INTERNAL MEDICINE

□ CASE REPORT □

# Seizures and Syncope Due to Complete Atrioventricular Block in a Patient with Acute Myocarditis with a Normal Left Ventricular Systolic Function

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## Abstract

A 43-year-old man was admitted to our hospital presenting with seizures and syncope. He had a history of a cold with a fever of 39℃ occurring three days earlier. Electrocardiography (ECG) showed complete atrioventricular block (AV block) with a maximum pause of 32 seconds, for which temporary pacing was performed. Echocardiography showed mild hypertrophy of the left ventricle (LV) with a normal ejection fraction of 61%. Coronary angiography showed normal coronary arteries. Then, an endomyocardial biopsy was performed, the results of which indicated a diagnosis of acute myocarditis. After admission, the complete atrioventricular block disappeared together with normalization of the LV wall thickness.

**Key words:** Adams-Stokes syndrome, acute myocarditis, right bundle branch block, syncope

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## Introduction

Myocarditis, inflammation of the myocardium, is associated with a number of etiologies and complications. Although conduction disturbances, such as complete atrioventricular block (AV block), may occur as serious complications of myocarditis, AV block is generally considered to be a rare complication, especially in the absence of severe forms of myocarditis with cardiac decompensation. We herein report the case of an adult patient with seizures and syncope related to complete AV block due to acute myocarditis without cardiac decompensation.

## Case Report

A 43-year-old man was transferred by ambulance to our hospital to treat convulsions and syncope. He had three seizures in the ambulance. The electrocardiography ECG monitor in the ambulance showed complete AV block and an escape rhythm with a heart rate (HR) of 45 beats per minute (bpm). The monitor did not work during the seizures because the seizures induced electrical artifact in the ECG. The patient had experienced a cold with flu-like symptoms and a fever of 39℃ three days earlier. He had no past medical history, and chest X-ray and ECG performed at his medical check-up were both normal. His family history was unremarkable.

On admission, the patient's blood pressure was 110/70 mmHg, his pulse rate was 40 bpm and his temperature was 37.4℃. A physical examination revealed no abnormal findings. Chest X-ray obtained with the patient in the supine position showed cardiomegaly with clear lung fields (Fig. 1A). ECG showed complete AV block, left axis deviation (LAD), right bundle branch block (RBBB) and negative T waves in leads I, $aV_L$ and $V_{3\text{-}6}$ (Fig. 1B). Ultrasound cardiography showed mild hypertrophy of the left ventricle (LV) (an interventricular septum (IVS) of 11 mm and a posterior wall thickness of 11 mm), a normal LV ejection fraction of 61% and slight pericardial effusion (Fig. 1C).

The laboratory data obtained on admission are shown in Table. The hemoglobin level and white blood cell counts

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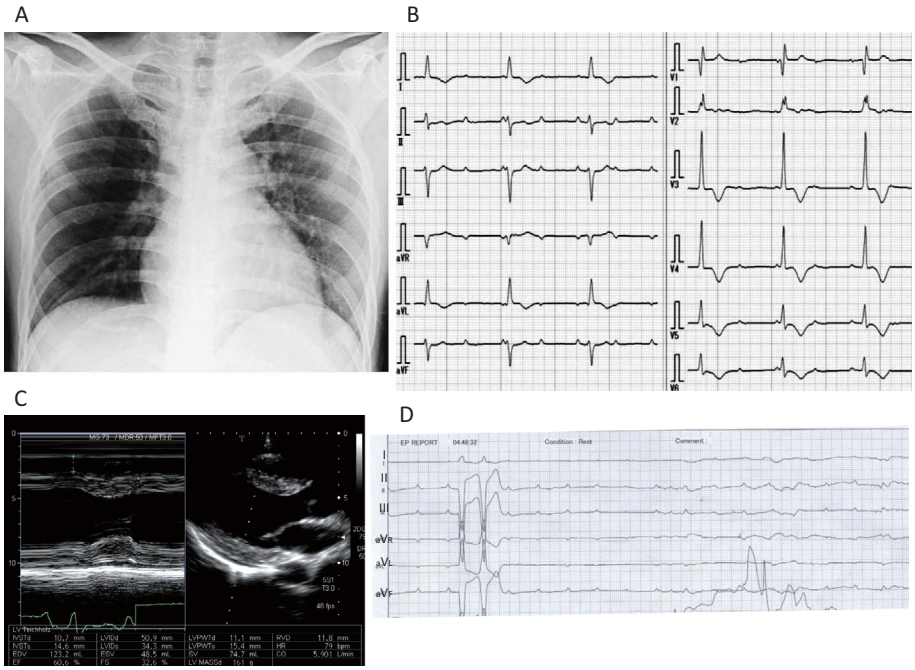


