



A 63-Year-Old Man With a Persistent Pulmonary Infiltrate and Pleural Effusion*

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CASE REPORT

A 63-year-old man was referred for evaluation of a nonresolving pneumonia. Six weeks prior to referral, he was seen by his internist with complaints of a nonproductive cough, shortness of breath, and subjective fevers and chills. He had lost 20 lb since the onset of symptoms, and had increasing stiffness and pain in both hands over this same time period. A chest radiograph revealed a right, peripheral lower lobe infiltrate, and he was treated with a 10-day course of gatifloxacin. His symptoms persisted despite treatment. Ceftriaxone was added, and he was continued on gatifloxacin for an additional 14 days. Two weeks prior to referral, symptoms continued and night sweats and right-sided pleuritic chest pain developed. A repeat chest radiograph revealed progression of the right lower lobe infiltrate and a small right-sided pleural effusion. He was placed on clindamycin and prednisone, 20 mg bid, and referred for additional evaluation.

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The patient's medical history was notable for hypertension and well-controlled diabetes mellitus. Medications included enalapril, hydrochlorothiazide, and glipizide. He had a 40-pack-year smoking history, although he quit 8 years ago.

On presentation, the patient had a nonproductive cough. Temperature was 37.2°C, and respiratory rate was 18 breaths/min with normal oxygen saturation. Inspiratory crackles were noted in the right mid-lung field, with dullness to percussion at the right lung base. His joints were without deformities or synovitis. Treatment with steroids was discontinued.

Notable laboratory results included: peripheral WBC count, 12.9/ μ L (neutrophils, 76%; lymphocytes, 14%; monocytes, 9%; eosinophils, 1%); hematocrit, 33%; platelets, 699/ μ L; creatinine, 1.0 mg/dL; total protein, 6.4 g/dL; and albumin, 2.6 g/dL. Urinalysis was normal. A rheumatologic evaluation yielded a Westergren sedimentation rate of 120 mm/h; C-reactive protein of 106 mg/L (normal range, 0 to 8 mg/L); negative anti-neutrophil cytoplasmic antibody panel; positive antinuclear antibody (titer of 1:320 with a speckled pattern); negative anti-double strand DNA; positive serum rheumatoid factor with a titer of 320 IU/mL (normal range, 0 to 39.9 IU/mL); and positive anti-cyclic citrullinated peptide (titer > 200 U). Routine sputum Gram stain and culture results were negative. Several days after corticosteroids were discontinued, significant morning stiffness of the hands, wrists, and elbows developed, and swelling and tenderness of the wrists and metacarpal-phalangeal joints occurred. The peripheral eosinophil count had increased to 1,090/ μ L (total peripheral WBC count, 12.2/ μ L; eosinophils, 9%).

Chest radiography revealed consolidation of the right middle and lower lobes, with a wedge-like distribution in the lateral aspect of the middle lobe (Fig 1). CT angiogram of the chest revealed no

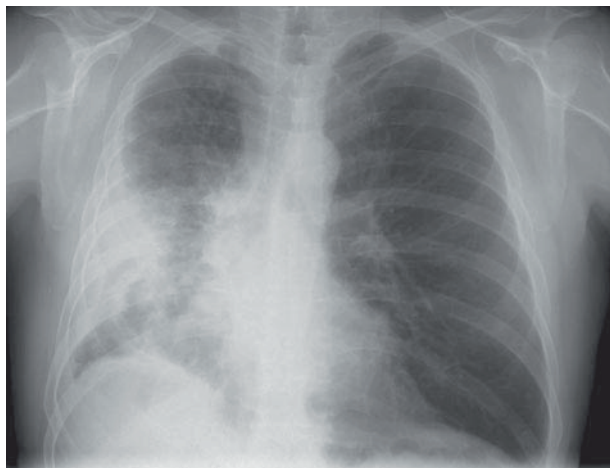


FIGURE 1. Chest radiograph demonstrating consolidative changes in the right middle and lower lobes, with a wedge-like distribution in the lateral aspect of the right lung field.

pulmonary emboli, and demonstrated consolidation with air bronchograms in the right middle and lower lobes and a moderate right pleural effusion (Fig 2). Bilateral hand and wrist radiographs revealed peri-articular osteopenia and subtle, marginal erosions at the distal-interphalangeal joints suggestive of an inflammatory arthritis.

