately ingested paraquat. He subsequently developed multi-organ injury involving the kidneys, liver, and lungs, along with complications such as spontaneous pneumothorax and pneumomediastinum. Management was largely supportive, even with early intervention, the prognosis was poor. ***Conclusion:*** There is increased regulation in the availability of paraquat to the public, due to its high toxicity but still paraquat poisoning continues to be a major public health issue, particularly in South Asia. This case highlights the need for increased awareness, early diagnosis, and multidisciplinary management in addressing paraquat poisoning.

Keywords: Paraquat poisoning, gastrointestinal distress, spontaneous pneumothorax.

INTRODUCTION

Paraquat poisoning is a serious toxicological issue worldwide, though case reports from India are relatively less (1).Paraquat, scientifically identified as 1,1′-dimethyl-4,4′-dipyridylium, is a highly toxic, corrosive liquid with a distinctive odor, primarily used as an agricultural herbicide (2). Upon ingestion, paraquat tends to accumulate in vital organs such as the lungs, liver, and kidneys, with the lungs being the most severely affected (3). Its toxic effects include local corrosive injury to the oral cavity and gastrointestinal tract, along with systemic complications such as metabolic acidosis, acute kidney and liver damage, pulmonary fibrosis, and acute respiratory distress (4).

In rare cases, paraquat poisoning can lead to spontaneous pneumothorax or pneumomediastinum, conditions collectively known as Daisley Barton Syndrome (5). These rare lung injuries represent a critical manifestation of paraquat toxicity, marked by rapid progression and significant clinical severity. In this report, we present a case of Daisley Barton Syndrome in a 27-year-old male emphasizing the clinical presentation, diagnostic complexities, and management of this rare and life-threatening condition following paraquat poisoning.

RESULTS

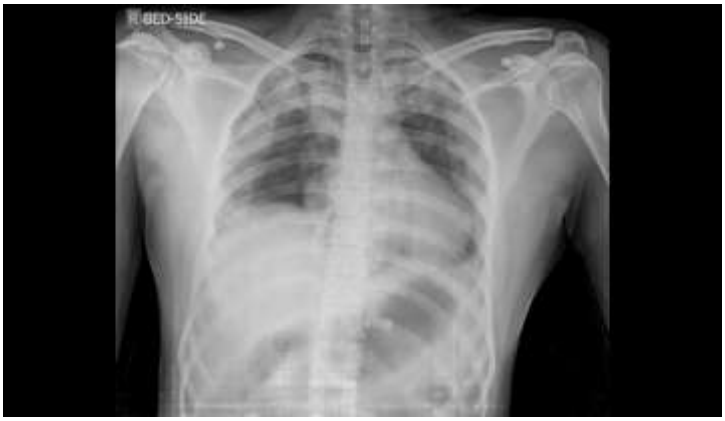
Case Presentation

A 27 years old male with no known comorbidities was brought to the emergency department of our institute after having ingested 250mL paraquat at his residence on Day 8. He was treated conservatively with iv fluids and gastric lavage with activated charcoal at multiple hospitals and transferred to our hospital. Upon admission he complained of exertional breathlessness and reduced urine output which was “Modified Medical Research Council (MMRC)” Grade II that worsened to Grade III. It was not associated with chest pain, hemoptysis, orthopnea and cough.

