Dyspnea and Hemoptysis

CASE PRESENTATION

An 83-year-old white woman presented with dyspnea on exertion, nonproductive cough, hemoptysis, and fatigue. Past medical history included diastolic heart failure, mitral regurgitation, coronary artery disease, paroxysmal atrial fibrillation, a pacemaker, hyperlipidemia, hypothyroidism, and hypertension. Medications included alprazolam, amiodarone 200 mg, amlodipine, aspirin, atenolol, furosemide, isosorbide mononitrate, levothyroxine, ramipril, pravastatin, and warfarin. Surgical history was unremarkable. Family history was positive for coronary artery disease. Social history included a 45 pack year smoking history (she quit 15 years ago), 2 vodka tonics nightly, and no illegal drug use.

Physical Assessment

Here vital signs were as follows: blood pressure = 116/60 mm Hg, pulse = 73 beats/min, respirations = 16/min, afebrile, oxygen saturation of 94% on room air, and body mass index = 24.2. The patient was in no acute distress. Head, neck, and neurologic examinations were unremarkable. Her lungs had bibasilar crackles with nonlabored respirations. A cardiac examination revealed normal rate and rhythm, 1/6 apical systolic murmur, and no jugular venous distention. Her abdomen was nontender without masses, bruits, or hepatosplenomegaly. Her extremities revealed trace ankle edema with normal pulses.

DIAGNOSTICS

Laboratory Values and Radiology

Brain natriuretic peptide and serum creatinine were mildly elevated. A complete blood count and iron panel revealed iron deficiency anemia. Thyroid stimulating hormone, electrolytes, and prothrombin time were normal.

Echocardiogram

An echocardiogram to evaluate for atrial fibrillation revealed sinus rhythm, rate of 61, and a mildly prolonged QTc. An echocardiogram to evaluate heart failure status revealed normal systolic function, stage III-IV diastolic dysfunction, moderate mitral regurgitation, severe biatrial enlargement, and mild tricuspid regurgitation.

Chest X-ray

A chest x-ray (CXR), ordered because of her continued dyspnea, revealed new infiltrates in the right middle and upper lobes and left lower lobe (Figure 1).

IMAGE OF THE MONTH

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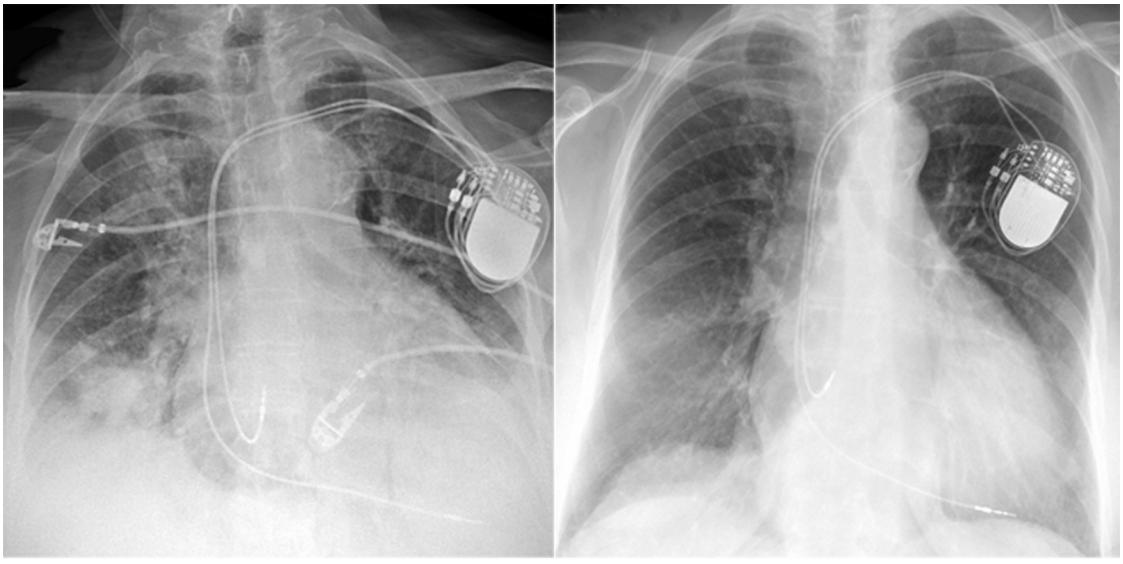
Pulmonary Function Studies

This study was ordered because of symptoms and amiodarone use for 4 years. It revealed normal flow rates, total lung volume, and forced vital capacity. However, the forced expiratory volume in 1 second/forced vital capacity ratio dropped from 71% to 32%, and the diffusing capacity worsened from moderate to severe over one year.

Bronchoscopy and Chest Computed Tomographic Scan

Because of the results of her CXR and pulmonary function study (PFT), amiodarone was discontinued, and a pulmonary referral was made. A chest computed tomographic (CT) scan revealed bilateral, diffuse, mixed interstitial and alveolar infiltrates in the right lower lobe, and tiny pleural effusions (Figure 2). Bronchoscopy revealed inflammatory changes but no malignancy or infection. Her laboratory values ruled out connective tissue disorders.

Figure 1. (Left) At the time of diagnosis, there were infiltrates in the right middle and upper lobes and the left lower lobe. (Right) Two months later, after prednisone and withdrawal of amiodarone, there is remarkable clearing of the infiltrates.



Cultures ruled out bacterial, viral, and fungal pneumonia. Lung biopsy revealed bronchiolitis obliterans organizing pneumonia.

Etiology of Amiodarone-induced Pulmonary Toxicity

Amiodarone is a class III antiarrhythmic recommended for ventricular fibrillation and unstable ventricular tachycardia.[1,2] Because of its efficacy in maintaining sinus rhythm, it is commonly used in atrial fibrillation,[1] but this is not a Food and Drug Administration—approved use.[3] Amiodarone-induced pulmonary toxicity (AIPT) often manifests as bronchiolitis obliterans organizing pneumonia, also known as cryptogenic organizing pneumonia. The etiology is an inflammatory process with connective tissue deposition in the distal bronchioles and alveoli, creating an organizing pneumonia.[4,5] Because the etiology is inflammatory, it is typically reversed with drug discontinuation and corticosteroids.[6] Its incidence is 0% to 10%, and the mortality rate is 9% (chronic), 20% to 30% (hospital admission), and up to 50% in cases developing acute respiratory distress syndrome.[4,5]

DIAGNOSIS

Clinical Presentation

Common symptoms include dyspnea on exertion, a nonproductive cough, rare hemoptysis, a low-grade fever, weight loss, malaise, bibasilar rales, and pleuritic chest discomfort. Acute cases

Figure 2. The chest CT scan revealed bilateral, diffuse, mixed interstitial and alveolar infiltrates in the right > left lower lobe caused by amiodarone toxicity (1-4), pacemaker wires (5), and breast implants (6).