

Case Report

A Bird's Tale: A Case of Acute Eosinophilic Pneumonia

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Abstract

Acute eosinophilic pneumonia is an uncommon respiratory condition whose etiology is unclear. Reports have identified cases caused by environmental inhalational exposures that lead to an overwhelming immune response. Here we present an unreported case of a 46-year-old female who developed acute eosinophilic pneumonia after avian exposure.

Keywords: Acute eosinophilic pneumonia; Bird exposure; Respiratory disease; Eosinophilic pneumonia

1. Introduction

Eosinophilic pneumonia refers to a spectrum of diseases that have the potential to cause eosinophilic infiltration of the lung parenchyma as well as the peripheral blood. There are infectious causes of pulmonary eosinophilia including parasitic and fungal as well as systemic vasculitis. Here we report a rare case of acute eosinophilic pneumonia secondary to avian exposure.

2. Case Presentation

A 46-year-old female with no past medical history or previous medications presented to the hospital for 1 week of shortness of breath with exertion. She reported that she was being treated for pneumonia by her primary care

physician with oral augmentin 875 mg twice a day and oral azithromycin 500mg on the first day and 250mg for four days thereafter. She denied fevers, chills, sweats, abdominal pain, nausea, vomiting, history of travel, ingestion of uncooked meats, unprocessed water or animal exposure. She denied smoking cigarettes, alcohol use or recreational drug use. She was employed as a caregiver who looked after the elderly.

On physical examination the patient was afebrile, however, saturating at 90% on room air. Patient appeared fatigued, often having to take breaks between sentences. Auscultation of her lungs revealed decreased breath sounds at the bases bilaterally. The remainder of the exam was unremarkable. Lab values revealed hemoglobin 10.3 gm/dL, hematocrit 30%, white blood cells $14.0 \times 10^9/L$ (with 10% eosinophils), and platelets $536 \times 10^9/L$. Computed tomography (CT) of the chest revealed bilateral tree in bud opacities, right and left lower lobe pneumonia, and hilar lymphadenopathy (Figure 1). The following laboratory tests were obtained to evaluate for unresolving atypical pneumonia with high risk features including legionella antigen, streptococcus urine antigen, fungal serologies, QuantiFERON gold, and HIV screening, which later returned negative. The patient was started on cefepime 2 gm IV q12h and vancomycin 1 gm IV q12h.

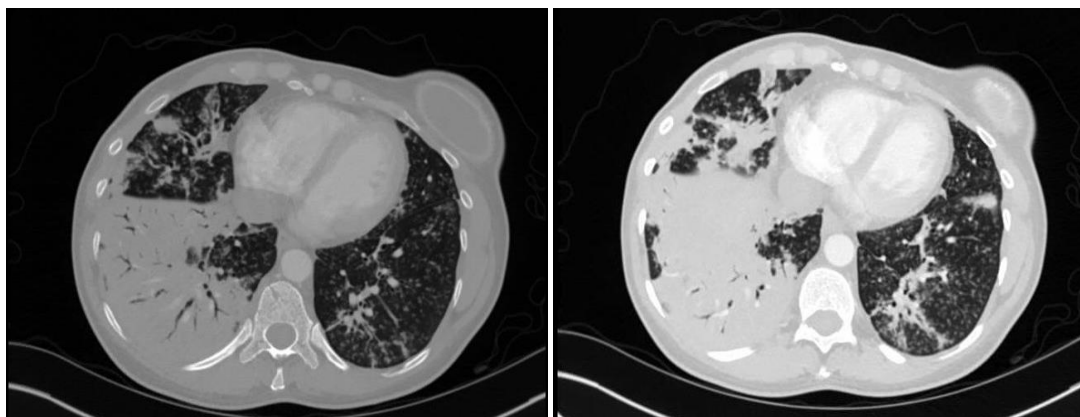


Figure 1: CT scan of the chest, axial view, with tree in bud opacities, bilateral opacities, and hilar lymphadenopathy.

