BACKGROUND

- Cocaine is one of the most abused drug in the United States.
- Cocaine is known to affect most organ systems, most commonly the cardiovascular, nervous, and respiratory system.
- The pulmonary manifestation of cocaine are diverse and potentially fatal. These may include “crack lung”, barotrauma, talcosis, organizing pneumonia, bullous emphysema, pulmonary infarction, cardiacogenic edema, and eosinophilic pneumonia.
- Pulmonary cocaine toxicity diagnosis is often missed as it requires knowledge of its various radiological manifestations.

CASE SUMMARY

A 44-year-old female with hypertension, chronic cocaine abuse and a stable right lung giant bulla presented with acute encephalopathy. She was afebrile with no leukocytosis. Chest X-ray showed a stable right lung bulla and computerized tomography (CT) abdomen/pelvis with IV contrast revealed colitis. She was started on empiric antibiotics while undergoing infectious and autoimmune workup for encephalopathy. She was COVID-19 positive which was thought not to be contributing to her acute illness. Urine drug screen was positive for cocaine. Patient’s respiratory status worsened on day two. A computed tomography angiography (CTA) chest showed no pulmonary embolism, however, there were new bilateral multifocal ground glass and consolidative pulmonary densities. The patient was intubated and moved to the intensive care unit. She underwent bronchoscopy with bronchoalveolar lavage (BAL) which ruled out diffuse alveolar hemorrhage, cultures and cytology were negative. She underwent an extensive work up for severe persistent respiratory failure and encephalopathy which was unrevealing. Hospital course was further complicated by ventilator-associated pneumonia progressing to multisystem organ failure. She was transitioned to comfort measures and eventually expired.

DISCUSSION

Cocaine lung toxicity have distinct presentations and are often missed by clinicians. The diagnosis is challenging and requires cocaine exposure, along with radiological findings.

In our patient, the giant emphysematous pulmonary bullae was chronic and was deemed to be caused by cocaine as other etiologies were ruled out when it was first identified. The mechanism of cocaine causing lung bullae is thought to be from both alveolar destruction and pulmonary vascular constriction.

The acute presentation of respiratory failure in our patient was most likely to be “crack lung.” Crack lung is a cocaine induced alveolar damage that usually manifests 48 hours after consumption and progresses rapidly to respiratory failure as seen in our patient. Crack lung has various patterns on CT chest including ground glass opacities, consolidations, halo sign, smooth septal thickening, crazy-paving pattern, centrilobular nodule, paraseptal emphysema and tree-in bud pattern.

Cocaine also increases the risk of pulmonary infections by reducing mucociliary clearance and immune cell count, this was a late complication in our patient.

Awareness of the various manifestations of cocaine toxicity in the pulmonary system can help clinicians make a diagnosis earlier, though management remains mainly supportive.

REFERENCES


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