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ARRHYTHMIAS

BEGINNER

CASE REPORT: CLINICAL CASE

Heart Brake

An Unusual Cardiac Manifestation of COVID-19

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ABSTRACT

A 49-year-old man presented with worsening high-grade fevers, dry cough, and shortness of breath. He tested positive for severe acute respiratory syndrome-coronavirus-2 and was noted to have bradycardia with intermittent high-degree atrioventricular block. However, cardiac biomarkers and echocardiographic findings were normal, thus making this an unusual and interesting manifestation of myocardial involvement of this novel coronavirus. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:1252–5) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 49-year-old man presented to the emergency department with acute-onset high-grade fevers accompanied by dry cough and shortness of breath that had been ongoing for a week before presentation. He denied any associated nausea or vomiting, diarrhea, sore throat, congestion, or skin rash. Of note, he had recently returned from a high-prevalence area for coronavirus disease-2019 (COVID-19) within the United States and was in self-quarantine. He was monitoring his symptoms; however, when his shortness of breath was not improving with his asthma medications (albuterol inhaler and cetirizine), he presented to the emergency department. On arrival, he was noted to be febrile at 102.5°F, he was tachypneic to 22 breaths/min, he was normotensive at 125/75 mm Hg, his heart rate was 75 beats/min, and he was saturating 98% oxygen on room air. Physical examination was remarkable for decreased breath sounds bilaterally.

LEARNING OBJECTIVES

- To anticipate and diagnose conduction disturbances associated with the novel coronavirus.
- To understand the mechanism responsible for high-degree AV block associated with COVID-19 without evidence of overt myocarditis.

PAST MEDICAL HISTORY

His past medical history was significant for mild intermittent asthma.

DIFFERENTIAL DIAGNOSIS

Our patient's clinical presentation was concerning for viral or bacterial lower respiratory tract infection.

INVESTIGATIONS

An electrocardiogram revealed normal sinus rhythm with normal PR (172 ms) and QRS (94 ms) intervals

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Case Reports* author instructions page.

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(Figure 1). No acute ST-T wave changes were noted. A single-view chest radiograph showed blunted costophrenic angles bilaterally with concern for right middle lobe opacity (Figure 2). Laboratory test results were remarkable for leukopenia to 3,900 cells/μl with lymphopenia (absolute lymphocyte count was reduced at 900 cells/μl). Serum chemistry results, including liver function tests, were normal. Arterial blood gas revealed acute respiratory alkalosis, with pH of 7.55, partial pressure of carbon dioxide of 25 mm Hg, partial pressure of oxygen of 94 mm Hg, and bicarbonate of 22 mmol/l on room air. The results of a nasopharyngeal respiratory panel (adenovirus, non–COVID-19 coronavirus, influenza A and B, human metapneumovirus, parainfluenza virus 1 to 4, respiratory syncytial virus, *Bordetella pertussis, Mycoplasma pneumoniae,* and *Chlamydophila pneumoniae*) were negative. Given the high pre-test probability for COVID-19, a nasopharyngeal swab for severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) was collected, and the patient was admitted to the medical floor, given the concern for viral or bacterial pneumonia. During his hospital course, he was noted to have evidence of bradycardia with intermittent high-degree AV block and AV dissociation; the ventricular rate was <20 beats/min (telemetry strips in Figures 3 and 4). He was symptomatic with diaphoresis during these episodes but remained hemodynamically stable. Pacemaker pads were placed; however, he did not require transcutaneous pacing. Cardiac biomarker levels, including troponin I (<0.012 ng/ml; normal range 0 to 0.034 ng/ml) and N-terminal pro-B-type natriuretic peptide (38.3 pg/ml; normal range 0 to 125 pg/ml), were normal. A transthoracic echocardiogram showed a normal ejection fraction, normal diastolic function, no wall motion abnormalities, and no significant valvular disease (Videos 1, 2, and 3). Inflammatory markers were mildly elevated; the ferritin level was 571 mg/ml (normal range 0 to 400 ng/ml), and C-reactive protein was elevated at 1.2 mg/dl (normal range 0 to 0.9 mg/dl). The procalcitonin level was negative at 0.039 ng/ml (normal range 0 to 0.080 ng/ml), and thyroid hormone levels were within normal limits. His nasopharyngeal swab tested positive for SARS-CoV-2 ribonucleic acid.

