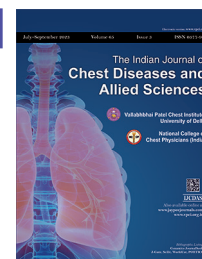


CASE REPORT

Isolated Spontaneous Pneumomediastinum Associated with Paraquat Poisoning: A Case Report

Sanjay Fotedar¹, Karthik A Shiv², Vikas Bhatti³

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ABSTRACT

Paraquat bipyridal herbicide, accidental and suicidal ingestion of the compound is associated with fatal toxicity. The corrosive nature of paraquat results in local effects including oral, esophageal, and gastric ulceration with esophageal rupture at times. Oxygen reactive species generation results in systemic manifestations characterized by MODS, acute liver and kidney injury, metabolic acidosis, acute respiratory distress syndrome, and pulmonary fibrosis. Spontaneous pneumothorax (Daisley Barton syndrome) with or without associated pneumomediastinum and subcutaneous emphysema has been reported with this poisoning and represents poor prognostic markers. An adult male was referred to our Institute for Management of MODS with impending respiratory failure with a history of paraquat ingestion, contrast-enhanced computed tomography (CECT) thorax revealed Pneumomediastinum with bilateral pulmonary infiltrates. The patient was managed accordingly and continuous renal replacement therapy (CRRT) was planned for acute renal shut-down, patient expired on the next day of admission. Isolated pneumomediastinum without subcutaneous emphysema and pneumothorax is an uncommon presentation associated with paraquat poisoning. Authors report a case of pneumomediastinum associated with paraquat poisoning with the aim to stress on pulmonary complications associated with paraquat. It is recommended that in patients with unrecognizable etiology of pneumomediastinum with or without pneumothorax, paraquat poisoning should be kept as one of differential diagnosis.

Keywords: Bipyridal herbicide, Case report, Fatal toxicity, Paraquat, Spontaneous pneumothorax, Pneumomediastinum.

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ABBREVIATIONS USED IN THIS ARTICLE

CECT = Contrast-enhanced computed tomography; CRRT = Continuous renal replacement therapy; RTPCR = Reverse transcription polymerase chain reaction; ROS = Reactive oxygen species.

INTRODUCTION

Paraquat, a liquid herbicide is associated with suicidal and accidental poisoning with fatal outcomes. The ingestion of paraquat results in reactive oxygen species (ROS), which disrupts cell membrane.^{1,2} In addition to local corrosive injury to the mucosa of GIT, multiple organ dysfunction leads to acute kidney injury, pulmonary fibrosis, metabolic acidosis, and ARDS. Pneumomediastinum is defined as the presence of air in the mediastinum and its presentation can be isolated or in association with pneumothorax depending on the underlying cause. Spontaneous pneumomediastinum without any associated pulmonary disease is usually seen in young adults and secondary is found in chest trauma, esophageal rupture, and lung diseases. Spontaneous pneumothorax with or without pneumomediastinum is reported to occur in paraquat poisoning with isolated pneumomediastinum as a rare presentation. Paraquat poisoning is associated with high mortality as no specific antidote and treatment has been described.^{3,4}

CASE DESCRIPTION

A 34-year-old male was referred to our Institute, a Tertiary Care Center in North India with a history of ingestion of 50 mL of 24% paraquat compound 2 days back for management. The patient developed impending respiratory failure which was initially

¹⁻³Department of Medicine, Pandit Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India

Corresponding Author: Sanjay Fotedar, Department of Medicine, Pandit Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India, e-mail: drsanjayfotedar@gmail.com

