back-leakage of chyle into the pericardial space. This mechanism would appear to be similar to that postulated by Thomas and McGoon,* venous obstruction causing blockage of the thoracic duct.

In general, the treatment for chylopericardium includes (1) surgical drainage of the fluid, (2) prevention of reaccumulation by pericardiectomy and ligation of the thoracic duct above the diaphragm, and, finally, (3) diets containing medium-chain triglycerides (since their source of fat can be a valuable adjunct in the clinical management of these patients).

REFERENCES

Myocardial Dysfunction and Hemolytic Anemia in a Patient with Mycoplasma pneumoniae Infection*

Henry Maresh, M.D.; Joseph J. Klimek, M.D.; and Richard Quintiliani, M.D.

A patient with evidence of myocardial abnormalities and hemolytic anemia is described, in whom the responsible pathogen appeared to be Mycoplasma pneumoniae (as indicated by a 64-fold rise in complement-fixation titers, and by a change in cold-agglutinin titers from 1:8 to 1:4,096). Both cardiac and hematologic problems occurred during the recovery phase from pneumonia and were associated with marked deterioration in the patient's clinical status. Electrocardiographic and serum enzymatic changes mimicked the patterns seen in acute myocardial infarction.

Mycoplasma pneumoniae is a common respiratory pathogen that may be associated with a variety of other complications, including erythema multiforme, meningitis, peripheral neuropathy, and hemolytic anemia.1 Myocarditis has also been previously described in association with infection with this organism, but the manifestations have been minor, both in the electrocardiographic change (nonspecific abnormalities of the ST-T wave) and in signs of cardiac dysfunction. In this communication, we present a patient who developed signs and symptoms of myocarditis during the course of infection with Mycoplasma pneumoniae associated with severe hemolytic anemia.

CASE REPORT
A 49-year-old white woman was hospitalized for evaluation of increasing shortness of breath, a productive cough of moderate amounts of yellowish-brown sputum, and fever of four days' duration. There was no prior history of cardiac disease.

On physical examination, the patient's oral temperature was 39°C (102.2°F), the pulse was 132 beats per minute, the blood pressure was 150/80 mm Hg, and the respiration rate was 30/min. The veins in the neck were flat at 45° in the semirecumbent position. On auscultation of the chest, diffuse rhonchi and rales were heard bilaterally. A soft grade1/6 systolic ejection murmur was heard along the left sternal border. The physical examination was otherwise unremarkable.

On admission, the hematocrit reading was 44 percent, and the white blood cell count (WBC) was 13,600/cu mm, with 68 percent polymorphonuclear cells and 20 percent band cells. The platelets were estimated to be normal. The serum electrolyte levels were as follows: sodium, 122 mEq/L; potassium, 3.8 mEq/L; and chloride, 89 mEq/L. Simultaneously determined urinary levels were sodium, 156 mEq/L; potassium, 39 mEq/L; and urinary osmolality, 602 mOsm/kg H2O. Arterial blood gas levels with the patient on a 10-L oxygen rebreathing mask were as follows: pH, 7.35; carbon dioxide tension, 36 mm Hg; oxygen pressure, 58 mm Hg; and calculated carbon dioxide content, 21 mEq/L, with oxygen saturation of 87 percent. Initial titers of cold agglutinins were positive at a 1:8 dilution. The chest roentgenogram revealed bilateral basilar interstitial infiltrates, and the electrocardiogram exhibited sinus tachycardia with borderline first-degree atrioventricular block (Fig 1).

Because of a presumptive diagnosis of viral pneumonia with a possible superimposed bacterial infection, the patient was treated with cephalixin sodium (3 gm intravenously per day), methylprednisolone (80 mg/day), and oxygen therapy, which resulted in gradual improvement. On the sixth day of hospitalization, the therapy with cephalixin was discontinued, but the methylprednisolone dosage was maintained at 20 mg/day.

