Dyspnea and chronic bronchitis in a man with ulcerative colitis

January 01, 2007 | Pneumonia [1], Respiratory Diseases [2], Infection [3], Obesity [4], Asthma [5] 
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The authors report a case of idiopathic eosinophilic pneumonia in a man with sclerosing cholangitis and ulcerative colitis associated with features of sarcoidosis.

The case
A 44-year-old man was referred to the pulmonary clinic for evaluation of worsening dyspnea, recurrent episodes of bronchitis, and copious sputum production over the past 3 months. He also reported low-grade fever and weight loss. The patient was a lifelong nonsmoker. His medical history was significant for ulcerative colitis diagnosed 25 years earlier. Because of severe pancolitis, total colectomy for definitive treatment of ulcerative colitis was done 6 years before presentation. During the course of ulcerative colitis, sclerosing cholangitis developed (thought to be secondary to the colitis), resulting in cirrhosis. He did not have other extraintestinal signs of ulcerative colitis, such as uveitis or skin or musculoskeletal manifestations.

The patient was evaluated for possible liver transplantation. His medications included esomeprazole and ferrous sulfate. Physical examination demonstrated a temperature of 36.7ºC (98ºF), heart rate of 88 beats per minute, blood pressure of 100/60 mm Hg, respiration rate of 16 breaths per minute, and oxygen saturation of 98% on room air. Lung auscultation revealed scattered crackles. Findings from cardiac and abdominal examinations were unremarkable.

A chest radiograph showed diffuse micronodular opacities and airspace disease in the right middle lung zone. A CT scan of the chest revealed small cavitory nodules, peripheral patchy infiltrates, and interlobular septal thickening (Figure 1). Diffuse mediastinal and hilar lymphadenopathy was also noted (Figure 2).

Results of pulmonary function tests showed forced expiratory volume in 1 second (FEV₁) of 1.89 L (44% of predicted), forced vital capacity (FVC) of 2.90 L (58% of predicted), FEV₁:FVC ratio of 65%, and single-breath carbon monoxide- diffusing capacity (DlCO) of 17 mL/min/mm Hg (61% of predicted). Laboratory findings are summarized in the Table.

Bronchoscopy with bronchoalveolar lavage (BAL) and transbronchial biopsy was performed. The BAL differential cell count showed 30% macrophages, 34% neutrophils, 32% eosinophils, and 4% lymphocytes (Figure 3A). The BAL cytologic examination results were negative for fungal elements, Pneumocystis jiroveci, and viral cytopathic changes. BAL cultures were negative for bacteria (including Legionella), viruses, mycobacteria, and fungi.

A transbronchial biopsy specimen showed noncaseating gran- ulomatous inflammation of lung parenchyma, with infiltration of eosinophils in surrounding lung parenchyma (Figure 3B). Special stains were negative for pathogenic organisms. Tests of 3 stool samples for parasitic ova and cysts yielded negative results.

The diagnosis of chronic eosinophilic pneumonia was made. Therapy with oral prednisone, 30 mg/d, was started. After 2 weeks, the patient's dyspnea and cough improved markedly. A repeated CT scan of the chest after 1 month of prednisone therapy showed complete resolution of pulmonary infiltrates (Figure 4).

At follow-up 2 weeks later, a complete blood cell count showed a white blood cell count of 8.7 3 10^3/L with 2.2% eosinophils. The corticosteroid dosage was gradually reduced. One year after presentation, the patient was asymptomatic and continued taking 5 mg of prednisone a day; his chest radiograph and DICO were stable.

Discussion
This case report describes an unusual presentation of pulmonary disease in a patient with a history of ulcerative colitis. Hallmarks of this disease are peripheral and pulmonary eosinophilia and noncaseating granuloma on lung biopsy. Noncaseating pulmonary granulomas are characteristic of sarcoidosis.
Although the association of sarcoidosis and ulcerative colitis has been described in the literature,\(^1\text{-}^3\) marked peripheral and alveolar eosinophilia are highly atypical for sarcoidosis.\(^4\text{-}^6\) Pulmonary eosinophilia and granulomas can be seen in allergic bronchopulmonary aspergillosis (ABPA) and Churg-Strauss syndrome. The lack of history of asthma, normal serum IgE level, negative skin reaction to *Aspergillus*, normal erythrocyte sedimentation rate, and absence of bronchiectasis on a chest CT scan make the diagnosis of ABPA and Churg-Strauss syndrome unlikely.\(^7\)

Certain medications are common causes of pulmonary eosinophilia. Sulfasalazine lung toxicity with eosinophilia and pulmonary infiltrates is well recognized. Our patient had not been taking sulfasalazine since his total colectomy 6 years earlier. On presentation, he was being treated with esomeprazole and ferrous sulfate, neither of which is associated with pulmonary eosinophilia.\(^8\text{-}^9\) Parasitic, fungal, and mycobacterial infections were excluded.

Peripheral and alveolar eosinophilia is consistent with a diagnosis of chronic eosinophilic pneumonia. Fever, weight loss, and productive cough are also characteristic of this disease. Peripheral infiltrates on a chest CT scan, rapid resolution of pulmonary infiltrates with corticosteroid therapy, and no evidence of other causes of alveolar eosinophilia strongly suggested that chronic eosinophilic pneumonia was the main cause of the patient's pulmonary disease.

Noncaseating granulomas characteristic of sarcoidosis rarely have been reported in patients who have chronic eosinophilic pneumonia.\(^10\text{-}^11\) We have seen only 1 other reported case in the literature in a patient with ulcerative colitis. BAL fluid interleukin (IL)-4 and IL-5 and serum rheumatoid factor levels have been reported to correlate with disease activity. In our patient, the level of rheumatoid factor was normal; we did not measure IL-4 or IL-5 in BAL fluid. In this setting, rapid and complete response to corticosteroids is characteristic.\(^10\text{-}^11\)

We believe that our patient had chronic eosinophilic pneumonia concomitant with sarcoidosis. The association of ulcerative colitis with sarcoidosis and the association of sarcoidosis with chronic eosinophilic pneumonia have been suggested in previous case reports. Our case, however, presents features of all 3 diseases in 1 patient. An unknown common pathophysiologic pathway may be responsible.

References:

**REFERENCES**


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