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CASE REPORT

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Lethal mediastinal emphysema caused by inhalation of paraquat: a case report

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Abstract

This paper reports on a 22-year-old male presenting with persistent chest pain accompanied by mediastinal emphysema. We firstly considered mediastinal emphysema induced by community acquired pneumonia. Pathogen detection was performed but no positive results were found. Based on the results of a subsequent lung CT scan, paraquat poisoning was suspected. Although there was no trace of paraquat in the blood, the nebulizer masks used by patient at home was found to be positive for paraquat. The diagnosis was ultimately established as paraquat poisoning via inhalation with mediastinal emphysema. This case report explores the clinical manifestations, diagnostic challenges, and treatment complexities of inhaled paraquat poisoning, emphasizing the importance of recognizing this rare poisoning route and its atypical symptoms.

Keywords Paraquat, Poisoning, Inhalation, Mediastinal emphysema, Pulmonary

Background

Paraquat (1,10-dimethyl-4,40-bipyridinium dichloride) is a pyridine herbicide highly toxic to humans [1]. Paraquat induces pulmonary fibrosis and multi-organ failure, causing a rapid clinical course with high mortality rate [2]. Ingestion is a common route of fatal poisoning [3]. Contrarily, lethal mediastinal emphysema resulting from paraquat inhalation remains unreported. Herein, we report a case of lethal inhaled paraquat poisoning, a rare mode of exposure, presenting primarily with mediastinal emphysema as the initial symptom. We emphasize on delineating this unusual mode of poisoning and its atypical clinical manifestations to enhance awareness regarding paraquat toxicity through this novel route of exposure.

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Case report

A 22-year-old male presented to our emergency department on February 6, 2024, complaining of "persistent chest pain for 6 days, worsening over the past day." The chest CT of the patient indicated multiple pneumatosis in the neck soft tissue space, right supraclavicular and shoulder regions, mediastinum, interlobar fissure, and interstitial spaces around the bronchioles and blood vessels of both lungs (Fig. 1A, C). The patient was interned in the emergency department. Four days later, the patient's symptoms worsened, and CT showed aggravation of mediastinal emphysema (Fig. 1B, D).The patient developed tachypnea, with a temperature of 36.7 °C, blood pressure of 156/71 mmHg, heart rate of 117 beats/min, and respiratory rate of 29 breaths/min. He appeared mentally distressed, with subcutaneous emphysema palpable in the neck and crepitus. The remaining physical examination was unremarkable. Arterial blood gas analysis showed pH 7.407, $PaCO_2$ 50.3 mmHg, PaO_2 78.48 mmHg (FiO_2 80%), and lactate 1.3 mmol/L. Blood biochemistry revealed aspartate transaminase (AST) of 64 U/L, alanine transaminase (ALT) of 81 U/L, total

