Imaging Findings in Eosinophilic Lung Diseases

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Learning objectives

Eosinophilic lung diseases (ELD, Table 1 on page ) encompass a variety of rare clinical entities characterized by tissue and blood eosinophilia. The objective of this study is to present the spectrum of typical and atypical imaging manifestations in seven patients with blood eosinophilia (>500 cells/ml).

Background

The series includes seven patients (3 males, 4 females, range=28-72y.o), out of whom 4 were smokers (5-40pack/years). Patients presented with wheezing, productive or non-productive cough, fatigue, fever and shortness of breath. All patients were evaluated with blood and pulmonary function tests, chest radiograph and lung HRCT. Bronchoalveolar lavage (BAL) was performed in three patients and open lung biopsy in one.

Imaging findings OR Procedure details

Peripheral eosinophilia was found in all patients, while BAL disclosed a marked increase in eosinophil count (>20%). Surgical biopsy disclosed infiltrates of eosinophils in alveolar airspaces and interstitial tissue consistent with eosinophilic pneumonia.

HRCT disclosed subpleural and peribronchovascular patchy areas of consolidations and ground-glass opacity that had upper lung zone predominance in four patients (Figures 1, 2, 3, 4) associated with peribronchovascular nodules in five patients (Figures 2, 3, 5, 6, 7). A non-smoker post-menopausal woman disclosed numerous cysts with no zonal predominance associated with patchy ground-glass opacity and linear atelectasis (Figure 6). Bronchial wall thickening was present in two patients (Figures 1, 4).

Clinical symptoms and peripheral eosinophilia regressed or resolved in all patients, following corticosteroid treatment.

ELD are a diverse group of pulmonary disorders associated with peripheral or tissue eosinophilia. The defining diagnostic criteria of these disorders include one of the following: i) peripheral eosinophilia associated with pulmonary opacities, ii) pulmonary eosinophilia demonstrated on lung biopsy and iii) eosinophil (EOS) count >20% in BAL fluid. [1,2]

ELD may be either idiopathic or secondary to known underlying processes. Idiopathic ELD include simple pulmonary eosinophilia (SEP), acute eosinophilic pneumonia (AEP),
chronic eosinophilic pneumonia (CEP) and idiopathic hypereosinophilic syndrome (IHS). ELD of known etiology encompass allergic bronchopulmonary aspergillosis (ABPA), bronchocentric granulomatosis (BG), parasitic infections, drug-induced pulmonary eosinophilia, as well as eosinophilic vasculitis, namely allergic angiitis and Churg-Strauss syndrome (CSS). A large variety of pulmonary disorders may also be associated with occasional blood eosinophilia of a minor degree, including asthma, Pneumocystis jirovecii pneumonia and mycobacterial infections, rheumatoid disease, Wegener granulomatosis, idiopathic pulmonary fibrosis, Langerhans cell histiocytosis, non-small cell lung carcinoma and lymphoma. However these pathologic processes are not considered as ELD, in which tissue eosinophilia is by definition significant. [3]

Diagnostic approach of ELD requires a thorough medical history including duration and severity of symptoms, medication, chemical/occupational exposures and smoking habits. A history of asthma is mandatory for CSS and ABPA, though it is also present in about 50% of CEP cases. Traveling history is related to tropical ELD, whereas extrapulmonary manifestations characterize CSS and IHS. Blood eosinophilia, defined as eosinophil count >250 cells/ml is almost always present in ELD with the exception of AEP which may initially lack this feature. Serologic tests are important for the diagnosis of ABPA, parasitic infections and allergic angiitis. BAL fluid analysis invariably reveals increased EOS count >20% in ELD and sputum analysis may demonstrate eosinophil-derived Charcot-Leyden crystals. Pulmonary function tests may show either a restrictive pattern in AEP, CEP and parasitic ELD or an obstructive pattern in ABPA and CSS. Lung biopsy may be necessary to confirm the diagnosis of CSS and BG, though it is generally not required for the diagnosis of ABPA, IHS, drug or parasitic induced ELD. [3,4,5]

