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**Respiratory Diseases**

# Severe Acute Asthma, Subcutaneous Emphysema, Pneumomediastinum and Pneumothorax Related to Cocaine: A Rare Clinical Case

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**Abstract** **Case Report**

Cocaine is one of the most widely used and trafficked illicit drugs in the world. Snorted or smoked cocaine can cause a variety of pulmonary complications ranging from bronchospasm to destruction of the alveolar-capillary membrane. We report the case of a 19 year old female patient, asthmatic since the age of 12 with non compliance to treatment, admitted for severe acute asthma associated with soft tissue emphysema, pneumomediastinum and right pneumothorax, secondary to her first consumption of snorted cocaine, requiring intubation and ventilation in intensive care with good radio-clinical evolution under symptomatic treatment.

**Keywords**: Chest pain, subcutaneous emphysema, pneumomediastinum, pneumothorax, asthma, cocaine, crack cocaine, psychoactive substances, drugs.

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## INTRODUCTION

Cocaine is an alkaloid with unique chemical properties and stands as one of the most widely used and trafficked illicit drugs globally. Primarily consumed by young adults, it is sought after for its sympathomimetic effects on the body and can be inhaled, smoked, or injected intravenously for recreational purposes. Cocaine abuse, whether occasional or chronic, is linked to various severe pathologies, particularly pulmonary issues ranging from bronchospasm to the destruction of the alveolar-capillary membrane and acute lung lesions. We present a case of a patient admitted to intensive care due to severe acute asthma associated with subcutaneous emphysema, pneumomediastinum, and right pneumothorax, all secondary to inhaled cocaine.

## OBSERVATION

We present the case of a 19-year-old patient, a chronic smoker with a history of occasional alcohol consumption, known to have asthma since the age of 12, and non-compliant with treatment. The patient was admitted to the emergency department following the sudden onset, the day before her consultation, of intense and persistent mid-chest pain associated with chest tightness and wheezing that did not respond to Salbutamol. A few hours later, the patient developed a sensation of facial swelling, which progressively worsened, and was later complicated by altered consciousness.

Upon admission, the clinical examination revealed a drowsy patient with desaturation of 80% in the open air. Bilateral cervical, thoracic, abdominal, and brachial subcutaneous crepitations were observed, with a positive Hammam's sign. Pulmonary auscultation revealed bilateral wheezing.

Facing acute severe asthma, the patient was intubated, ventilated in intensive care, and administered systemic corticosteroids with magnesium sulfate infusion. The blood count was normal, with a white blood cell count of 10660/µL and a CRP of 16 mg/L. The thoracic CT scan (Figure 1) showed extensive emphysema dissecting the subcutaneous tissues in the cervical and thoracoabdominal regions, with moderate pneumomediastinum and a pneumothorax on the right, without pneumopericardium.

