



# Syncope due to complete atrioventricular block and treatment with a transient pacemaker in acute rheumatic fever

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## Abstract

Various rhythm and connection disorders can be seen in the acute phase of acute rheumatic fever. First degree atrioventricular block, one of the minor signs of acute rheumatic fever, is the most common connection disturbance in this disease. Complete atrioventricular block, which seriously affects the conduction pathways, is rare in the literature. A 15-year-old boy was admitted because of syncope caused by complete atrioventricular block and a temporary pacemaker was employed because of symptomatic complete atrioventricu-

lar block. The transient pacemaker treatment was terminated due to recovery of complete atrioventricular block on the third day of antiinflammatory treatment. Acute rheumatic fever should be kept in mind as a possible cause of acquired complete atrioventricular block. Connection disturbances in acute rheumatic fever improve with antiinflammatory treatment. Transient pacemaker treatment is indicated for patients with symptomatic transient complete atrioventricular block.

**Keywords:** Acute rheumatic fever, complete atrioventricular block, transient pacemaker

## Introduction

Acute rheumatic fever (ARA) is an autoimmune, multi-system disease that develops in response to group A streptococcus pharyngitis. Carditis is the most important major finding of the disease. Rheumatic heart disease, which is a complication of carditis, continues to be an important public health problem in developing countries (1).

Carditis in ARA is pancarditis. However, endocarditis, which leads to valvular regurgitation, is observed much more frequently in clinical practice. Conduction pathways may be affected in relation to pancarditis. Prolongation in the PR interval on electrocardiography (ECG) is a minor diagnostic finding of rheumatic fever. Complete atrioventricular block (AVB) with serious involvement of the conduction pathways has been reported

only as case presentations in the literature (2). In this article, a patient who presented with syncope due to complete AVB and his treatment are presented.

## Case

A 15-year-old male patient was admitted to the emergency department because of syncope. The patient, who had joint pain in the last days, had tonsillopharyngitis three weeks ago. On physical examination, his consciousness was closed and he had severe bradycardia (33 beats/min). The other physical examination findings were as follows: body weight: 49 kg (10-25 percentile), body temperature: 37.2°C, respiratory rate: 20/min, and blood pressure: 105/60 mm Hg. On auscultation, a 2°/6° systolic ejection murmur was heard. The ventricular rate was found as 33/min and complete AVB was observed in ECG (Picture 1). Echocardiographic ex-

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amination revealed first-degree mitral valve regurgitation and very mild aortic valve regurgitation. Systolic function of the left ventricle was found to be normal (EF: 58%, FS:30%) and dilatation was not found in the left ventricle (left ventricular end-diastolic diameter 41 mm, z score -1.37; left ventricular systolic diameter 28.8 mm, z score: 0.07).

Laboratory findings were as follows: white blood cell count: 13,470/mm<sup>3</sup>, acute phase reactants (erythrocyte

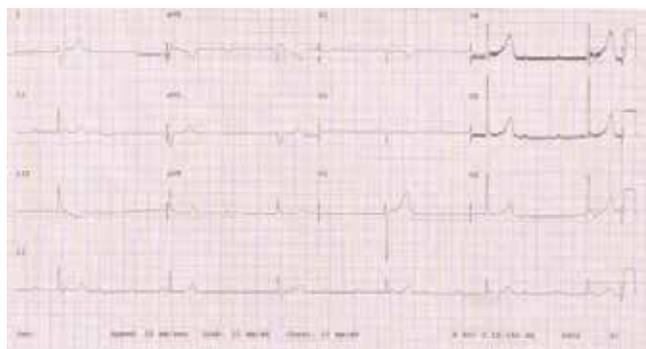
sedimentation rate: 26 mm/h, C-reactive protein: 37 mg/L) and antistreptolysin-O titer: 1270 IU/mL (increased). Troponin T and creatinine kinase-MB levels were found to be normal.