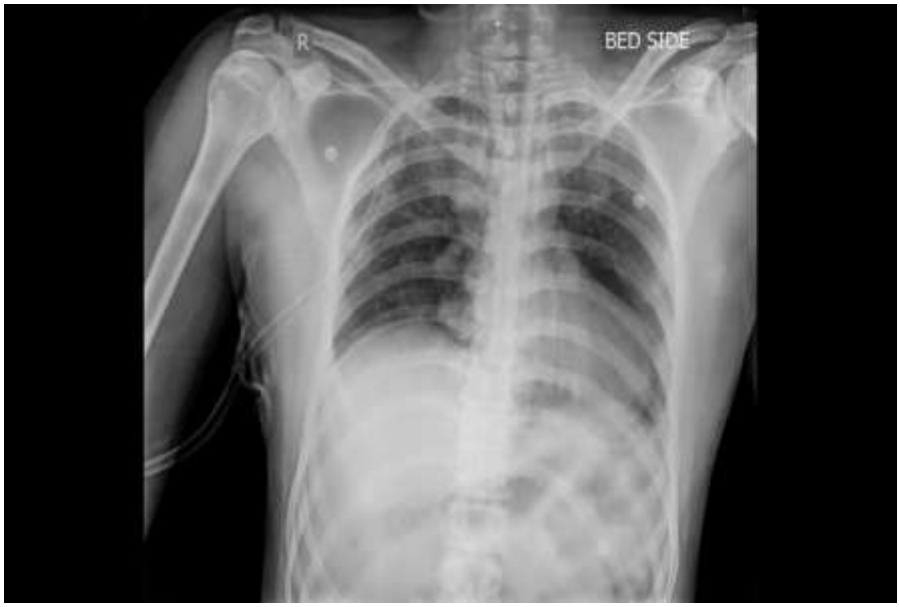
On *examination*, the patient was conscious. His vitals were as follows:
Heart rate 94 beats per minute, Oxygen saturation 75% in room air and improved to 92% with 5L oxygen using facemask, blood pressure 110/80 mmHg, and respiratory rate 26 breaths per minute. On general examination, pallor and icterus was present. Respiratory system examination showed bilateral crepitations in the infra axillary and mammary regions. Other system examinations were normal. Local examination revealed multiple erosions and whitish adherent plaques with hemorrhagic crusting over lips and scrotal area suggestive of toxic spill excoriations.
Laboratory investigations revealed neutrophilic leukocytosis, elevated urea, and elevated creatinine, hyperbilirubinemia and transaminitis. ABG suggestive of Type 1 Respiratory failure.

Table/Fig 1: The patient's blood work-up.

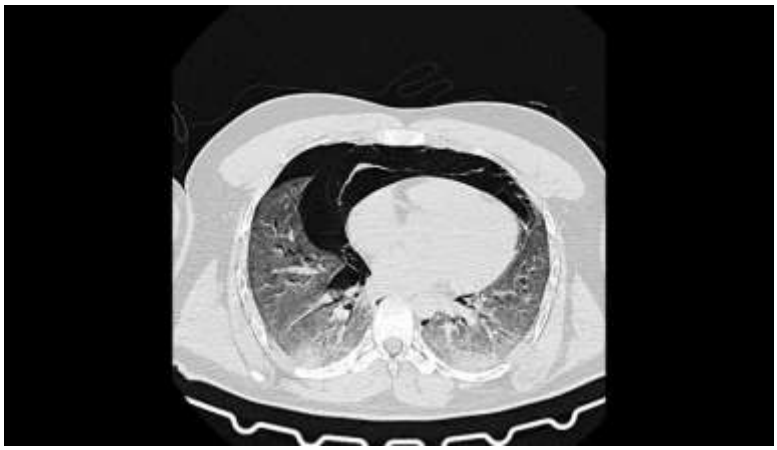
Days of Admission	Day 1	Day 7	Day 21	Reference ranges
Complete Blood counts				
Hemoglobin	13.1	14.4	11.6	Males: 13-17g/dl
WBC	5530	15,710	10,690	4000-11000
Platelets	2, 27,00	3,86,000	1,52,000	1,50,000-4,10,000
Renal Function test				
BUN/ Urea	299/140	131/61	44/21	BUN- 6 - 20mg/dl Urea- 17- 43 mg/dl
Sr Creatinine	13.5	1.9	0.6	0/5-1.2 mg/dl
Liver Function Test				
T Bilirubin	18.44	27.85	5.07	0.5- 1.0 mg/dl
Direct Bilirubin	7.36	14.08	2.12	Upto 0.3 mg/dl
AST	271	538	99	<31 IU/L
ALT	421	1275	524	<34 IU/L
ALP	271	294	233	30-120 IU/L
GGT	461	592	374	<38 U/L
Albumin	3.3	3.1	2.8	3.5- 5.2 g/dl
Globulin	3.3	3.3	3.4	2.5 - 3.0 g/dl
Total Protein	6.6	6.4	6.2	6.6 - 8.3 g/dl



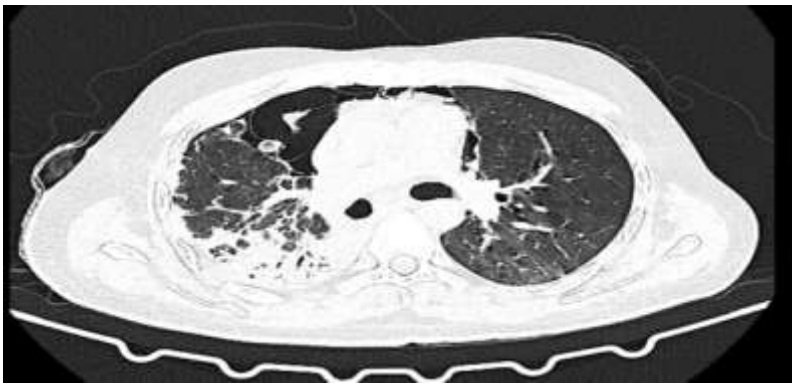
Table/Fig 2: Initial Pneumomediastinum along paracardiac region



Table/Fig 3: Day 2- After ICD insertion



Table/Fig 4: CT chest (Day 1) suggestive of Bilateral pneumothorax with moderate pneumomediastinum along with features of aspiration pneumonitis.



