Complete heart block in COVID-19 without prior cardiac disease

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ABSTRACT

Cardiac involvement in COVID-19 is not rare but underdiagnosed. We report a 78-year-old man with COVID-19 and complete heart block, timely managed by teamwork involving internist, cardiologist, and intensivist. This case highlights the importance of involvement of the conducting system of the heart in COVID-19 that needs immediate life-saving intervention, especially in community.

Keywords: Cardiovascular manifestations, coronavirus disease 2019, cytokine storm, heart block

Introduction

Complete heart block (CHB), also known as third-degree AV nodal block, results when no atrial impulses reach the ventricle. It can occur due to idiopathic fibrosis, myocardial ischemia (acute or chronic), amyloidosis, sarcoidosis, endocarditis, collagen vascular disorders, myocarditis, and so forth. Myocarditis or myocardial injury due to viral infections is a well-known phenomenon and has been associated with coxsackievirus, parvovirus-B19, HHV-6, EBV, and hepatitis viruses. Direct invasion of the infectious agent can lead to myonecrosis, and host immune responses get activated.¹ This can lead to arrhythmia. Viral infections have also been postulated to cause arrhythmias due to other mechanisms such as altered intercellular coupling, abnormal conduction due to cardiac fibrosis, abnormal calcium handling, and so on. However, the prevalence and pathogenesis of COVID-19-related arrhythmia especially CHB have been unclear due to the lack of specific diagnostic modalities to assess myocarditis. More importantly, primary physician should have suspicion of heart block among COVID-19 patients.

We report a case of an elderly man without prior cardiac disease who developed CHB, possibly secondary to COVID-19 infection.

Case Report

A 78-year-old man presented to the emergency with a 5-day history of mild to moderate continuous fever associated with chills and rigors and a 2-day history of insidious onset, gradually progressive shortness of breath that was associated with episodes of lightheadedness. Episodes lasted for a few minutes but were not associated with loss of consciousness, blurring of vision, double vision, or a ringing sensation in the ears. There was no history of orthopnea, chest pain, or palpitation. He had no other co-morbidity, no history of alcohol intake, or smoking. He had had no prior contact with healthcare.

On arrival, his blood pressure was 138/78 mmHg, pulse rate 37 beats per minute, and respiratory rate of 36 breaths per minute with 88% SpO2 on room air. Restricted systemic examinations (wearing personal protective equipment) were unremarkable. An electrocardiogram (ECG) done was suggestive
of CHB [Figure 1a]. Cardiology consultation was done for temporary pacemaker insertion (TPI). Post-procedure X-Ray was done and showed TPI in situ. Post-TPI insertion ECG was performed. The heart rate was set at 80 per minute, mode ventricular pacing, ventricular sensing, inhibition response (VVI), Voltage of 5 mv. Therapeutic anticoagulation was initiated with injection low-molecular-weight heparin 60 mg S/C BD. Screening transthoracic echocardiogram (2D) showed no structural pathology including normal ejection fraction and no clot, vegetation, pericardial effusion, and regional wall motion abnormalities. He had no history of receiving any atrioventricular node blocking agents. Nasopharyngeal swab for Covid-19 RT-PCR was found to be positive. Baseline arterial blood gas and electrolytes were normal. After stabilization, a detailed echocardiogram revealed mild LV systolic dysfunction and global LV hypokinesia with an LVEF of 45–50%.

