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Case Report

Fatal Triad: Spontaneous Pneumothorax, Pneumomediastinum, and Pneumorrhachis in a Case of Paraquat Ingestion

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Abstract

Objective: Dependency on agriculture and the unregulated sale of paraquat makes it an easy alternative for homicidal and suicidal use in developing nations. It kills by multiorgan failure, predominantly pulmonary fibrosis, and ARDS. We report a case of alleged paraquat ingestion with spontaneous pneumothorax, spontaneous pneumomediastinum, and pneumorrhachis. Aim is to reinforce the importance of a high index of suspicion in early diagnosis when the above findings are present with ARDS in absence of trauma and a history of alleged substance ingestion.

Case Presentation: A 35-year-old male presented with loose stool occasionally bloody, oral ulcers, yellow discoloration of eyes with fever, and decreased urine output for three days after consuming some substance with his seafood. On examination, he had yellow discoloration of eyes and oral mucosa along with multiple ulcers on the buccal region as well as the dorsum of the tongue and lateral margins with generalized subcutaneous emphysema. In addition, decreased air entry in the bilateral lung field and muffled heart sounds were present. He had an acute hepatorenal failure and severe metabolic acidosis with respiratory failure. Urine tested positive for myoglobin and muscle enzymes (creatinine kinase and lactate dehydrogenase (LDH)) were raised. He was intubated and shifted to the intensive care unit. Injectable N-acetyl cysteine (NAC) for acute liver failure was started with empirical antibiotics and intravenous fluids. We supplemented thiamine and vitamin K, and hemodialysis was done in view of progressive renal failure. Radiological evaluation showed spontaneous pneumothorax, pneumomediastinum, and pneumorrhachis which were managed conservatively. His respiratory parameters worsened despite maximal ventilatory support. Renal failure and metabolic acidosis worsened in spite of hemodialysis. He succumbed to his illness on day five of admission and seven days after toxin ingestion.

Conclusion: We recommend that the sale of paraquat be restricted and regulated to avoid its use for suicidal and homicidal purposes. More research is required to find measures to intervene early and prevent pulmonary fibrosis. We propose that paraquat toxicity be considered early in a patient with the triad in an atraumatic setting with acute respiratory distress syndrome (ARDS).

Keywords: Paraquat toxicity, Pneumorrhachis, Pulmonary fibrosis, Respiratory Distress syndrome

Introduction

Paraquat is a commonly used herbicide in agriculture-dependent economies like India. It is easily available due to widespread agricultural applications as a corrosive liquid with trade names "Aspire-24", kattar, trap, Grammo in 24% SL concentration. Although it has been reformulated with emetic to discourage accidental ingestion, it still remains 100% fatal in suicidal, and homicidal ingestions. Even an amount as little as 10 ml is adequate to cause death (1). Bioavailability of paraquat is documented to be less than 5% in humans but due to rapid absorption its peak plasma concentration is reached within an hour of ingestion. It is not metabolized in the body and has almost exclusive renal excretion (2).

The toxic effect of paraquat on the body, in essence, is by its action on the lungs. After active uptake by the alveolar lining, it disrupts the integrity of the alveolar-capillary barrier facilitating pathological events leading to fibrin deposition and ultimately pulmonary fibrosis. The earliest evidence of alveolar injury is seen within 24 hours of ingestion reaching its peak in 3-4 days |(3).

Pneumorrhachis, a rare phenomenon described in 1977, has been linked to other causes besides trauma. Among the well-documented non-traumatic benign causes, violent cough in acute bronchitis, upper respiratory tract infection, and primary spontaneous pneumothorax are documented

in the literature (4). Spontaneous pneumomediastinum and pneumothorax have been reported in acute paraquat poisoning due to their toxic effects on the lungs (5,6). We report the case of a 35-year-old single male with paraquat ingestion presenting with spontaneous pneumothorax, pneumomediastinum, and pneumorrhachis.

Case Presentation

A 35-year-old single male, farmer by profession, without any known co-morbidities, was brought to the emergency department of our tertiary care with complaints of loose stool with occasional blood, hoarseness of voice, and difficulty speaking. He also complained of oral ulcers, neck pain, generalized weakness, yellow eyes, pain and tightness in both lower limbs, and swelling of the whole body. He had a fever with decreased urine output for three days after allegedly consuming some substance with his seafood.

Upon arrival, he was conscious and oriented, maintaining spo2 of 88-92% at room air, blood pressure was 110/70 mm Hg and heart rate of 110/min. On examination, he had a puffy face, yellow discoloration of eyes and oral mucosa along with multiple ulcers on the dorsum of the tongue, and lateral margins. There was crepitation upon palpation in the face, and neck extending up to bilateral lower limbs suggesting generalized subcutaneous emphysema. Air entry was decreased in the bilateral lung and cardiac examination revealed muffled heart sounds. He had no signs of trauma.