Table/Fig 5: CT chest (Day 21) showing mild to moderate increase in pneumomediastinum, ICD tube on right side, significant reduction of pneumothorax with peribronchovascular opacities and subpleural atelectatic bands in Superior segments of both lobes.

DISCUSSION

Daisley Barton Syndrome is a rare and severe form of paraquat poisoning, primarily resulting from the herbicide's toxic effects on the respiratory system (6). This condition is characterized by spontaneous pneumothorax and pneumomediastinum. The pathophysiology of paraquat toxicity is driven by the generation of reactive oxygen species (ROS), leading to oxidative stress and extensive cellular damage (6).

Paraquat causes injury mainly by generating reactive oxygen species (ROS) and hydroxyl radicals, which compromise cell membrane integrity (7). Although the liver, kidneys, and pancreas can be affected, the lungs are particularly vulnerable due to the active transport of paraquat into type 1 and type 2 alveolar epithelial cells (7,8). ROS-induced damage to these cells impairs gas exchange and reduces surfactant levels. This loss of surfactant raises surface tension within the alveoli, resulting in their rupture, leading to pneumothorax and pneumomediastinum. Additionally, pulmonary hypertension and collagen buildup in the alveoli elevate alveolar pressure, further contributing to pneumothorax (7,9). The occurrence of pneumothorax and pneumomediastinum in paraquat poisoning is referred to as "Daisley Barton syndrome" (7,10).

The patient initially presented with acute kidney injury (AKI), a common early manifestation of paraquat toxicity attributed to its nephrotoxic properties and systemic oxidative stress (11). Emergent hemodialysis was initiated, a standard intervention for toxin clearance in poisoning-related AKI. However, while hemodialysis facilitates paraquat elimination in the initial phase, its efficacy diminishes once the toxin undergoes intracellular sequestration and systemic distribution.
A key feature of paraquat toxicity is lung damage, which was evident in this patient. He developed aspiration pneumonitis along with bilateral pneumothorax and pneumomediastinum, significantly worsening his condition. Paraquat-induced pulmonary injury results from oxidative stress damaging the alveolar epithelium, ultimately leading to fibrosis, inflammation, and respiratory failure (12). The progression of pneumothorax and pneumomediastinum required prolonged intercostal drainage, highlighting the severity of lung involvement. Although pleurodesis was considered, it was not pursued due to uncertain benefits in this case.

Hepatic dysfunction also emerged as a notable complication, as indicated by elevated bilirubin and transaminase levels. This likely resulted from both direct liver toxicity caused by paraquat and systemic oxidative damage. Supportive care, including hepatoprotective agents and antioxidants such as N-acetylcysteine, was administered, leading to some improvement in liver function over time.

Despite intensive supportive measures, the patient ultimately succumbed to respiratory failure. This case highlights the significant challenges associated with paraquat poisoning, given the lack of a definitive antidote. Early and aggressive decontamination, antioxidant therapy, and organ support play critical roles in management. However, once pulmonary fibrosis and respiratory failure develop, the prognosis remains poor.

Given that paraquat ingestion is frequently linked to self-harm, psychiatric support is a crucial component of management. The patient received psychiatric counseling, emphasizing the need for a multidisciplinary approach in poisoning cases. This case represents the devastating impact of severe paraquat poisoning, the limitations of available treatment strategies, and the importance of preventive measures. Strengthening regulatory control of paraquat and enhancing mental health support are key steps in reducing self-poisoning incidents.

CONCLUSION

Daisley Barton Syndrome is an uncommon yet severe consequence of paraquat poisoning, marked by the occurrence of spontaneous pneumothorax and pneumomediastinum. This case emphasizes the swift deterioration of multiple organ systems, particularly the lungs and kidneys, despite intensive supportive treatment. The absence of a specific antidote highlights the critical need for early medical intervention, a multidisciplinary treatment approach, and preventive measures such as stricter regulations and enhanced mental health support. Raising awareness and ensuring prompt diagnosis are essential to improving patient outcomes in cases of paraquat toxicity

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