Myopericarditis as an Initial Presentation of Meningococcemia

Unusual Manifestation of Infection with Serotype W135

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Acute meningococcemia is a dramatic clinical syndrome from infection with the gram-negative diplococcus, Neisseria meningitidis. Although pericarditis may complicate the course of meningococcemia, it is distinctly unusual as a presenting sign. A case of disseminated meningococcemia presenting as acute myopericarditis is reported. The serotype isolated, type W135, was a sporadic cause of N. meningitidis in the Boston area. Although the patient had meningitis, bacteremia, and myopericarditis, his course was uncomplicated with early institution of antibiotic therapy.

Meningococcemia is a characteristic clinical syndrome due to infection with the gram-negative diplococcus, Neisseria meningitidis. The human nasopharynx is the natural reservoir of Neisseria [1], and overt clinical illness occurs when the initial nasopharyngeal infection invades locally and progresses to bloodstream invasion. The most common clinical syndromes are meningococcemia and acute purulent meningitis [2]. Unusual complications of N. meningitidis infections include suppurative arthritis, isolated purulent pericarditis, and panophthalmitis. Although pericarditis complicates the clinical course of meningococcal disease, it rarely is the presenting sign [3]. We herein report an unusual case in which acute myopericarditis was the initial clinical manifestation of meningococcemia.

CASE REPORT

A 46-year-old Hispanic man presented to our hospital for evaluation of fever and pleuritic chest pain. The patient was in excellent health until several days prior to admission when he noted a sore throat. One day before admission, he had fever to 40°C and knifelike inspiratory chest pain with radiation to the back. Simultaneously, he noted a mild bitemporal headache. His only cardiac risk factor was a 40 pack/year smoking history.

On examination, he was a thin, ill-appearing Hispanic man. The systolic arterial pressure was 82 mm Hg with a pulsus paradoxus of 5 mm Hg. The heart rate was 130 beats/minute, respiratory rate was 24 breaths/minute, and the rectal temperature was 38°C. The skin was clear, and the oropharynx was erythematous and dry. Rales were heard at the right lung base. Cardiac examination revealed a normal first heart sound, physiologically split second heart sound, and no detectable murmur or rub. Neurologic examination was remarkable for an alert, oriented, Spanish-speaking man. Anterior neck flexion was full to one inch from the sternum.

On initial laboratory examination, urine was benign, electrolyte values were normal, creatine phosphokinase level was 88 IU/ml (normal less than 180 IU/ml), and white blood cell count was 11,100/mm³ with 49 percent polymorphonuclear leukocytes, 44 percent band forms, and 7 percent lymphocytes. The prothrombin time was 12.3 seconds, and the partial

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thromboplastin time was 24.8 seconds. Arterial blood gas determination revealed a partial pressure of oxygen of 75 mm Hg, a partial pressure of carbon dioxide of 36 mm Hg, and a pH of 7.36. Chest radiographic findings were interpreted as showing normal heart size, no parenchymal infiltrates, and evidence of old granulomatous disease. Electrocardiography revealed sinus tachycardia with P-R depression and diffuse ST segment elevation most prominent in the anterior leads consistent with pericarditis (Figure 1). Two-dimensional echocardiography showed hyperdynamic function with normal heart size and configuration. Specifically, there was no pericardial effusion and valves were normal in appearance.

The patient was admitted to the cardiac intensive care unit and received vigorous parenteral fluid administration. His blood pressure responded to hydration, but his temperature spiked to 40°C, and the patient became confused. Blood, urine, and cerebrospinal fluid culture specimens were taken. Lumbar puncture demonstrated an opening pressure of 200 mm H2O. The cerebrospinal fluid glucose value was 79 mg/dl, protein value 32 mg/dl, red blood cell count 2/mm³, and white blood cell count 3/mm³. Cytospin analysis of the cerebrospinal fluid revealed a predominance of lymphocytes and monocytes; specifically, no polymorphonuclear leukocytes were identified. Results of Gram stain and India ink stains were negative.

The patient was empirically treated with cefamandole and given high-dose aspirin for presumed pericarditis. Within 12 hours, the cerebrospinal fluid culture specimens were growing N. meningitidis; additionally, results of three of three blood cultures were positive for the same organism on the second hospital day. Therapy was immediately changed to high-dose penticillin G (24 million units per day) intravenously. Typing of the Neisseria species revealed serotype W135.

