Massive Pulmonary Hemorrhage in an Adolescent

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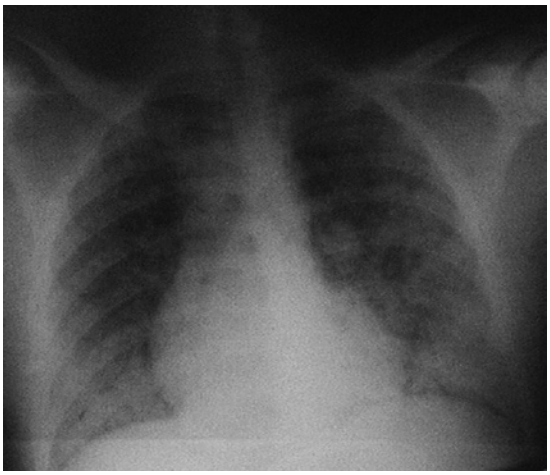
Cocaine has become one of the most frequently abused substances. More than 30 million Americans are estimated to have taken cocaine at least once, and 5 million reportedly use it regularly [1]. Infants, children and adolescents are also exposed. It is estimated that almost 6% of high school seniors in the United States have used cocaine and crack, a cocaine derivative [2]. Cocaine and crack are among the most common drugs encountered in the pediatric emergency department. Cocaine-related medical complaints continue to increase. The inhalation of cocaine can induce a wide variety of acute pulmonary disorders, such as alveolar hemorrhage, acute pulmonary edema, interstitial pneumonitis and fibrosis, pulmonary hypertension, pulmonary barotraumas, emphysema, foreign body granuloma, cocaine-related obliterative bronchiolitis, and gas exchange abnormalities [3].

Although diffuse alveolar hemorrhage associated with dyspnea and hemoptysis is a common manifestation of cocaine abuse, this phenomenon is not given sufficient attention in the literature. Indeed one pathological study demonstrated occult alveolar hemorrhage at autopsy in 58% of crack users, suggesting that occult alveolar hemorrhage occurs frequently in subjects using crack.

Patient Description

A 17 year old healthy adolescent was admitted to the pediatric intensive care unit. During the preceding month he began to experience hemoptysis while coughing, accompanied by epigastric pain and sometimes blood-stained vomiting. In the 2 days prior to his admission he became dyspneic with general weakness, and the bloody cough was exacerbated. He smoked 40 cigarettes a day but denied alcohol and drug abuse. He did not have allergies, prior hospital admission or an unusual family history.

Upon arrival he looked pale and exhausted, dyspneic, with a respiratory rate of 30/minute, blood pressure 110/60, tachycardia 110 beats/min, and body temperature 38.4°C. On auscultation, his heart sounds were normal but bilateral chest inspiratory crackles were detected. The rest of the physical examination was unremarkable. Chest X-ray revealed diffuse lung infiltrates with a normal cardiac shadow [Figure]. Laboratory tests demonstrated erythrocyte sedimentation rate 10 mm/hour, hemoglobin 5.7 g/dl, leukocytes 13.4 k/ul and platelets 439 k/ul. Glucose, electrolytes, kidney and liver function tests were all normal. Prothombin time was 18.9 seconds (mildly prolonged); activated partial thromboplastin time and fibrinogen were normal.



Chest X-ray on admission illustrates bilateral lung infiltrate with normal cardiac shadow.

He was admitted to the pediatric intensive care unit. Due to hypoxemia, high flow oxygen was administered through a facial mask with reservoir. The patient required blood products transfusion, 1500 ml packed cells and a similar quantity of fresh frozen plasma. Cefuroxime was administered intravenously and roxythromycin orally. A few hours later he became even more dyspneic and hypoxemic, with mental deterioration manifesting as irritability and confusion. He was sedated with propofol, intubated and mechanically ventilated. During laryngoscopy and tube insertion a large amount of red blood was aspirated from the trachea.

A computed tomography scan with angiography of the chest revealed normal pulmonary arteries and diffuse alveolar infiltrates without any other pathology. Echocardiography excluded cardiac pathology. Gastroduodenoscopy demonstrated a normal upper gastrointestinal tract. Larygoscopy was normal. Other laboratory tests included serology for human immunodefeciency virus and antineutrophil cytoplasmic antibodies, both of which were negative; complement (C3,C4) was normal. Toxicology tests of urine were positive for cocaine, marijuana and methamphetamine.

Two days later, suctions through the tracheal tube were clean and a bronchoscopy was normal. The patient was extubated on the third day of admission, and a repeated chest X-ray demonstrated a significant improvement. His hemoglobin level stabilized at 10 g/dl and the

coagulation tests normalized. Later on, the patient admitted to drug abuse in the previous 2 months.

Comment

Cocaine abuse, and particularly crack smoking, is extremely addictive. Users experience pleasant sensations that are quickly replaced by dysphoria, which may lead to self-recrimination, agitation, anxiety, or symptoms of clinical depression. Through its release of catechols into the blood, cocaine may cause an adrenergic storm with stimulatory central nervous system, cardiovascular (acute myocardial infarction, dilated cardiomyopathy, myocarditis or cardiac arrhythmias), renal and respiratory effects.

Although hemoptysis occurs frequently in crack users, the pathogenesis of diffuse alveolar hemorrhage related to its use remains unclear [4]. Possible mechanisms that induce alveolar hemorrhage are vasoconstriction of the pulmonary vascular bed, which may result in anoxic epithelial or endothelial cell damage producing alveolar hemorrhage and edema, and direct toxic effect of the inhaled substances on the alveolar epithelium leading to injury [4]. There are some suggestions of an immunological mechanism.

In the presented case we could not confirm any other diagnosis apart from the toxicity related to cocaine. In view of the rapid improvement in the patient's condition and the negative infection workup, an infectious etiology was very unlikely. Negative serological study, low sedimentation rate, clinical resolution without a specific therapy, normal complement levels, normal urinalysis, and no other organ involvement, exclude connective tissue or immunocomplex diseases. Neither the patient nor any of his relatives had a history of epistaxis or mucosal and cutaneous hemangiomas, which reduced the likelihood of arteriovenous malformations. Cardiovascular workup did not reveal pulmonary emboli or cardiac diseases. Mild prolongation of prothrombin time, which normalized later, did not explain the clinical picture. The prolongation could be due to consumption coagulopathy secondary to prolonged hemorrhage. Other entities like idiopathic pulmonary hemosiderosis or hemosiderosis related to milk allergy are diseases of infancy and childhood and are not compatible with our patient's uneventful medical history.

Although his urine tested positive to both marijuana and methamphetamine, we were unable to find an association between the use of these substances and pulmonary hemorrhage. Tashkin [5] described the airway effects of marijuana, cocaine and other inhaled illicit agents. Of all these substance, only cocaine caused pulmonary hemorrhage.

Since cocaine is one of the most popular drugs and its abuse is related to multiple clinical effects including pulmonary hemorrhage, we suggest that cocaine be considered in similar cases. The clinical course in cocaine-induced pulmonary hemorrhage is usually benign, and management is supportive. Thus, keeping in mind cocaine as a cause of pulmonary hemorrhage can save extensive workup.

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