

Complete Heart Block In Mumps Myocarditis

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Abstract

Myocarditis is a relatively common complication occurring in Mumps (epidemic parotitis). Inflammation of the myocardium causes complications in some of these cases. Observation of complete heart block in patients of acute mumps myocarditis is a rare instance. We report a case of complete heart block in a patient suffering from mumps.

Key Words

Myocarditis, Mumps, Complete Heart Block.

Introduction

Epidemic parotitis was first reported in the 5th century BC by Hippocrates and the possibility of myocarditis secondary to systemic mumps infection was identified in 1918 (1). Electrocardiographic manifestations were described in 1944 and complete heart block as an electrocardiographic manifestation was first reported in 1943 (2).

Case Report

The patient, a 12 years old school boy, was in a good state of health when he noted pain in left side of neck which was followed by swelling in the same region. The local physician's consultation was taken the next day who noticed bilateral parotid swelling, enlargement of submandibular gland and tender lymph nodes at the angle of left jaw (Fig 1). Two days later he developed fever of 104 F and pulse of 35/min. was recorded. He was referred to the department of cardiology of our institute. On the basis of these complaints, the patient was admitted and upon admission physical examination revealed a well developed, well nourished boy of the stated age. The temperature was 102 F; the pulse rate was 25/min; the respiratory rate was 26/min; and the blood pressure was 110/70 mm Hg. The other positive findings of the physical examination were as follows: swelling of the right parotid gland, intermittent cannon 'a' waves in jugular venous pulse, and changing intensities of heart sounds. An electrocardiogram showed complete heart

block with the atrial rate of 115/min; and ventricular escape rate of 22/min.(Fig.2).



Fig: 1 Bilateral Parotid swelling

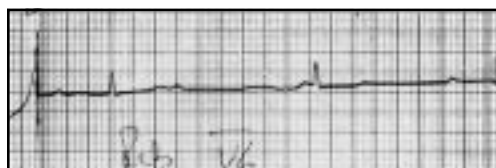


Fig: 2 ECG showing complete heart block

The laboratory data revealed a total leukocyte count of 9,520/mm³ with 83% polymorphs, 15% lymphocytes, and 2% monocytes, and a normal erythrocyte sedimentation rate; Urine analysis showed no abnormality; blood sugar, blood urea nitrogen, serum electrolytes, proteins, calcium, alkaline phosphatase and serum transaminases were normal; blood culture was negative; chest roentgenogram was normal. Mumps specific IgM ELISA was 1:20 and rose to 1:80 after one week later.

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Other viral serology tests were negative. The serum amylase level was 260 μ /L with a normal lipase level of 3.0 μ /L and creatine phosphokinase increased from 320 μ /L on day 1st to 595 μ /L on day 3rd of admission. Echocardiogram was normal.

The therapy was entirely symptomatic for parotitis and fever, supported by temporary pacing lead placed in right ventricle and connected to external generator. The patient became afebrile on 3rd day; resumed sinus rhythm on the 4th day; and was discharged on 10th day after admission. However, four days after discharge he was again brought to our hospital with history of 'recurrent syncopal attacks. A plan of putting in the temporary pacing lead was made but before patient could be shifted to the cath lab. for the same he was declared dead.

Discussion

The diagnosis of acute mumps myocarditis complicated by complete heart block in the patient appears justified. The clinical picture was compatible; IgM ELISA for mumps and creatine phosphokinase was elevated. The electrocardiographic abnormality persisted in this patient for five days only. It is regrettable that we were unable to acquire additional periodic observation on this patient. It appears that the mumps myocarditis is nonspecific in nature and gives rise to symptoms and signs which differ in no respect from myocarditis from other causes (3). Biopsy evidence of myocarditis has been reported in 8%-60% of patients having life threatening arrhythmias and normal ventricular function (4). The reported changes in mumps occur in 4.4 to 15.4%. The exact incidence of complete heart block and its relation to severity of myocarditis remains unanswered. However, all types of rhythm disturbance could occur including torsades-de pointes tachycardia (5), lone atrial fibrillation (6) and heart blocks. The combination of tachycardia and atrioventricular (AV) block in a patient is suggestive of inflammatory myocarditis (7). Structural alteration conducive to tachycardia may occur due to inflammation, patchy fibrosis, microaneurysms; or may even result from more subtle alterations in sarcolemmal permeability induced by the virus. It is important to determine whether myocarditis is the cause of arrhythmia because the prognosis may be favorable. However, the management of patients of myocarditis and ventricular arrhythmias is problematic. The arrhythmia is not inducible in the laboratory in two thirds of such cases and though immunosuppressive

therapy may be helpful in some, sudden death can occur (8). Similarly, myocarditis in some have predilection for conduction system and present with AV blocks. It accounted for 8-12 % of the cases of sudden death (9).

The prolongation of PR interval and related bradyarrhythmias appear either to result from increased vagal tone, a response similar to that observed in acute rheumatic carditis, or related to myocardial inflammation itself. In patients of acute rheumatic fever, heart failure seems to result from valvulitis and not myocarditis. Myocardial biopsy is not diagnostic in these patients. Very rarely, intense inflammation of the papillary muscle may generate a pseudo-tumor in the ventricle, confusing the clinical diagnosis (10).

To conclude from this case report and others in literature, the awareness of this complication in mumps, may lead to its increasing recognition and treatment, and reduce morbidity and mortality in these patients. Moreover, complete heart block may occur intermittently, so these patients should be followed electrocardiographically for many weeks to manage this complication.

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