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Case Report

Acute Pulmonary Injury after Inhalation of Free-Base Cocaine: A Case Report

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Introduction: Many patients presenting to urban emergency departments (EDs) have chief complaints directly related to the use of illicit drugs. Given the reluctance of patients to admit to their use of cocaine, it is important for the emergency medicine provider (EMP) to recognize key epidemiologic principles as well as features of the history, physical examination, and diagnostic studies that suggest the sequelae of cocaine abuse.
Case Presentation: We describe our assessment of an otherwise healthy 47-year-old man with the acute onset of pleuritic chest pain accompanied by hypoxia, radiographic evidence of diffuse alveolar hemorrhage (DAH), and an elevated creatine phosphokinase (CPK) level. The patient vehemently denied active cocaine abuse. No clear pulmonary, cardiac, or infectious explanations for his signs and symptoms were readily apparent. Ultimately, after further workup and urine toxicology screening, the cause of this patient's chest pain and hypoxia was determined to be DAH related to his recent inhalation of crack cocaine. The patient was treated with systemic corticosteroids and improved.
Conclusion: Nearly 41% of patients who present to the ED because of complications of inhaled cocaine use are experiencing pleuritic chest pain, and more than half have an elevated CPK concentration. As many as 40% of these patients deny using the drug when asked. These data are important for EMPs to know when formulating a differential diagnosis for patients presenting with pleuritic chest discomfort.

Key words: *chest pain, cocaine, inhalation injury, pulmonary disease*

Introduction

In 2008, the National Institute on Drug Abuse's National Survey on Drug Use and Health estimated that 1.9 million Americans were current cocaine users and, of that group, 359,000 used crack cocaine. Cocaine use was cited as a factor in one-fourth of the nearly 2 million emergency department (ED) visits for drug misuse or abuse.[1]

Trauma caused by drug abuse receives greater attention but cocaine use has a significant relationship with non-trauma conditions such as chest pain (CP) and dyspnea. Hollander and colleagues reported that up to 25% of patients with nontraumatic CP tested positive for cocaine. Of that group, only 72% admitted to recently using the drug.[2] Of ED patients with new-onset bronchospasm, the percentage who used cocaine was 2.5 times higher than that of cocaine users in a matched group of non-asthmatics.[3]

Emergency medicine providers (EMPs), especially those working in urban settings, are likely to encounter patients with cocaine-related complaints. It is important for EMPs to be aware of the clinical signs and symptoms of cocaine use and to follow a systematic approach in the evaluation of patients who present to the ED with those indicators.

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Case Report

A 47-year-old man presented to the ED after the acute onset of dyspnea and sharp, pleuritic, substernal CP. These symptoms started acutely with no clear precipitating event. He was not experiencing vomiting, diaphoresis, or radiation of the pain. Deep inspirations worsened the pain but no other aggravating or relieving factors were identified.

The patient's medical history was unremarkable. His surgical history included exploratory laparotomy 10 years earlier secondary to gunshot wounds of the abdomen and chest. Those injuries caused no long-standing sequelae. He took no medications and had no known allergies. His social history revealed that he smoked 10 cigarettes/day and used marijuana twice a week. He denied alcohol use. The patient reported a distant history of cocaine abuse but denied use in the past 2 years. He had no family history of chronic lung disease.

On initial examination, the patient appeared healthy but in moderate respiratory distress. He used accessory muscles to breathe while sitting in the tripod position. His vital signs were temperature, 37.6 °C; blood pressure, 111/85 mmHg; heart rate, 120 beats/minute; respiratory rate, 28 breaths/minute; and oxygen saturation, 87% on room air. The oxygen saturation improved to 94% with a nonrebreather mask run at 15 liters/minute. The physical examination was remarkable for tachycardia, tachypnea, and coarse breath sounds throughout the bilateral lung fields. The patient's pupils were 6 mm in diameter, equal, and reactive. Facial and oropharyngeal examinations revealed no burns or areas of discoloration.

An electrocardiogram showed sinus tachycardia without an acute injury pattern. A chest radiograph (CXR) showed no abnormalities (Fig. 1). Laboratory findings were significant for mild leukocytosis ($16 \times 10^3/\mu l$) and an elevated creatine phosphokinase (CPK) level (997 IU/L). A urine toxicology test was requested.

Vancomycin, 1 g, and piperacillin/tazobactam, 4.5 g, were administered intravenously because of the patient's respiratory distress and the possibility of an infectious etiology.

There was a high pretest suspicion for pulmonary embolism (PE); therefore, computed tomographic angiography of the lungs was obtained. The results showed widespread bilateral ground-glass opacities, suggesting diffuse alveolar hemorrhage (DAH). No PE was evident. There were no findings of an infectious process (Fig. 2).