**Figure 1.** **A) Chest X-ray showing cardiomegaly without lung congestion. B) On admission, ECG showed complete AV block, left axis deviation (LAD), right bundle branch block (RBBB) and negative T waves in leads I, $aV_L$ and $V_{3\text{-}6}$. C) Echocardiography showed mild left ventricular (LV) hypertrophy, a normal LV ejection fraction of 61% and mild pericardial effusion. D) After admission, ECG showed a long pause of 32 seconds due to complete AV block with a sinus rate of 100 beats/min.**

**Table.** **Laboratory Data**

| | | | | | |
|---|---|---|---|---|---|
| WBC | 8,400/μL | TP | 6.8 g/dL | CK | 576 mg/dL |
| Neutrophil | 67.5% | Na | 140 mEq/L | CRP | 5.2 mg/dL |
| Eosinophil | 0.2% | K | 3.6 mEq/L | Troponin T | >2.0 ng/mL |
| Lymphcyte | 20.5% | Cl | 103 mEq/L | H-FABP | (+) |
| Monocyte | 10.8% | BUN | 24.4 mg/dL | BNP | 151 pg/mL |
| RBC | $464\times10^4$/ μL | Cr | 1.0 mg/dL | ACE | 17.3 U/L |
| Hb | 14.5 g/dL | AST | 131 IU/L | | |
| Hct | 42.2 % | ALT | 74 IU/L | | |
| PLT | $19.4\times10^4$/μL | LDH | 415 IU/L | | |

WBC: white blood cell, RBC: red blood cell, Hb: hemoglobin, Hct: hematocrit, PLT: platelets, TP: total protein, BUN: blood urea nitrogen, Cre: creatinine, AST: aspartate aminotransferase, ALT: alanine aminotransferase, LDH: lactate dehydrogenase, CK: creatine kinase, CRP: C-reactive protein, H-FABP: heart type fatty acid-binding protein, BNP: brain natriuretic peptide, ACE: angiotensin converting enzyme

were normal. The serum level of C-reactive protein was 5.2 mg/dL, and the serum levels of creatine kinase (CK), aspartate aminotransferase and lactate dehydrogenase were elevated on admission. The serum was positive for troponin T and the serum level of brain natriuretic peptide was 151 pg/mL.

No prominently increased levels of circulating virus antibodies were found using paired titers, including coxsackie virus A and B, echovirus, adenovirus and the influenza A and B viruses.

After admission, temporary pacing was immediately performed to treat complete AV block. During the procedure, the patient had a seizure with a maximum pause of 32 seconds on ECG due to the complete AV block (Fig. 1D). The complete AV block disappeared on the third day after admission without any medication administration. At that time, ECG demonstrated that the LAD had disappeared. Additionally, negative T waves were seen in leads II, III and $aV_F$, and poor R wave progression was seen in the precordial leads (Fig. 2). On the seventh day after admission, the negative T waves had disappeared, although RBBB was observed (Fig. 2).

The CK serum levels were the highest at the time of admission and then gradually decreased thereafter (576 IU/L on admission, 139 IU/L on the 3rd day and 77 IU/L on the 7th day).

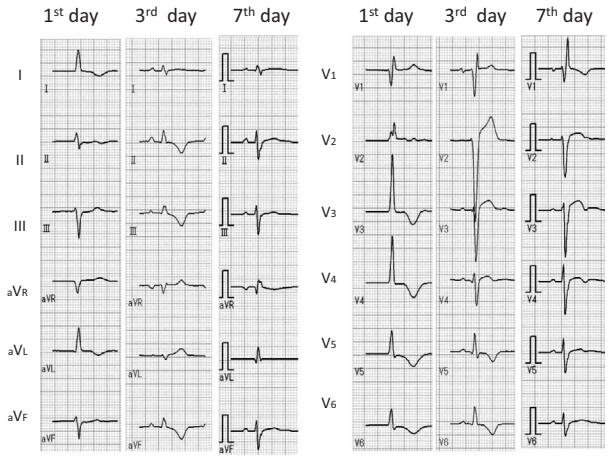


**Figure 2. The time course of the patient's ECG. On the third day after admission, ECG showed the disappearance of the LAD, the presence of negative T waves in leads II, III and $aV_F$ and poor R wave progression in the precordial leads. By the seventh day after admission, the negative T waves had disappeared, although RBBB was still seen.**

