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ST ELEVATION IN THE COVID-19 ERA: A DIAGNOSTIC CHALLENGE

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Abstract—Background: Severe acute respiratory syndrome coronavirus 2 induces a marked prothrombotic state with varied clinical presentations, including acute coronary artery occlusions leading to ST-elevation myocardial infarction (STEMI). However, while STEMI on electrocardiogram (ECG) is not always associated with acute coronary occlusion, this diagnostic uncertainty should not delay cardiac catheterization. Case Reports: We present 2 cases of patients with COVID-19 that presented with STEMI on ECG. While both patients underwent cardiac catheterization, a delay in time to intervention in the patient found to have acute coronary artery occlusion may have contributed to a poor outcome. Why Should an Emergency Physician Be Aware of This?: These cases highlight the fact that while not all COVID-19 patients with STEMI on ECG will have acute coronary artery occlusions, there is continued need for prompt percutaneous coronary intervention during the severe acute respiratory syndrome coronavirus 2 pandemic. © 2020 Elsevier Inc. All rights reserved.

Keywords—COVID-19; ST elevation myocardial infarction; thrombosis

INTRODUCTION

There have been myriad consequences resulting from the outbreak of the novel severe acute respiratory syndrome coronavirus 2 causing the pandemic characterized by the coronavirus disease 2019 (COVID-19). The impact on cardiovascular care has been multifaceted and profound. The direct impact of COVID-19 on the cardiovascular system has been described with 1 study observing an in-hospital mortality rate of 51.2% in patients with myocardial injury vs. 4.5% in patients without myocardial injury (1). The prothrombotic, proinflammatory state caused by COVID-19 has been associated with the development of an acute COVID-19 cardiovascular syndrome (ACovCS) that has been described as most commonly presenting as acute cardiac injury with cardiomyopathy, ventricular dysrhythmias and hemodynamic instability in the absence of obstructive coronary artery disease (CAD) (2). We present 2 cases of patients with symptoms compatible with COVID-19 (both of whom would go on to test positive for the disease) who presented to the emergency department (ED) with ST-segment elevation myocardial infarction (STEMI) on their initial electrocardiograms (ECGs). These cases show the challenge in managing patients with STEMI during the COVID-19 pandemic as the differences between COVID-19 patients with ACovCS and coronary artery occlusions from CAD can be extremely difficult to identify.

CASE REPORTS

Case 1

A 51-year-old man with a medical history of hypertension, CAD, hyperlipidemia, previous MI, and diabetes
presented to the ED for chest pain and shortness of breath. The patient described chest pain similar to his previous MI, with onset 1 h before arrival. Vital signs on presentation showed a blood pressure of 165/112 mm Hg, a heart rate of 110 beats/min, a respiratory rate of 22 breaths/min, a temperature of 36.9°C, and pulse oximetry of 91% on nonrebreather mask (NRB). Chest radiography (CXR) and a 12-lead ECG were performed as well as a bedside cardiac ultrasound. The ECG revealed 1- to 2-mm ST elevations in leads V5 and V6 (Figure 1). CXR revealed multifocal airspace disease consistent with COVID-19 pneumonia. Point-of-care cardiac ultrasound revealed severely depressed left ventricular ejection fraction (LVEF) by visual estimation, with a previously documented LVEF of 35–40%. In consultation with interventional cardiology the patient initially underwent a medical management strategy because of concerns that the primary cause of the presentation was infection from COVID-19. The patient received 325 mg aspirin, 40 mg lasix, and 1 mg nitroglycerin intravenously. The patient was then given a bolus dose of heparin and started on a continuous heparin infusion. Initial high sensitivity troponin was 175 ng/L. A repeat ECG obtained approximately 90 min after presentation showed >2 mm ST elevation in leads V5, V6, II, III, and arteriovenous fistula. There were reciprocal ST segment depressions in leads I, aVL, V1, V2, and V3 (Figure 2). The interventional cardiology team re-evaluated the patient, who was then intubated and taken emergently to the cardiac catheterization laboratory (CCL). Angiography revealed 100% occlusion of the proximal circumflex with Thrombolysis In Myocardial Infarction grade III flow after procedure; there was also severe left anterior descending coronary artery disease. The patient then went into cardiac arrest (pulseless electrical activity) before the right-sided coronary circulation could be assessed. The CCL team performed cardiopulmonary resuscitation for 33 min before the patient died. A coronavirus polymerase chain reaction swab was positive.

