*Brief Report*

# Eosinophilic Pleural Effusion: Is It Always Nondiagnostic?

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The presence of eosinophils in the pleural effusion is generally considered nondiagnostic. It usually indicates that the patient has had a previous thoracentesis and that air or blood has come in contact with the effusion. Idiopathic acute eosinophilic pneumonia is characterized by acute onset of pulmonary symptoms with hypoxemia, pulmonary infiltrates, eosinophils in bronchoalveolar lavage fluid, and prompt response to steroid therapy. We report a patient who presented with symptoms of acute pneumonia in which the presence of increased eosinophils in the pleural effusion indicated eosinophilic pneumonia.

*Key words.* Eosinophils; pleural effusion; pulmonary eosinophilia; bronchoalveolar lavage fluid.
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The presence of eosinophils in the pleural fluid is generally considered to be of little diagnostic value.[1–4] Eosinophils in the pleural fluid usually indicate that air or blood has leaked into the effusion from a previous thoracentesis.[2,5] We describe a patient with pleural fluid eosinophilia.

## Case Report

A 22-year-old white woman presented to the emergency department with a 1-day history of shortness of breath, fever, dry cough, myalgias, and pleuritic chest pain. She was a nonsmoker and denied alcohol, illicit drugs, or any change in her habits or environmental exposures. She had a temperature of 104°F, respiratory rate of 28 breaths per minute, pulse rate of 128 beats per minute, and blood pressure of 118/72 mm Hg. Examination of her chest revealed dullness to percussion and diminished breath sounds over the right lung base and bilateral inspiratory crackles. Chest roentgenogram demonstrated diffuse bilateral alveolar and interstitial infiltrates and a possible small right-sided pleural effusion (Figure 1). Laboratory data revealed an elevated white cell count of 29,900/$mm^3$ with 70% segmented neutrophils, 1% band neutrophils, 7% lymphocytes, 6% monocytes, and 1% eosinophils. Arterial blood gas analysis on room air was pH 7.47, $Paco_2$ 34 mm Hg, $Pao_2$ 59 mm Hg, and $O_2$ saturation 91%.

Treatment was begun with supplemental oxygen and intravenous (IV) erythromycin and cefuroxime for presumed community-acquired pneumonia. The patient's temperature continued to spike to 104°F during the next 2 days. A high-resolution computed tomographic (CT) scan was obtained to evaluate for the possibility of a complicated parapneumonic pleural effusion. The CT scan confirmed diffuse patchy alveolar infiltrates and small bilateral pleural effusions (Figure 2). On the third hospital day, a diagnostic thoracentesis was done. Pleural fluid examination revealed a pH 7.57, pleural fluid protein to serum protein ratio of 0.7, lactate dehydrogenase ratio 0.7, and pleural fluid glucose of 65 mg/dL. The white cell count was 3640/$mm^3$ with 19% eosinophils, 17% neutrophils, 43% macrophages, and 15% lymphocytes. A bronchoscopy was performed because of the possibility of eosinophilic pneumonia causing eosinophilic effusion. Bronchoalveolar lavage fluid yielded a total cell count of 970/mL, with 40% eosinophils, 28% macrophages, 20% lymphocytes, and 4% neutrophils (Figure 3). Pulmonary function tests showed forced vital capacity (FVC) of 3.19 L (69% predicted), forced expiratory volume in 1 second ($FEV_1$) of 2.48 L (67% predicted), $FEV_1$/FVC 78%, total lung capacity of 4.13 L (71% predicted), and diffusion capacity of 69% predicted. All bacterial, viral, and fungal

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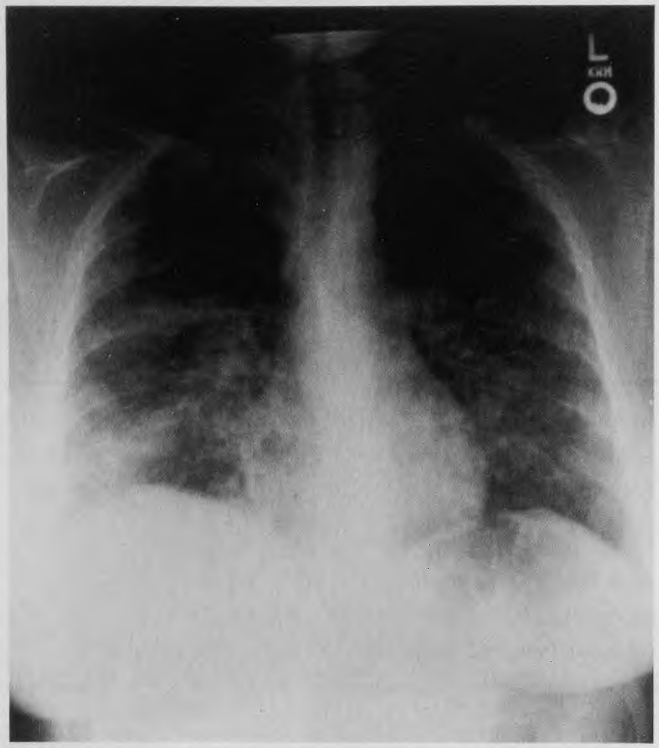

Figure 1. Admission posterior-anterior chest roentgenogram showing bilateral diffuse air space and interstitial infiltrates with a small right-sided pleural effusion.

cultures and stains from bronchoalveolar lavage fluid, pleural fluid, and blood were negative. The patient was started on IV corticosteroid therapy (methylprednisolone 60 mg IV every 6 hours) with prompt resolution of fever and improvement in gas exchange and clearance of chest

