An unusual case of acute respiratory distress syndrome in a young man

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Abstract

Acute Respiratory Distress Syndrome (ARDS) is a complex and progressive lung injury that results in respiratory failure, often caused by pulmonary or systemic disorders. We present a 32-year-old male who developed ARDS following intranasal inhalation of cocaine. Diagnosis was established thorough clinical evaluation, identification of characteristic ultrasonographic patterns, and careful exclusion of other potential causes. The patient was managed with conservative medical therapy, including non-invasive ventilation, bronchodilators, antibiotic therapy, and comprehensive supportive care. Despite initial concerns about the severity of his condition, the patient's outcome was favourable, reflecting the diverse presentations and potential reversibility of ARDS. This case highlights two main aspects: i) the critical role of emergency physicians in recognizing and managing ARDS, even when the underlying cause is not immediately apparent; and ii) the importance of considering illicit drug use as a potential trigger during the initial assessment of respiratory complications.

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Key words: ARDS, cocaine, drug abuse, emergency medicine, non-invasive ventilation, ultrasonographic patterns, PoCUS.

Contributions: AC, MDS, BP, GM, and MG designed the project and wrote the paper. AC, CP, and MG searched the literature for retrievable papers. MDS, CP, RDG, and MG critically reviewed the paper. All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

Patient consent for publication: the patient gave his written consent to use his personal data for the publication of this case report and any accompanying images.

Availability of data and materials: all data underlying the findings are fully available.

Received: 30 August 2024.
Accepted: 7 November 2024.
Early view: 23 December 2024.

Emergency Care Journal 2025; 21:12989
doi:10.4081/ecj.2025.12989

Introduction

Acute Respiratory Distress Syndrome (ARDS) is a severe, life-threatening diffuse inflammatory lung injury. It is characterized by poor oxygenation, pulmonary infiltrates, and acute onset, with microscopic findings of massive capillary endothelial and alveolar damage.[1] In January 2024 a modern definition of ARDS by a consensus conference of 32 critical care ARDS experts set new diagnostic recommendations, i.e.: i) maintaining bilateral opacities as major diagnostic criterion, while adding ultrasound as an imaging modality, especially in resource-limited areas; and ii) in that latter setting, do not require positive end-expiratory pressure, oxygen flow rate, or specific respiratory support devices.[2]

ARDS progresses through distinct phases: an initial one of alveolar-capillary damage, a proliferative phase with partial recovery of lung function and a final fibrotic phase, which terminates the acute disease. Inflammation, apoptosis and necrosis of pulmonary epithelial and endothelial cells cause an increased alveolar-capillary permeability, leading to alveolar edema and proteinosis with hypoxemia due to impair gas exchange.[3-5] A hallmark of ARDS is the non-uniform pattern of lung injury throughout different regions, with some segments being more severely affected than others. This leads to reduced regional lung compliance, often more pronounced in the lower portions of the lung. The variable injury pattern implies that oxygenation strategies might improve oxygen diffusion in affected alveoli, although these can also elicit "volutrauma" and "atelectrauma" in spared pulmonary tissue.[1] ARDS can result from pulmonary infection or aspiration, as well as extrapulmonary/systemic sources e.g. sepsis, trauma, massive transfusion, drowning, drug overdose, fat embolism, inhalation of toxic substances, and pancreatitis. These extra-thoracic conditions initiate an inflammatory cascade that culminates in pulmonary injury. The diagnosis of ARDS is based on acute onset, bilateral lung infiltrates on chest imaging (of non-cardiac origin), and a PaO2/FiO2 ratio < 300 mm Hg, with subtypes classified by severity. Patients with ARDS often develop pulmonary artery vasoconstriction and may progress to pulmonary hypertension. ARDS has a high mortality rate therefore a timely diagnosis is crucial to start the available treatment options.[3-5] Herein, we describe the very peculiar (and previously not so commonly reported) case of a patient admit-

ted to the Emergency Department for an ARDS related to cocaine assumption.