Case 2

A 67-year-old man with no reported medical history presented with a 3-day history of worsening dyspnea. He had vague chest pain that resolved on the day before presentation. Vital signs at presentation showed a blood pressure 147/79 mm Hg, a heart rate of 113 beats/min, a respiratory rate of 30 breaths/min, a temperature of 36.1°C, and oxygen saturation of 78% on room air that improved to 95% on 15 L/min by nonrebreather mask. An ECG demonstrated >2 mm ST segment elevations in the lateral leads with inferior ST segment depressions (Figure 3); no previous ECG was available for comparison. CXR showed bibasilar interstitial airspace opacities; point-of-care cardiac ultrasound showed normal right and left ventricular systolic function by visual estimation and no regional wall motion abnormalities. Interventional cardiology was consulted and recommended emergent cardiac catheterization, which the patient initially declined. Laboratory testing revealed a blood glucose of 518 mg/dL and anion gap of 34 consistent with diabetic ketoacidosis, creatinine of 1.43 mg/dL, high-sensitivity troponin 6853 ng/L, D-dimer >35.2 mg/L, fibrinogen 646 mg/dL, and ferritin

Figure 1. Initial electrocardiogram of case 1.
996.1 ng/mL. Computed tomography pulmonary angiography was negative for pulmonary embolism. The patient ultimately agreed to cardiac catheterization and was given 5000 units of heparin and 325 mg oral aspirin. He was intubated and taken to the CCL where angiography revealed 35% stenosis of the distal left main artery, 20% stenosis of the mid-LAD, 60% stenosis of the proximal first obtuse marginal artery, and 90% stenosis of the proximal posterior descending artery; no intervention was performed. His COVID-19 polymerase chain reaction nasal swab was positive; he received methylprednisolone, remdesivir, azithromycin, and ceftriaxone for pneumonia, enoxaparin 100 mg twice daily, and 81 mg daily aspirin. He stabilized with medical management in the intensive care unit. He was extubated on hospital day 6 and found to have a new left-sided facial droop. His symptoms resolved and neuroimaging did not show any findings of cerebral ischemia, leading to a diagnosis of transient ischemic attack. He was transferred out of the intensive care unit on day 7, requiring oxygen by nonrebreather mask to maintain his oxygen saturation >90%. In-hospital comprehensive echocardiography was not performed because of concerns over the spread of COVID-19 infection. On hospital day 14, while awaiting discharge to a skilled nursing facility, he suffered a cardiac arrest and died.

Figure 2. Repeat electrocardiogram of case 1 (90 min after the initial electrocardiogram).

Figure 3. Initial electrocardiogram of case 2.
DISCUSSION

While the pathophysiology of COVID-19 remains incompletely understood, inducement of a prothrombotic state has been recognized as a contributor to adverse outcomes. This fact makes the diagnosis of acute myocardial infarction more complex in the COVID-19 era because there can be substantial overlap in symptoms and results of diagnostic tests in patients with acute coronary artery occlusion and microthrombotic disease. The cases presented herein highlight the diagnostic and therapeutic challenges faced by emergency physicians and cardiologists when caring for these patients.

Both patients herein presented with a constellation of symptoms consistent with COVID-19 pneumonia. However, case 1 occurred much earlier in the pandemic when case volume was lower and less was known about the disease and its transmissibility, thereby lending greater exigency to maintaining provider safety. Therefore, while the patient presented with symptoms and ECG findings concerning for acute coronary occlusion, the decision was made to initially manage this case medically with close observation in the ED. When his condition deteriorated, he was taken promptly to the CCL where he was found to have multivessel CAD requiring percutaneous coronary intervention (PCI). The adverse outcome in this case highlights the importance of prompt PCI in cases of suspected STEMI, as recommended in a joint statement from the Society for Cardiovascular Angiography and Interventions, American College of Cardiology, and the American College of Emergency Physicians on April 20, 2020 (case 1 occurred >3 weeks before the release of this statement) (3).

Conversely, case 2 presented with primary respiratory symptoms and with STEMI on ECG but absence of chest pain. The patient was taken urgently to the CCL and found to have nonobstructive CAD with no PCI performed. This finding is consistent with a recent case series in which 9 of 18 COVID-19 patients with STEMI on ECG underwent coronary angiography but only 6 patients were found to have obstructive disease (4). In contrast, however, the patient in case 2 had D-dimer and troponin levels markedly greater than the group with noncoronary myocardial injury. This finding suggests that while elevation of these markers portend a worse prognosis, laboratory testing by itself cannot reliably identify patients requiring emergent PCI. This lends further support to the joint society statement that expeditious transfer to the CCL is indicated in cases of suspected STEMI (3).

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

These cases highlight the extraordinary challenges of caring for patients with COVID-19, because despite appropriate treatment, both patients died. Morbidity and mortality from COVID-19 is high, likely multifactorial, and varies from patient to patient. Therefore, while no single treatment may prevent poor outcomes, aggressive efforts should be made to deliver interventions with known efficacy—in this case, PCI for STEMI—to maximize the chances of survival.

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