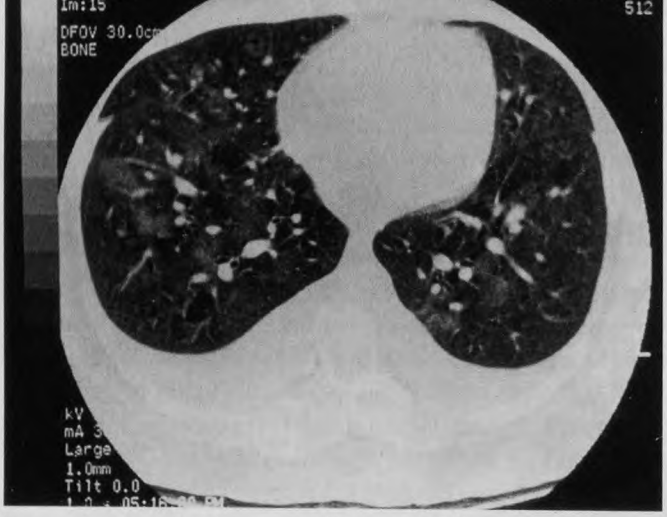


Figure 2. Computed tomographic scan of the chest with contrast showing diffuse patchy interstitial infiltrates and small bilateral pleural effusions.

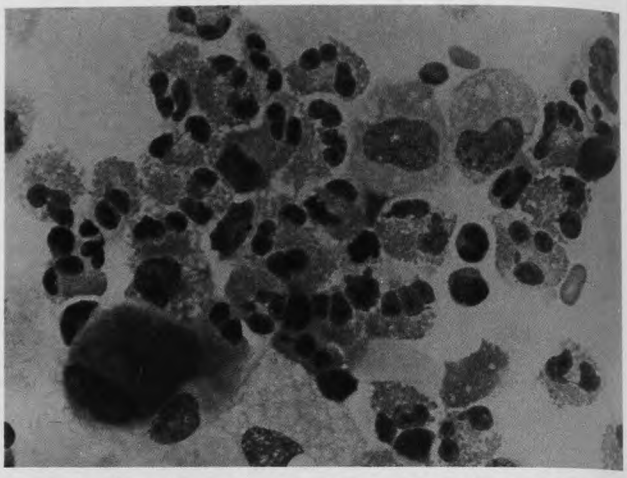

Figure 3. Bronchoalveolar lavage cytocentrifuge preparation stained with modified Wright-Giemsa showing sheets of eosinophils. The differential count showed 40% eosinophils.

roentgenographic abnormalities. Prednisone was tapered over 6 weeks, and the patient has continued to do well during 4 months of follow-up without evidence of recurrent disease.

## Discussion

Idiopathic acute eosinophilic pneumonia in the setting of acute respiratory failure was first described in two cases in 1986.[6] Idiopathic acute eosinophilic pneumonia has been well characterized in patients with acute onset of pulmonary symptoms with severe hypoxemia, diffuse pulmonary infiltrates without any evidence of infection, history of asthma, or atopy. The diagnosis is confirmed by the presence of eosinophilia on bronchoalveolar lavage or lung biopsy. Peripheral blood eosinophilia is characteristically absent in patients with acute eosinophilic pneumonia. Normally, eosinophil counts in bronchoalveolar lavage fluid are less than 1%; however, in most series of acute eosinophilic pneumonia, the bronchoalveolar lavage eosinophil count ranges from 20% to 80%. A bronchoalveolar lavage eosinophil count over 20% usually can be considered diagnostic in the appropriate clinical setting. Pulmonary function studies in the acute phase typically reveal a restrictive pattern and a reduced diffusing capacity, as in our patient.[11] The signs and symptoms resolve rapidly with corticosteroids without evidence of recurrence when steroids are discontinued.[7–10]

Other causes of eosinophilia in bronchoalveolar lavage fluid include simple pulmonary eosinophilia (Löffler's syndrome), hypersensitivity reactions to drugs, parasitic infections, chronic eosinophilic pneumonia, asthma, allergic bronchopulmonary aspergillosis, and Churg-Strauss syndrome.

Pleural fluid eosinophilia has been considered a nondiagnostic finding most commonly representing foreign body (air or blood), benign asbestos effusion, Hodgkin's disease, drug reactions, or parasitic infection within the pleural space.[2,4,5] It has been suggested that in the absence of parasitic infection, pneumothorax, or pulmonary embolus, the workup of pleural fluid eosinophilia should be nonaggressive.[5] The cause of 25% of eosinophilic effusions is unclear.[2] Pleural fluid eosinophilia is a good prognostic indicator because these effusions rarely become infected.[2]

Although acute eosinophilic pneumonia is commonly associated with small pleural effusions, thoracentesis is rarely performed. Only two cases have been previously reported in the literature.[1,11] Greenburg et al[1] described a patient with large bilateral pleural effusions with pleural fluid eosinophilia in the setting of hypoxemic respiratory failure. Ogawa et al[11] described a series of five patients with acute eosinophilic pneumonia. All patients had small pleural effusions; diagnostic thoracentesis was obtained from one of the patients, documenting pleural fluid eosinophilia. An elevated pH is associated with pleural fluid eosinophilia and is consistent with release of basic eosinophil granule contents.[9]

Our case suggests that pleural fluid eosinophilia is a manifestation of acute eosinophilic pneumonia. Since the steroid therapy is associated with a dramatic response, the presence of eosinophils in the pleural fluid should direct the physician to bronchoalveolar lavage to confirm the diagnosis.

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