Acute Rheumatic Carditis Manifesting As Complete Heart Block At Initial Presentation In A Young Male – A Rare Case Report

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Abstract — A 26-year male came to the emergency department with a history of syncope along with fever and joint pains of two weeks duration. The electrocardiogram showed a complete heart block (CHB). On further workup, the CHB appeared secondary to Acute Rheumatic Carditis (ARC). The CHB recovered over a week with anti-inflammatory therapy. This case shows that ARC can manifest with a complete heart block and syncope, which is reversible.

Index Terms — acute rheumatic carditis; complete heart block; corticosteroids; syncope.

I. INTRODUCTION

Acute Rheumatic Fever (ARF) manifests as a delayed autoimmune response to group A beta-hemolytic Streptococcal pharyngitis. It manifests as a generalized inflammatory process involving predominantly the heart and joints, along with the involvement of the central nervous system, the subcutaneous tissue, and the skin. All the manifestations and the cardiac conduction abnormalities except for the carditis (valvular involvement) are self-limiting and leave no significant residual damage. Among the conduction abnormalities, the first-degree atioventricular block is most common, followed by the second-degree atioventricular block. The complete heart block (CHB) is rare [1]. ARF commonly affects children between ages 5 and 15 years [1]. The occurrence of ARF as the initial episode in adults is rare. Here we report a case of complete heart block as a manifestation of acute rheumatic carditis at initial presentation in a young male along with the review of the literature.

II. CASE PRESENTATION

A 26-year male came from an outside hospital with a history of syncope. Investigations confirmed a complete heart block. A temporary pacemaker from a trans-venous route helped in managing the arrhythmia.

He reported being having fever and joint pains for last two weeks. It was associated with chills and rigors. The joint pains were migratory, involving the large joints (ankle, knee, and then the wrist). There were no associated with skin lesions, seizures. Next week, he developed palpitations and shortness of breath, followed by two episodes of presyncope and syncope, for which he visited a local hospital. He was diagnosed having a complete heart block and referred for further management.

Physical examination revealed the following; heart rate of 45 per min (see Fig. 1), blood pressure was 100/70 mm Hg, and fever 39 °C. Intermittent cannon waves in Jugular venous pulse, mildly swollen right wrist, and ankle joints noticed. Cardiovascular system examination revealed a pansystolic murmur at apex.

On investigation, he had a raised total leucocyte count (TLC) 18,000 cells/mm3 with differentials N 86, L 13, M01. E01. He had a hemoglobin of 12.3 gm/dl. The electrocardiogram showed a complete heart block with a heart rate of 45/min, varying ventricular beat morphology between narrow complex and left bundle branch block (see Fig. 1).

Fig. 1. Electrocardiogram at admission showing complete atrioventricular block (p waves marching through the QRS complexes) and the ventricular escape rhythm morphology varying between narrow complexes (first two complexes of rhythm strip ) and broad complex of left bundle branch morphology (third and fourth complexes of rhythm strip).

The echocardiogram showed moderate mitral regurgitation (see Fig. 2), mild aortic regurgitation. LV ejection fraction – 55%. There was mild pericardial effusion with no evidence of tamponade. His ESR was 56/mm in the first hour. His CRP was 36.5mg/dl, and the ASO titer was >200 on sequential samples. His Chest X-ray PA view showed mild right pleural effusion with a normal cardiac silhouette. The positron emission tomography (PET) scan showed no hilar lymphadenopathy and increased cardiac uptake (see Fig. 3). Cardiac Magnetic Resonance imaging showed late gadolinium enhancement (LGE) in the
epicardium of inferior and left lateral walls and mid-myocardium of the middle and distal septum (see Fig. 4). The T2 weighted images showed no myocardial edema – normal myocardial perfusion at rest. The patient was negative for leptospirosis, brucella, Lyme disease serology. The final diagnosis was syncope due to complete heart block in the course of acute rheumatic carditis.

