

CASE REPORT

Reversible complete heart block in a patient with coronavirus disease 2019

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Abstract

Patients infected with novel coronavirus (SARS-CoV-2) can present with a variety of arrhythmias. We report an unusual case of reversible complete heart block (CHB) in the setting of acute coronavirus disease 2019 (COVID-19). A 23-year-old male with a history of Hodgkin's Lymphoma presented with dizziness and syncope. He was found to be in CHB associated with hypotension requiring a transvenous pacemaker. Methylprednisolone and remdesivir were started with rapid resolution of the CHB. Further study is needed to determine the mechanism of CHB in COVID-19. This case underscores the importance of including COVID-19 in one's differential diagnosis for acute CHB.

KEYWORDS

arrhythmia, complete heart block, COVID-19, myocarditis, transvenous pacing

1 | INTRODUCTION

The coronavirus disease 2019 (COVID-19) pandemic has had a profound global impact with over 120 million cases and 3 million fatalities worldwide.¹ Since the onset of this pandemic, cardiovascular complications have increased in prevalence.^{3,7} Myocardial involvement has been reported in 8%–28% of adults infected with SARS-CoV-2.⁵ Cardiac arrhythmias are common in patients with COVID-19.^{2,6,13} Supraventricular arrhythmias have been frequently reported, with atrial fibrillation being the most common. Ventricular arrhythmias have also been reported.³ Severe bradyarrhythmias are relatively rare with only a few cases reported in the literature.⁸ We report an unusual case of reversible complete heart block in a patient with acute SARS-CoV-2 infection and COVID-19 myocarditis.

2 | CASE REPORT

A 23-year-old male with a past medical history of Stage IIIB Hodgkin's lymphoma status post three cycles of brentuximab vedotin, doxorubicin, vinblastine, and dacarbazine who presented to the emergency

department (ED) complaining of dizziness and recurrent syncope. Initial vital signs were remarkable for a heart rate of 128, blood pressure of 114/63, and an oxygen saturation of 98% on 4 L nasal cannula oxygen. The initial 12-Lead ECG (ECG) demonstrated sinus tachycardia (HR 120) with a new right bundle branch block (RBBB) (Figure 1). Two weeks prior to this presentation, an ECG demonstrated sinus tachycardia (101 bpm) with normal intervals, narrow QRS, and QTc 448. In the ED, he developed complete heart block (Figure 2B, sinus rate 140 bpm, ventricular escape 40–58 bpm) with hypotension not responsive to fluid resuscitation. The patient was placed on an intravenous norepinephrine infusion. A transvenous temporary pacing (TVP) wire was inserted. The patient was not taking any atrioventricular (AV) nodal blocking agents.

Labs revealed an elevated high sensitivity Troponin-T (hs-TnT) 3.06 ng/ml, ferritin 6246 ng/ml, LDH 726 IU/L, D-dimer 1557 ng/ml, and C-reactive protein (CRP) 6.39 mg/dL. SARS-CoV-2 polymerase chain reaction (PCR) testing was positive with a low cycle threshold of 14, suggestive of a high SARS-CoV-2 viral load. SARS-CoV-2 IgG antibodies were negative, consistent with acute COVID-19 infection. Subsequent hs-TnT trended down to 2.88 ng/ml. Left heart catheterization demonstrated angiographically normal coronary arteries (Figure 3A).

