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【 CASE REPORT 】

# Paroxysmal Atrioventricular Block in a Relatively Young Patient with COVID-19

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**Abstract:**
Cardiac involvement has been reported in patients with coronavirus disease 2019 (COVID-19). We herein report a 41-year-old man who presented with recurrent paroxysmal atrioventricular block without showing significant cardiac injuries or comorbidities. The patient was diagnosed with COVID-19 and admitted to our hospital, where he was noted to have paroxysmal atrioventricular block. Cardiac biomarkers, echocardiography, and cardiac magnetic resonance imaging findings were fairly normal. An endomyocardial biopsy performed before the implantation of a permanent pacemaker revealed mild myocardial fibrosis without inflammatory infiltrates. The unusual myocardial involvement of the novel coronavirus was suspected.

**Key words:** COVID-19, bradycardia, atrioventricular block

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## Introduction

Although the hallmarks of coronavirus disease 2019 (COVID-19) are respiratory symptoms, the involvement of other systems, including the cardiovascular system, has been documented (1). In one study, approximately 16.7% of COVID-19 patients had cardiac arrhythmias (2); however, the effects of the disease on the conduction system of the heart have not been well reported.

We herein report a relatively young patient who developed recurrent paroxysmal atrioventricular block without features of significant myocardial injury or other comorbidities and required permanent pacemaker implantation.

## Case Report

A 41-year-old-Japanese man presented with a 6-day history of a high-grade fever, cough, and dyspnea. Although he had never undergone a medical checkup, it turned out that he had diabetes mellitus but no other significant comorbidities. He had never experienced syncope or dizziness.

On arrival at our hospital, he had a high-grade fever (38.5 °C) and tachycardia (120 beats/min); he weighed 118.0 kg and had a body mass index of 35.6 kg/m$^2$. Chest computed tomography revealed multifocal bilateral infiltrates (Fig. 1). His clinical features were highly suggestive of COVID-19, and an antigen test for severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) was performed, which yielded a positive result. His electrocardiogram on admission showed a normal sinus rhythm with normal PR (190 ms) and QRS (92 ms) intervals. No acute ST-T changes were noted (Fig. 2). His blood tests were remarkable with elevated liver enzyme levels [glutamic oxaloacetic transaminase (GOT) 151 U/L, glutamic pyruvic transaminase 187 U/L], inflammatory markers [C-reactive protein (CRP) 4.2 mg/dL], and HbA1c (7.8%). Other blood tests, including white blood cell counts, electrolytes, and his thyroid function, were normal.

Treatment with intravenous infusion of dexamethasone (6.6 mg/day) was initiated, and the disease course was good, except for recurrent paroxysmal atrioventricular block detected by telemetry from the second day of admission. Bradycardia lasted only a few seconds and caused no symp-

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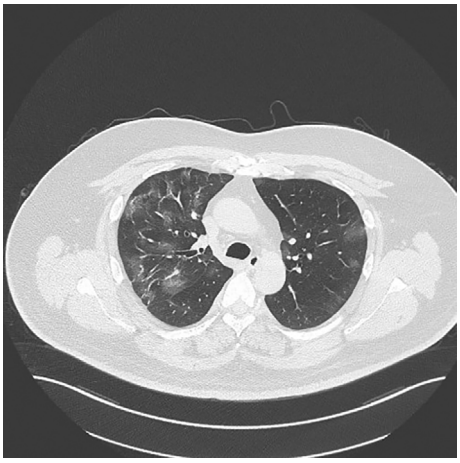

**Figure 1. Computed tomography of the chest showing multifocal bilateral ground-glass opacity pulmonary lesions.**

toms; thus, he was closely followed up without implanting a pacemaker. In the course of treatment, he showed no findings that indicated significant myocardial injury or comorbidity.

The level of serum troponin I on days 5 and 10 of admission were normal (2 pg/mL), and N-terminal prohormone of brain natriuretic peptide (NT-pro BNP) on day 10 was normal (55.3 pg/mL). Transthoracic echocardiography on day 10 showed a normal ejection fraction, with no wall motion abnormalities and no significant valvular disease. Cardiac magnetic resonance (CMR) imaging on day 10 revealed no myocardial edema or delayed myocardial enhancement (Fig. 3). Nevertheless, paroxysmal atrioventricular block occurred recurrently, and he experienced about 5.6. s of asystole without symptoms on day 10 of admission (Fig. 4). These episodes of atrioventricular block occurred regardless of whether he was awake or sleeping and occurred spontaneously in the absence of triggers that might stimulate parasympathetic activity, such as vomiting, micturition and intense coughing.

The patient was not on any medications with negative chronotropic effects. We therefore decided to implant a permanent pacemaker on day 10. Before implanting the pacemaker, we performed coronary angiography and an endomyocardial biopsy of the right ventricular septum. Coronary angiography revealed no significant lesion. A histological examination revealed mild myocardial fibrosis but no significant inflammatory infiltrates (Fig. 5). After the pacemaker was implanted, he continued to improve clinically and was discharged home on day 15.