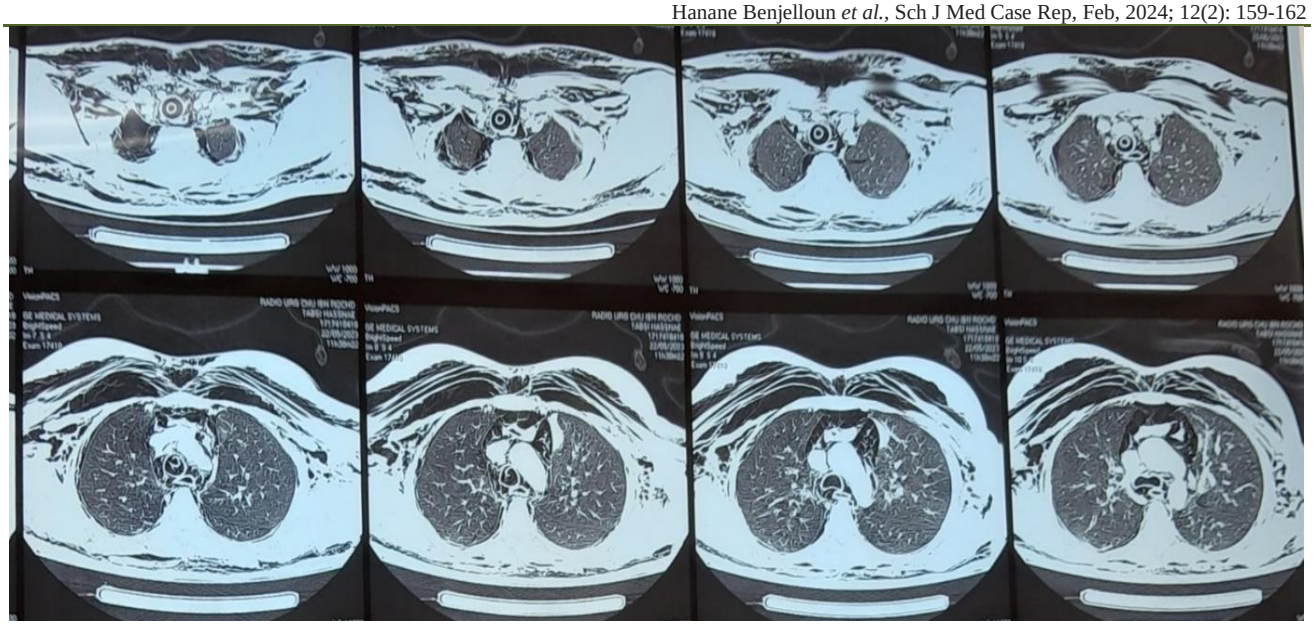

**Figure 1: Chest CT scan in axial section and parenchymal window, showing subcutaneous emphysema, moderate pneumomediastinum and right pneumothorax**

The patient was extubated after 48 hours and then placed on oxygen therapy with nebulized Salbutamol, followed by oral corticosteroids and asthma treatment with high-dose inhaled corticosteroids and Formoterol. The peak expiratory flow was estimated at 340 L/min. A chest X-ray (Fig 2a) taken on day 3 of hospitalization revealed a continuous diaphragm sign and a fine opaque border delimiting the mediastinal elements medially. We also note the obvious thoracic distension and subcutaneous cervico-thoraco-abdominal emphysema.

After the patient was reassured, she admitted that the chest pain had occurred suddenly and immediately after the first consumption of snorted cocaine (2 lines of powdered cocaine) while performing the Valsalva maneuver to increase the effect of the substance absorbed.

The diagnosis was, therefore, nasal cocaine consumption, causing barotrauma following the Valsalva maneuver and resulting in bronchospasm leading to severe acute asthma, responsible for intra-thoracic gas effusions.

On the 7th day of hospital stay, the patient progressed well with symptomatic treatment. The control Peak Expiratory Flow (PEF) was 450 L/min. The chest X-ray was normal (Fig 2b).

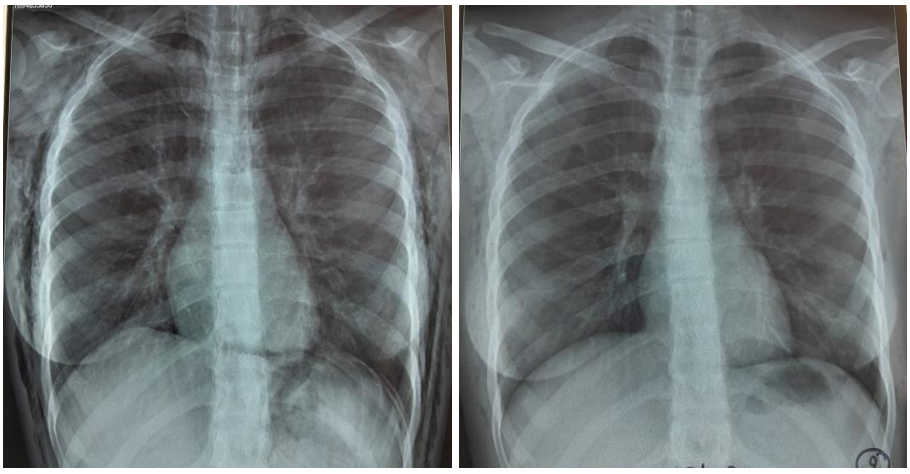

**Figure 2: Chest X-ray on admission (a) and at D7 of hospitalization (b)**

The patient was discharged, emphasizing the importance of therapeutic education for her asthma and stressing the significance of quitting smoking and cocaine. She was referred to consultations in addiction medicine and allergology.

