CASE OF THE MONTH

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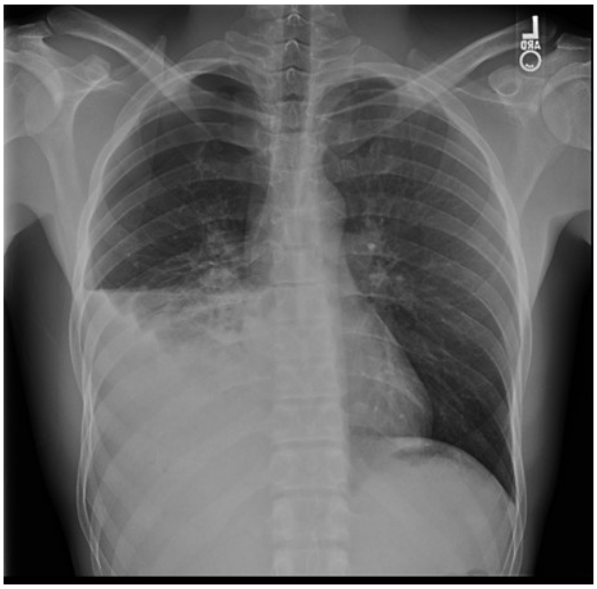
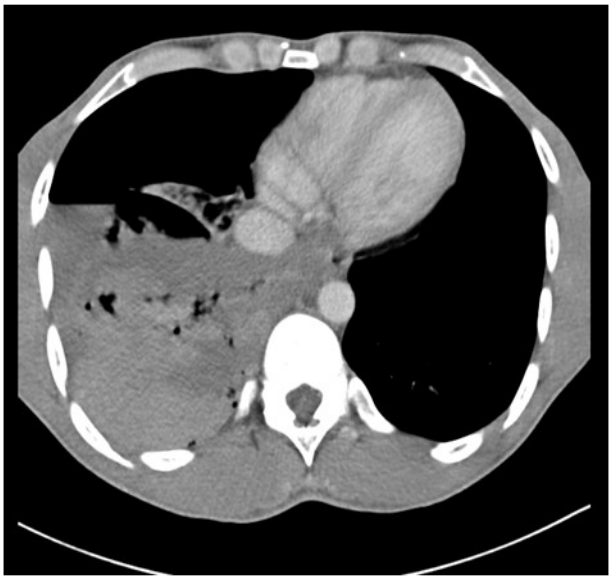
A previously healthy 26 year old African American male presented to the ED with the acute onset of right-sided posterior thoracic pain and shortness of breath over the previous four hours. He described the pain as severe, sharp and exacerbated by deep inspiration and reported that it radiated to the RUQ and anterior chest wall. He denied fever, chills, cough, trauma to the chest wall or a history of similar episodes in the past. He denied the use of IV drugs and had no known exposure to TB; however, he did admit to a history of tobacco use (1/2 ppd for two years), a history of inhaling cocaine (most recently two days ago) and a history of incarceration several years prior to this presentation.

Physical exam on admission demonstrated that he was alert and fully oriented. Vitals were WNL except for tachypnea with shallow respirations; his O2 Sat was 97%. He was lying very still for his exam and grimaced with any movement. The ENT exam was unremarkable and his trachea was midline though asymmetric chest motion was noted, with diminished chest expansion on the right. In the supine position, his right chest was tender to palpation, hyperresonant to percussion anteriorly and dull to percussion laterally. He was mildly tender to palpation in the RUQ. The remainder of his exam was normal.

Admission labs revealed a WBC of 6.5, Hgb 10.5, Hct 31.7, MCV 92.3 and Platelet count of 143. Coagulation studies were normal. BMP was normal except for BUN 26 and Cr 1.4; LFTs, serum amylase and serum lipase were all normal. Cardiac enzymes were normal and a UA was unremarkable. ABGs on supplemental oxygen via nasal cannula revealed pH 7.3, pCO2 45, pO2 111, HCO3 24, BE –0.6 and O2 sat of 98.7%. A rapid HIV was negative and a PPD later proved to be negative. A CXR and CT Chest revealed a right hydropneumothorax, possibly representing a large empyema (see images below).





Empiric IV antibiotics were ordered and a chest tube was placed with the immediate return of 1 liter of bloody fluid, free of clots. Pleural fluid studies were not consistent with empyema but, rather, suggested a hemothorax: the fluid HCT was 30.1%, the gram stain was negative and cultures, including mycobacteria, were all negative. Cytology of the pleural fluid revealed RBCs but was negative for malignant cells. Following placement of the chest tube and initiation of IV hydration, his Hgb remained stable, renal function normalized and the lung (cont)

(cont) re-expanded without evidence of underlying mass or infiltrate. The patient remained afebrile and had no leukocytosis throughout his hospital stay; the chest tube was removed without complication and he was discharged to home.

DISCUSSION:

The recreational use of cocaine can be traced back to early civilizations. Originally documented by the ancient Moche tribe of Peru over 2500 years ago, the practice of liberating cocaine from coca leaves (using lime produced from roasted sea shells) was still occurring in Venezuela in 1499, at the time of Amerigo Vespucci. Cocaine's ability to alter physiologic activity was clear to even the early users of the drug. Cieza de Leon wrote "When I inquired of certain Indians why they keep their mouths ever filled with that herb…..they say that they have little sense of hunger and feel great vigor and strength." [1] The ability of cocaine to induce such feelings of euphoria is likely responsible for its popularity in modern-day culture.

Cocaine can be introduced to the body in a multitude of ways. Popular methods include inhalation, intravenous injection or mucosal absorption (intranasal, sublingual, intravaginal or rectal). Inhalation results in a more rapid onset of action and a shorter time to peak effects than other methods [2]. The mechanism by which the drug is introduced is an important consideration when evaluating a patient for pathologic effects.

Pulmonary complications are common in patients who inhale cocaine. Cough is present in 26-61% of subjects and hemoptysis develops in up to 26% [4]. Bleeding may result from rupture of submucosal vessels or directly from the alveolar-capillary membrane. Autopsy series have revealed that 85% of patients who died from cocaine intoxication had evidence of pulmonary hemorrhage [5]. Pneumothorax, pneumomediastinum and pneumopericardium may occur less frequently but are also well-documented complications of cocaine inhalation. In one study of 71 crack smokers who presented to an ER with chest pain, two were found to have pneumomediastinum, one had a pneumothorax and one had a hemopneumothorax, directly attributed to the cocaine inhalation [4].

The manner in which the cocaine is inhaled and the techniques used to increase absorption across the pulmonary -capillary barrier can predispose the patient to barotrauma. Users attempt to introduce cocaine into the pulmonary system by maximal inhalation; once the peak inspiratory volume is achieved, users resort to one of several methods to enhance absorption of the drug into the bloodstream. These techniques include the Valsalva maneuver and the forceful exhalation by a smoking partner into the mouth of the user [3]. Additionally, severe coughing may be induced by irritation from the cocaine powder. All three of these factors trigger a sudden increase in intra-alveolar pressure that may exceed the elastic potential of the parenchyma, resulting in alveolar rupture [3].

Treatment of pneumothorax or hemothorax due to cocaine inhalation is similar to that for spontaneous pneumothorax: chest tube placement, supplemental oxygen and supportive care, including pain management. Of course, cessation of cocaine use is an important part of the therapy and a referral to a drug abuse treatment program should be considered. [3]

REFERENCES:

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