Patients with ELD present with lung opacities with or without pulmonary symptoms, accompanied by blood or tissue eosinophilia. Imaging findings in chest radiographs are generally nonspecific. Although lung HRCT demonstrates more characteristic findings, a considerable overlap exists among the various ELD and atypical appearances may also occur, thus precluding a final diagnosis to be based merely on imaging features. The common HRCT findings of SEP, CEP, ABPA, AEP and CSS are summarized in Table 2 on page . [3,6,7]

The radiographic manifestations of SEP (Figures 3, 4, 5) consist of transient and migratory nonsegmental consolidations with ill-defined margins and peripheral distribution that typically clear spontaneously within 1 month. HRCT reveals peripheral ground glass opacities (GGO) or consolidations with middle and upper lung zones predominance, as well as single or multiple nodules with surrounding GGO. [3,6,8]

Bilateral reticular densities or patchy consolidations and pleural effusions are the predominant radiographic findings in AEP. HRCT demonstrates bilateral patchy
conglomerations and GGO, frequently accompanied by interlobular septal thickening and poorly defined nodules. [3,9,10]

**CEP (Figures 1, 2, 7)** is manifested radiographically in less than 50% of the cases as peripheral consolidations with mid-upper zone predominance, which has been described as the "photographic negative" of pulmonary edema. HRCT typically demonstrates peripheral nonsegmental consolidations and occasionally GGO, nodules and reticular opacities. Linear band like opacities paralleling the pleural surface may be seen in CT scans performed more than 2 months after the onset of symptoms. Pleural effusion is observed in less than 10% of cases. [3,6,11]

Radiographic findings of **IHS** consist mainly of focal or diffuse interstitial or alveolar opacities, with most of them being related to severe cardiac failure. HRCT shows nodules with or without a surrounding ground glass halo and focal or diffuse areas of GGO. [3,12,13]

**ABPA (Figures 8, 9)** may demonstrate transient, pulmonary opacities in chest radiographs or homogenous, tubular, gloved-finger areas which affect mainly the upper and central lung zones and are related to mucoid impaction. Lobar or segmental atelectasis is not uncommon. CT findings in ABPA are basically related to mucoid impaction and manifest as bronchiectasis as well as centrilobular nodules or branching linear densities. In about 30% of patients, the impacted mucus is highly opaque or demonstrates frank calcification. [3,7,14,15]

**BG and drug related ELD (Figure 10)** generally lack specific imaging findings and may present as non-segmental consolidations, GGO, nodules and irregular linear densities, affecting mainly the lung periphery. In about 30% of BG, imaging findings resemble ABPA. [3,16,17]

**Parasitic ELD** show a wide spectrum of imaging findings ranging from an SEP-like pattern (Loeffler Syndrome) seen with Ascaris lumbricoides infection [1,3] to reticulonodular opacities and small/medium sized nodules with surrounding GGO halo seen in Wuchereria bancrofti infection and schistosomiasis. [1,3,18,19] Moreover, schistosomiasis may induce obliterator arteriolitis and pulmonary hypertension. Pleuropulmonary paragonimiasis (PP) (Figure 11) manifestations include focal airspace consolidations or GGO and linear opacities, while lasting disease may result in thin-walled cysts, mass like consolidations, nodules and bronchiectasis. The nodules in PP are usually subpleural and poorly marginated and may be associated with adjacent focal pleural thickening, pleural effusion or pneumothorax. PP may mimic lung cancer both in HRCT and positron emission tomography (PET) imaging. [3,20,21]
CSS (Figure 12) usually appears as bilateral nonsegmental consolidations or reticulonodular opacities on radiographs. Common features in HRCT include subpleural GGO or consolidation with a lobular distribution, centrilobular nodules, bronchial wall thickening and interlobular septal thickening. Occasionally, hilar lymphadenopathy and pleural or pericardial effusion may be present. [3, 22, 23]

Conclusion

HRCT is an indispensable part in the investigation of blood eosinophilia. Eosinophilic lung diseases may manifest with a variety of imaging findings. Integration of clinical, laboratory and HRCT findings facilitates prompt diagnosis and patient management.

Personal Information

References