



## Case report

## A case of cocaine-induced eosinophilic pneumonia: Case report and review of the literature

Felix Reyes, MD<sup>a,\*</sup>, Vytas Vaitkus, DO<sup>b</sup>, Mohammad Al-Ajam, MD<sup>b,c</sup><sup>a</sup> Department of Internal Medicine, SUNY Downstate Medical Center, Brooklyn, NY, USA<sup>b</sup> Department of Pulmonary and Critical Care Medicine, SUNY Downstate Medical Center, Brooklyn, NY, USA<sup>c</sup> Division of Pulmonary and Critical Care Medicine, Department of Veterans Administration NY Harbor Healthcare System, Brooklyn Campus, Brooklyn, NY, USA

## A B S T R A C T

Cocaine is a commonly abused recreational drug in the United States. An adult man developed non-specific pleuritic chest pain, pharyngitis and odynophagia after inhaling cocaine. Initial laboratory results revealed eosinophilia. Bronchoalveolar lavage also showed eosinophilia in the lavage fluid. These findings suggested the diagnosis of eosinophilic pneumonia. Chest imaging revealed scattered bilateral opacities and interstitial infiltrates. After initiation of systemic corticosteroids, the patient reported symptomatic resolution and radiographic clearance was achieved at 2 months follow up.

## 1. Introduction

Cocaine is one of the most commonly abused drugs in the United States. Despite having millions of chronic and occasional users, the reported cases of cocaine-induced eosinophilic pneumonia remain a handful [1,2]. Eosinophilic lung disease can be identified by an increased number of eosinophils in the lung tissue or BronchoAlveolar Lavage (BAL) fluid of a patient who has pulmonary symptoms or infiltrates on chest imaging.

## 2. Case presentation

A 59-year-old male presented to Emergency Department complaining of pleuritic chest pain for 10 days prior to presentation. The pain was described as reproducible and band like across the lower chest with worsening during deep inspiration. He also reported associated pharyngitis and odynophagia. The patient endorsed daily cocaine use, was an active smoker with a 12-pack years history, and occasional alcohol consumer. He was an employed electrician. He denied sick contacts, recent travel or other complaints prior to presentation. The patient had no history of lung disease and denied childhood asthma, he was born in Trinidad and Tobago but moved to the United States in his 20s. The patient reported having no pets or birds or mold in his apartment. There was no significant family history for cancer, connective tissue diseases or pulmonary diseases.

On physical examination the patient had red conjunctiva and denied chest tenderness upon palpation. Expiratory wheezes were auscultated

in all lung fields with basilar rales and crackles noted at the bases.

Urine toxicology at the time of admission was positive for cocaine and cannabis (THC), but negative for other illicit drugs. Laboratory studies revealed a white blood count of 9.2/ $\mu$ L with elevated serum eosinophils 11.8% (ref 0–7, Absolute Eosinophil Count: 1.1 cells/ $\mu$ L). HIV testing was negative.

Chest X-ray showed bilateral nodular opacities in the lungs, most prominent within mid to lower lung zones, a stable cardio-mediastinal silhouette and no pleural effusion were noted (Fig. 1). Computed Tomography (CT) imaging of the chest revealed: small mediastinal lymph nodes with innumerable nodular densities in the pulmonary parenchyma scattered throughout both lungs, majority of which demonstrated a surrounding area of ground glass opacity (Halo sign). Some consolidations were also noted, the largest of which was located lateral to the left upper lobe and measured 3.6  $\times$  3.1 cm with presence of air bronchograms (Fig. 2).

Urine Legionella, M. Pneumonia, and Aspergillus antibodies were negative. Procalcitonin level was negative.

Blood cultures revealed no growth after 5 days. Sputum analysis revealed normal flora but elevated WBC count (> 25 in a low power field) and acid-fast bacilli (AFB) showed no organisms. CMV, ANA, Anti-GBM, Anti-centromere, anti-SCL-70, ANCA, myeloperoxidase, proteinase-3-antibody, atypical pANCA, perinuclear ANCA, Cytoplasmic AB, Histoplasma were all negative. Angiotensin was found to be within normal limits.

His eosinophil count peaked at 14.4 (WBC: 10.3, AEC 1.5). He underwent diagnostic bronchoscopy which evidenced normal mucosa,

\* Corresponding author.

E-mail address: [felix.reyes@downstate.edu](mailto:felix.reyes@downstate.edu) (F. Reyes).

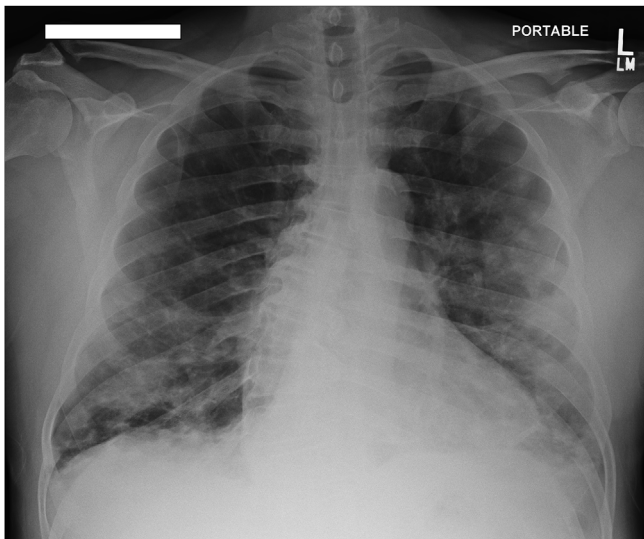


Fig. 1. Chest X-ray showing bilateral nodular opacities in the lungs.