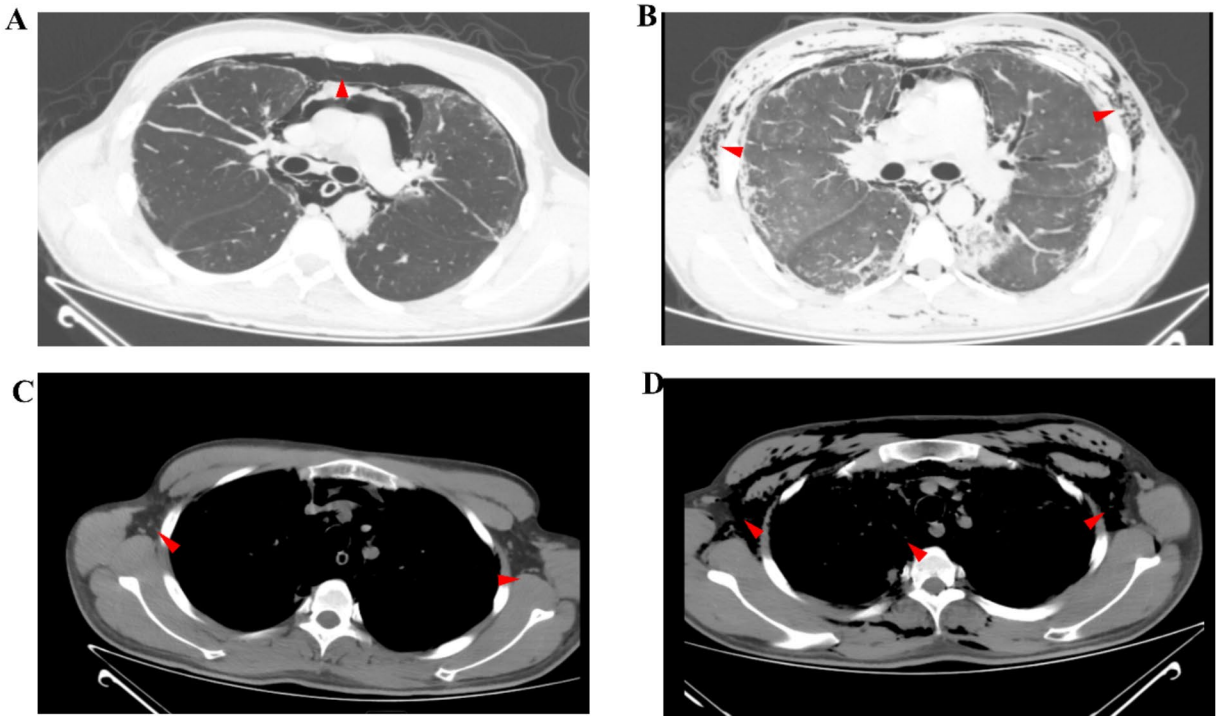

Fig. 1 Patient's chest CT scan. **A**: February 6th chest window imaging; **B**: February 10th chest window imaging; **C**: February 6th mediastinal window imaging; **D**: February 10th mediastinal window imaging. The red arrow points to the lesion

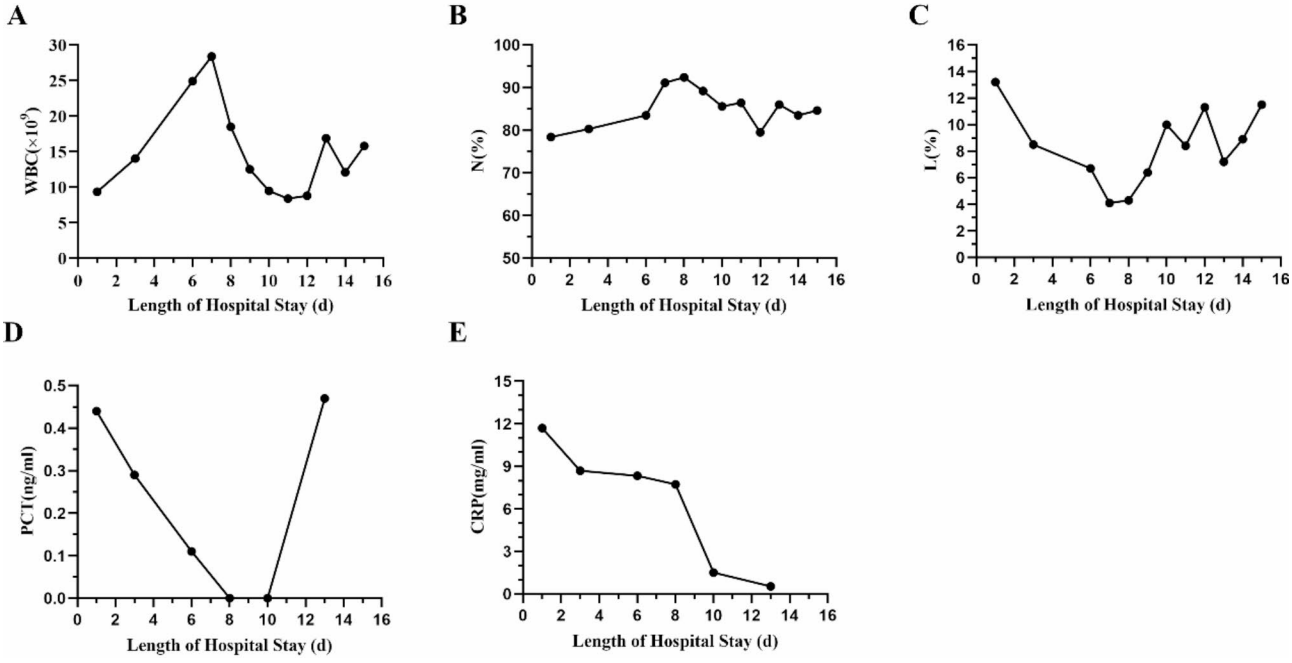

Fig. 2 Inflammatory biomarkers. **A**: White blood cell count (WBC) = 3.5–9.5 × 10^9. **B**: Neutrophil percentage (N(%)) = 40.0–75.0%. **C**: Lymphocyte percentage (L(%)) = 20.0–50.0%. **D**: Procalcitonin (PCT) = 0.04–0.5 ng/ml. **E**: C-reactive protein (CRP) = 0–11 mg/ml

bilirubin (TBIL) of 26.2 μmol/L, lactate dehydrogenase (LDH) of 752 U/L, creatinine (Cr) of 172 μmol/L, and blood urea nitrogen of 13.6 mmol/L (Fig. 2a and d). Thereafter, he was transferred to the thoracic surgery ward. Based on the clinical findings, we initially considered a diagnosis of mediastinal emphysema induced by community-associated pneumonia. Pathogen detection was performed but no positive results were obtained.

