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CASE REPORT

Unusual Cardiac Presentation of COVID-19 with Significant Sinus Pauses

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Abstract

There is growing evidence of cardiac complications due to corona virus disease 2019 (COVID 19). Our case is a case of a young patient with COVID-19 and symptomatic sinus pauses.

Keywords: Sinus pauses, COVID 19, Bradycardia

1. Introduction

Since the diagnosis of the first case of corona virus disease (COVID-19) in China in Dec 2019, the virus has spread rapidly, and declared pandemic by WHO in March 2020. Respiratory symptoms predominate the presentation because the virus binds to ACE2 receptors through its spike protein (S protein) after being activated by trans-membrane protease serine 2 [1].

In a recent study of 138 patients who were hospitalized with COVID-19, approximately 16.7% developed arrhythmia [2,3]. This is a case of a young patient with COVID-19 and symptomatic sinus pauses.

2. Case Presentation

A 40 years old man with history of type II diabetes mellitus and hypertension was presented to the emergency department of our hospital with his first episode of syncope while having lunch followed by another episode 1 h later. He did not have similar episodes in past. He was having fever and dry cough for the last three weeks. He had close contact with a family member who is SARS-CoV-2 positive. His reverse transcriptase polymerase chain reaction (RT-PCR) test was positive on two occasions separated by 12 h. At home, he was taking metformin, sitagliptin, and Irbesartan. He reported no use of herbal supplements, illicit drugs, unusual dietary consumption or alcohol use. He was not taking any AV nodal blocking agents.

On arrival to the emergency department, his vitals were as follows, heart rate 21 bpm, blood pressure 80/50 mmHg, respiratory rate 21/min, temp 36.5 °C, oxygen saturation 98% on room air. Physical examination including respiratory and cardiovascular systems were unremarkable.

Electrocardiogram (ECG) showed long pauses with ventricular escape rhythm (Fig. 1). He received 1 mg of atropine which temporarily increases his heart rate to the 70s but soon his heart rate drifted down, hence was started on dopamine infusion. Following dopamine his ECG revealed normal sinus rhythm at 65 b/min with right bundle branch block (RBBB), left axis deviation, and left anterior fascicular block (Bifascicular block) (Fig. 2). Unfortunately, he had no previous ECG for comparison. Bed-side temporary transvenous pacemaker was inserted via left internal jugular vein under full personal protective equipments with droplet

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precautions. The temporary pacemaker was kept at VVI 40/min.

His chest X-ray which was done in supine position with inadequate inspiration showed possible basal consolidation. His laboratory investigations revealed high D-Dimer 1.18 ug/ml (N:0–0.5 ug/ml), high fibrinogen 5.05 g/L (N: 2–4 g/L), ESR 25, von Willebrand factor (vWF) antigen 57% (N:50–160%), CRP 16.76 mg/L (N:0–5 mg/L), Ferritin 731 ng/ml (N:30–400 ng/ml), Troponin-I 0.12 ug/L (N:0–0.3 ug/L). There was mild neutropenia 37% (Normal range 40–70%). His creatinine kinase and creatinine kinase –MB, renal profile, hemoglobin and platelets were all normal. His NT-pro brain natriuretic peptide (NT-proBNP) was 44 pg/ml.

He was shifted from the emergency department to an assigned COVID 19 field intensive care unit (FICU). He was treated with ceftriaxone intravenously and enoxaparin subcutaneously. His 2D ECHO was done within 48 hrs from the admission and was completely normal. He used the pacemaker briefly in the first 24 hrs but subsequently, he maintained a stable rhythm throughout hospital stay until discharge after 13 days. The temporary pacemaker was removed on day four. He was evaluated few weeks after discharge with 48 h Holter which showed normal sinus rhythm (maximum heart rate 116/min and minimum heart rate 57/min with no pauses). His ECG was still

Abbreviations

COVID 19	coronavirus disease 2019
SERS COV 2	severe acute respiratory syndrome corona virus 2
ACE 2	Angiotensin converting enzyme 2
CHB	Complete heart block