The patient was shifted to the intensive care unit, intubated, and attached to ventilatory support. He was managed conservatively with adequate intravenous fluids, proton pump inhibitors (PPI), liquid sucralfate, and empirical antibiotics. Primary lab results revealed acute hepatorenal failure with severe metabolic acidosis and type I respiratory failure (Table 1). His urine tested positive for myoglobin and muscle enzymes, and both creatinine kinase and lactate dehydrogenase (LDH), were raised. N-acetyl cysteine (NAC) was added for acute liver failure and the patient was hemodialyzed in view of acute kidney injury, decreased urine output, and metabolic acidosis. Chest x-ray (CXR) revealed bilateral pneumothoraxes with pneumomediastinum, and generalized subcutaneous emphysema (Figure 1). High-resolution computerized tomography (HRCT) chest revealed aspiration pneumonitis, pneumothorax, and pneumomediastinum with pneumorrhachis (Figures 2A and 2B). General surgery and cardiothoracic surgery consultation were taken. Conservative management and continual monitoring were advised.

His renal failure progressed from oliguria to anuria and metabolic acidosis worsened despite multiple rounds of hemodialysis. His respiratory failure did not improve despite maximal ventilatory support and serial CXR showed increasing pulmonary infiltrates. Eventually, the patient developed nasal and oral mucosal bleeding

Table 1. Summarized laboratory results of the patient during course of hospital stay

	Day 1	Day 3	Day 5	Reference range
Hemoglobin (g/dL)	17.8	16.8	15.9	13-16
PCV (%)	57	49	45	40-50%
Platelet (10^9/L)	149	156	153	150-410×10^9
TLC (10^9/L)	10	9.2	6.7	4-10×10^9
Serum bilirubin (mg/dL)	11	12.2	14.6	0.2-1.0
SGOT (IU/L)	238	410	480	5-40
SGPT (IU/L)	286	330	440	5-35
Albumin (g/L)	3.2		2.4	3.8-5.5
Na (mEq/L)	138	135	138	135-145
K (mEq/L)	5.6	6.4	5.8	3.5-5.5
Urea (mg/dL)	263	287	306	15-45
Creatinine (mg/dL)	10.3	9.8	10.1	0.8-1.8
LDH		673		
CK-MM (IU/L)		89		
CRP (mg/L)	44		58	0-5
ESR	14		44	0-10 mm 1st h 0-20 mm 2nd h
pH	7.2	7.0	6.9	7.35-7.45
pCO_2 (mm Hg)	38.5	47.7	44	35-45
pO_2 (mm Hg)	77	98	98	80-100
HCO_3 (mEq/L)	14.5	12.6	10	21-28
Lactate (mEq/L)	1.7	2.7	3.4	0-2

PCV, pack cell volume; TLC, total leucocyte count; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic-pyruvic transaminase; LDH, lactate dehydrogenase; CKMM, creatinine kinase; CRP, C reactive protein; ESR, erythrocyte sedimentation rate; pCo2, partial pressure of carbon dioxide; pO2, partial pressure of oxygen; HCO3, arterial blood bicarbonate.

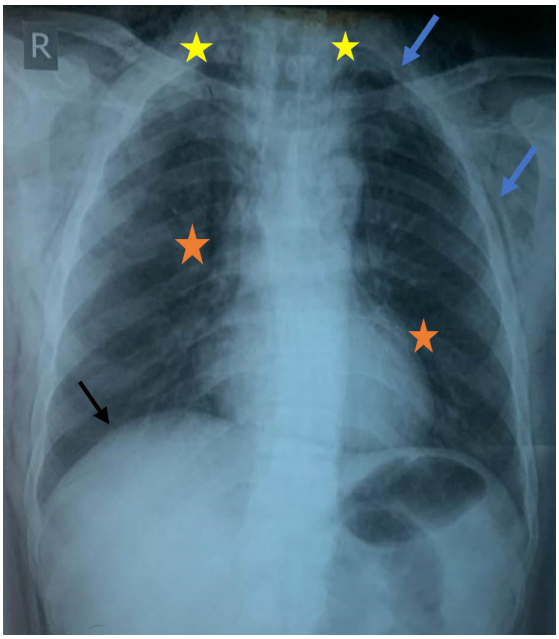
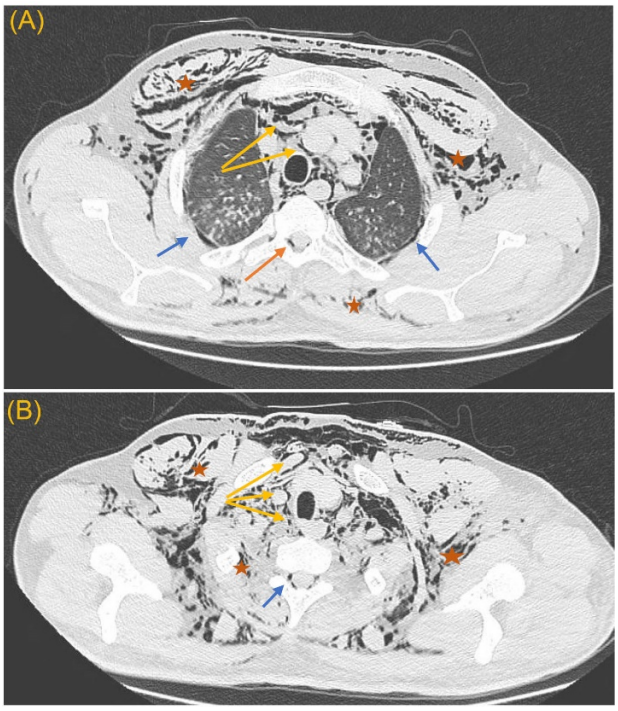