The patient's condition worsened gradually; he was transferred to ICU on February 16, 2024, where he was placed on mechanical ventilation and administered ECMO therapy. Initially, we considered that the mediastinal emphysema might be caused by an infection and conducted microbiological tests, which yielded negative results. Additionally, the blood routine tests and inflammatory markers did not support the presence of an infection (Fig. 2). Furthermore, we excluded other potential causes of mediastinal emphysema. After the cause of pneumomediastinum could not be determined, paraquat poisoning was suspected through an emergency physician consultation. However, paraquat was not detected in blood. After further inquiry and reviewing the patient's online shopping record, the patient was discovered to have purchased "methyl violet 98%" (a raw material for paraquat synthesis) in April 2023, and had first experienced chest pain with dyspnea in October 2023. Finally, paraquat was detected in the nebulizer used by the patient, and residues were found in the reagent bottles, indicating intermittent exposure to a paraquat environment. This suggests that mediastinal emphysema was due to paraquat inhalation toxicity. Unfortunately, the patient's family refused further treatment, and the patient was transferred to a palliative care hospital, where they passed away on April 28th.

Discussion

Modes of exposure for paraquat poisoning include the gastrointestinal tract, respiratory tract, and skin contact [4], with concentrated solution ingestion being the most common [5]. Here, the patient suffered from inhalation poisoning, which made identification challenging due to the absence of prominent indications of poisoning in the medical history. For example, the patient had no evident signs of erosion symptoms in the oral cavity or pharynx, with mediastinal emphysema with subcutaneous emphysema being the sole clinical manifestation. Moreover, a definitive diagnosis of paraquat poisoning often relies on serum toxicology testing, further complicating diagnosis in concealed poisoning cases. Despite banning paraquat in many regions worldwide, it remains accessible in the pesticide market. Therefore, in clinical practice, possibility of paraquat poisoning cannot be overlooked. Particularly regarding rare poisoning routes and atypical symptoms, healthcare professionals should exercise caution.

Mediastinal emphysema is the accumulation of air within the mediastinum, which may or may not be accompanied by subcutaneous emphysema. This can be spontaneous or secondary to iatrogenic, traumatic, or gastrointestinal perforation, among other factors [6, 7]. It is a severe complication in paraquat poisoning. Corrosive effects and using ventilators can contribute to mediastinal emphysema directly [8, 9]. Paraquat can induce reactive oxygen species generation and inflammatory responses [10], which can indirectly lead mediastinal emphysema. Approximately 20% patients develop mediastinal emphysema, with mortality rate approaching 100%, becoming a specific prognostic indicator of mortality in paraquat poisoning [9, 11, 12]. Considering the inhalational nature of this case and the absence of injuries caused by gastric lavage or ventilator use, we are more inclined to consider mechanisms related to the lungs, including alveolar exudation, fibrosis, and increased alveolar tension and shear forces, causing bronchovascular rupture and subsequent mediastinal emphysema.

Therefore, the inhalational route of paraquat poisoning renders the onset and symptom presentation of patients more insidious. Compared to other exposure routes, this toxic pattern may not manifest immediate prominent symptoms but present with delayed onset or atypical symptoms, including mediastinal emphysema, making the diagnosis more challenging. This condition can cause mortality due to diseases including interstitial pulmonary fibrosis. Our report highlights the need for healthcare professionals to enhance their understanding of rare poisoning routes and their atypical symptoms to improve diagnostic and treatment outcomes for such cases.

Thanks

Thanks to all the doctors and nurses who provided medical services for this case.

Author contributions

First Author: Qing Tang wrote the main manuscript text(Emergency Medicine Department, Tianjin Medical University General Hospital,300041,Email:3119713921@qq.com) Other authors: Jiaqi Xu and Hao Wang prepared all figures (Emergency Medicine Department, Tianjin Medical University General Hospital,300041,Email: 437714393@qq.com)Corresponding Author: Lijun Wang (Emergency Medicine Department, Tianjin Medical University General Hospital,300041,Email: 641998933@qq.com).

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Data availability

Due to patient privacy concerns, the data supporting the results of this study are not publicly available but can be obtained from the corresponding author upon reasonable request. The data are stored in a controlled access data repository at Tianjin Medical University General Hospital.Corresponding author email: wanglijun211022@tmu.edu.cn.

Declarations

Consent to publish

Informed consent has been obtained from the patient's family members.

Conflict of interest

The authors declare that they have no financial interests/personal relationships which may be considered as potential competing interests.

Clinical trial number

Not applicable.

Competing interests
The authors declare no competing interests.

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