His serum creatine phosphokinase level rose to 1,120 IU/ml 20 hours after admission, with an MB creatine phosphokinase isoenzyme value of 14 percent. Serial electrocardiography showed evolution consistent with pericarditis (Figure 2). He had several brief episodes of nonsustained monomorphic ventricular tachycardia, which were suppressed with intravenous procainamide.

Defervescence occurred after four days of penicillin therapy; technetium pyrophosphate scanning at this time showed no focal areas of increased myocardial uptake. Symptoms of pericarditis resolved over the first seven days of hospitalization. The procainamide was terminated after 10 days, with subsequent 24-hour electrocardiographic monitoring showing only an isolated ventricular premature depolarization. Results of repeated echocardiography were essentially unchanged from results of the study performed on the day of admission.

He was discharged after 14 days of parenteral antibiotics, receiving no medications. His close contacts were given a two-day course of rifampin prophylaxis.

COMMENTS

Herein we report a dramatic case of meningococcemia presenting as acute myopericarditis. The initial diagnosis of pericarditis was made on the basis of the combination of stabbng inspiratory chest pain and characteristic electrocardiographic changes [4]. There was no pericardial effusion. Serum cardiac enzyme values were transiently abnormal, consistent with myocardial necrosis. Clinically, left ventricular function remained stable with no deterioration noted on serial echocardiography. The patient showed signs of myocardial irritability with transitory episodes of ventricular tachycardia. The overall clinical course was uncomplicated after initiation of appropriate therapy with high-dose penicillin.

Pericarditis and myocarditis have been reported separately as complications of meningococcemia. In an autopsy series of fulminant meningococcemia, Herdmann and Eark [5] found a 78 percent incidence of myocarditis (by
histologic evidence of infiltration with inflammatory cells), although other series place the incidence much lower, at 22 to 46 percent \cite{6,7}. Since many organs are affected, it is not unreasonable to suspect the myocardium to be involved—particularly in cases of overwhelming sepsis. The clinical importance of myocarditis in survivors has not been assessed prospectively, and it is not known when (in the course of infection) myocarditis occurs.

The incidence of pericarditis complicating meningococcal disease is not known, and the rare anecdotal case reports suggest it is an uncommon occurrence. In the pre-antibiotic era, Herrick \cite{8} reported an incidence of 4.3 percent (12 of 280 patients) of pericarditis complicating meningococcemia. Lipton \cite{9} reviewed 8,844 cases of meningococcemia treated with sulfonamides and found no case of pericarditis. Wolf and Birbara \cite{10} noted only two instances of pericardial effusion, both of which were diagnosed postmortem, in a series of 112 cases of bacteriologically confirmed meningococcal infection.

More recently, symptomatic pericarditis was reported in six patients by Morse et al \cite{11}. The clinical onset of pericarditis occurred after the signs of meningitis had cleared or markedly improved, and the pericardial symptoms responded to either salicylate therapy or glucocorticoids. One patient required pericardiocentesis for a sterile exudate. As cultures revealed N. meningitidis serotype C in each instance, the authors speculated that the pericarditis was a consequence of the specific immunologic response to that serotype.

Pierce and Cooper \cite{12} reported a different spectrum of meningococcal pericarditis in five selected patients. Pericardial involvement was suspected at presentation with meningococcemia because of characteristic electrocardiographic changes or the presence of a friction rub. However, only one patient initially complained of chest pain, whereas symptomatic pericarditis developed two to 20 days after admission in the other four. Enlargement of the cardiac silhouette was noted on chest roentgenography in all five patients. Serotyping was not consistently performed. Pierce and Cooper concluded that there were two types of pericarditis associated with meningococcemia—early and late. The early pericarditis was presumably due to true pericardial infection secondary to bacteremia, whereas late pericarditis occurring during convalescence was secondary to a localized hypersensitivity phenomenon. This view has been echoed in a recent review of purulent pericarditis \cite{13}.

In the pre-antibiotic era, pericarditis complicating meningococcemia predicted a very poor prognosis with a case-fatality rate of 83 percent versus only 25 percent in patients without apparent pericardial involvement \cite{8}. Whether pericarditis affects prognosis in the post-antibiotic era is not known.

In summary, we describe a patient with acute myopericarditis as the initial presentation of meningococcemia. This patient presented with typical features of pericarditis including chest pain and electrocardiographic changes in the absence of pericardial effusion or cutaneous signs of meningococcemia. We can only speculate on the pathogenesis of the myopericarditis, which is most likely related to early bactereemic seeding of the pericardium. The initial assessment of clinical pericarditis should include consideration of possible bacteriologic causes that would necessitate the use of antibiotics as part of the therapeutic regimen.

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