Case Report

Delayed pneumomediastinum: A rare subacute complication of paraquat poisoning

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Abstract

Paraquat (PQ) is a non-selective widely used weedicide. Paraquat poisoning has a high mortality rate with no available antidote as of now. It is rare in Bhutan, but mostly discovered in the southern part of the country. Patients usually present with multi-organ failures such as respiratory, liver, and renal failures. This is a case report of a 24-year-old female referred with a history of paraquat ingestion. Initially, she complained of epigastric and retrosternal chestpain with excoriation of the oral mucosa. She later developed acute kidney injury and respiratory distress. Chest CT revealed pneumomediastinum, massive bilateral pneumothoraces, and subcutaneous emphysema. Although there have been few reported cases of pneumomediastinum with or without pneumothorax and subcutaneous emphysema, most of them have an acute presentation (less than 1 week).

Keywords: Paraquat, Poisoning, Complication, Pneumomediastinum, Pneumothorax.

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Case summary

A 24-year-old female was admitted to the emergency department (ED) with an alleged history of deliberate self-harm in which she consumed around 15-20 ml of PQ at her home. She was initially seen at her local hospital and was referred to the district hospital on the same night. In the district hospital, the patient was given intravenous (IV) fluids, H2 blocker, antiemetic, steroids, and IV antibiotics (ceftriaxone and metronidazole). On admission at ED, she complained of retrosternal and epigastric pain (burning type) and shortness of breath on exertion. She had a history of migraines and no past psychiatric history. There was no fever, nausea, vomiting, diarrhea, or cough. Urine output was adequate at the time of admission. On examination, she was afebrile and anicteric with mild facial swelling. There was excoriation of oral mucosa and around the lower lip. Her GCS was 15/15 and all her vital signs were stable. The auscultation of the lung revealed few bilateral basal crepitations. The neurological, cardiovascular, and abdominal systems were unremarkable. Her blood report at the time of the admission showed neutrophil leukocytosis (WBC-21930 μ/mL, neutrophil-89.2%). Her renal function test showed raised urea and creatinine level, about 165 mg/dL and 4.7 mg/dL, respectively, and low potassium (3.3 mEq/L) suggestive of acute kidney injury (AKI). The rest of her investigation was unremarkable.

Initial standard CXR anteroposterior (AP) view revealed patchy infiltration involving bilateral lower lobes with pleural effusion. No evidence of pneumothorax, pneumomediastinum, or subcutaneous emphysema (Figure 1).

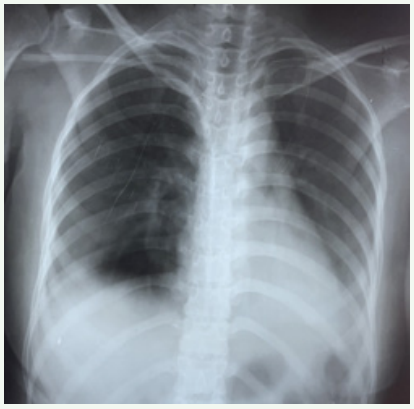

Figure 1. *Chest X ray anteroposterior(AP) view showing patchy infiltration involving bilateral lower lobes with pleural effusion.*

She was admitted to the medical ward where they established a central line and urgent hemodialysis was performed. IV steroids and ceftriaxone were continued. With regular hemodialysis and potassium chloride (KCl) solution, her AKI and hypokalemia improved. The subsequent abdomen ultrasound showed bilateral increased renal cortical echoes, in line with kidney insult, and bilateral pleural effusion. Echocardiography showed mild left ventricular hypertrophy (LVH) and tricuspid regurgitation (TR) with small pericardial effusion. The systolic function was normal.

Then on the 20th day of the admission, she developed respiratory distress and was given high flow oxygen. The urgent computed tomography of the chest was performed which revealed massive subcutaneous emphysema, bilateral pneumothoraces, and pneumomediastinum with collapse-consolidation of both lungs (Figure 2).