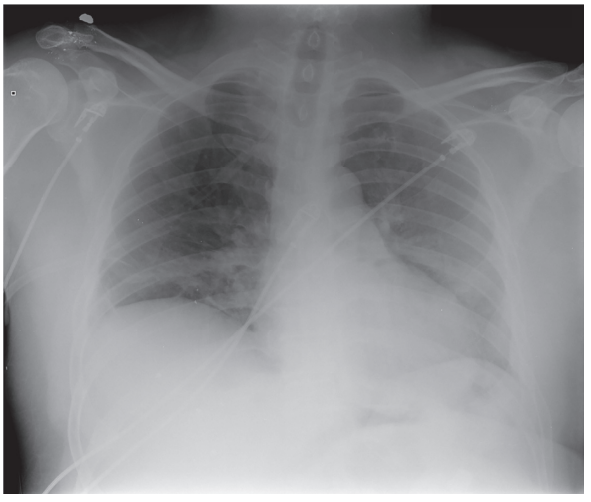

Fig. 1. Chest radiograph demonstrating no acute pulmonary abnormalities.

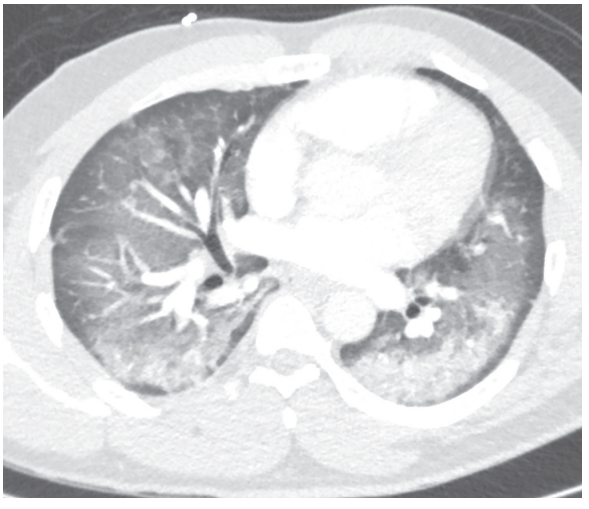

Fig. 2. Computed tomographic angiography revealing widespread bilateral ground glass opacities.

The patient was reexamined and his social history was revisited. He remained in moderate respiratory distress without any significant change in his physical examination. He continued to vehemently deny cocaine abuse as well as risk factors for the human immunodeficiency virus. His urine toxicology screen returned positive for cocaine. When confronted with these results, the patient admitted to smoking crack cocaine immediately prior to the onset of his symptoms. His ultimate diagnosis was acute pulmonary injury secondary to inhaling cocaine, colloquially known as "crack lung."

The antibiotics were discontinued and methylprednisolone, 125 mg, was administered. The patient

was admitted to the intensive care unit and improved rapidly. Transthoracic echocardiogram showed normal valve function, no pericardial effusion, and an ejection fraction of 65%.

Shortly after admission, the patient was discovered smoking crack cocaine and subsequently left the hospital against medical advice. The timeline of the patient's hospital course is presented in Table 1.

Although the patient was given an appointment for substance abuse counselling, he was lost to follow-up. We contacted his family but they did not know his whereabouts. Records reveal that the patient was later seen in an ED after being found unresponsive in the correctional facility where he was incarcerated. A urine toxicology screen at that time was negative for cocaine. The patient survived and no cause for the event was recorded in the medical record.

Table 1. Timeline of significant clinical events

Time (relative to arrival)	Event
-60 min	Reported onset of symptoms
	Arrival to the ED & initial evaluation
+ 8 min	ECG obtained
+ 30 min	CXR obtained
+ 35 min	Antibiotics administered
+ 50 min	Serum lab values available
+ 1 hr 40 min	CTA chest completed showing DAH
+ 5 hr 10 min	Urine toxicology reveals cocaine use
+ 5 hr 20 min	Patient admits to inhalation cocaine use
+ 5 hr 30 min	Antibiotics discontinued
+ 5 hr 40 min	Systemic corticosteroids administered
+ 8 hr 35 min	Admitted to the ICU
+ 9 hr to 24 hr	Patient improves
+ 24 hr 10 min	Echocardiogram completed
+ 25 hr 15 min	Patient found smoking crack cocaine in the ICU
+ 26 hr	Leaves AMA

ED, emergency department; ECG, electrocardiogram; CXR, chest radiograph; CTA, computed tomographic angiography; DAH, diffuse alveolar hemorrhage; ICU, intensive care unit; AMA, against medical advice.

