reconstruction utilizing a prosthetic heart valve and Dacron. These principles of management are well-described by others and were used in our patient successfully. The extent of anular erosion and myocardial abscesses are unpredictable at surgery and may require radical debridement and suitable reconstruction. We have introduced a modified technique of aortoseptoplasty to reconstruct the membranous septum and aortic outflow tract in a six-year-old boy with pneumococcal infective endocarditis.

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Pulmonary Toxicity of Amiodarone*

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Mild pleuroparenchymal fibrosis associated with amiodarone pulmonary toxicity is reported in a 63-year-old white man; partial radiographic resolution and complete symptomatic resolution with decreasing the daily dosage to 200 mg permitted continued anti arrhythmic therapy.

Since amiodarone hydrochloride was released as an investigational drug in the United States and Britain for the treatment of recurrent ventricular arrhythmia, several reports of pulmonary toxicity believed attributable to amiodarone have been published. This experience of pulmonary toxicity may be a dose-related phenomenon. In Europe, a much longer experience with lower dosages (200 mg/day) used for antianginal effects has produced only one report of pulmonary toxicity.

Among 47 individuals begun on therapy with amiodarone in this institution in the past two years, we have observed two cases of pulmonary toxicity, one with some unique features that we hope may suggest a means of continuing therapy despite side effects in selected individuals.

CASE REPORT

A 63-year-old white man was managed on therapy with amiodarone (400 mg daily) from January 1981, for recurrent sustained ventricular tachycardia refractory to multiple other antiarrhythmic drugs. In July 1982, he was admitted to another hospital for abdominal pain and found to have right-sided apical infiltrates on the roentgenogram on admission; a chest x-ray film in April of that year had been normal. Tranbronchial biopsy performed there was reportedly normal.

The patient was admitted to the University of Illinois Hospital in September 1982, with complaints of loss of weight, weakness, and fatigue with a nonproductive cough and some shortness of breath. The roentgenogram (Fig 1) now showed bilateral apical infiltrates with thickened adjacent pleura. Measurements of pulmonary volume were interpreted as a moderate restrictive defect (forced vital capacity [FVC], 42 percent of predicted; ratio of the forced ex-

FIGURE 1. Bilateral apical pleuroparenchymal fibrosis.
Steroid Therapy in Cardiac Sarcoidosis*

Increased Left Ventricular Contractility Concomitant with Electrocardiographic Improvement after Prednisolone

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A 23-year-old woman was referred to our clinic because of pulmonary edema. The electrocardiogram on admission demonstrated eventual QS pattern in leads I and aVL with a poor progression of the R-wave amplitude in the precordial leads. The left ventricle was remarkably dilated with severely diminished contractility. Biopsy of the inguinal lymph nodes, liver, and myocardium confirmed the diagnosis of generalized sarcoidosis. After administration of prednisolone, the left ventricular dimension decreased, and contractility increased concomitant with an improvement of the electrocardiographic abnormalities without any aneurysm forming. We conclude that steroids may keep some jeopardized myocardium viable and that initiation of steroid therapy in an early phase does not result in formation of an aneurysm.

Steroids for the treatment of cardiac sarcoidosis are not conclusive, although sarcoi granuloma in the heart is responsive to the therapy. This is because the disappearance of sarcoi granuloma from cardiac muscle might result in formation of a ventricular aneurysm, which may be a cause of congestive heart failure or sudden death.

Herein we present a case of cardiac sarcoidosis in which steroid administration increased left ventricular contractility concomitant with an improvement of electrocardiographic abnormalities and was not associated with formation of an aneurysm. The clinical course of the present case may shed light on the etiology of aneurysmal formation after steroid therapy.

Case Report

A 23-year-old Japanese woman was referred to our clinic on Feb 10, 1982 with a one-week history of orthopnea with cough and foamy sputum. She delivered twins on Dec 27, 1981, and had had hypertension, proteinuria, and general edema for one month before delivery; however, these disappeared soon after delivery, and the patient had been doing well. She experienced cough and paroxysmal nocturnal dyspnea on Feb 5, 1982, with the symptoms gradually intensifying.

On physical examination the patient was in severe respiratory distress. The blood pressure was 130/70 mm Hg; the pulse rate was 121 beats per minute and regular; the body temperature was 37.4°C, and the respiratory rate was 28/min. The veins in the neck were distended at 40°, and hepatosplenic reflex was demonstrated. Diffuse moist rales and an S4 gallop rhythm were heard. Hepatomegaly (three fingerbreadths) with tenderness on percussion was noted in the abdominal examination. Pretibial edema was present on both legs.

References


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