## Discussion

We herein report a patient with COVID-19 who developed recurrent paroxysmal atrioventricular block that required implantation of a permanent pacemaker. To our knowledge, this is the first report of a COVID-19 patient who underwent CMR imaging and an endomyocardial biopsy before a permanent pacemaker was implanted. Disorders of the atrioventricular node occur occasionally in critically ill COVID-19 patients (3, 4). However, our patient showed only moderate symptoms of COVID-19. Kir et al., reported a similar case of transient atrioventricular block in a young patient who presented with normal transthoracic echocardiography findings and troponin I levels (5). In our case, in addition to these examinations, CMR and an endomyocardial biopsy were performed, and none of these examinations indicated significant cardiac involvement.

The telemetry strip on day 10 showed Wenckebach type atrioventricular block that developed into paroxysmal atrioventricular block, and the sinus rate slowed down progressively during ventricular asystole, indicating a vagal response (6). Given these findings, it is very unlikely that SARS-CoV-2 directly injured the myocardium; instead, it may have caused autonomic dysfunction. The myocardium is innervated by sympathetic and vagal parasympathetic nerve fibers. The wide expression of angiotensin-converting enzyme 2 (ACE2) in nerve tissues and the neurotropic nature of SARS-CoV-2 could make the cardiac nerve fibers a favorite prey (7). Previous reports regarding COVID-19-induced bradycardia have shown that the usefulness of a permanent pacemaker differs among cases (3, 4, 8, 9). In our patient, whether the possible autonomic dysfunction was permanent or temporary remains uncertain; however, the patient recurrently developed paroxysmal atrioventricular block, and the episodes occurred after 10 days of admission; whether or not the conduction disturbances were reversible was thus unclear. According to the pacemaker recordings made following his discharge, ventricular pacing was required intermittently even at three months after discharge. Although our patient did not show any symptoms related to bradycardia, he developed 5.6 seconds of asystole while he was awake. We therefore ultimately decided to implant a permanent pacemaker following the guidelines of Japanese Circulation Society. Although we did not perform the procedure this time, the adenosine triphosphate test could have clarified the necessity of implanting a permanent pacemaker (10).

Since this patient did not show any symptoms attributed to bradycardia, we cannot exclude the possibility that he latently had an underlying disorder of cardiac conduction. Nevertheless, considering his relatively young age and age-appropriate activity, it is plausible that he newly developed conductive disturbance after contacting COVID-19. In addition, the CMR and endomyocardial biopsy results did not indicate cardiomyopathy, which can cause conduction disturbance, such as cardiac sarcoidosis, giant cell myocarditis, and cardiac amyloidosis. We did not perform an electrophysiological study to determine the site of the conduction block. However, considering the involvement of the parasympathetic activity, the block site is presumed to be within the atrioventricular node.

At present, data regarding the neuroinvasive potential of SARS-CoV-2 with subsequent autonomic dysfunction are