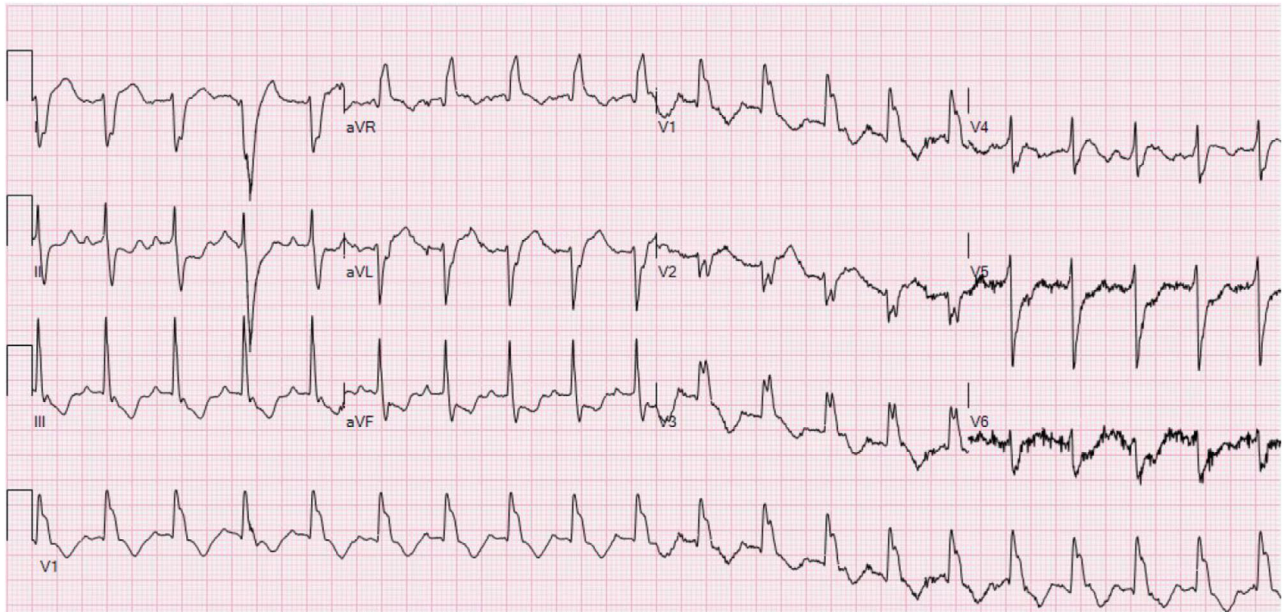


FIGURE 1 Initial EKG on presentation to emergency department [Color figure can be viewed at wileyonlinelibrary.com]

Right heart catheterization revealed RA 14, RV 39/13, PA 35/19, Mean PA 25, wedge pressure 18, left ventricular end-diastolic pressure (LVEDP) 19, cardiac output 3.1 L/min, and a severely depressed cardiac index of 1.74 L/min/m. The advanced heart failure team was consulted, and milrinone 0.25 mcg/kg/min was started. High dose intravenous methylprednisolone (1 g daily) and remdesivir were initiated for suspected acute COVID-19 myocarditis. He was admitted to the cardiac intensive care unit for close monitoring and further management.

Transthoracic echocardiogram (TTE) revealed a reduced ejection fraction (EF) of 35%–40% with moderate global hypokinesia, moderate concentric left ventricular hypertrophy, borderline dilation of the right ventricle (RV), mild RV hypertrophy, normal RV systolic function, no significant valvular disease, and a small pericardial effusion (Figure 3B). A TTE obtained 15 months prior to this presentation had shown an EF of 55%–60% with normal RV systolic function and normal diastolic filling pattern. Within 24 hours, the CHB resolved with improvement in hemodynamics. The patient was successfully titrated off all inotropic and pressor support. The RHC and TVP were removed. Endomyocardial biopsy was discussed, but given the prompt resolution of symptoms, continued hemodynamic stability, and absence of any further CHB or ventricular arrhythmias, it was ultimately not performed.

He completed a 5-day course of remdesivir and was started on a prolonged prednisone taper. Cardiac MRI (cMRI) was completed on hospital day 5 and revealed an EF of 47%, with no delayed gadolinium enhancement. With progressive clinical improvement, the patient was discharged home with close outpatient follow-up. A follow-up TTE completed 1 month after hospital discharge demonstrated full myocardial recovery (LVEF 55%–60%), normal RV systolic function, and trace pericardial effusion.

3 | DISCUSSION

Our patient developed reversible complete heart block in the setting of acute COVID-19 myocarditis. Few cases of complete heart block in patients with COVID-19 have been recently reported in the literature. Complete heart block has been reported both in patients with and without any pre-existing conduction disease.^{8,9,10} Cases of transient heart block in the setting of COVID-19 have been reported with subsequent resolution with clinical improvement.¹⁰ However, persistent heart block requiring pacemaker implantation despite resolution of COVID-19 has also been reported.^{8,9,11}

The mechanism of arrhythmias and heart block in COVID-19 remains unclear. This patient exhibited rapid resolution of CHB and rapid clinical improvement with early administration of high dose steroids and anti-viral therapy with remdesivir. Acute myocardial injury and cardiomyopathy is associated with an increased risk of arrhythmia. Acute myocarditis continues to be an important cause of AV block in young patients.

