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Case Report

Crack lung with toxic cerebral vasculitis: Case report ☆

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ABSTRACT

Crack is the most potent form of cocaine. It directly affects lungs if inhaled and the damage may include barotrauma, acute pulmonary edema, alveolar hemorrhage, bronchiolitis obliterans with organizing pneumonia, or vasculitis. The diagnosis of cocaine-related lung damage is based on clinical symptoms and radiological findings. When young individuals develop respiratory symptoms, investigation into cocaine use is necessary. We report the case of a young man with a history of cocaine use who presented for respiratory and neurological symptoms revealing crack lung and toxic cerebral vasculitis.

Introduction

Cocaine remains the leading cause of drug-related emergency room visits. Respiratory and neurological symptoms are the most revealing signs of this intoxication. Crack lung is an acute respiratory syndrome characterized by the development of diffuse alveolar damage and hemorrhagic alveolitis within the first 48 hours of cocaine use. Diagnosis of cocaine-induced lung disease is challenging for clinicians and radiologists.

Case presentation

A 37-year-old man with a history of recurrent crack cocaine use experienced a significant increase in cocaine consumption 3 days before admission. He presented to the emergency room with shortness of breath and confusion. There was no personal or family history of pulmonary disease. On examination, Glasgow Coma Scale was recorded at 11, with a temperature of 39°C, a pulse rate of 145 beats/min, blood pressure measuring 130/70 mmHg, a respiratory rate of 32 breaths/min,

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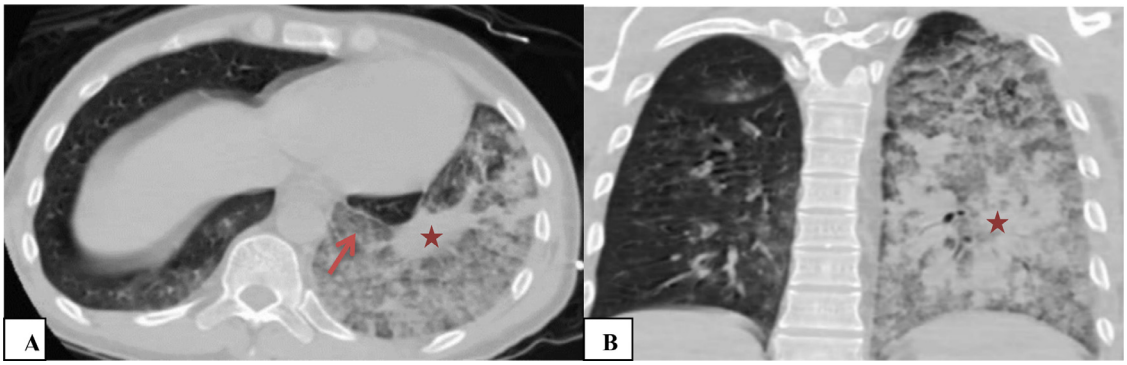

Fig. 1 – CT chest scan in axial (A) and coronal (B) sections showing a diffuse ground-glass opacities and interlobular septal thickening, configuring a "crazy-paving" pattern (arrow) in the left lung, associated with ipsilateral consolidation (star).

and an oxygen saturation of 67 % on room air. Auscultation detected inspiratory crackles in the left basilar area, but the remainder of the physical examination revealed no other abnormalities.

Given the presence of If ill-defined severe and acute respiratory failure with fever, a contrast CT scan and SARS-CoV2 swab along with routine blood tests are required.

Laboratory tests showed only increases in C-reactive protein (70 mg/dL) and procalcitonin levels (9.3 ng/mL). D-dimer was within normal limits, white blood cells were normal, there was mild neutrophilia, and no eosinophilia. HIV test was negative.

Chest computed tomography excluded signs of pulmonary embolism or cardiac hypertrophy and showed diffuse ground-glass opacities and basal consolidation in the left lung, as well as air bronchograms and interlobular septal thickening, configuring a "crazy-paving" pattern. There was no pleural effusion (Fig. 1). These specific findings, consistent with hemorrhages, and the negative result of SARS-CoV2 swab favored the diagnosis of “crack lung.”