ABBREVIATIONS AND ACRONYMS

ACE 2 = angiotensin-converting enzyme 2

AV = atrioventricular

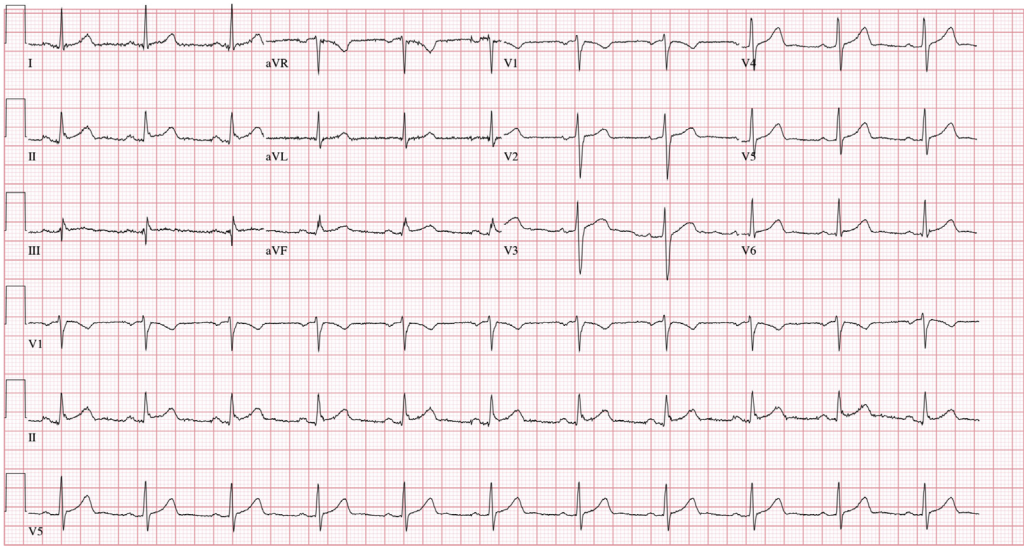
COVID-19 = coronavirus disease-2019

SARS-CoV-2 = severe acute respiratory syndrome-coronavirus-2

MANAGEMENT

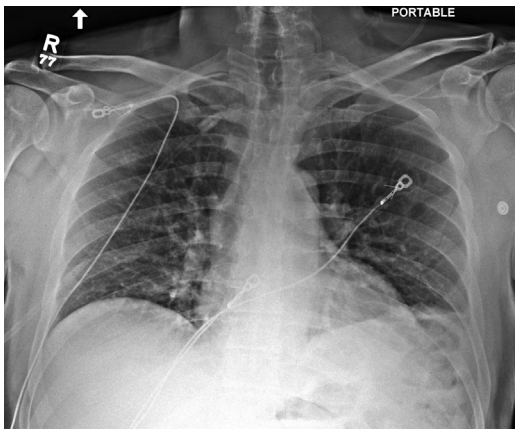
Given the patient's underlying asthma, which predisposed him to an increased risk for pulmonary

FIGURE 1 Initial Electrocardiogram Findings



A 12-lead electrocardiogram on presentation showing normal QRS complex and PR interval.

FIGURE 2 Initial Chest Radiograph



Anteroposterior single-view chest radiograph on presentation showing blunted costophrenic angles bilaterally and a right middle lobe opacity.

complications, he was started on a course of azithromycin and hydroxychloroquine after consultation with infectious disease doctors.

FOLLOW-UP

He was monitored as an inpatient because of worsening hypoxemia for 5 days. The rest of his hospital course remained uneventful. No further evidence of conduction disease was noted on telemetry, and the QTc interval remained normal while he was receiving hydroxychloroquine. He continued to improve clinically. He was discharged home with a plan to continue self-quarantine, and outpatient follow-up was arranged.