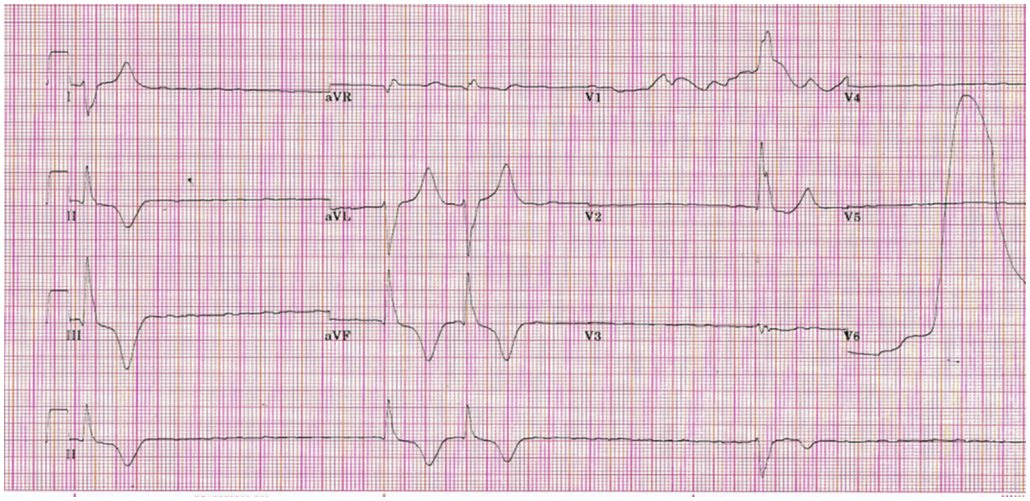

Fig. 1. ECG sinus pauses HR 21 b/min with ventricular escape rhythm.

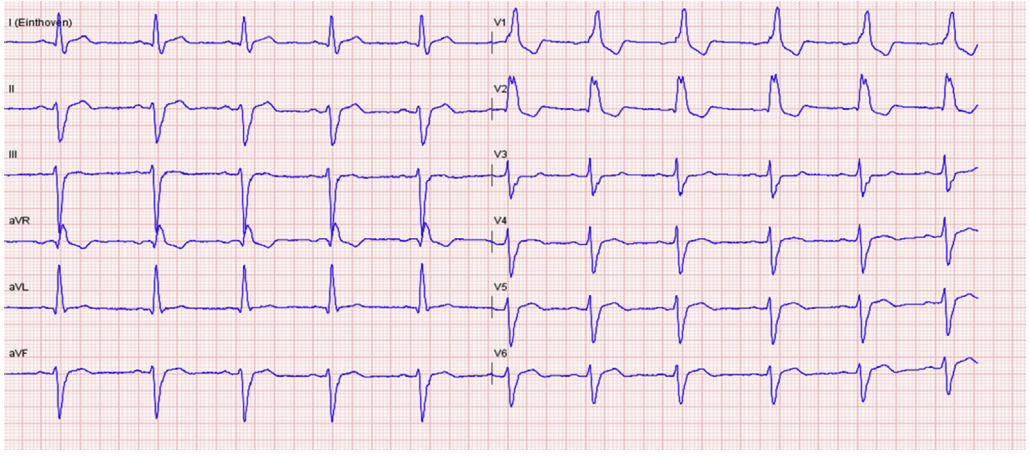

Fig. 2. ECG – post Atropine and on dopamine infusion.

showing bifascicular block. He underwent treadmill exercise which was unremarkable.

3. Discussion

Although most patients with COVID-19 present with mild respiratory illness, involvement of other organs is common including the cardiovascular system with manifestations that include myocardial injury, myocarditis, and arrhythmias. Arrhythmic complications from COVID-19 include various forms of tachyarrhythmias and bradyarrhythmias. One case report described the occurrence of asystole in the setting of severe COVID-19 [4]. Cardiac involvement with high troponin level is associated with worse clinical outcomes.