Figure 1. CXR showing pneumothorax, pneumomediastinum, and subcutaneous emphysema. CXR posterior-anterior (PA) view showing air streaking in soft tissue and muscle plane of chest wall and neck (blue arrows), minimal apical pneumothoraxes (yellow stars), continuous diaphragm sign (black arrow) with air outlining heart (orange stars)



Figures 2. HRCT showing generalized emphysema, pneumomediastinum, and pneumorrhachis. (A) HRCT chest showing bilateral pneumothoraces (blue arrows) and air outlining trachea and large vessels (yellow arrow). Air in the spinal canal outlining the thecal sac can be seen (orange arrow). Air is also visualized in anterior and posterior subcutaneous tissue (stars). (B) At the level of neck air in the subcutaneous tissue and intramuscular plane (stars), air outlining thyroid, trachea, and vascular structures (yellow arrows), air around the spinal canal (blue arrow).

and was managed conservatively with tranexamic acid, vitamin K, fresh frozen plasma, and packed red blood cell transfusion. Despite the above measures, he did not show any signs of improvement and succumbed to his illness on day five of admission and seven days after alleged toxin ingestion.

Discussion

The dependency on agriculture and unregulated sale of paraquat make it an easy alternative for both homicidal and suicidal use in developing nations. In the absence of any effective modalities and guidelines for treatment, death is the most common outcome in almost ninety percent of cases (5). Adding to this, high case fatalities are delayed referral to a higher center, multiple organ involvement, and early rapid pulmonary involvement causing lung fibrosis and severe respiratory failure (1).

Initially, the development of pneumothorax and pneumomediastinum in paraquat toxicity was attributed to toxin-associated pulmonary hypertension. Later it was highlighted that paraquat extensively damages pulmonary endothelium as well as epithelium predominantly type II pneumocytes causing lung injury followed by fibrosis (5,7). Air escape from damaged alveoli due to paraquat-induced acute lung injury seems to cause pneumomediastinum and pneumothorax. Other proposed mechanisms are esophageal erosion due to corrosive effects of paraquat on mucosa, esophageal mucosal damage because of severe vomiting bouts, multiple gastric lavages, or barotrauma due to mechanical ventilation to the already damaged lung (6,8). More recent studies propose early pneumomediastinum as the predictor of mortality while others recognize it as a marker of paraquat poisoning in the absence of any recognizable cause (6,9).

Pneumorrhachis can be iatrogenic, traumatic, and nontraumatic. It has been documented to occur in inhalational abuse of ecstasy and marijuana. Mostly it is found with air in other areas of the body especially pneumomediastinum, pneumothorax, pneumopericardium, and subcutaneous emphysema (10). A few case reports have documented pneumomediastinum and pneumothorax in cases of paraquat poisoning but pneumorrhachis has not been mentioned so far. Our patient had pneumomediastinum, pneumothorax, and pneumorrhachis with generalized subcutaneous emphysema. Postmortem autopsy revealed corrosive damage to mucosa without any esophageal rupture with extensive pulmonary injury and fibrosis.

Conclusion

Despite paraquat being a commonly abused herbicide, we lack definitive diagnosis and treatment guidelines to manage it. Paraquat poisoning should be suspected early in all cases of spontaneous pneumothorax, pneumomediastinum, and pneumorrhachis in an atraumatic setting with ARDS (acute respiratory distress syndrome). Early suspicion is key to diagnosis and effective management for a better outcome.

Authors' Contribution

Conceptualization: Zainab Mehdi, Daljinderjit Kaur, Narinder Kaur.
Data curation: Zainab Mehdi, Daljinderjit Kaur, Narinder Kaur.
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Competing Interests

None.

Ethical Approval

Informed consent statement was obtained from the patient for the publication of this report.

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