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/m². 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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who underwent CMR imaging and an endomyocardial biopsy. 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 was 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 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...
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 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.
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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