One purported mechanism of cardiac involvement is direct myocardial infiltration of the COVID-19 virus via the Angiotensin-Converting Enzyme 2 (ACE2) receptors, which are expressed throughout the myocardium.⁴ A more likely mechanism is a post-infection inflammatory response, owing to the rapid resolution with high dose steroid therapy. SARS-CoV-2 mRNA has been confirmed in postmortem analysis of myocardial specimens of human patients who had COVID-19 with elevated troponin and pro-BNP levels.^{2,15} The binding of SARS-CoV-2 to the ACE2 receptor has been shown to mediate myocardial inflammation and damage by down-regulating the ACE2 pathway and increasing levels of angiotensin II.^{12,14} In our case, the early initiation of high dose steroids and remdesivir likely resulted in rapid attenuation of the systemic inflammatory response associated with acute COVID-19

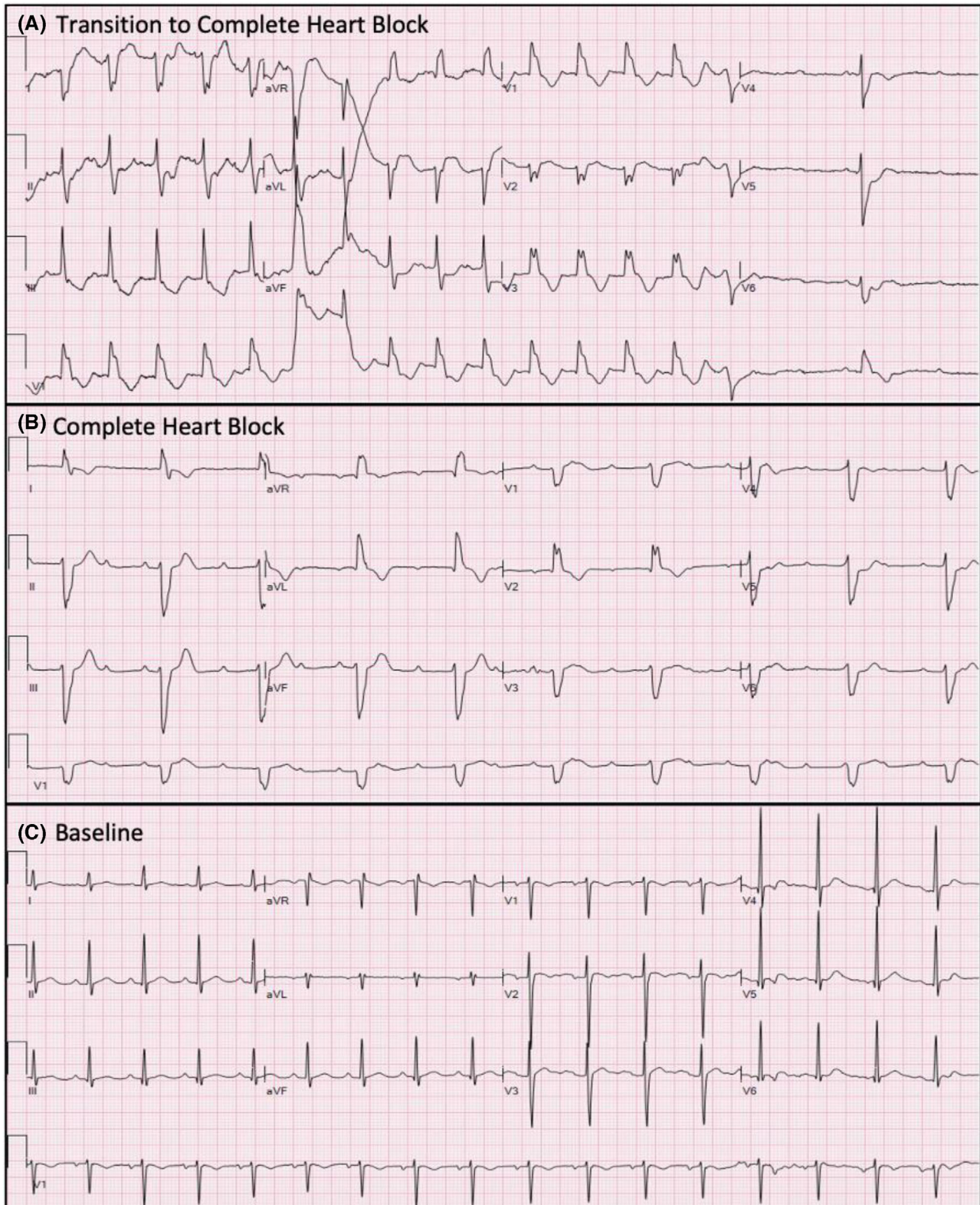


FIGURE 2 12 Lead EKGs. (A) Transition to complete heart block, (B) Sinus rhythm with complete heart block (sinus rate 140, ventricular escape 58), (C) Baseline EKG (2 weeks prior to presentation) showing sinus tachycardia, HR 101 [Color figure can be viewed at wileyonlinelibrary.com]

