Peripheral Lung Infiltrates in a Young Woman*

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A 40-year-old woman was referred for evaluation of fever, cough, and chest pain. The patient had been entirely well until four months prior to presentation, when she developed fevers, left-sided chest pain, and a cough productive of minimal amounts of rust-colored sputum. The sputum production cleared after two days, but the fever persisted. A chest roentgenogram demonstrated a “ground-glass” lingular infiltrate.

Empiric antibiotic therapy with erythromycin followed by a cephalosporin failed to achieve complete resolution of the patient’s symptoms. Two weeks after discontinuation of the antibiotics, her cough worsened, and she was seen by a pulmonologist; at this time the lingular infiltrate had resolved, but an ill-defined density was seen in the left lower lobe. She received another course of erythromycin, but reported no improvement in symptoms. Laboratory data at that time revealed an elevated erythrocyte sedimentation rate (ESR) and peripheral blood eosinophilia of 18 percent. Physical examination demonstrated wheezing. She received some benefit from prednisone started at a dose of 10 mg/d, but her symptoms returned shortly after the drug dosage was tapered three weeks later. Her wheezing and cough then responded to inhaled beta-agonists and corticosteroids, but her malaise was so profound that she could not continue to work.

The patient’s medical history revealed no childhood pneumonia or asthma. She never smoked tobacco or used drugs. A recent PPD test was negative, and she denied exposure to tuberculosis. There was no significant travel or occupational exposure history.

Physical examination was unremarkable. Laboratory studies revealed an ESR of 40 mm/h (normal, 0 to 20 mm/h), an absolute eosinophil count of 1,288/µl (normal, <480/µl), and a total IgE level of 272 IU/ml (normal, 10 to 150 IU/ml). Pulmonary function tests and arterial blood gas values were normal. A chest roentgenogram was obtained (Fig 1).

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**Diagnosis: Chronic eosinophilic pneumonia**

Chronic eosinophilic pneumonia (CEP) is an idiopathic pulmonary eosinophilic syndrome characterized clinically by symptoms of cough, fever, dyspnea, and malaise and histologically by an intra-alveolar exudate of eosinophils and histiocytes. Up to 40 percent of patients with CEP develop wheezing, many with no prior history of asthma. Physical findings include crackles more frequently than wheezes. Laboratory data are nonspecific, but leukocytosis with eosinophilia and an elevated ESR are supportive. Sputum eosinophilia is present in fewer than half of all cases of CEP, but bronchoalveolar lavage fluid commonly has a marked increase in the percentage of eosinophils. Pulmonary function tests most frequently demonstrate a gas exchange impairment with a widened alveolar-arterial oxygen gradient; it is notable that the patient presented here did not manifest abnormalities of gas exchange at the time of physiologic assessment. Spirometry reveals reduced forced vital capacity and FEV₁ in a large fraction of patients. Restrictive, obstructive, and mixed patterns may be seen in patients with spirometric abnormalities.

The chest roentgenogram most commonly demonstrates a peripheral infiltrate, but the "photographic negative of pulmonary edema" pattern was found in only one fourth of cases of CEP in a recent review by Jederlinic and colleagues. Our patient demonstrated this classic but far from invariable finding, at least in the left hemithorax (Fig 1). Jederlinic et al also noted that the radiologic infiltrates were not migratory as reported in some earlier studies, but rather progressed and regressed in one area. The chest roentgenogram is often supportive of the suspected diagnosis of CEP, but in cases in which the clinical picture or the chest roentgenographic findings are not consistent, high-resolution computed tomography (HRCT) is very helpful in identifying the peripheral location of the infiltrates. Infiltrates may also be identified on HRCT in areas of the lungs that appear spared on chest roentgenogram (Fig 2).

After treatment with corticosteroids, the infiltrates may appear completely resolved on chest roentgenogram but remain on HRCT (Fig 2). This helps explain why patients with chronic eosinophilic pneumonia need prolonged therapy with corticosteroids despite rapid improvement in symptoms upon the initiation of treatment. In the review by Jederlinic and colleagues, 80 percent of patients had a short-term relapse, and many of those were still receiving tapering doses of corticosteroids at the time of relapse.

Some authors feel that the clinical criteria of peripheral pulmonary infiltrates, peripheral blood eosinophilia, and negative cultures for infectious organisms obviate a histologic diagnosis, and recommend a three- to five-day therapeutic trial of corticosteroids for the presumptive diagnosis of CEP. A prompt response to prednisone given at a dose of 40 to 60 mg daily argues strongly for CEP. Others feel, however, that the differential diagnosis of peripheral infiltrates on chest roentgenogram is broad, including bronchiolitis obliterans organizing pneumonia, other eosinophilic pneumonias, and sarcoidosis, and therefore feel that lung biopsy is more appropriate than the therapeutic trial of corticosteroids. Although there are differences in the management of CEP and these other disorders, it is unlikely that the brief trial of corticosteroids would provide the dramatic response that it does in CEP, making lung biopsy necessary when the trial fails anyway. In all cases of suspected CEP, the clinician is obligated to exclude the secondary lung eosinophilias, specifically pulmonary hypersensitivity reactions to either drugs or parasitic infestations.

**References**