Case Report

A 32-year-old man presented to the Emergency Department (ED) with acute respiratory distress, complaining of chest pain that had worsened in a few hours. He was an active smoker, not taking any medications and with an unremarkable medical history. On admission, he denied fever or cough. A low-flow oxygen therapy via nasal cannula was administered due to low SpO2 on ambient air (92%). He had a blood pressure of 155/80 mmHg, heart rate of 115 bpm (regular rhythm), and respiratory rate of 34 acts per minute. An ECG showed no ischemic changes, with sinus tachycardia. Arterial blood gas analysis revealed primary respiratory failure (pH 7.39, pCO2 41 mmHg, pO2 70 mmHg, HCO3- 22 mmol/L, p/F ratio 251, lactate 0.5 mmol/L). On physical examination, the patient was dyspnoeic with the use of accessory muscles. Auscultation revealed reduced breath sounds in the left lung base, but no other significant findings. A Point-of-Care Ultrasound (PoCUS) of the anterior chest revealed a prominent type A pulmonary anterior pattern (no interstitial damage), without signs of pneumothorax or pleural effusions. No ultrasonographic signs of aortic dissection, pericardial effusion, inferior vena cava dilation or right ventricular overload/dysfunction were detected. Chest X-ray and laboratory tests, including troponin, D-dimer, and C-reactive protein, were unremarkable. Since respiratory conditions worsened oxygen delivery was increased by switching to an oxygen mask, and intravenous methylprednisolone (1 mg/kg), magnesium sulphate (2g), and broncho-dilatator aerosol therapy were administered. A low dose of intravenous morphine (0.03 mg/kg) was used to alleviate respiratory distress and anxiety. An initial improvement was observed for about an hour, although subsequently the patient had a further worsening with anxiety, dyspnoea and orthopnoea. A second dose of intravenous morphine (0.06 mg/kg) was given, and the patient was re-evaluated. Thoracic auscultation revealed minimal crackles, and PoCUS performed on the posterior thoracic wall now showed a diffuse, confluent type B pattern (suggestive of an interstitial damage) with pleural irregularity. A trial of non-invasive ventilation (NIV) was initiated (PS 10 cmH2O, PEEP 6 cmH2O, FiO2 40%) to support the rapidly deteriorating respiratory dynamics with gradual improvement. Without finding any other cause of ARDS and suspecting a drug abuse, a new anamnestic re-evaluation was performed. The patient admitted an occasional use of intranasal cocaine a few nights before. After discussing the case with the intensivist team, the patient was admitted to the intensive care unit. After 24 hours from the admission, during which treatment with NIV was continued (resulting in an at least partial respiratory stabilization), a pulmonary Computed Tomography (CT) scan identified bilateral basal atelectasis (Figure 1) and Pulmonary Embolism (PE) in the left lobar artery (Figure 2). Even in the presence of residual atelectasis, we decided not to increase the ventilation pressure settings in order to avoid the occurrence of a pulmonary barotrauma. A venous doppler ultrasound of the lower limbs showed no deep vein thrombosis, and echocardiography documented normal biventricular function. The patient was treated with appropriate anticoagulation (apixaban 10 mg bid for 7 days followed by 5 mg bid), combined with antibiotic therapy (amoxicillin/clavulanic acid 2.2g qid and azithromycin 500 mg die). NIV was discontinued with a gradual decrease of oxygen supplementation followed by early weaning. The patient was subsequently transferred to the pneumology clinic and discharged one week after admission in good condition.

Discussion

In this case, the patient developed ARDS few days after an illicit drug abuse, *i.e.* crack cocaine, notoriously able to induce severe lung injury. Cocaine's harmful effects are twofold: it induces intense pulmonary vasoconstriction and exerts a direct toxic action on the alveolar cells and capillaries. These combined effects can rapidly precipitate a very severe life-threatening condition such as ARDS.[6] The connection between respiratory complications and the abuse of chemical inhalants / other substances is extensively documented in clinical practice. The most dangerous substances include cocaine, amphetamines, opiates, and benzodi-

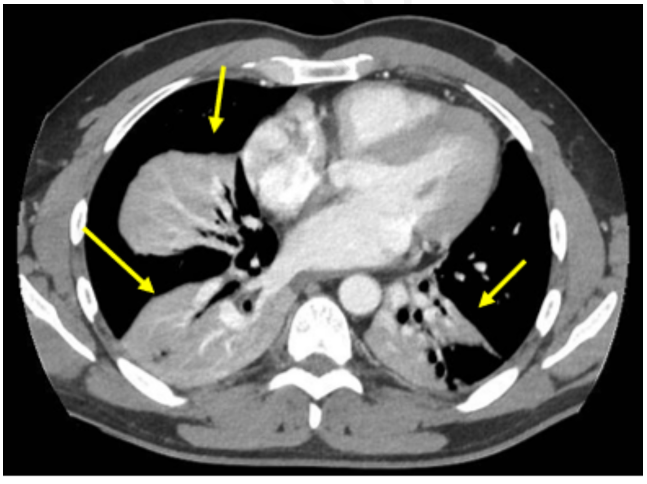

Figure 1. CT scan performed after 24 hours of NIV still showing basal bilateral atelectasis (yellow arrows). Note: CT: Computed tomography; NIV: Non-invasive ventilation.