BAL showed a clear hazy fluid with 40 WBC 35% eosinophils, diagnostic testing of the aspirate was negative for bacteria, viral or AFB infection.

Our patient reported symptomatic improvement after the initial administration of intravenous steroids. Prior to discharge the patient was started on oral corticosteroids with an outpatient taper: 60mg daily with a 10mg weekly decrease, until reaching 10mg daily. After reaching 10mg daily, the patient completed 2 weeks of 10mg daily and 2 weeks of 5mg daily.

Outpatient PFTs revealed no obstruction or restrictive defect. Follow up CT imaging two months after discharge revealed resolution of previously noted nodular opacities but persistence of ground glass opacities with interval decrease in size. The patient reported abstinence from cocaine use at 2 months follow up (Fig. 3).

### 3. Discussion

The most frequent pulmonary complaints reported by cocaine users are: dyspnea, cough, sputum production and non-specific chest pain [3].

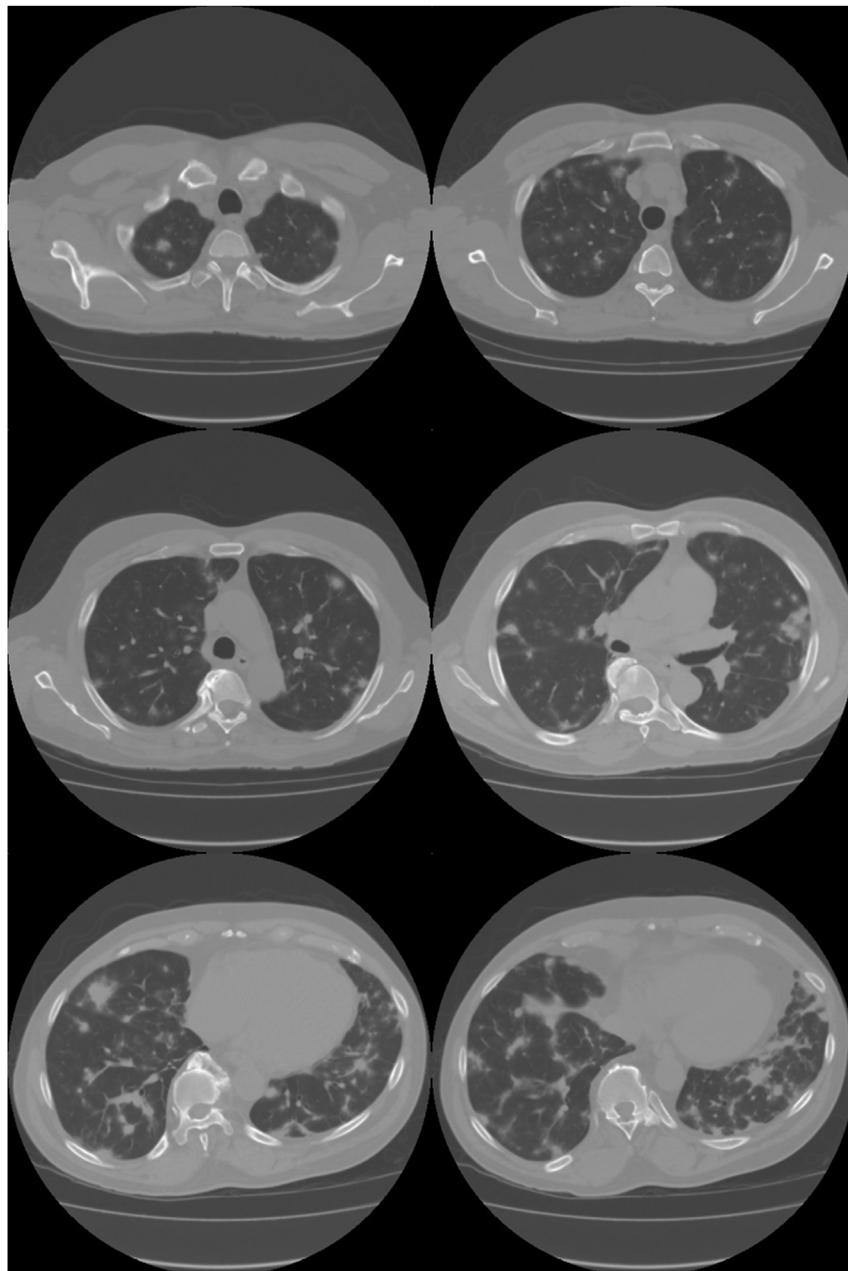


Fig. 2. Chest Computed Tomography (CT) imaging revealing: small mediastinal lymph nodes with innumerable nodular densities in the pulmonary parenchyma scattered throughout both lung.

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