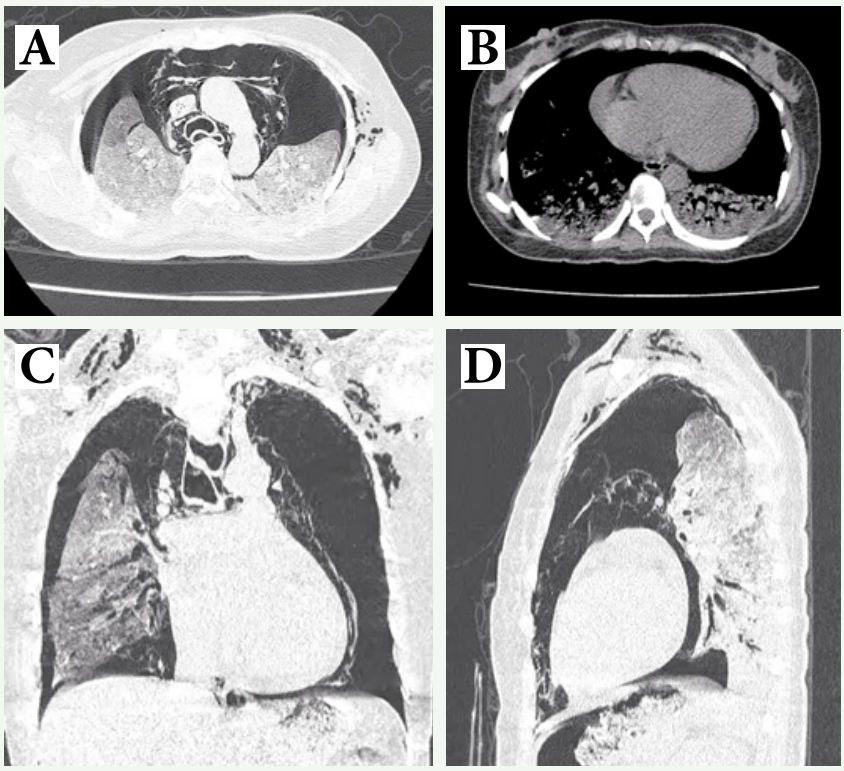

Figure 2. *Plain CT Chest A) axial (lung window), B) axial (mediastinal window), C) coronal (lung window) and D) sagittal (lung window) showing pneumo-mediastinum subcutaneous emphysema, bilateral pneumothoraces and with collapse-consolidation of both lungs.*

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The patient was immediately transferred to the adult intensive care unit (AICU) in view of impending type 2 respiratory failure. She was intubated and bilateral intercostal drainage (ICD) was inserted (Figure 3). However, the patient expired on the same day in the evening.

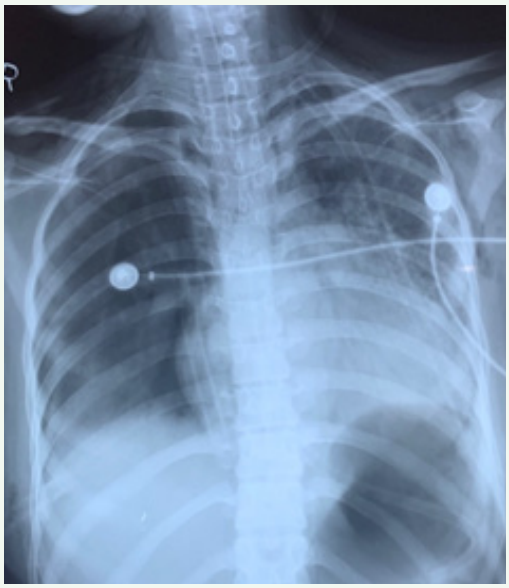

Figure 2. *Chest X-ray anteroposterior (AP view) showing bilateral pneumothoraces, pneumomediastinum, subcutaneous emphysema and consolidation in both lungs with endotracheal tube and bilateral intercostal drainage tubes.*

Discussion

Paraquat (N, N'-dimethyl-4, 4'-bipyridinium dichloride) is a non-specific weedicide that has broad-spectrum effects, rapid deactivation on contact with soil, and is relatively inexpensive. Due to these favorable characteristics, it is a widely used weedicide which is frequently abused with lethal consequences. It is a leading cause of fatal poisoning in many parts of Asia, Pacific nations, and the Americas [1]. Paraquat poisoning is rare in Bhutan but is mostly discovered in the southern part of the country. In Bhutan, PQ is not an approved weedicide according to National Plant Protection Centre (NPPC) [2].