Discussion

This case illustrates the need to maintain a wide differential diagnosis in the setting of undifferentiated acute dyspnea. The EMP was able to make the correct diagnosis by paying attention to several clues in the patient's history, physical examination, and diagnostic studies. The limitations of the case are that the patient's post-hospitalization course and his long-term outcome are unknown.

Numerous pulmonary complications secondary to cocaine inhalation have been described. The inhalation of cocaine can trigger acute processes such as bronchospasm, pneumothoraces, pulmonary infarction, eosinophilic lung disease, and DAH.[4] Long-standing use can result in emphysema, pulmonary hypertension, and bronchiolitis obliterans. The challenge faced by EMPs when treating cocaine-related pathology is the detection of the underlying disease process and identification of a primary cause, especially in patients who fail to disclose recent drug abuse.

The most common symptom of acute cocaine inhalation injury is pleuritic CP. Tashkin found that nearly 41% of patients who had inhaled cocaine experienced some type of CP.[5] Other complaints included wheezing, hemoptysis, dyspnea, and cough. Black sputum production was described by 40% of patients. Melanoptysis results from the inhalation of carbonaceous residue from the torches used to heat the crack cocaine.[4,6] Hemoptysis is caused by alveolar hemorrhage induced by vasoconstriction, pulmonary infarction, and cocaine-induced thrombocytopenia.[7,8] Marijuana use is frequently associated with the co-use of other drugs, including cocaine.[9-11]

Physical examination findings may also suggest the use of cocaine. The sympathomimetic effects cause tachycardia, hypertension, mydriasis, and possibly hyperthermia. Thermal injury caused by hot vapor might be visible in the upper airway. Wheezing, as a result of bronchospasm secondary to inhaled irritants, is present in 50% of crack users.[7,12]

The initial CXR might be unremarkable, but certain features suggest cocaine inhalation injury. These include a peripheral distribution of parenchymal opacities, bilateral lung involvement; and signs of barotrauma such as pneumothorax, pneumomediastinum, or subcutaneous emphysema.[6] For patients with significant pulmonary injury, computed tomographic imaging might be necessary for elucidation of the disease process. Extensive bilateral disease with ground-

glass opacities indicating noncardiogenic pulmonary edema or DAH might be seen. The cause of pulmonary edema is likely pulmonary capillary endothelial injury.[13] Bronchoalveolar lavage may reveal eosinophilia.[4]

A urine toxicology screen is the easiest and cheapest objective way to determine if a patient has used cocaine. The timing of the use and its contribution to the clinical presentation are more difficult to assess. Urine testing for cocaine's major metabolite, benzoylecgonine, can be positive for 3 days after acute exposure and remain positive for 2 weeks in chronic abusers.[11]

Elevated CPK is presumed to be secondary to skeletal muscle injury, although whether this is due to ischemia or increased muscle activity is not clear. Even with an elevated CPK level, most of these patients do not complain of myalgias.[14,15]

Although aspects of the medical history, physical examination, and radiographic and laboratory findings suggest inhalation injury, the ultimate diagnosis of pulmonary injury from crack cocaine use is clinical. Relevant cardiac, infectious, and other pulmonary causes should be entertained, even in the face of a positive toxicology screen for cocaine. The final diagnosis can be made only after these causes have been eliminated.

The treatment of patients with pulmonary injury secondary to cocaine abuse is largely supportive. The majority of patients show improvement within 24 hours of the drug being discontinued. Using high-dose steroids is controversial but case reports suggest it might be beneficial.[8-10,16] Antibiotics are not indicated; if they were initiated empirically, they should be discontinued.

Crack lung should be considered when patients with or without known pulmonary or cardiac disease present to the ED with CP or dyspnea. This cause must be considered even if the patient denies cocaine use, since nearly 40% of ED patients who test positive for cocaine initially deny its use.

Findings of new-onset wheezing, pleuritic CP, other illicit drug use, fever, tachycardia, hypertension, and hypoxia all should raise the EMP's concern for cocaine inhalation injury. An elevated CPK and chest imaging showing peripheral distribution of parenchymal opacities should further raise suspicions.

Toxicology screening may corroborate the suspected diagnosis of crack lung. A toxicology screen can remain positive for 2 weeks after cocaine use; therefore, a positive screen cannot be used as the sole diagnostic criterion.

Maintaining a high suspicion for inhalation cocaine injury in the right clinical setting will help the EMP make the correct diagnosis. Treatment can thereby be directed toward supportive therapy and corticosteroid use, and away from antibiotics.

Conflict of Interest

The authors declare that they have no competing interests.

Acknowledgment

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