Most reports of amiodarone lung toxicity have emphasized the associated fibrosing alveolitis, and the drug information package insert does not address pleural involvement as an adverse effect of amiodarone. Our report illustrates an unusual pleuropulmonary manifestation of amiodarone toxicity that should alert clinicians, as this association has previously not been sufficiently emphasized and could be mistaken for exacerbation of congestive heart failure.

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Reversible Valvulitis in Wegener’s Granulomatosis*

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A patient with Wegener’s granulomatosis sustained a myocardial infarction. Subsequent echocardiography showed aortic valvulitis, which resolved with therapy with cyclophosphamide and prednisone.

Wegener’s granulomatosis is characterized by granulomatous vasculitis of the upper and lower respiratory tract, by disseminated vasculitis, and by necrotizing glomerulonephritis. Clinical involvement of the heart is infrequent and usually takes the form of pericarditis. Valvular involvement has not been diagnosed before death but has been described in postmortem reports. The following case report describes a patient with Wegener’s granulomatosis in whom echocardiography revealed aortic valvulitis which resolved with medical therapy.

CASE REPORT

A 20-year-old man had a six-month history of nasal congestion. One week prior to admission, he developed a nonproductive cough, and on the day of admission, he experienced hemoptysis. Upon admission on Nov 20, 1984, physical examination revealed a patient in no acute distress, with a temperature of 38.4°C (101.1°F), pulse rate of 120 beats per minute, blood pressure of 130/70 mm Hg, and respiration rate of 18/min. The nasal mucosa had crusted exudates and ulcerations. Chest auscultation demonstrated diffuse coarse rhonchi and an S4 gallop.

Laboratory tests revealed the following values: hemoglobin, 11.5 g/dl; hematocrit reading, 35.5 percent; reticulocyte count, 0.3 percent; white blood cell count, 15,200/cu mm; platelet count, 762,000/cu mm; erythrocyte sedimentation rate (Westergren), 80 mm/hr; blood urea nitrogen, 10 mg/dl; serum creatinine, 1.2 mg/dl; serum sodium, 135 mEq/dl; serum potassium, 4.3 mEq/dl; serum chloride, 102 mEq/dl; and serum bicarbonate, 26 mEq/dl. The urinalysis had 1+ protein, 60 red blood cells per high-power field, 10 to 12 white blood cells per high-power field, and two red blood cell casts. The chest roentgenogram revealed multiple thick-walled cavities in both pulmonary fields, and sinus films revealed mucosal thickening of the maxillary sinuses. Cultures of blood, fungal septic culture studies, and the tuberculin skin test were negative. Biopsies of the nasal mucosa revealed necrotizing granulomatous inflammation with giant cells, and cultures and special stains of the material from biopsy were negative for mycobacteria and fungi. A necrotizing glomerulonephritis with crescents was seen on renal biopsy.

Wegener’s granulomatosis was diagnosed, and on Dec 5, therapy with cyclophosphamide (140 mg/day) and prednisone (70 mg/day) was started. One day later, the patient experienced retrosternal pain in the chest which lasted 15 minutes. Initial and serial electrocardiograms, as well as cardiac enzyme levels, were normal. On December 10, the patient once again experienced chest pain, which lasted one hour and was associated with diaphoresis and shortness of breath. An ECG revealed new ST-segment depression of 2 to 3 mm in the anterolateral leads, and serial ECGs were consistent with an acute anterolateral myocardial infarction. Serum levels of creatine kinase demonstrated an appropriate rise and fall, with a peak level of 610 IU (normal, less than 280 IU) and 9 percent MB band. The patient had no further chest pain, but a mild sinus tachycardia persisted into the second week after infarction. A multiple gated acquisition radionuclide angiogram showed an ejection fraction of 49 percent, and an echocardiogram on Dec 18 revealed a large area of increased reflectance which moved with the noncoronary cusp of the aortic valve (Fig 1). Serial cultures of blood were sterile. The patient remained asymptomatic, and the sinus tachycardia slowly resolved. By early January 1985, the nasal mucosa

Reversible Valvulitis in Wegener’s Granulomatosis (Gerbracht, Savage, Scharrf)
Diffuse Triple-vessel Coronary Artery Spasm Complicated by Idioventricular Rhythm and Syncope*

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A 70-year-old man presented with diffuse triple-vessel coronary arterial spasm accompanied by ST segment elevation in the inferior and anterior leads when the severity of pain moderated. At the beginning, he noted throat and chest pain followed by syncope. Atropine, norepinephrine, and lidocaine were administered therapeutically. The initial electrocardiogram showed an idioventricular rhythm without ST segment deviations, which made the prompt diagnosis of coronary arterial spasm difficult.

Coronary arterial spasm is an important etiologic mechanism in the pathogenesis of myocardial ischemia. It usually involves the same part of one vessel, and rarely involves different coronary arteries at the same time. Simultaneous involvement of all three major coronary arteries is rather uncommon. There have been only two reported cases with spontaneous three-vessel coronary artery spasm documented by coronary arteriography. In these cases, coronary spasm was identified as a reversible segmental narrowing accompanied by ST segment elevation in both inferior and precordial leads. In this report, we describe a patient showing spontaneous triple-vessel spasm with diffuse distribution complicated by idioventricular rhythm and syncope.

CASE REPORT

A 70-year-old man who had experienced cerebellar hemorrhage at the age of 65 years without serious complications noted burning pain.

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FIGURE 1A (upper). Left parasternal long-axis two-dimensional echocardiogram showing large area of increased reflectance involving noncoronary cusp of aortic valve. B (lower). Left parasternal short-axis view showing similar involvement.

cleared. A chest roentgenogram on Jan 3 was normal, and the sinus films revealed 90 percent resolution of the maxillary mucosal thickening. Serial echocardiograms showed decreasing size of the aortic valvular vegetation, and a study on Feb 14 revealed complete resolution.