PQ intoxication results in multi-systemic toxicity with early manifestation seen in the gastrointestinal tract, kidney, liver, and lungs [3]. The toxic effect of PQ is thought to be caused by the production of free radicals which damage cell membrane resulting in a wide spectrum of cellular, structural and functional abnormalities [4].

PQ poisoning can either be accidental and deliberate, although in most cases it is deliberate [5]. Following ingestion, early symptoms of PQ poisoning include burning sensation in the throat, abdominal pain, nausea, vomiting, and diarrhea. There can be excoriation and ulcerations of the tongue, oral mucosa, and esophagus due to its caustic effect [6]. If the patient has ingested 4mg/kg or more, it can lead to multi-organ failures such as renal, liver, and respiratory failures. Likewise, in our case, the patient complained of retrosternal and epigastric pain (burning type), shortness of breath on exertion with excoriation of oral mucosa and around the lower lip. Later on, she developed oliguria with a deranged renal function test suggestive of acute kidney injury.

The most common affected system in PQ ingestion is the respiratory system. This could be due to the increased concentration of PQ and low concentration of antioxidant levels in the lungs [4,7]. Following ingestion of PQ, initially, there is a destructive phase characterized by damage of alveolar epithelium, hemorrhage, and congestion which will manifest as diffuse ground-glass opacities and consolidation on imaging. In some rare cases, a patient can have an acute presentation of pneumomediastinum with or without pneumothorax and subcutaneous emphysema as demonstrated Daisley et al [4], Im et al[6] and Chen et al [8]. Our patient initially complained of shortness of breath on exertion. CXR revealed patchy infiltration in bilateral lower lobes which could be due to consolidation. There was no evidence of pneumothorax, pneumomediastinum, or subcutaneous emphysema then.

It was only on the 20th day of admission that she developed respiratory distress. The computed tomography of the chest revealed pneumomediastinum, massive subcutaneous emphysema, and bilateral pneumothoraces with collapse-

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consolidation of both lungs. The available literature reported that pneumomediastinum occurs mostly within 1 week of PQ ingestion [4,6,8]. However, there were 2 reported cases of pneumomediastinum that developed in the 2nd week while there were 2 cases of pneumothorax and 1 case of subcutaneous emphysema that developed after 2 weeks of PQ ingestion according to Im et al [6]. Therefore, that makes this a rare case scenario in which the patient developed pneumomediastinum after 2 weeks of PQ ingestion. Although one could argue that patients might have developed pneumomediastinum during the first 2 weeks as no follow-up CXR or CT was done then, those pneumomediastinums would have been small or insignificant as the patient was clinically stable without respiratory distress.

There are various theories explaining the pathogenesis of pneumomediastinum and pneumothorax. According to Im et al, it is caused by pulmonary interstitial emphysema with bullae formation when ruptures give rise to pneumothorax and subcutaneous emphysema [6]. Maunder et al postulated that it is probably caused by rupture of alveoli into bronchovascular sheath due to alveolar over-distension [9]. Another obvious cause would be esophageal perforation due to the caustic nature of PQ. In our patient, there was no evidence of esophageal perforation. However, there was air within bronchovascular sheath suggesting PQ-induced alveolar injury with subpleural bullae formation and subsequent rupture in accordance with Im et al. hypothesis.

Following the diagnosis, the patient was intubated, and bilateral ICD tubes were inserted. The patient expired the same day around the evening. This is in line with other literature which states that the presence of pneumomediastinum indicates poor prognosis [6,10]. The other poor prognostic CT findings include increased lung segment involvement, the extent of disease characteristics visualized in CT, and speed of progression from baseline [11].

Conclusion

Pneumomediastinum, pneumothorax and subcutaneous emphysema are rare subacute complications of paraquat poisoning and are associated with poor prognosis. Nevertheless, one needs to anticipate and recognize these complications for proper management.

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