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## COVID-19 presenting as complete heart block: A case report

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### ABSTRACT

**Rationale:** COVID-19 has a wide range of clinical presentations requiring a high index of suspicion for diagnosing patients presenting with extrapulmonary manifestations. Among them, patients with cardiovascular involvement have a high mortality.

**Patient's concerns:** A 50-year-old male patient with COVID-19 infection presented with multiple syncopal episodes, myalgia, and mild respiratory symptoms.

**Diagnosis:** Mild COVID-19 infection with complete heart block.

**Interventions:** Temporary pacing followed by permanent pacemaker insertion 10 days after the onset.

**Outcomes:** The patient was managed as per COVID-19 protocol in an isolation ward, and his condition improved but remained pacemaker dependent until a repeat RT-PCR for COVID-19 tested negative, after which he was shifted back to the cardiac care unit for permanent pacemaker insertion. The patient was discharged after inflammatory markers were normal and clinical condition was completely stable.

**Lessons:** COVID-19 has a wide range of clinical presentations, and extrapulmonary manifestations, especially, cardiovascular involvement can not be ignored.

**KEYWORDS:** COVID-19; Heart block; Cardiac; Coronavirus; Case report

### 1. Introduction

Viral infections are a common cause of myocarditis[1]. Apart from pneumonia and acute respiratory distress syndrome (ARDS), severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has a wide range of extra-pulmonary manifestations. As more literature

continues to be published, many atypical manifestations of COVID-19 are now being recognized. However, till date, little information is available about the cardiac implications of COVID-19. Herein, we presented an unusual case of complete heart block (CHB) with syncopal attacks as the presenting feature of COVID-19 infection.

### 2. Case report

This study was supported by the Ethical Committee of Government Medical College and Hospital. Written informed consent was obtained from the patient for publication of this case

#### Significance

COVID-19 has a wide range of clinical manifestations which necessitates high index of suspicion for diagnosing patients presenting with any of the extrapulmonary manifestations. Among them, patients involving cardiovascular system have a high mortality. This case represents a novel clinical scenario of symptomatic complete heart block secondary to COVID-19 infection which highlights the importance of variability in presenting complaints and need to be vigilant to timely diagnose and manage.

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report and accompanying images.

A 50-year-old male, daily wage worker, presented to the emergency with 2 syncopal episodes over the preceding 24 h. Attacks occurred out of the blue without any aura or an obvious precipitant, lasting for a few seconds with spontaneous complete recovery. There was no history of exertional onset of attacks, no associated complaints of chest pain, palpitations, urinary incontinence, severe headache, focal neurologic deficits, ataxia, and diplopia before or during the syncopal event. He experienced myalgias and low-grade fever measuring 37.3°C on one or two occasions in the last 3 days. There was no history of similar episodes in the past. He did not smoke or consume alcohol, and his past history was only significant for mild intermittent asthma.

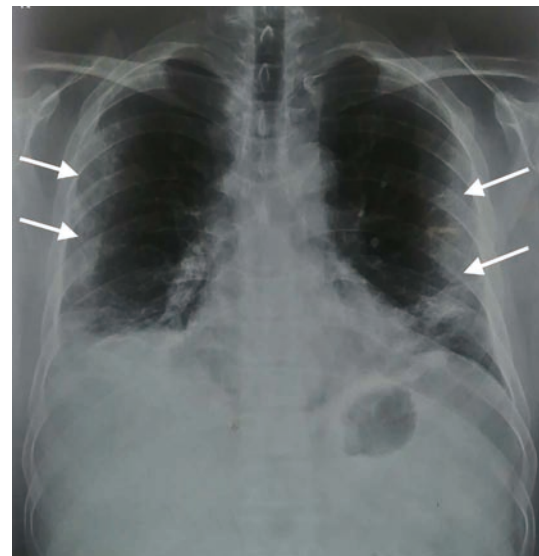
On presentation, he was afebrile; blood pressure was 100/60 mmHg (normal range: 90/60 mmHg-120/80 mmHg); capillary oxygen saturation was 94% (normal: above 95%) on room air; the respiratory rate was 24 breaths/min (normal range: 12-18 breaths/min) and random blood sugar was 126 mg/dL (normal range: below 200 mg/dL). The pulse was feeble and regular with a rate of 36/min (normal range: 60-110/min) and electrocardiography (ECG) revealed complete heart block (CHB) (Figure 1). No murmurs were heard, and there was no evidence of fluid overload. The rest of the systemic examination did not reveal any abnormality.