Oxygen and naloxone were administered, resulting in an increase in blood oxygen saturation to 99%, but there was no improvement in mental status. Subsequently, a brain MRI was performed showing increased signal intensity and diffusion restriction involving the cerebral cortex in the left parietal region, as well as an irregular appearance of the arteries of the WILLIS polygon, creating a pearl necklace sign (Fig. 2). The diagnosis of a toxic cerebral vasculitis was retained

Subsequent urine toxicology results confirmed the presence of cocaine. Following the complete resolution of symptoms and normalization of inflammatory markers, the patient was discharged from the hospital after 7 days. At discharge, a 7-day course of oral prednisone therapy and 5 weeks of radiographic monitoring were recommended.

Discussion

Cocaine is one of the most commonly used illicit drugs worldwide. It remains a serious global problem due to its highly addictive nature [1]. Its use can lead to various acute and chronic pulmonary damages including eosinophilic alveolar pneumonia, pulmonary edema and hemorrhage or bronchiolitis obliterans organizing pneumonia, pneumothorax, pulmonary hypertension and thromboembolic disease [2].

Lung involvement may result from thermal damage to the airways, direct cytotoxicity, and induction of inflammatory damage, barotrauma, or vasoconstriction leading to ischemia [3].

Crack lung is an acute pulmonary syndrome involving diffuse alveolar damage within the first 2 days of smoking crack cocaine, presenting with symptoms such as chest pain, cough, hemoptysis, and shortness of breath which may progress to respiratory failure [1]. Cocaine overdose causes also neurological effects which may manifest as headaches, seizures, cerebral vasculitis, stroke and brain atrophy in the long term [4].

Acute cocaine use appears to increase the risk of major cerebrovascular accidents (hemorrhagic or ischemic) in young adults, although cocaine-induced cerebral vasculitis is rarely described. As patients often have additional risk factors, establishing a clear causal relationship can be challenging [5].

Diagnosing cocaine-induced lung disease remains difficult for clinicians due to nonspecific clinical features and chest images that can mimic several other conditions if no context of cocaine use is known.

Smoking cocaine can lead to edema (cardiogenic or not) and alveolar hemorrhage, evident in CT by diffuse or multifocal lung injury with ground-glass opacities, consolidation, and interlobular septa thickening, pleural effusion, and cardiac enlargement silhouette. Other forms of chest involvement include barotrauma, which may manifest as pneumothorax, pneumopericardium, pneumomediastinum, or subcutaneous emphysema and is usually diagnosed by Chest X-Rays. In cases where CXR results are inconclusive, high-resolution CT (HRCT) can aid in accurate diagnosis.

Another complication often associated with drug use is organizing pneumonia, which manifests as central and peripheral consolidation and structural distortion on HRCT. Bullous emphysema is reported to occur in 2%-4% of drug users, primarily in the upper lung area, and usually affects young men [6].

Community-acquired pneumonia and septic pulmonary embolism are common infectious pulmonary complications in drug users [6]. The computed tomography findings in

