tion in the right upper lobe, which cavitated and spread subsequently to the right lower pulmonary fields.

Amphotericin B and flucytosine are the most effective agents presently available for treatment of systemic infection with *T. glabrata*. Amphotericin B is more active in vitro, inhibiting all strains at a concentration of 1\(\mu \text{g/ml}\) and killing all strains at 2\(\mu \text{g/ml}\), but is more toxic and requires intravenous administration and hospitalization. Flucytosine is generally nontoxic and is well distributed in the tissues, the tissue level being equal to or higher than that in the serum. In a concentration of 0.24\(\mu \text{g/ml}\), it inhibited in vitro 82 percent of *T. glabrata* strains. While receiving flucytosine, the patient had marked symptomatic improvement and radiographic clearing, but sputum cultures continued to contain a few colonies of *T. glabrata*. Acute disseminated infections and urinary-tract infections due to *T. glabrata* have been treated with flucytosine alone, amphotericin B alone, or the two combined. There was no precedent as to length of treatment for pulmonary infection with *T. glabrata*. The fact that our patient’s disease spread when therapy was stopped after 3 months suggests that a long duration of treatment is desirable, as in tuberculosis.

*Torulopsis glabrata* should be considered a potential pulmonary pathogen, even in a normal host. Specific treatment with amphotericin B alone, flucytosine alone, or the two drugs in combination is indicated early in the course of the disease, and duration of treatment should be sufficiently long to prevent progression of infection and permanent damage to the lung.

**REFERENCES**


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**Detection of Covert Myocardial Sarcoidosis by Scalene Node Biopsy**

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The antemortem diagnosis of myocardial sarcoidosis is rare in patients without overt signs of the disease. Two patients are presented to alert physicians to the value of early scalene node biopsy when sarcoidosis could be the cause of marked disturbances in cardiac conduction. The first patient, aged 29 years, had first, second, and third degree atrioventricular block and intermittent left and right bundle-branch block. The second, aged 59 years, had second degree atrioventricular block and complete right bundle-branch block. Both had diagnoses of sarcoidosis based on scalene node biopsy. The cardiac conductive disturbance improved, and the symptoms disappeared with steroid therapy.

The antemortem diagnosis of myocardial sarcoidosis is made infrequently. Moreover, the diagnosis of myocardial sarcoidosis in patients without lymphadenopathy or other clinical or chest roentgenographic signs is an unusual event. This is a report of two patients who had marked cardiac conductive disturbances without overt evidence of sarcoidosis. Yet they were shown at scalene node biopsy to have the disease, and their abnormalities in cardiac conduction improved with steroid therapy.

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While histologic confirmation of sarcoid infiltration of the conductive system is lacking, the favorable response to steroid therapy supports the diagnosis.

These cases are presented to alert physicians to the potential value of early scalene node biopsy in patients with severe cardiac conductive disturbances when sarcoidosis could be the cause.

**CASE REPORTS**

**Case 1**

A 29-year-old woman had a two-week history of easy fatigability, occasional light-headedness, and intermittent fluttering of her heart. Physical examination revealed a grossly irregular pulse of 50 beats per minute. There was neither cardiac enlargement nor murmur. Variable intensity and degree of splitting of the first and second heart sounds were found. No lymphadenopathy, hepatomegaly, or skin lesions were present.

The findings from routine laboratory tests, including sedimentation rate, serum calcium level, and protein electrophoresis, were normal. The patient's chest roentgenogram was normal, without hilar adenopathy. Her electrocardiogram revealed variable atrioventricular block, intermittent left and right bundle-branch block (Fig 1), as well as premature atrial and ventricular contractions, and short runs of supraventricular tachycardia.

Because of a suspicion of myocardial sarcoidosis, a right scalene lymph node biopsy was performed on the morning after admission. Granulomatous lymphadenitis consistent with sarcoidosis was found (Fig 2). Ophthalmologic examination showed no sign of sarcoidosis. Routine skin tests were negative. The patient was started on therapy with prednisone (80 mg/day), resulting in a return to sinus rhythm with only a residual right bundle-branch block and no ectopic beats. Prednisone therapy was tapered over 18 months to a maintenance dose of 7.5 mg/day. Twenty-one months after hospitalization, the ECG changed to rate-dependent left bundle-branch block and first-degree atrioventricular block. Prednisone therapy was increased to 80 mg/day. The patient's most recent electrocardiogram (Fig 3), at 28 months, reveals only left anterior hemiblock.

**Case 2**

A 50-year-old man was admitted to the hospital with a three-month history of episodes of rapid, forceful heartbeats, as well as one episode of near syncope. The vital signs were normal, except for an irregular heart rate of 100 beats per minute. The findings from cardiac examination were unremarkable. There was no adenopathy, hepatomegaly, or skin lesions.

Findings from routine laboratory tests, including sedimentation rate, serum calcium level, and protein electrophoresis, were normal. Roentgenograms of the chest were also normal. The ECG showed second degree atrioventricular block, complete right bundle-branch block, and a superior axis (Fig 4), as well as paroxysms of supraventricular tachycardia.

A right scalene node biopsy showed noncaseating granulomata with early fibrosis consistent with sarcoidosis (Fig 5). Ophthalmologic examination revealed a small focus of periphlebitis in an inferior temporal vein, suggestive of sar-
Sarcoidosis. Routine skin tests were negative.

Therapy with prednisone (80 mg/day) was begun. Within a few days, the patient was free of palpitations, and the ECG reverted to first degree atrioventricular block with persistent complete right bundle-branch block, superior axis, and no ectopic beats. Eight months later, the ECG was unchanged, and the patient remained free of palpitations.

**DISCUSSION**

Recurrent Stokes-Adams attacks or sudden death may be the only clinical manifestation of sarcoidosis. Bernstein and associates first reported myocardial involvement with the disease in 1929. The first death from myocardial sarcoidosis was reported by Gentzen in 1937. By 1971, a total of 70 autopsied cases had been reported. Fifty more cases from the British Isles were reported in 1974. Antemortem diagnosis was infrequent because of the paucity of clinical clues to the illness. The diagnosis of myocardial sarcoidosis was usually considered only when the chest roentgenogram showed the typical hilar configuration in a patient with cardiac conductive disturbances.

Our cases demonstrate that sarcoidosis may affect the heart without other clinical or radiographic manifestations. Suspicion should be aroused by significant cardiac conductive disturbances in an otherwise normal heart of a person who is a bit young for the typical case of LeV's or Lenegre's disease. Evidence of sarcoidosis in the scalene node would seem to accompany cardiac involvement.

While the results of corticosteroid therapy have generally been disappointing in myocardial sarcoidosis, favorable outcomes have been reported. Such therapy might well be most effective in the early stages of the disease, before fibrosis of the cardiac conductive system occurs. Occasional instances of spontaneous improvement of rhythm disturbances have occurred, but most patients have gone on to die, usually suddenly. Pacemaker therapy for myocardial sarcoidosis prevents death from asystole but will not diminish ventricular irritability or heart failure caused by diffuse myocardial infiltration with sarcoid granulomata. Prompt corticosteroid therapy is indicated, whether or not a pacemaker is used. Diagnosis by scalene lymph node biopsy allows the institution of early therapy in cases of myocardial sarcoidosis.

**REFERENCES**