DISCUSSION

SARS-CoV-2 invades cells through the angiotensin-converting enzyme 2 (ACE2) receptor, which is abundant in the heart (1). Among 138 patients hospitalized with COVID-19 in Wuhan, China, acute cardiac injury (defined if the serum levels of cardiac biomarkers were above the 99th percentile upper reference limit or new abnormalities were shown on electrocardiography and echocardiography) was diagnosed in 7% patients, and approximately 16.7% patients were noted to have cardiac arrhythmias (2). COVID-19 involvement of the heart has ranged from asymptomatic myocardial injury to acute coronary syndrome, mild to fulminant myocarditis, stress cardiomyopathy, and cardiogenic shock; however, the mechanism of cardiac involvement is not exactly clear (3). Furthermore, underlying cardiovascular disease or risk factors and myocardial injury have been shown to portend poor prognosis in these patients (4). In this case, we present a patient with moderate COVID-19 infection who showed evidence of transient conduction disturbances with high-degree atrioventricular (AV) block. High-degree AV block is known to be an uncommon presentation of acute myocarditis in adults, more commonly seen in cardiac sarcoidosis and giant cell myocarditis (5). However, because our patient did not have any other overt evidence of myocardial involvement, with normal cardiac biomarkers and a normal echocardiogram, his presentation is unusual and interesting. It is possible that COVID-19 may have caused subclinical myocarditis leading to high-degree AV block in this case. ACE2 receptors are abundant in the heart and are present in multiple cell types, including macrophages, endothelial cells, smooth muscle cells, and cardiomyocytes (6). Further, animal models have shown the presence of ACE2 receptors in sinoatrial nodal cells in rats (7), and conduction disturbances and ventricular fibrillation have been noted with overexpression of the ACE2 receptor in experimental mice models (8). Hence, another possibility is that isolated involvement of the AV node and infra-Hisian conduction system by SARS-CoV-2 may have caused transient high-grade AV block. Whether this block is secondary to direct viral involvement or is an autoimmune response is unknown at this time. Our patient did not have a recurrence of these conduction disturbances after he was started on supportive

FIGURE 3 Telemetry Strip Showing Bradycardia With High-Degree Atrioventricular Block

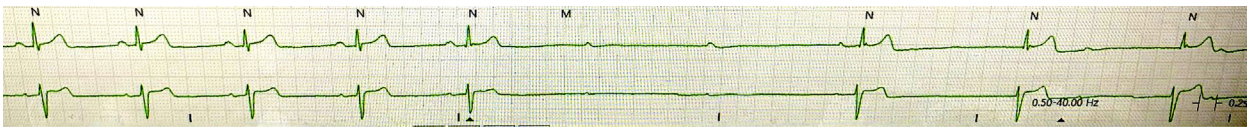
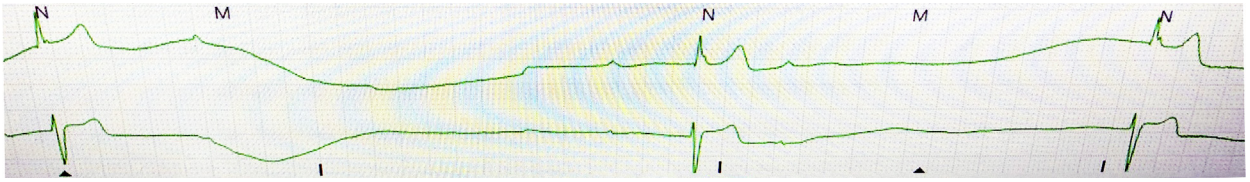


FIGURE 4 Telemetry Strip Showing High-Degree Atrioventricular Block With Slow Escape Ventricular Rates <20 Beats/Min



treatment with azithromycin and hydroxychloroquine. This outcome may have reflected recovery from the viral infection or the anti-inflammatory effects of the medications, or both.

CONCLUSIONS

We reported an unusual presentation of a young patient without any significant cardiac comorbidities or underlying conduction disease who presented with transient high-degree AV block that was associated with a diagnosis of moderate COVID-19. Conduction disturbances in our patient were likely the result of subclinical myocarditis or isolated involvement of the AV node and infra-Hisian block secondary to infection with this novel coronavirus. We need to maintain a high index of suspicion for cardiovascular complications in these patients. Conduction disturbances were transient in the case presented here. However, with increasing severity of disease, persistent conduction disturbances could require pacing.

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KEY WORDS bradycardia, cardiomyopathy, complication, COVID-19

APPENDIX For supplemental videos, please see the online version of this paper.