On the tenth day of hospitalization, the patient noted worsening of her shortness of breath and orthopnea. Physical examination now revealed a grade 3/6 pansystolic murmur at the lower left sternal border and apex, an S4 gallop rhythm, diffuse pulmonary rales, and distended neck veins.
without peripheral edema. The chest roentgenogram showed improvement of the interstitial infiltrate seen on admission but also showed borderline cardiomegaly. Over a period of 12 hours, the ECG evolved from a pattern of tachycardia with peaked T waves in the anterior precordial leads to changes consistent with an acute lateral-wall myocardial infarction (Fig 2). The hematocrit reading, which was 38 percent on the tenth day of hospitalization, fell to 25 percent over the next two days, with a WBC of 28,000/cu mm and a platelet count of 1,065,000/cu mm. Spherocytes and basophilia were noted on the peripheral blood smear. The reticulocyte count was 6.5 percent, and a small amount of urinary hemoglobin was detected. Titers for cold agglutinins were now positive at 1:4,096 at 4°C and at 1:256 at 37°C, with erythrocyte anti-I antibodies demonstrated. The direct Coombs' test was positive to C3 complement. The serum iron level, the total iron-binding capacity, and the levels of folate and vitamin B12 were normal.

On the 11th day of hospitalization, the creatine phosphokinase level, which was 48 units/L on admission, rose to 273 units/L (normal, 50 to 200 units/L). The lactic dehydrogenase level was 638 units/L (normal, 100 to 250 units/L); the level of serum glutamic transaminase was 46 units/L (normal, 3 to 23 units/L); and the level of serum glutamic pyruvic transaminase was 30 units/L (normal, 3 to 20 units/L). Renal function remained normal.

The patient was given digoxin, and six units of warmed packed red blood cells were transfused to maintain the hematocrit reading at 30 percent. Five days after the apparent infarction, the ECG revealed only nonspecific T-wave changes, with resolution of the pattern of infarction (Fig 3). The patient improved without further complications and was
discharged on the 22nd day of hospitalization.

Two months after discharge, only nonspecific T-wave changes remained. The complement-fixation titer for *Mycoplasma pneumoniae*, which was negative on admission, rose to 1:64. Acute and convalescent serologic testing for adenovirus, influenza A and B, psittacosis, respiratory syncytial virus, Q fever, herpes, and mumps showed no evidence of recent infection from these organisms.

**DISCUSSION**

Myocardial involvement associated with infection with *Mycoplasma pneumoniae* was first reported by Rosner and associates, who described an 18-year-old patient who developed complete atrioventricular block during an upper-respiratory-tract illness. This patient had a change in the complement-fixation test consistent with recent infection with Mycoplasma, but levels of cold agglutinins and serum cardiac enzymes were normal. In a series of 18 patients with asymptomatic myocarditis associated with viral-like illnesses reported by Lewis et al., two patients had serologically proven infection with Mycoplasma. The myocarditis in these two patients was manifested only by abnormalities of the ST-T wave on the ECG and occurred concurrently with respiratory symptoms.

The cold-agglutinin hemolytic anemia associated with infections with *Mycoplasma pneumoniae* characteristically occurs in the second and third weeks of the illness, with the appearance of cold agglutinins in significant titers (> 1:512). The cold agglutinins are IgM antibodies with anti-I erythrocyte antigen specificity and have a high thermal maximum in those patients who develop hemolytic anemia. El Khatib and Lerner recently reported serologically proven infection with Mycoplasma in a patient with asymptomatic myopericarditis and hemolytic anemia associated with a markedly elevated titer of cold agglutinins. Of significant note is the fact that the myocarditis and hemolytic anemia occurred simultaneously on the 22nd day of illness, after resolution of an acute pneumonitis. At no time was the patient's anemia severe enough to warrant transfusion.

Our patient initially had severe pneumonia due to *Mycoplasma pneumoniae* and associated with the syndrome of inappropriate secretion of antidiuretic hormone. The pneumonia appeared to be resolving clinically and radiographically, when the patient experienced sudden clinical deterioration associated with electrocardiographic changes simulating an acute myocardial infarction and a concomitant cold-agglutinin hemolytic anemia. An infarction cannot be entirely ruled out without the benefit of coronary arteriograms; however, the clinical course of increasing dyspnea without associated chest pain, persistent tachycardia, a transient murmur of mitral insufficiency, and extensive electrocardiographic changes which were transient in nature and associated with only minimal cardiac enzymatic elevations are perhaps more consistent with acute myocarditis. The cold-agglutinin hemolytic anemia appeared during the second week of hospitalization, with a markedly elevated titer of cold agglutinins (1:4,096), which had significant activity at 37°C (1:256) and was of characteristic anti-I antibody specificity. The patient experienced no myalgias during her illness.