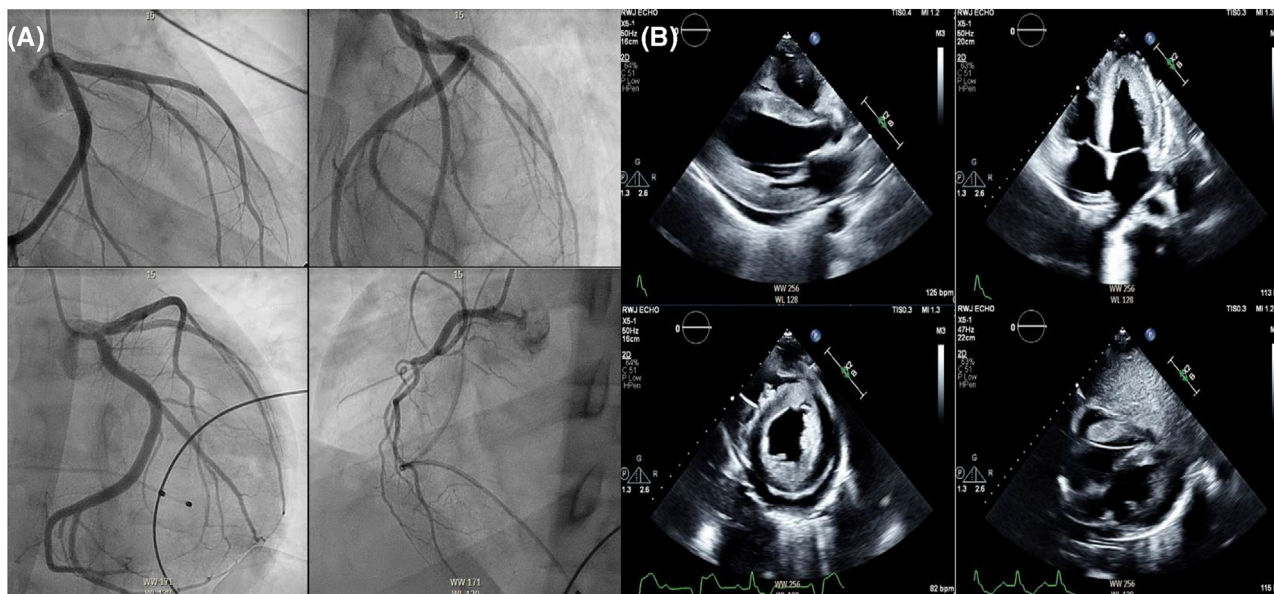


FIGURE 3 Left heart catheterization and transthoracic echocardiogram
(A) Left heart catheterization with angiographically normal coronaries
(B) Initial transthoracic echocardiogram on hospital day 1
 [Color figure can be viewed at wileyonlinelibrary.com]

infection and enabled prompt resolution of the complete heart block. Further study is needed to elucidate the underlying mechanism of conduction disease and heart block in patients infected with SARS-CoV-2. The impact of pre-existing cardiac conduction disease and concomitant co-morbidities on the risk of developing heart block in COVID-19 needs further investigation.

4 | CONCLUSIONS

Complete heart block is a relatively rare complication of SARS-CoV-2 infection. Additional studies are needed to determine the short and long-term impact of SARS-CoV-2 infection on the cardiac conduction system, underlying mechanism of CHB, risk factors for development heart block in COVID-19, and prognostic implications. This case underscores the importance of including COVID-19 in one's differential diagnosis for patients presenting in CHB. As in this case, a multi-specialty team-based approach involving electrophysiologists, advanced heart failure specialists, interventional cardiologists, and infectious diseases specialists can help facilitate optimal management in challenging cases of COVID-19 complicated by heart block, myocardial injury, and myocardial dysfunction.

CONFLICT OF INTEREST

No conflict of interest to be reported.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed for this case report.

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