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managed at a secondary health care facility with gastric lavage, PPI, and symptomatic treatment. The patient develops oxygen desaturation, hypotension, and decreased urinary output. The patient was not maintaining oxygen level at room air. On examination the patient was conscious, respiratory rate 24/m, hemodynamically unstable with blood pressure 80/40 mm Hg, afebrile, mild pallor, chest diffuse bilateral crepitations, Hb 10 gm, TLC 14000, DLC 80/25/3/2/0, AST 60 IU, ALT 65 IU, blood urea 80, serum creatinine 3.4. In view of current COVID-19 pandemic, reverse transcription polymerase chain reaction (RTPCR) was done and tested negative for COVID-19 virus. Contrast-enhanced computed tomography (CECT) thorax was planned to assess the involvement of lungs and associated complications which revealed bilateral diffuse opacities and pneumomediastinum without pneumothorax and

subcutaneous emphysema (Figs 1 to 3). The patient was managed in ICU settings with high flow oxygen, nebulization, injection of piperacillin plus tazobactam 4.5 mg twice daily, and injection of azithromycin 500 mg IV once daily besides symptomatic treatment and presser agents. Patients had developed anuria so continuous renal replacement therapy (CRRT) was planned with strict monitoring of input and output parameters. On the next day of admission, the patient had cardio-respiratory arrest and could not resuscitate.

DISCUSSION

Paraquat, an herbicide is reported as a common self-poisoning agent because of its easy availability in India and other areas of Southeast Asia. The fatality rate is reported about 50–90% due to the non-availability of specific antidotes and treatments till now. The oral route is the most common route of poisoning, accidental or intentional. Superoxide radicals and other reactive oxygen species produced by paraquat are responsible for direct cell damage by disrupting the cell membrane.^{1,2} Paraquat exerts local systemic effects characterized clinically by ulceration of the oral cavity, tongue, and GI mucosa and affecting the other organ systems resulting in hepatotoxicity, nephrotoxicity, pancreatitis, cardiogenic shock, respiratory failure, and the culminating event being MODS. The histopathological findings in fatal cases include congestion of pulmonary parenchyma, edema, alveolar hemorrhage, and pulmonary fibrosis. The severity has been graded clinically as mild, moderate, and severe (fatal) depending upon the amount of paraquat compound ingested corresponding to less than 30, 40, and more than 40 mg/kg body weight.^{2–4}

Paraquat is actively taken up by type I and II pneumocytes resulting in higher concentration than in plasma. The type I pneumocytes function results in impaired oxygenation and capillary function and increased surface tension and fluid accumulation due to damaged type II pneumocytes. Remodeling of lung parenchyma sets in due to pathological healing in response to inflammation, cytokine release, and collagen deposition resulting in early-onset lung fibrosis. Macklin proposed the most accepted mechanism. The damage of type II pneumocytes results in secondary atelectasis, emphysematous changes, and rupture due to increased surface tension and formation of sub-pleural or peripheral bullae which rupture resulting in pneumothorax and pneumomediastinum as a consequence of escape of gases along connective tissue planes and vascular sheaths.^{3,4} The early development of pneumomediastinum has been described as a specific mortality predictor with a fatality rate of almost 100%. Our patient died within three days of ingestion of the paraquat compound. The pneumomediastinum, pneumothorax, and subcutaneous emphysema is also attributed to esophageal erosion and perforation due to the corrosive effect of paraquat, with gastric lavage, severe vomiting and mechanical ventilation also contributing factors.^{5,6}

Management mainly consists of symptomatic treatment. To date, no specific antidote/treatment is available. Early induction of activated charcoal hemoperfusion combined with continuous venovenous hemofiltration has been reported to prolong survival by delaying the onset of systemic complications. N-acetyl cysteine, pulse therapy with corticosteroids, cytotoxic agents such as cyclophosphamide, nitric-oxide supplementation, antioxidants (vitamin C/E) and paraquat antibodies use has been described in the literature with the varied outcome. Mild to moderate cases of poisoning cyclophosphamide and corticosteroids are known to offer