Differential diagnoses of acute coronary syndrome, electrolyte imbalance, drug effect, or infectious etiology were considered for the CHB. The patient denied using any atrioventricular blocking agents or having previous heart problems. He hadn't taken any regular medications so the possibility of drug-induced arrhythmias was also ruled out. Reviewing his previous ECG which was done one year ago, showed normal sinus rhythm..

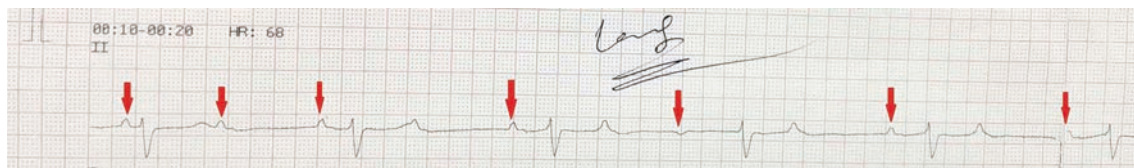
In compliance with the local guidelines regarding the ongoing COVID-19 pandemic, his nasopharyngeal swab was subjected to reverse transcription-polymerase chain reaction (RT-PCR), and it tested positive. Chest X-ray was done, suggestive of bilateral

peripheral infiltrates involving 25% lung fields (Figure 2). Transthoracic echocardiography showed normal biventricular wall motion, left ventricular ejection fraction of 55% without significant valvular heart disease or pericardial effusion. Figure 3 demonstrates a repeat ECG showing ventricular-paced rhythm after temporary pacing was done.

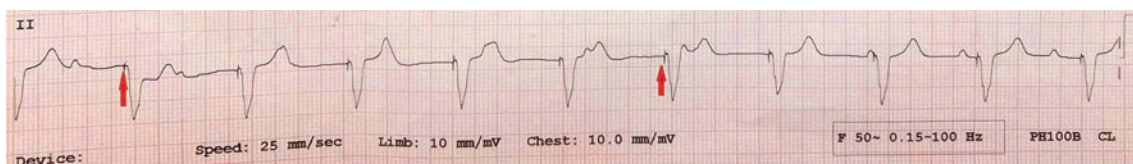
Admission labs were significant for mildly deranged liver functions with aspartate transaminase elevated to 87 U/L (normal range: 5-40 U/L), alanine transaminase elevated to 53 U/L (normal range: 5-35 U/L) and deranged renal functions suggestive of acute kidney injury with raised urea of 99 mg/dL (15-45 mg/dL) and creatinine 1.8 mg/dL (0.8-1.2 mg/dL). Serum electrolytes were normal. Creatine kinase-MB isoenzyme, depicting myocardial injury, was slightly raised with a value of 39 IU/L (normal range: 5-25 IU/L). The blood counts were normal with hemoglobin of 12.8 g/L, total leucocyte count of  $6.2 \times 10^9/L$  and platelet count of  $167 \times 10^9/L$ .