Thoracentesis of the pleural fluid revealed: pH 7.37; lactate dehydrogenase, 109 IU/L (serum normal range, 100 to 175 IU/L); total protein, 3.5 mg/dL; glucose, 297 mg/dL; WBC count, 133 cells/

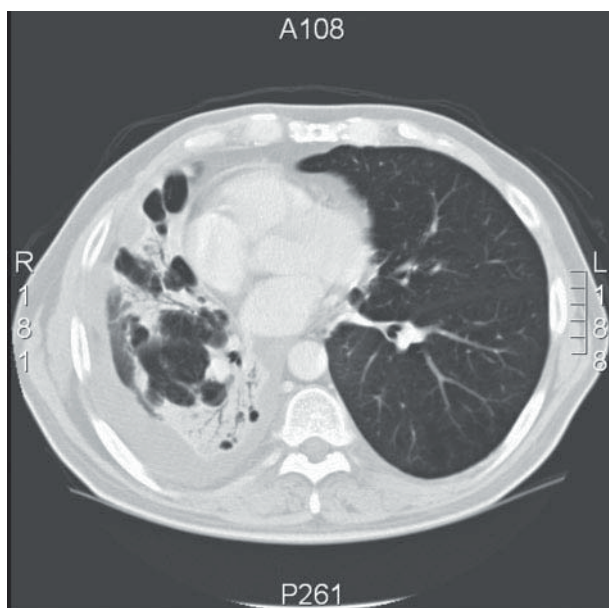


FIGURE 2. CT scan of the thorax demonstrating right middle and lower lobe peripherally based consolidation with a moderate-sized pleural effusion.

μL ; and WBC count differential, 11% neutrophils and 50% eosinophils. Pleural fluid culture results were negative. Bronchoscopy did not reveal any bronchial lesions. The BAL fluid culture findings (for bacteria, fungus, and acid-fast bacilli) and cytology were negative. The cell differential on the BAL fluid revealed: neutrophils, 95%; lymphocytes, 4%; and large mononuclear cells, 1%.

DISCUSSION OF THE CASE

What Is the Cause of This Patient's Inflammatory Arthritis?

Although this patient initially presented with pulmonary symptoms, further examination (after corticosteroids were discontinued) revealed an acute, symmetric inflammatory process that involved his hands, wrists, and elbows. After further discussion of these symptoms with the patient, he stated that he had experienced mild morning stiffness, and pain in both hands several months prior to his presentation. In addition to clinical symptoms, laboratory data revealed a high-titer rheumatoid factor and a high-titer anti-cyclic citrullinated peptide, and hand and wrist radiographs that were consistent with an inflammatory arthritis. This constellation of findings established the diagnosis of rheumatoid arthritis.¹

In a Patient With Rheumatoid Arthritis and a Unilateral Middle- And Lower-Lobe Infiltrate With an Associated Pleural Effusion, What Pulmonary Diseases Are Included in the Differential Diagnosis?

Rheumatoid arthritis is a chronic, systemic, inflammatory disease that typically affects the diarthrodial joints. Extra-articular disease is common and may be present in as many as 75% of patients with severe rheumatoid arthritis.² A number of pleuropulmonary manifestations have been associated with this disease, including interstitial lung disease (usual interstitial pneumonia, nonspecific interstitial pneumonia, lymphocytic interstitial pneumonia, organizing pneumonia, and chronic eosinophilic pneumonia), airway disease (bronchiectasis, bronchiolitis obliterans, follicular bronchiolitis, and diffuse panbronchiolitis), rheumatoid nodules, pulmonary vascular disease, and pleural disease (pleuritis), as well as drug-induced lung disease.² One study³ of patients with rheumatoid arthritis and joint disease of < 2 years in duration reported that 58% of patients had findings consistent with interstitial lung disease, and only male gender was statistically associated with clinically significant disease.

While there are a number of pleuropulmonary

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