He received Benzathine Penicillin 12 lakh units deep intramuscularly. Anti-inflammatory therapy with Aspirin 600 qds was started along with prophylaxis for gastric ulcers. Prednisolone 60mg once daily was given for five days.

The complete heart block improved to first-degree atrioventricular block (see fig.5) in the next five days, and the temporary pacemaker removed. After tapering for two weeks, Prednisolone stopped. The aspirin was decreased to 300 mg qds after two weeks of the resolution of the swelling of joints and continued for the next ten weeks. He received Penicillin prophylaxis once every three weeks for secondary prevention.

At three months of follow up, his electrocardiogram showed a normal PR interval. The echocardiogram showed mild mitral regurgitation, with normal left ventricular function. There was no pericardial effusion.

III. DISCUSSION

The electrocardiogram is a valuable investigation in acute rheumatic fever (ARF). The most common finding is a prolonged PR interval. Increased vagotonia is the reason behind prolongation of PR interval rather than the carditis [1].

The most extensive series of conduction abnormalities in ARF comes from Filberbaum et al. in 1960, who documented rhythm abnormalities from sinus arrest to atrial, junctional tachycardia and complete atrioventricular block [2].
In about 50% cases, the etiology of the complete atrioventricular block is unclear in young (age <55 years). The most common causes found are as a complication to cardiac surgery, congenital atrioventricular block, congenital heart disease, or as a complication of radiofrequency ablation, myocarditis, cardiomyopathies, and inflammatory diseases. In our patient, the presence of fever, increased leukocyte count, and raised CRP, led to the consideration of inflammatory disease. The recent onset of symptoms and no previous heart disease rules out the complication of cardiac surgery and congenital etiology [3].

Prolongation of the PR interval seen in almost 70% of cases [1,4]. The second degree AV block - Mobitz type I was the next common abnormality seen with an incidence of 1.5% [1] – 2.6% [4]. The complete AV block occurs in 0.6% of cases [1] and 4.6% of cases [4]. The documented cases of complete AV block in the literature due to acute rheumatic carditis to date are 31 [5] and frequently seen in the pediatric age group 19/31 (61.2%) [5]. The AV block was transient, lasting from a few minutes to days, seven patients required permanent pacemaker implantation [5]–[7]. Our patient was 26 years old, had transient CHB recovered over five days after using prednisolone.

Treatment of the conduction system involvement with ARF is in the same lines as the ARF treatment for carditis. The inflammation of the conduction system leading to the complete heart block hypothesis holds good as the patient improves with anti-inflammatory agents, and the response monitored by a decrease in inflammatory markers like C-reactive protein levels [4]. In patients with symptomatic advance AV block temporary pacemaker placement to be done.

Patients with ARF should receive treatment for Streptococcal infection with or without evidence of pharyngitis. [8]. The treatment includes either oral Penicillin for ten days or a single dose of intramuscular Penicillin [8]. Aspirin in high doses (80–100 mg/kg in divided doses/day) is the drug of choice when there is arthritis. The resolution of the swelling of joints in 48 hours of aspirin is considered the hallmark of ARF [8]. The role of systemic corticosteroids is less clear [8]. Carano and colleagues, as well as Hubail patient, received steroids as part of his treatment regimen [5].

Secondary prevention for recurrent ARF should follow after treatment of the acute phase. Three weekly intramuscular injections are preferred. For patients with a history of carditis (including conduction system involvement), the treatment duration is at least ten years after the initial attack or until age 40 years, whichever is longer. There are some suggestions to receive lifelong prophylaxis in patients with a history of severe carditis [8].

IV. Conclusion

Acute rheumatic fever manifesting as an initial episode in a young male is rare. Carditis manifests as valvulitis, with mitral being more involved than the aortic. The most common conduction abnormality is the prolongation of PR Interval, and few cases with CHB are not uncommon. The CHB correlates with the degree of inflammation as monitored by CRP and the ASO titer levels. It is usually transient, though rarely some need permanent pacemaker implantation.

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