On the sixth day after admission, cardiac catheterization and an electrophysiological study were performed. Coronary angiography (CAG) showed no significant stenosis, and left ventriculography (LVG) demonstrated a normal ejection fraction of 62.1% (Fig. 3). An endomyocardial biopsy was also performed to evaluate the cause of the complete AV block. Histological studies of the biopsy specimen demonstrated lymphocytic infiltration, interstitial edema, interstitial fibrosis and myocyte necrosis, although there were no findings of specific types of myocarditis such as giant cells or eosinophil infiltration (Fig. 4). These findings suggested a diagnosis of acute lymphocytic myocarditis.

Permanent pacemaker implantation was not required because a treadmill test did not induce AV block and the electrophysiological study (EPS) suggested a preserved AV conduction function, as indicated by an (AA) of 792 ms, an (AH) of 100 ms, an (HV) of 49 ms, a sinus node recovery time of 1,069 ms and a Wenckebach point of 100 bpm. Twenty days after admission, after observing normalization of the LV wall thickness and disappearance of the pericardial effusion on UCG, the patient was discharged from the hospital.

Follow-up performed approximately two months after admission showed a normal chest X-ray. Additionally, follow-up ECG showed persistence of the RBBB with amelioration of the R wave progression and follow-up UCG showed a normal LV systolic function without pericardial effusion (Fig. 5).

## Discussion

Cardiac arrhythmias such as complete AV block can occur with nonspecific and specific myocarditis, including that due to sarcoidosis. Previous reports have shown that Adams-Stokes attacks or syncope can occur due to the sudden onset of complete AV block in patients with acute nonspecific myocarditis, especially children and young adults (1-16). There are eight previous reports of seizures occurring due to complete AV block with acute myocarditis in children and adolescents (1, 7-10, 14-16) and only one precise case in adults (13). This indicates that children and young adults are particularly vulnerable to these complications.

Our patient experienced seizures due to complete AV block with cardiac arrest of 32 seconds duration resulting from acute myocarditis that was successfully treated with a temporary pacemaker. Complete AV block in this patient was thought to be an isolated feature that could be rapidly and fully addressed if diagnosed early and treated with emergency pacemaker implantation (17). However, deaths from complete AV block leading to ventricular arrest have been reported (18, 19), and sudden cardiac death may be the initial presentation of myocarditis in some patients with complete AV block or ventricular tachycardia.

The outcomes of patients with complete AV block complicated by myocarditis are variable. Most patients recover normal heart function after suffering from myocarditis with complete AV block. The incidence of persistence of complete AV block reported in previous studies is 22% to 27% (1, 2).

The myocardial damage that occurred in our patient was mild and no typical cardiac symptoms were observed, such as dyspnea due to heart failure, chest pain related to the myocarditis itself or associated pericarditis. Although the complete AV block observed in the present case was severe, it disappeared three days after its occurrence, with EPS showing normal AV conduction after the patient's recovery. Previous reports have demonstrated that the average time for recovery from complete AV block in children is 3.3 days (2). Arima et al. (13) reported an adult case of myocarditis involving seizures due to complete AV block. However, their patient had fulminant myocarditis, persistent complete AV block and a recurrence of myocarditis. Previous studies on the clinical and experimental histopathology of myocarditis suggest that myocardial interstitial edema and neural tissue damage are implicated in the transient conduction disturbances of acute myocarditis (20, 21). This suggests that transient complete AV block occurring in patients with acute myocarditis, as in our case, can develop even in the absence of severe myocarditis and has the potential to cause direct damage to the AV conduction system. In this case, the precise myocardial damage area could not be evaluated because the patient did not undergo other imaging studies such as myocardial scintigraphy or gadolinium-enhanced cardiac magnetic resonance imaging (cMRI). Therefore, the presence of injury or lesions around the AV node could not be determined.