A transient pacemaker electrode was placed in the right ventricle by way of the right femoral vein because symptomatic complete AVB was present. The pacemaker was adjusted with VVI mode and a rate of 75/min (Picture 2). A diagnosis of ARA was made with carditis as one major finding and increased acute phase reactants and arthralgia as two minor findings with a supportive finding of increased antistreptolysin-O titer. Oral methylprednisolone was given at a dose of 16 mg three times a day as anti-inflammatory treatment. Benzathine penicillin G (1,200,000 IU, intramuscular) was administered and recommended to be administered every 21 days as prophylactic treatment. On the third day of steroid treatment, ECG showed first-degree AVB (PR 0.28 s, heart rate 110/min) (Picture 3). Thereupon the transient pacemaker treatment was terminated. Acute phase reactants became normal on the fourth day of anti-inflammatory treatment. After two weeks of methylprednisolone treatment, the dose of this drug was tapered. In the second week, oral acetylsalicylic acid was initiated at a dose of 3.5 g/day. In the third week of treatment, first-degree AVB was also recovered. At the end of treatment, an ECG examination revealed that aortic valve regurgitation was recovered, but the first-degree mitral valve regurgitation continued. Written informed consent was obtained from the patient's family.

**Discussion**

In the acute phase of ARA, many rhythm and conduction disorders may be observed independent of valve involvement, and these disorders generally recover with anti-inflammatory treatment (3).

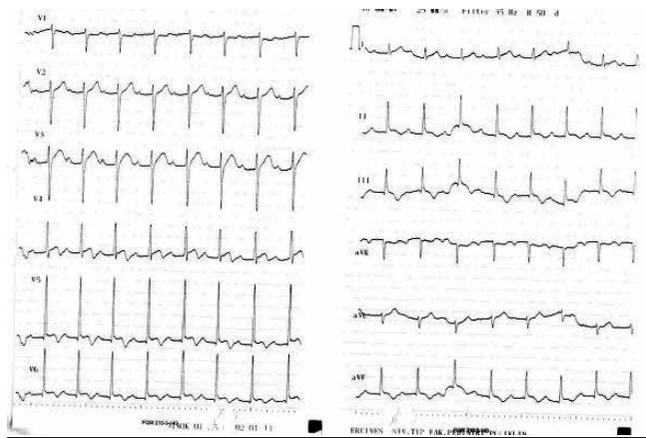
The most common conduction disorder in ARA is prolongation of the PR interval in ECG. In our retrospective incidence study involving 624 patients with ARA between 1998 and 2011, we found first-degree AVB in 17.4% of the subjects (1). This patient was our first patient who presented with complete AVB in our clinical practice. Carano et al. (4) recently reported a case similar to ours and reviewed the literature related to the issue. Nineteen of 25 subjects with complete AVB reported in the literature were in the childhood age group and seven of these presented with syncope episodes.



**Picture 1. Complete atrioventricular block with a ventricular rate of 33/min on electrocardiogram**



**Picture 2. Wide QRS on electrocardiography due to pacemaker**



**Picture 3. First-degree complete atrioventricular block on electrocardiography**

A transient pacemaker was employed in five of these seven patients. Atrioventricular blocks of all subjects whose outcomes were reported were recovered with anti-inflammatory treatment in eight days at the latest. In our case, complete recovery occurred on the third day, in accordance with the literature.

In children, complete AVB is most commonly observed as a result of a developmental disorder of atrioventricular node and structural cardiac diseases. The most common congenital diseases associated with complete AVB include atrioventricular septal defect (isolated or associated with left atrial isomerism) and corrected congenital transposition of the great arteries. Maternal autoimmune diseases (especially anti-SS-A/Ro and anti-SS-B/La antibodies) may lead to development of AVB. In addition, AVB may also develop as a result of damage to the atrioventricular node with surgical or catheter interventions. Infections, myopathies, and genetic mutations constitute other rare causes of complete AVB (5, 6).

Pacemaker indications in children were classified in the guideline for device-based therapy of cardiac rhythm disorders published in 2008. As in our patient, symptomatic complete AVB is included in complete AVB class 1 (recommended) (7). A transient pacemaker was employed in our patient because it was predicted that complete AVB would be recovered with anti-inflammatory treatment.

Acute rheumatic fever very rarely leads to complete AVB in children. Conduction disorders due to ARA recover with anti-inflammatory treatment. Treatment with a transient pacemaker is indicated in patients with symptomatic complete AVB due to acute rheumatic fever.

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