She underwent a diagnostic bronchoscopy where the right and left lung lobes were examined to the level subsegmental bronchi and were unremarkable. Right middle lobe bronchoalveolar lavage was performed. After further discussion with our patient she reported cleaning bird droppings for one of her clients, she was unaware of the type of birds. During the subsequent days, the patient required about 2-3 L of oxygen at rest without tolerating wean. Cultures from bronchial lavage were negative for bacterial, fungal or mycobacterial infections. Cytology returned from bronchoalveolar lavage which revealed 29% eosinophils. The patient was started on oral prednisone 40 mg daily and reported symptomatic improved of breathlessness. She was discharged on 3L of oxygen at rest and exertion. She was also advised to avoid exposure to birds. On 5-month outpatient follow-up she had tapered off

systemic glucocorticoids with improvement of her symptoms. Repeat CT of the chest revealed resolution of the tree in bud opacities (Figure 2).

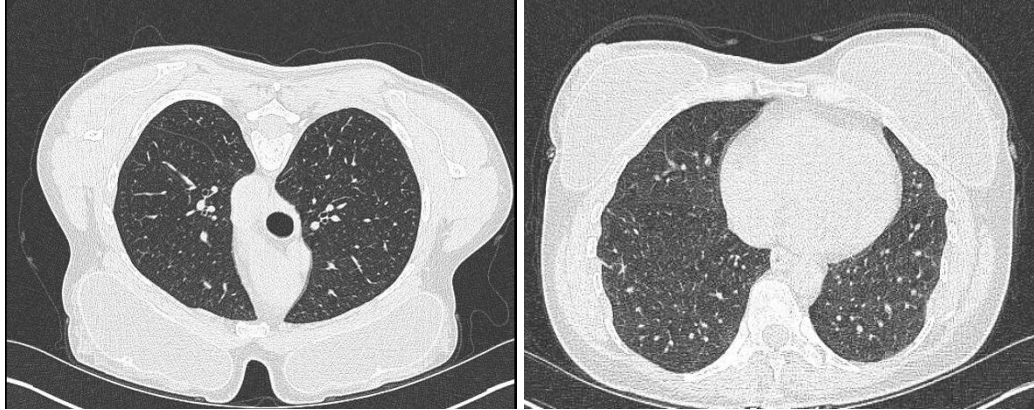


Figure 2: CT scan of the chest, axial view, with resolution of bilateral opacities after five months of glucocorticoid therapy.

3. Discussion

Acute eosinophilic pneumonia is a diagnostic dilemma as its etiologies often remain unidentified [1, 2]. There has been hypothesis that the underlying alveolar damage, interstitial eosinophilia, and vascular inflammation of the pulmonary parenchyma are the result of a hypersensitivity reaction by an inhaled insult [3]. This can include outdoor inhalational exposures, smoking, inhalation of cocaine and heroin [4]. A retrospective study of 22 patients was performed to evaluate the characteristics of acute eosinophilic pneumonia. Patients presented with hypoxemia, 14 requiring intubation with mechanical ventilation and all undergoing bronchoalveolar lavage which showed elevated eosinophils [5]. The diagnosis of acute eosinophilic pneumonia can be made in the absence of a lung biopsy if the patient presents with an acute febrile event, hypoxemia, non-specific opacities on chest imaging, and when other etiologies of eosinophilia are not present.

Our patient experienced acute new-onset hypoxic respiratory failure which failed to improve with antibiotics alone. After bronchoalveolar lavage returned for marked eosinophils this led us back to question our initial diagnosis and seek further history.

4. Conclusions

This case highlights the unfamiliarity in regards to potential etiologies of acute eosinophilic pneumonia, particularly avian exposure which is not explored in literature. When a patient presents with acute hypoxic respiratory failure with non-specific imaging, a thorough history should be undertaken.

Acknowledgements/Disclosures

No conflicts of interests to disclose.

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