In the present case, seizures and syncope were the primary symptoms. Mahoney et al. stated that Stokes-Adams seizures may be the only clinical manifestation of myocarditis with complete AV block in children (22). Therefore, in some cases, the diagnosis of acute myocarditis may be diffi-

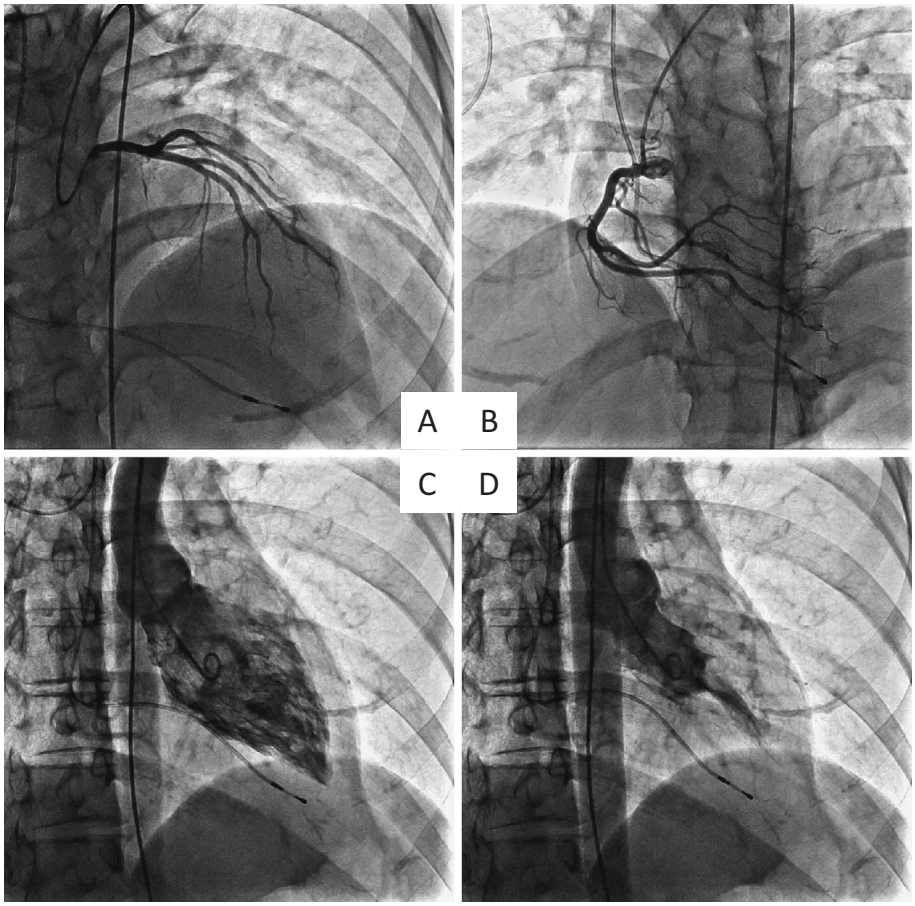


**Figure 3.** **Coronary angiography (CAG) showed no significant stenosis, and left ventriculography (LVG) demonstrated a normal ejection fraction of 62.1% (A, CAG of the left coronary artery; B, CAG of the right coronary artery; C, the end-diastolic phase of LVG; D, the end-systolic phase of LVG).**

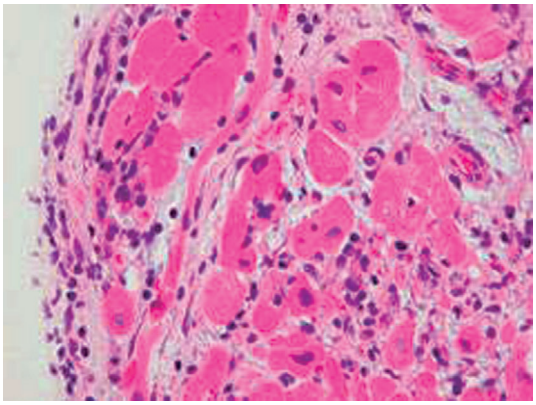

**Figure 4.** **A microphotograph of the endomyocardial biopsy of the left ventricle demonstrating lymphocytic infiltration, interstitial edema, interstitial fibrosis and myocyte necrosis. However, there were no findings of specific myocarditis such as giant cells or eosinophil infiltration (Hematoxylin and Eosin staining, magnification ×100).**

cult to make using only ordinary non-invasive examinations. In our case, UCG showed a normal LV systolic function with mild LV hypertrophy due to myocardial edema associated with the myocarditis and slight pericardial effusion. An endomyocardial biopsy was performed to determine the diagnosis of acute myocarditis.