The association of myocarditis with markedly elevated titers of cold agglutinins and hemolytic anemia suggests an immune basis for this complication. In patients with primary atypical pneumonia, Thomas et al. have described cross-reacting antibodies directed primarily against pulmonary tissue, but with occasional reactivity to other tissues, including the heart; however, Kerr and Bridges noted an absence of γ-globulin deposits in the cardiac lesions of chicken embryos infected by *Mycoplasma synoviae*, suggesting a direct toxic effect of the organism.

Although it is difficult to arrive at any definitive
conclusions from these data from animals, the clinical presentations suggest that there may be two distinct forms of myocardial involvement associated with infections with Mycoplasma. The first form of myocarditis occurs early in the infection, concomitant with pulmonary parenchymal involvement, and may be related to a direct toxic effect of the infectious agent. The second form, as perhaps demonstrated in our patient, occurs later during the recovery phase and appears to be an immunologically related phenomenon associated with cold hemaggutinin disease.

An alternative explanation for the cardiac involvement in our case should also be considered. Maisel et al6 presented autopsy evidence of multiple intravascular thromboemboli in a fatal case of infection with Mycoplasma pneumoniae associated with cold hemaggutinin disease. Our patient also had a marked thrombocytosis, which can be associated with an increased incidence of arterial and venous thrombotic complications, particularly in patients with qualitatively normal platelets unassociated with hematopoietic malignant disease.*

Thus, it can be speculated that transient embolic occlusion or partial occlusion of the coronary arteries by agglutinated red blood cells or platelets produced the abnormal electrocardiographic and serum enzymatic changes in our patient; however, as noted previously, the rapidity of evolution and the eventual resolution of electrocardiographic changes seem to mitigate this possibility.

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CHRONIC DERMATOMYOSIS WITH INTERMITTENT TRIFASCICULAR BLOCK* 413

Chronic Dermatomyositis with Intermittent Trifascicular Block*

An Electrophysiologic-Conduction System Correlation

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A case of dermatomyositis associated with recurrent syncope and complete heart block is described. Right bundle branch block and left anterior hemiblock with intact atrioventricular conduction then persisted over a five-year period. His bundle studies demonstrated a prolonged Hisventricle (H-V) interval. Postmortem examination confirmed the diagnosis of dermatomyositis as well as a fibrotic cardiomyopathy. The pathologic findings of marked fibrosis of both bundle branches, especially the right bundle branch and the anterior portion of the main left bundle branch, correlated well with the electrocardiographic and electrophysiologic findings.

Chronic deramatomyositis is a disease of unknown etiology characterized by inflammation and degeneration of skeletal muscles and cutaneous abnormalities. Clinical evidence of heart disease secondary to chronic dermatomyositis is infrequent and usually nonspecific.1-8 Four previous cases of complete heart block occurring with polymyositis1-7 have been reported, with detailed pathologic descriptions of the conduction system in two.5,6 In this communication, we report a case of chronic dermatomyositis associated initially with intermittent complete heart block and then with a five-year history of the stable bifascicular block pattern of right bundle branch block and left anterior hemiblock.8 This is believed to be the first report of this entity studied by both intracardiac electrophysiologic studies and serial sections of the conduction system.

CASE REPORT

In November 1967, a 47-year-old black man initially had severe pain and weakness of both thighs as well as discoloration of his skin. The concentrations of serum glutamic oxalacetic transaminase (SGOT), creatinine phosphokinase, and aldolase were markedly elevated. A skin and muscle biopsy was diagnostic of chronic dermatomyositis and demonstrated chronic dermatitis and skin atrophy, as well as severe focal

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