Complete Heart Block from Mycoplasma Pneumoniae Infection

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SUMMARY. Complete heart block (CHB) in infants and children is usually congenital. Non-surgical acquired CHB is rare. Occasionally, transient acquired CHB is seen in association with viral myocarditis. We describe here an unusual case of transient CHB in a 12-year-old boy with endomyocardial biopsy-proven myocarditis and evidence of Mycoplasma pneumoniae infection.

KEY WORDS: Complete heart block — Myocarditis — Mycoplasma pneumoniae

Infants and children with acute myocarditis may present with various electrocardiographic abnormalities, ranging from ST-T changes to tachyarrhythmia and complete heart block (CHB). Similarly, the clinical manifestations have a wide spectrum (e.g., chest pain to congestive heart failure and sudden death).

CHB is usually congenital in infants and children [16]. Nonsurgical acquired CHB is rare. Occasionally, transient acquired CHB is seen in association with viral myocarditis [10, 11, 18, 19, 20].

We report here a 12-year-old patient with CHB and endomyocardial biopsy-proven myocarditis and serologic evidence of Mycoplasma pneumoniae infection.

Case Report

History

A 12-year-old black male was admitted to Wyler Children’s Hospital Intensive Care Unit through the Emergency Room where he presented with a history of shortness of breath, chest pain, fever, and vomiting. He was apparently in good health 7 days prior to admission when he complained of upper respiratory infection (URI).

Two days prior to admission he was seen in a local hospital emergency room, he was diagnosed as having URI and sent home on 650 mg acetaminophen every 4 h as needed. At that time his heart rate was 100 beats/min. His past medical and family histories were noncontributory.

Physical examination revealed a lethargic but oriented child with a heart rate of 32 beats/min, a respiratory rate of 28/min, a blood pressure of 100/40 mmHg (supine), and a temperature of 38°C (oral). A mild erythematous pharynx was noted. The abdomen was soft. A 1-cm tender liver edge was palpable at the mid-clavicular line below the right costal margin. Both lungs were clear to auscultation and percussion. Cardiovascular examination revealed a quiet precordium with precordial maximal impulse at the sixth left intercostal space just outside the mid-clavicle line without any palpable heave or thrill. Both the first and second heart sounds were soft and no heart murmur or rub were heard.

In the emergency room, the electrocardiogram revealed CHB, an atrial rate of 90 beats/min with a wide QRS ventricular escape rhythm at 30 beats/min and RBBB pattern. Atropine at 0.4 mg i.v. increased the atrial rate (Fig. 1) from 90 to 160 with no effect on the ventricular rate. Intravenous isoproterenol drip (0.2 μg/kg/min) increased the ventricular rate to 58 beats/min and induced ventricular tachycardia that was well tolerated. The ventricular tachycardia was then controlled with lidocaine. A temporary transvenous pacemaker catheter was inserted and he was paced at a rate of 80 beats/min with significant clinical improvement.

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In the intensive care unit the patient received the following medications: Lasix 20 mg b.i.d. daily, digoxin 0.125 mg daily, erythromycin 250 mg Q6H for 10 days, and prednisone 20 mg t.i.d. beginning the 11th day for 1 week followed by 20 mg daily for 2 more weeks. Heart block disappeared spontaneously in 48 h and his rhythm returned to sinus with 1:1 conduction (Fig. 3). His cardiac function by echocardiogram and heart size on chest x-ray have returned to normal. He underwent a myocardial biopsy (Fig. 4) during this acute phase of the illness. The patient was discharged on medications 2 weeks from admission. Since discharge he has remained asymptomatic and maintained a sinus rhythm on Holter monitor on many occasions.
Fig. 1. An electrocardiogram showing CHB with a ventricular rate of 30 beats/min. Note the blocked P waves are seen in lead V1. Also, this electrocardiogram is showing evidence of left-axis deviation with RBBB.

Fig. 2. Chest x-ray showing a generalized cardiomegaly with a transvenous pacemaker catheter tip in the right ventricle.

Fig. 3. An electrocardiogram taken 48 h after the one shown in Fig. 1. It shows a normal sinus rhythm with left-axis deviation and LBBB.

Fig. 4. Myocardial biopsy showing diffuse infiltration of lymphocytes and histocytes with myocyte necrosis. Interstitial edema is present with some early collagen deposit. Viral inclusions are not detected.

Laboratory Data

Mycoplasma immuno gamma-M globulin (immuno fluorescent antibody) dilution titer was 10 in the acute stage and was increased to 20 in the convalescent stage. The IGG dilution titer was less than 8 (IFA test). The following viral acute and convalescent titers were negative: Coxsackie B (1–6), influenza A and B, parainfluenza (1–3), adenovirus, cytomegalovirus, rheovirus, respiratory syncitial virus (RSV), and mumps. The nasopharyngeal viral culture and bacterial culture for group A streptococcus were negative.

Collagen profile including antinuclear antibodies, double-strand DNA, and rheumatoid factors were negative. The cardiac enzymes were elevated. Creatine phosphokinase and lactate dehydrogenase were as high as 1036 and 592 U, respectively, and the sedimentation rate was 32 mm/h.

Discussion

In 1944, Eaton et al. [6] described a pathologic agent thought to cause atypical pneumonia. In 1962, Chanock et al. [4] identified this agent as mycoplasma, which is now known as Mycoplasma pneumoniae. It was isolated from patients with pneumonia and upper respiratory tract infections in school-age children. Rarely has this organism affected the cardiovascular system. Myocarditis from M. pneumoniae (Eaton agent) was reported in 1970 by Lewes and Rainford [14]. Gerzén et al. [8] in 1972 identified M. pneumoniae as the cause of myocarditis in several patients. Pönkä in 1979 [20] reported 560 patients with serologically proven M. pneumoniae infection, 25 (4.5%) had carditis, 19 perimyocarditis, and six pericarditis, none presented with CHB. Friedli et al. [7] in 1977 reported an 18-month-old infant presenting with a syncopal episode preceded by URI-like symptoms found to have CHB, presumably due to M. pneumoniae.