Imaging of Eosinophilic Lung Diseases

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KEYWORDS

- Eosinophilic pneumonia Eosinophilic granulomatosis with polyangiitis
- Allergic bronchopulmonary aspergillosis
 Hypereosinophilic syndromes

KEY POINTS

- The classic "photographic negative of pulmonary edema" pattern is only seen in a minority of patients with chronic eosinophilic pneumonia.
- Simple pulmonary eosinophilia is characterized by peripheral eosinophilia, minimal respiratory symptom, and transient pulmonary opacities.
- Eosinophilic granulomatosis and polyangiitis, formerly known as Churg-Strauss syndrome, typically
 occur in patients with history of asthma, allergic rhinitis, and/or sinusitis.
- Hyperdense mucus is pathognomonic for allergic bronchopulmonary aspergillosis.
- Drug-induced eosinophilic pneumonia can have clinical presentations and imaging features similar to acute eosinophilic pneumonia, chronic eosinophilic pneumonia, or rarely, eosinophilic granulomatosis and polyangiitis.

INTRODUCTION

Eosinophilic lung diseases encompass a varied group of pulmonary diseases that characteristically feature peripheral or tissue eosinophilia. The clinical presentation of these disorders varies markedly, and patients may be asymptomatic or experience life-threatening respiratory illness at the time of diagnosis.¹

Eosinophilic lung disease traditionally can be diagnosed when one of the following criteria are met: (1) peripheral eosinophilia in the presence of opacities on a chest radiograph, (2) surgical or transbronchial lung biopsy demonstrating tissue eosinophilia, or (3) increase in the percentage of eosinophils in bronchoalveolar lavage (BAL) fluid.²

Imaging findings, particularly with thin-section computed tomography (CT), can sometimes

suggest the diagnosis of an eosinophilic lung disease. In many cases, given the relatively rare nature and nonspecific clinical presentation of these diseases, the findings on CT may be the first clue to the diagnosis and may prompt further diagnostic workup. Alternatively, in patients with known peripheral eosinophilia, imaging can provide information regarding presence, severity, and distribution of pulmonary involvement, potentially narrow the differential possibilities, and serve as a guide for BAL or biopsy.

IMAGING PROTOCOLS

Standard chest CT protocol is preferably with thin slice thickness (<1.5 mm) and high spatial resolution image reconstruction algorithm.

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DIAGNOSTIC CRITERIA

- Blood, tissue, or BAL fluid eosinophilia
- Pulmonary opacities

Acute Eosinophilic Pneumonia

The clinical diagnosis of acute eosinophilic pneumonia (AEP) is made in patients with an acute febrile illness lasting fewer than 5 days, hypoxemic respiratory failure, diffuse opacities on chest radiograph, and greater than 25% eosinophils in BAL fluid. Blood eosinophils may be normal or mildly elevated at initial presentation² but subsequently increase in the days after treatment.^{3,4} The absence of a concurrent infection is necessary for the diagnosis.^{2,5} Although the clinical presentation and imaging of findings of AEP may mimic acute respiratory distress syndrome (ARDS), the BAL fluid should demonstrate high neutrophils in ARDS.²

The cause of AEP has not been identified, but there is a reported association with cigarette smoking, particularly new-onset cigarette smoking.^{4,6-9} The illness has also been reported to occur after inhalation of toxins¹⁰ and use of certain medications.^{11,12}

The chest radiograph findings in patients with AEP vary and include bilateral septal thickening and patchy or diffuse opacities.^{4,13}

CT commonly shows ground-glass opacities, consolidation, interlobular septal thickening, bronchial wall thickening, and pleural effusions.¹⁴ A CT pattern of crazy-paving with thickening of the interlobular septa and intralobular lines in the setting of ground-glass opacities¹⁵ can be seen in patients with AEP¹⁴ (**Fig. 1**). When present, pleural effusions are most commonly bilateral. In most cases, there is no overall lung zone predominance in the cephalocaudal plane.^{14,16}

A peripheral distribution of the ground-glass and consolidative opacities has been described to occur in up to half of patients with AEP.¹⁴ Cardiomegaly is not a feature of the illness.¹⁷ In a minority of patients with AEP, lymphadenopathy may be observed at CT.¹⁶

AEP is extremely steroid responsive; however, there are patients who recover fully in the absence of corticosteroid treatment.¹⁸

Chronic Eosinophilic Pneumonia

Idiopathic chronic eosinophilic pneumonia (ICEP) is an uncommon entity with respiratory symptoms such as dyspnea and cough lasting more than 2 weeks. It is associated with alveolar eosinophilia 40% or greater at (BAL) differential cell count and/ or blood eosinophilia 1000/mm³ or more. Other known causes of eosinophilic lung disease must be excluded. The disease affects women twice as often as men. Up to 50% patients with ICEP have a history of asthma.¹⁹ In distinction from eosinophilic granulomatosis with polyangiitis (EGPA) or hypereosinophilic syndrome (HES), patients with ICEP usually do not have extrathoracic manifestations.¹⁹

The classic radiographic finding has been described as the photographic negative of pulmonary edema with diffuse peripheral opacities and ill-defined margins.^{20,21} However, this classic radiographic pattern is seen in fewer than one-third of patients.²² The opacities usually do not have lobar or segmental distribution, can be unilateral or bilateral, and are often in an apical or axillary location without basilar involvement. These opacities can disappear and reappear in the exact same locations.²⁰ Pleural effusion is not commonly seen.

CT often shows bilateral subpleural consolidation and ground-glass opacities and can be associated



Fig. 1. AEP in a 41-year-old male firefighter who presented with severe dyspnea 6 days following a significant episode of occupational smoke inhalation. BAL specimen showed a heavy eosinophilic infiltrate with 36% eosinophils. (A) Chest radiograph shows patchy linear and nodular opacities. (B) CT demonstrates patchy ground-glass opacities (*black arrow*), interlobular septal thickening (*white arrows*), and small bilateral pleural effusions.

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