The pathophysiology of cardiac involvement in COVID-19 is multifactorial. It can result from direct cardiotropic action of the virus mediated by angiotensin-converting enzymes inhibitor receptors (ACE-2) which are expressed in various organs including the cardiac myocites [5]. Cytokine storm syndrome is an exaggerated systemic inflammatory process that result from activation of various inflammatory markers including TNF (tumor necrosis factor)-α, IL (interleukin)-6, MCP (monocyte chemoattractant protein)-1, G-CSF (granulocyte-colony stimulating factor) and others [6]. This intense inflammatory response can affect various organs including the heart and associated with high mortality. Inflammatory cells and SARS-CoV-2 virus have been identified in the myocardium in autopsy studies, suggesting direct invasion of the heart in some cases [9].

Other theories that implicate cardiac involvement in COVID-19 include endothelial and coronary microcirculatory dysfunction resulting from damage of the cardiac pericytes. Hypoxemia with oxygen-demand mismatch, mediated by hypoxic respiratory failure and increased metabolic demand is another mechanism by which the virus can affect the heart, while hypercoagulable states and plaque destabilization may result in coronary events [7]. SARS-CoV-2 can affect various cardiac ion channels leading to alteration in cardiac conduction and calcium handling which may predispose to arrhythmias [8].

It has been shown that atrial fibrillation is the commonest tachyarrhythmias reported, while sinus bradycardia and complete heart block (CHB) are the most common bradyarrhythmias reported [10]. Ventricular tachyarrhythmias and pulseless electrical activity are less commonly seen.

Our patient presented with syncope secondary to significant sinus pauses. Coronary artery ischemia or myocarditis are not the cause of his presenting arrhythmias as there was no history of chest pain. His cardiac biomarkers were normal, and no evidence of ischemic changes in the ECG. Furthermore, his 2D ECHO revealed no regional wall motion abnormalities with normal left ventricular function. The mechanism of sinus pauses in this case is suspected to be due to inflammatory response based on high levels of D-dimers, CRP, and Ferritin. With treatment his inflammatory markers trended down and there was no recurrence of pauses on follow up.

4. Conclusion

COVID-19 was initially considered solely to affect the respiratory system leading to acute respiratory distress. However, with time it became obvious that its clinical spectrum expanded to involve other systems like the cardiovascular system. Arrhythmic complications resulting from cardiac involvement are well described. Most of the bradyarrhythmias reported as a complication of COVID-19 are CHB. Significant pauses are unusual presentation and are reported in the setting of severe COVID-19 with evidence of myocardial injury. Our case is rare as the patient presented with sinus pauses without risk factors for sinus node dysfunction and was clinically and hemodynamically stable during his hospital course with no evidence of myocardial injury. His sinus pauses completely resolved with treatment.

Author contribution

Conception and design of Study: Tarique S. Chachar, Salman K. Slais. Literature review: Tarique S. Chachar, Salman K. Slais. Acquisition of data: Adel K. Hamad. Analysis and interpretation of data: Tarique S. Chachar, Salman K. Slais, Abdulrahman Almadani, Adel K. Hamad. Research investigation and analysis: Tarique S. Chachar, Salman K. Slais. Data collection: Tarique S. Chachar, Adel K. Hamad. Drafting of manuscript: Tarique S. Chachar. Revising and editing the manuscript critically for important intellectual contents: Tarique S. Chachar, Salman K. Slais, Abdulrahman Almadani, Adel K. Hamad. Data preparation and presentation: Tarique S. Chachar, Salman K. Slais, Abdulrahman Almadani, Adel K. Hamad. Supervision of the research: Adel K. Hamad. Research coordination and management: Adel K. Hamad.

Conflicts of interest

The authors declare that the case report was reported in the absence of any commercial or financial

relationships that could be constructed as a potential conflict of interest.

Learning Objectives

1. Conduction system of the heart can be infected by COVID 19 as part of its disease process
2. To understand the pathology of complete heart block due to COVID 19 infection without evidence of Myocarditis
3. More Data is required to further characterize the potential effects of SARS-COV-2 on the cardiac conduction system

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