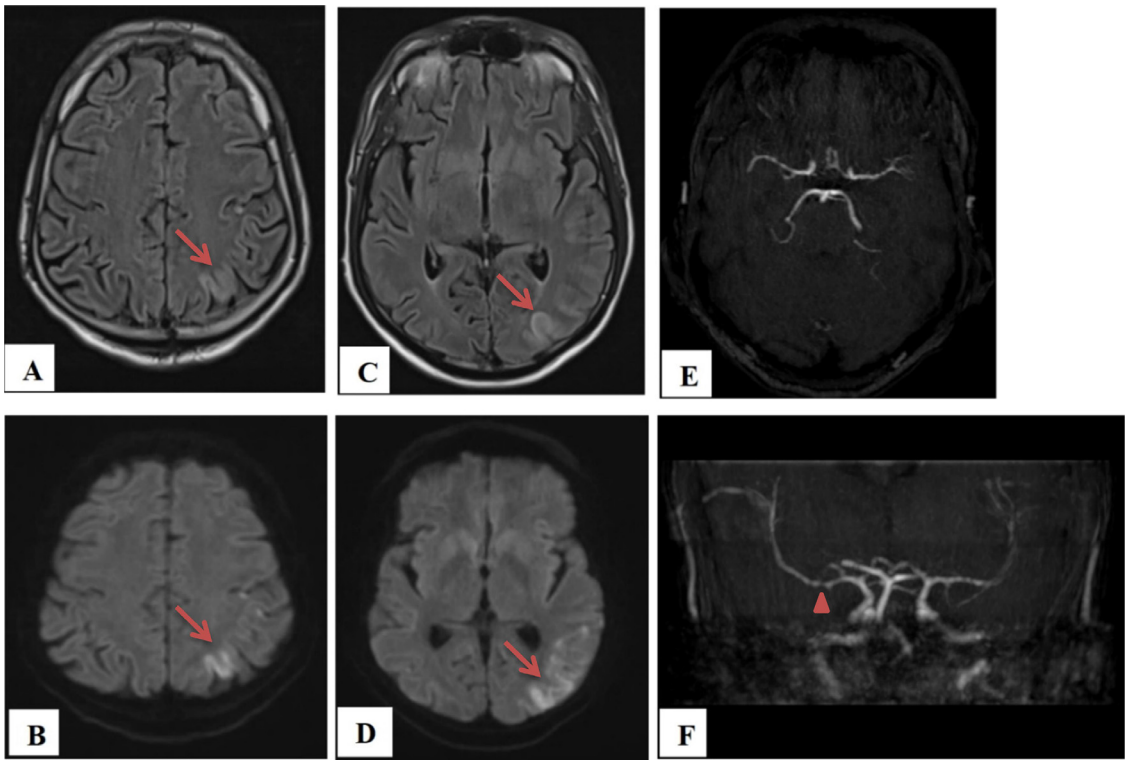

Fig. 2 – Cerebral MRI in axial section (A,B,C,D,E) and coronal section (F) in FLAIR (A,B) with DIFFUSION (C,D) sequences showing increased signal intensity and diffusion restriction involving the cerebral cortex in the left parietal region (Arrow) consistent with ischemic damage.

community-acquired pneumonia may exhibit variability, often dependent on the causative agent. Conversely, septic emboli typically manifest as numerous peripheral pulmonary nodules in various stages of cavitation, indicative of septic infarction [7]. Chest radiology is crucial in assessing pulmonary complications associated with drug consumption, although CT findings generally lack specificity.

Bronchoalveolar lavage can help rule out infection and diffuse alveolar hemorrhage and typically reveals large numbers of eosinophils, Charcot-Leyden crystals, alveolar macrophages containing hemosiderin, and elevated protein concentrations [8].

Diagnosing cocaine-related pulmonary disease primarily relies on a history of cocaine exposure, consistent radiographic findings, and the exclusion of other obvious causes of the findings. Currently, there are no characteristic clinical, laboratory, or radiologic findings for "crack lung". Therefore, when encountering acute hypoxic respiratory failure with bilateral pulmonary infiltrates, physicians should consider crack lung in the differential diagnosis.

Treatment for a crack lung mainly involves supplemental oxygen, bronchodilator therapy, and assisted ventilation if necessary. Cocaine-induced thrombosis may require the use of low-molecular-weight heparin [9].

This case highlights one of the severe pulmonary and neurological complications associated with cocaine use, underscoring the importance of considering illicit drug use as a possible diagnosis when treating patients with undiagnosed airspace disease.

Conclusion

Diagnosis of cocaine related lung or cerebral intoxication is challenging if there is no know context of cocaine use because clinical and radiological features are not specific. It should be considered in the differential diagnosis to direct investigations in search of other arguments.

Patient consent

I, the author of the article: «*CRACK LUNG WITH TOXIC CEREBRAL VASCULITIS: CASE REPORT*» approve that the patient gives his consent for information be to published in RADIOLOGY CASE REPORTS.

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