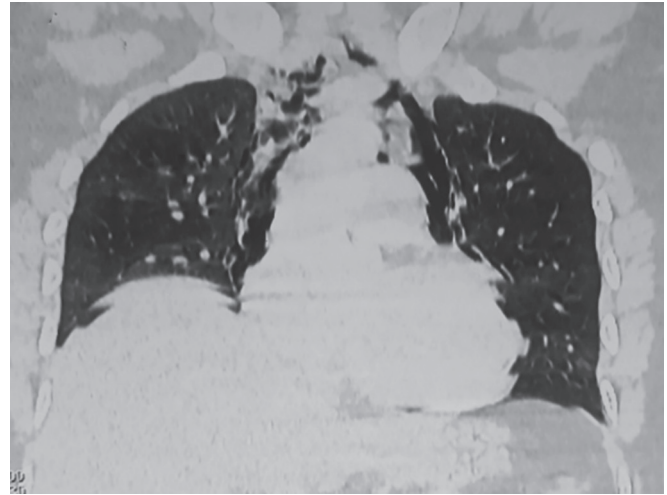


Fig. 1: Coronal section HRCT chest revealing, pneumomediastinum

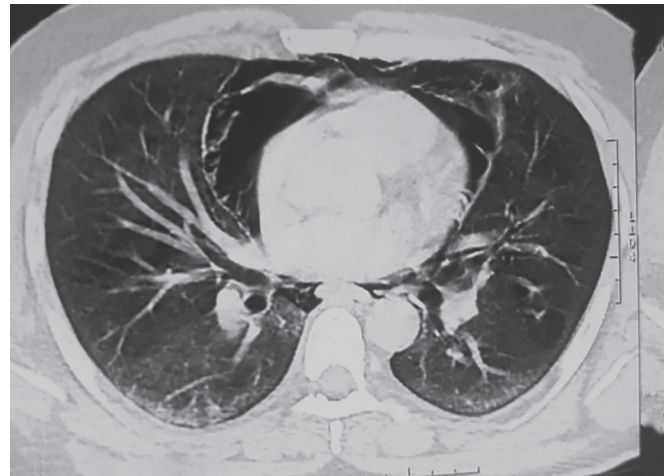


Fig. 2: Axial section HRCT chest, revealing mild inter- and intra-lobular septal thickening with fine reticular pattern, ground glass attenuation and pneumomediastinum

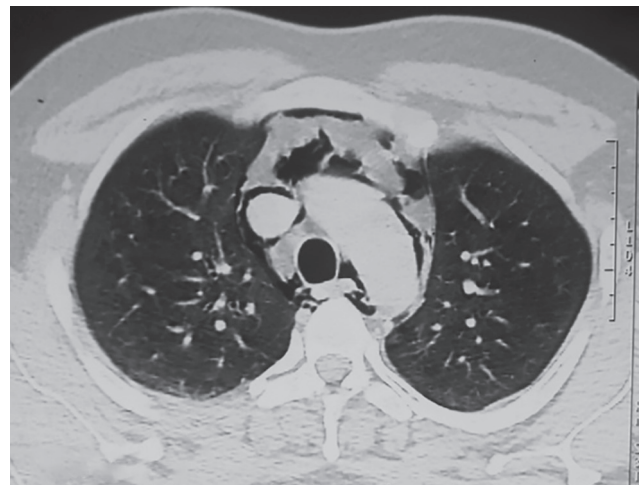


Fig. 3: Axial section HRCT chest revealing pneumomediastinum with presence of air around main vessels

some benefit by decreasing the severity of ongoing inflammation and pulmonary fibrosis, with lung transplantation also described in literature.^{6,7} Pulmonary manifestations of paraquat poisoning which include pulmonary fibrosis, pneumothorax, and pneumomediastinum increase morbidity and mortality. Diagnosis is made based on history, and physical examination and confirmed by serum paraquat level. Paraquat poisoning should be kept in mind while encountering pneumothorax, pneumomediastinum, or both in critical care settings with no obvious cause. Though the pulmonary complications pose a tough challenge in the management timely institution of comprehensive therapy is associated with better outcomes.

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