His initial laboratory work-up suggested lymphopenia (400 cells/cumm; normal range 800-5000), elevated levels of aminotransferases (SGOT 105.4 U/L; normal range 0-40) and SGPT 57.3 U/L; normal range 0-45), acute kidney injury (blood urea nitrogen 104.2 mg/dL; normal range 13-43, and serum creatinine 1.35 mg/dL; normal range 0.5-1). He was also found to have elevated levels of serum D-dimer (2.23 microgram/ml; normal range <0.50), ferritin (781.5 mg/ml; normal range 10-291), NT-pro BNP (70 pg/dL; normal range <125), and CPK-MB (140.3 U/L; normal range 0-1). He was also found to have elevated levels of serum D-dimer (2.23 microgram/ml; normal range <0.50), ferritin (781.5 mg/ml; normal range 10-291), NT-pro BNP (70 pg/dL; normal range <125), and CPK-MB (140.3 U/L; normal range 0-1). A high-resolution CT scan of chest showed areas of ground-glass attenuation with interspersed areas of consolidation and interstitial septal thickening in bilateral lungs, with findings suggestive of Covid-19 pneumonia–CORADS 6, CTSS -26/40 [Figure 1b]. He received oxygen support (6 liters per minute) via a face mask. Injection dexamethasone 6 mg IV OD was given. During an ICU stay of 14 days, the patient was weaned off oxygen and shifted to the Cardiology department. Temporary pacemaker was removed under fluoroscopic guidance and a permanent pacemaker was inserted through left subclavian venous access [parameters: RV, Mode ventricular pacing, ventricular sensing, inhibition response and rate-adaptive (VVIR), Threshold 0.7 V @ 0.4 ms, Impedance 907 Ohms, R wave 8.2 mV]. Leads were connected to pulse generator, placed in infraclavicular pocket, and sutured in layers. Successful VVIR pacemaker implantation was done using a generator.

The patient was discharged under stable medical condition after a 7-day stay in the Cardiology ward. He was asked to follow-up after 1 month, but lost unfortunately due to COVID-19 peak second wave effect.

**Discussion**

This case exemplifies a life-threatening cardiac complication possibly due to COVID-19 infection. Considering the lack of angina or anginal equivalents as well as the absence of ST-T changes or a new-onset LBBB in a patient without any risk factor for accelerated atherosclerotic cardiovascular disease (no history of diabetes, hypertension, smoking, family history of cardiac illness) barring age, the possibility of a non-ischemic cardiac pathology was entertained. This suspicion was further supported by the negative cardiac enzyme levels. Additionally, the patient had no history of receiving beta-blockers or digoxin or any other cardiac medicines. The patient also did not give any history of unintentional weight loss or constitutional symptoms suggestive of an occult malignancy, neither had features suggestive of hypothyroidism nor adrenal insufficiency. As a result, a diagnosis of viral-induced myocardial injury and secondary CHB was made. For community practitioners, it is easy to diagnose the same through a minimal test like ECG. However, early identification and proper referral is need of the hour.

A single-center cohort study in Wuhan, China with a total of 416 patients included in the final analysis revealed that a total of 82 patients (19.7%) had cardiac injury.[2] Arrhythmias have also been frequently reported in multiple studies but the incidence and association have not been well established.[3] Reports of the disease course complicated with CHB during hospitalization, with otherwise normal cardiac structure and function have been described in a case series of three cases.[4] Postulated mechanisms for conduction abnormalities in COVID-19 include hypoxemia, excessive inflammation associated with the cytokine storm, abnormalities of electrolytes, and direct invasion of cardiomyocytes. In the present case, this may be due to hypoxia and inflammation.

A pro inflammatory state and cytokine storm, myocyte injury, and ischemia due to microvascular involvement have been proposed as possible causes associated with Covid-19, which can predispose the heart to arrhythmias.[5] A systemic autopsy review says cardiac involvement in COVID-19 is rarely due to myocarditis (1.5%) but due to cardiac dilatation (20%), acute ischemia (8%), intracardiac thromb (2.5%), pericardial effusion (2.5%); SARS-CoV-2 was detected in 47% of studied hearts.[6] It is difficult for a primary physician to establish these diagnoses in a suspected cardiac ailment in COVID-19 cases. However, cardiac history and
examination with the help of bedside ECG and echocardiogram can easily diagnose all of them.

**Conclusions**

COVID-19 can manifest with life-threatening arrhythmia in the form of a CHB warranting immediate action such as insertion of a pacemaker. There can be a heart block during the first week of COVID-19 illness, especially in patients having raised inflammatory markers. Timely referral from local practitioner, management of arrhythmia with multispecialty teamwork including internist, cardiologist, and intensivist can be life-saving.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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