**Figure 2.** Chest X-ray of a 50-year-old male patient with COVID-19 showing peripheral lung opacities (white arrows).



**Figure 1.** Electrocardiogram of a 50-year-old male patient showing complete heart block (red arrows: P waves).



**Figure 3.** Paced rhythm after insertion of temporary pacemaker (red arrows: Pacer spikes).

The inflammatory marker profile revealed C-reactive protein of 21 mg/L (normal range: <5 mg/dL), lactate dehydrogenase of 802 IU/L (normal range: 235-470 IU/L), procalcitonin of 0.16 ng/mL (normal range: <0.10 ng/mL), ferritin of 362 ng/mL (normal range: 22-322 ng/mL), D-dimer of 5.1 mcg FEU/mL (normal range: <0.5 mcg FEU/mL), interleukin-6 of 0.10 pg/mL (normal range: <4.4 pg/mL). The arterial blood gases were normal except for slightly raised lactate. The patient was managed as per COVID-19 protocol in an isolation ward, and he improved but remained pacemaker dependent until a repeat RT-PCR for COVID-19 tested negative after which he was shifted back to the cardiac care unit for permanent pacemaker insertion. His inflammatory markers normalized before discharge.

### 3. Discussion

COVID-19 virus gains access to human tissues through the binding of its S-protein to the angiotensin-converting enzyme-2 (ACE-2) receptor. ACE-2 is highly expressed on the cardiac myocytes, vascular endothelial cells, and pulmonary epithelial cells; thus resulting in predominant cardiopulmonary symptomatology[2].

The cardiac manifestations are in general classified into mechanical (myocardial, pericardial, vascular, and valvular complications) and electrical (arrhythmias and conduction defects) dysfunction. Different arrhythmias in COVID-19 can be secondary to electrolyte imbalance, hypoxia of ARDS, or attributable to commonly used drugs like hydroxychloroquine, azithromycin, and lopinavir/ritonavir which may lead to QT prolongation[3].

Wang *et al.* reported acute cardiac injury in 7.2% of their 138 patients hospitalized with COVID-19 pneumonia out of which 16.7% of patients developed dysrhythmias[4]. In a global survey conducted by Heart Rhythm Society with more than 1 100 electrophysiology physicians representing 76 countries, atrial fibrillation was the most commonly reported tachyarrhythmia whereas severe sinus bradycardia and CHB were the most common bradyarrhythmias[5].

Kir *et al.* and Al-Assaf *et al.* in different case reports have discussed a similar case of COVID-19 infected middle-aged males with multiple episodes of bradycardia due to intermittent advanced AV blocks[6,7]. In another report by Krishna *et al.*, CHB with right bundle branch block morphology complicated the clinical course of a COVID-19 infected 46-year-old woman with no previous cardiac morbidity[8].

Increased morbidity and mortality have been noted in COVID-19 patients who develop brady-arrhythmias[9]. In a review that analyzed 45 publications about cardiovascular complications in COVID-19 patients, the conclusion was drawn that preexisting cardiovascular disease may be associated with a more severe

COVID-19 infection and a higher chance of requiring critical care[10].

Our patient had only mild systemic and respiratory symptoms typical of COVID-19 infection at presentation yet showed evidence of conduction disturbances with complete heart block, which is why it caught our attention. Hence, the possibility of isolated involvement of the AV node and infra-His conduction system by novel coronavirus should be considered which leads to the development of advanced heart blocks in COVID-19 infected patients during their illness.

This case represented a novel clinical scenario of symptomatic CHB secondary to COVID-19 infection which strengthens the fact that SARS-CoV-2 has a wide range of clinical presentations. We need to have a high index of suspicion for diagnosing patients presenting with a different set of complaints to hospitals in the current pandemic. Currently, there is insufficient literature on the cardiovascular manifestations of COVID-19 and long-term effects are unknown.

### Conflict of interest statement

The authors report no conflict of interest.

### Authors' contributions

J.A.: Concept, design, intellectual content, literature search, data acquisition, manuscript preparation, editing, and review; A.S.L.: Data acquisition, design, intellectual content, literature search, editing, and review; S.G.: Intellectual content, literature search, data acquisition, manuscript editing, and review; M.G.: Literature search, clinical studies, data acquisition, manuscript preparation, editing, and review; S.K.A.: Intellectual content, literature search, data acquisition, manuscript editing, and review.

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