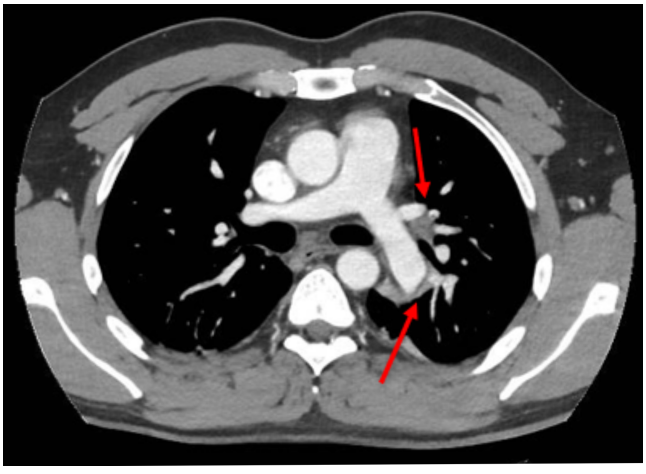

Figure 2. CT scan of the pulmonary embolism of the left lobar artery (red arrows) performed after 24 hours of NIV. Note: CT: Computed tomography; NIV: Non-invasive ventilation.

azepines, all of which have the potential to precipitate acute respiratory failure.[7] Pulmonary complications linked to substance abuse can manifest with various clinical pictures, one of the most dramatic being pneumothorax or pneumomediastinum, often triggered by smoking or inhaling cocaine. The mechanism underlying these conditions involves barotrauma, where the sudden changes in lung pressure, caused by forceful inhalation or snorting, lead to bronchospasm and a dangerous increase in alveolar pressure. This elevated pressure can break the alveolar lining causing air to escape into the pleural space or mediastinum, thus resulting in pneumothorax or pneumomediastinum, respectively.[8,9] In addition to pneumothorax and pneumomediastinum, other severe pulmonary conditions associated with cocaine use include acute pulmonary edema, where fluid accumulates in the alveoli, impairing gas exchange and facilitating respiratory distress. Cocaine can also cause alveolar haemorrhage, which further complicates respiratory function. Cocaine users are at risk of developing organizing pneumonia, eosinophilic pneumonia, or even pulmonary infarction due to the vasoconstrictive properties of the drug, which worsens blood flow microcirculation responsible for ischemic tissue damage.[10]

A particularly devastating syndrome related to cocaine use is "crack lung," a condition characterized by severe lung inflammation and haemorrhage that can rapidly progress to respiratory failure.[11] The toxic effects of crack cocaine are so potent that they can overwhelm the defence mechanisms of the lung, leading to widespread damage and, in many cases, the need for intensive medical intervention.[12] In this case, we attributed limited clinical relevance to the documented PE, as it appeared to be an incidental finding that did not account for the respiratory failure, given its size and the absence of cardiorespiratory impact. It is likely that the PE developed later, considering the initial D-dimer negativity on the first day, especially since thromboembolic disease is a known complication of cocaine use.[11]

In summary, the inhalation of crack cocaine poses a significant risk for a wide range of serious and potentially life-threatening pulmonary conditions. The recognition of these risks is crucial for clinicians when evaluating patients with acute respiratory symptoms, particularly in the context of substance abuse. Early identification and intervention can result in a critical difference in patient outcomes, highlighting the importance of considering substance abuse in the differential diagnosis of respiratory distress.

Conclusions

Cocaine-induced respiratory illness is a well-known, broad-spectrum condition. In our case, the correct diagnosis was established by repeated physical and ultrasonographic assessments as the clinical progression occurred. Once the clinical elements of ARDS were identified, a rapid review of the main possible causes prompted us to focus on investigations of the most epidemiologically likely condition. Furthermore, nearly 41% of patients who present to the ED with complications from inhaled cocaine use report chest pain, even without evidence of acute coronary syndrome, and about 40% of them deny drug use when asked. Given the serious nature of the disease, the high prevalence of cocaine abuse, the large number of young patients presenting with dyspnea (with or without actual respiratory failure), and the reluctance of patients to admit their use of cocaine, emergency physicians should consider illicit drug consumption as a risk factor for respiratory complications during the initial assessment.[13]

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