## DISCUSSION

Cocaine is the second most abused and trafficked illicit drug in the world, following marijuana. Consequently, it is unsurprising that it ranks as the most commonly abused illicit drug in emergency departments and is one of the leading causes of drug-related deaths [1]. Cocaine, in the form of white powder (cocaine hydrochloride), is either snorted or injected after dissolution. It is also available in crystal form known as Crack, primarily smoked with a potent addictive potential [2]. The cocaine circulating in the illicit market is seldom pure, often adulterated to increase its volume. Adulterants can escalate the risks associated with the sold cocaine, ranging from inert substances to pharmacologically active ones (phenacetin, diltiazem, caffeine, levamisole, lidocaine, etc.).

Karila *et al.*, [3] comprehensively outlined various somatic complications linked to cocaine use, encompassing cardiovascular, neurological, infectious (related to risk-taking behavior), respiratory, ENT (chronic nasal use), dermatological, gynecological, and obstetrical complications. Snorted or smoked cocaine can lead to numerous pulmonary complications, including gas effusions, asthma, pulmonary edema, diffuse alveolar hemorrhage, pulmonary infarction, eosinophilic lung disease, interstitial lung disease, obliterative bronchiolitis with organized pneumonia, pulmonary arterial hypertension, and thermal lesions of the airways [4]. Two primary complications, intra-thoracic gas effusions, and asthma, will be discussed in detail below, mirroring the presentation of our patient.

Intra-thoracic effusions are associated with barotrauma, resulting from the direct toxicity of cocaine or its impurities following repeated inhalation. Barotrauma occurs due to a sudden increase in intra-alveolar pressure, leading to the rupture of the alveolar wall and the diffusion of air through interstitial spaces, progressing along bronchovascular axes. If the alveolar wall ruptures directly into the pleural cavity, it can lead to pneumothorax. Air can also reach the epidural space, causing pneumorachis, as well as soft tissues, pericardium, and peritoneum. This phenomenon, known as the "Macklin effect," occurs during deep, forced inspiration with mouth and nose closure (Müller maneuver) or during forced expiration with a closed glottis and prolonged apnea (Valsalva maneuver). Coughing after inhalation can increase intra-alveolar pressure and promote alveolar rupture [4]. In 73% of cases, pneumomediastinum occurs in isolation, while in 27%, it is associated with one or more other gas effusions [5].

Among the bronchial complications secondary to cocaine use is asthma. Cocaine use, especially through the pulmonary route (snorting and/or smoking), can contribute to the development of asthma, showing a temporal relationship between the onset of use and the manifestation of asthma [6]. The prevalence of asthma is higher in cocaine users, often associated with poorer compliance [7]. In non-asthmatic individuals, smoked cocaine (crack) leads to a significant decrease in specific airway conductance. Studies on bronchial hyperreactivity and carbon monoxide diffusion in cocaine users yield discordant results [8]. However, cocaine use increases the risk of asthma exacerbation and prolonged hospitalization. During asthma exacerbation, cocaine users are more frequently admitted to intensive care, intubated, and may require invasive ventilation, as observed in our patient, sometimes leading to fatal outcomes [9]. In a study of 102 patients whose deaths were attributed to asthma, blood toxicology tests were positive for cocaine in 14% of cases [10]. Several pathophysiological mechanisms have been proposed to explain the effects of cocaine consumption on asthmatic disease. Despite cocaine's inhibition of pre-synaptic reuptake of dopamine and norepinephrine, producing a bronchodilator effect, its irritant effect on the bronchial mucosa surpasses the former, explaining bronchospasm in crack smokers. Additionally, repeated cocaine use diminishes local immune defenses, increasing susceptibility to lower respiratory infections and asthma exacerbations [11].

As cocaine is an illicit substance, establishing the causal link between pulmonary complications and cocaine use may be challenging if cocaine use is not disclosed. Toxicological analysis of urine can be a valuable diagnostic aid. It is essential to discontinue cocaine use to prevent pulmonary complications, as well as the numerous psychological and somatic issues caused by this substance. Therefore, all healthcare professionals should systematically offer assistance for cocaine cessation to individuals using this psychoactive substance.

## CONCLUSION

Injected, snorted, or smoked cocaine can lead to various pulmonary complications, some of which are severe, necessitating intensive care hospitalization. Fatal cases have been reported. Given that cocaine is an illicit substance, its use is often underreported. In cases of lung damage, especially gas effusions or exacerbation of asthma in a young and healthy individual, healthcare providers should consider the possibility of cocaine consumption to explore its potential responsibility and assess imputability. Discontinuing cocaine use remains essential to prevent both psychological and somatic complications.

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