Gadolinium-enhanced cardiac magnetic resonance imaging (cMRI) is able to detect myocarditis-related injuries. Prochnau et al. (23) reported that gadolinium-enhanced cMRI is useful for diagnosing acute myocarditis with complete AV block. However, their patient had persistent complete AV block, and MRI cannot be performed in patients with temporary pacing or permanent pacemaker implantation. Hence, endomyocardial biopsies may be clinically indicated for diagnosing complete AV block in patients with acute myocarditis. Endomyocardial biopsies also provide information regarding the post-treatment status of patients. Based on the guidelines related to regarding the use of endomyocardial biopsies in patients with myocarditis, endomyocardial biopsies are an important diagnostic tool for making a diagnosis of acute myocarditis, including giant cell myocarditis and eosinonphilic myocarditis, in order to decide whether to administer corticosteroids and/or immunosuppressants (24, 25).

The cause of myocarditis was not determined in the present case. Previous reports in adults have demonstrated that Lyme disease, a tick-borne spirochetal infection, is a cause of transient complete AV block. Lyme disease is diagnosed

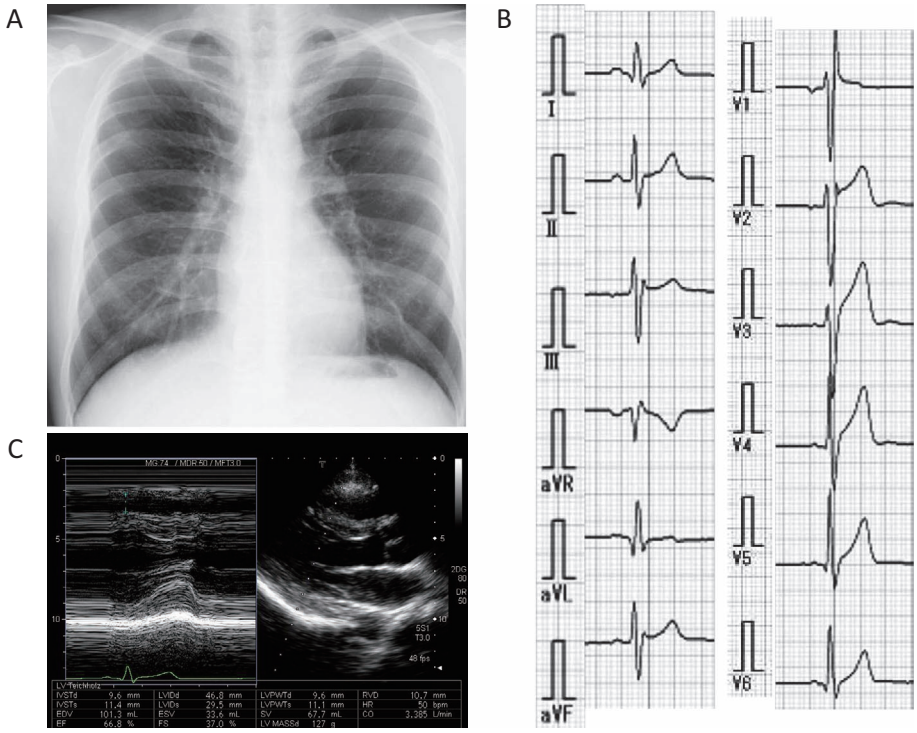


**Figure 5.** **Follow-up of the patient approximately two months after admission showed a normal chest X-ray (A). Follow-up ECG showed persistence of the RBBB and amelioration of the R wave progression (B), and follow-up UCG showed a normal LV systolic function without pericardial effusion (C).**

using serologic tests and treated with antibiotics (26). Therefore, we considered Lyme disease in the differential etiological diagnosis of our patient's symptoms. However, the patient's clinical course and the results of the endomyocardial biopsy were more suggestive of either a nonspecific or viral infection.

In conclusion, a diagnosis of acute myocarditis should be considered in patients presenting with seizures or syncope after the occurrence of flu-like symptoms, even in the absence of symptoms of heart failure, chest pain or signs of cardiac dysfunction.

**The authors state that they have no Conflict of Interest (COI).**

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