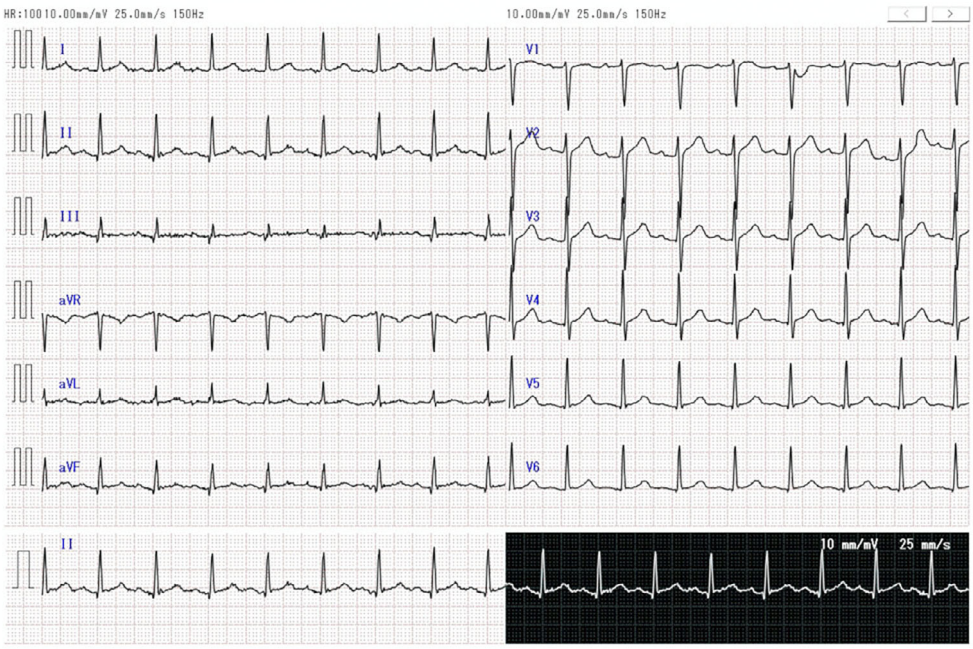


**Figure 2.** **A 12-lead electrocardiogram on presentation showing a normal QRS and PR interval.**

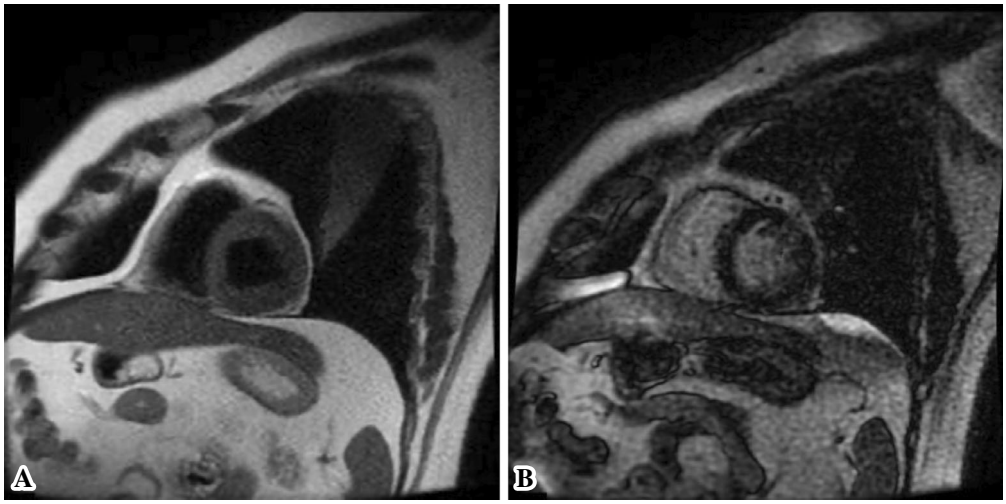


**Figure 3.** **(A) T2-weighted cardiac magnetic resonance imaging showing no obvious myocardial edema. (B) Late gadolinium enhancement is not seen.**

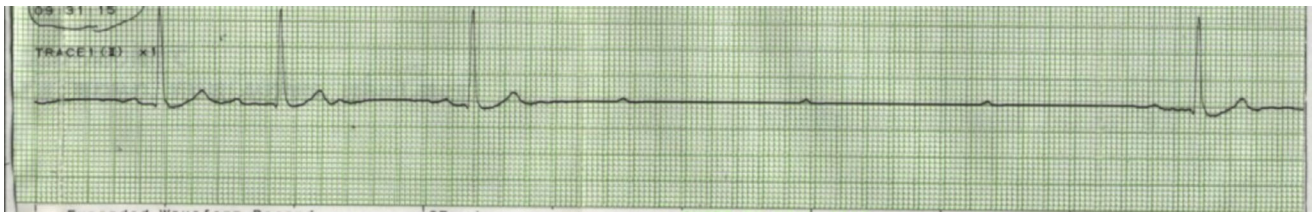

**Figure 4.** **Telemetry strip on day 10 showing about 5.6 s of asystole with advanced atrioventricular block. The sinus rate slowed down progressively during ventricular asystole. The time was 9:31 a.m., and the patient was awake.**

limited. We need to practice caution concerning potential conduction disturbances in COVID-19 patients, even when they are not critically ill and show no symptoms suggestive of significant cardiac involvement. These conduction distur-

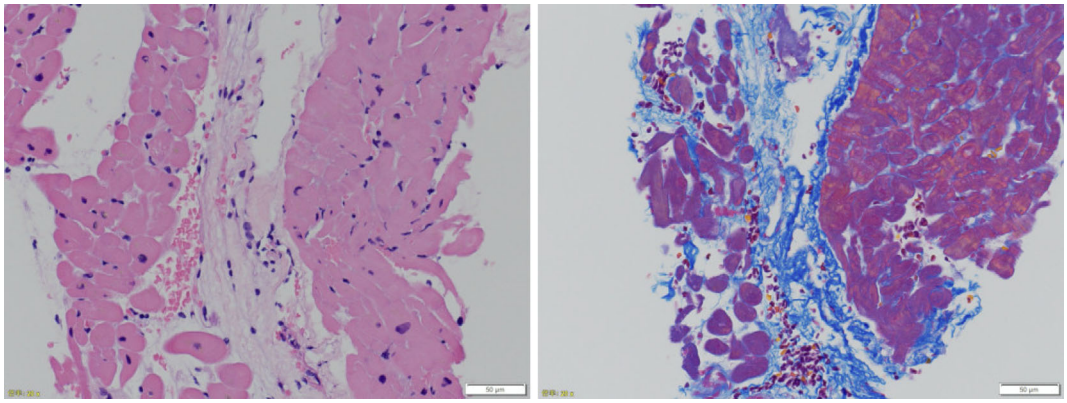

**Figure 5.** **(A) Hematoxylin and Eosin staining. No myocyte hypertrophy, disarrangement or lymphocyte infiltration are detected. (B) Azan staining. Mild perivascular exclusive myocardial fibrosis can be seen, but this is probably due to his diabetes mellitus, not myocarditis.**

bances can be recurrent or persistent; permanent pacemaker implantation might therefore be required for such patients.

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

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