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Cardiothoracic Imaging

COVID-19 infection complicated by acute ST-elevation myocardial infarction

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ABSTRACT

Clinicians should be aware of the potential for cardiovascular involvement in COVID-19 infection. Coronavirus disease-2019 (COVID-19) is a viral illness caused by severe acute respiratory syndrome-coronavirus-2. While it primarily causes a respiratory illness, a number of important cardiovascular implications have been reported. We describe a patient presenting with COVID-19 whose hospital course was complicated by ST elevation myocardial infarction requiring percutaneous coronary intervention. The goal is to help clinicians gain awareness of the possibility of cardiovascular disease in COVID-19 infection, and maintain a high index of suspicion particularly for patients with risk factors or a prior history of cardiovascular disease.

1. Introduction

COVID-19 is responsible for a disease outbreak which was first reported in Wuhan, China towards the end of December 2019 and has now been characterized as a pandemic by the World Health Organization. Besides the lungs, it has been reported to involve extra-pulmonary organ systems, particularly the cardiovascular system. Patients with pre-existing comorbidities including cardiovascular disease or risk factors for such are at higher risk of adverse events from COVID-19. Furthermore, patients without cardiovascular comorbidity are also at relatively increased risk for incidental cardiovascular complications.

2. History of presentation

A 62-year-old otherwise healthy male presented to the emergency department with a ten day history of fatigue, low grade fevers, cough, pleuritic chest pain as well as progressive shortness of breath, following known contact with proven cases of COVID-19 two weeks prior. At presentation, the patient was afebrile with a blood pressure of 195/102, respiratory rate of 18, and oxygen saturation of 89% on room air. His pulmonary and cardiovascular physical exams were otherwise unremarkable.

3. Past medical history

The patient’s medical history is positive for hypertension and coronary artery disease. He had a history of an ER visit 7 years prior which he presented with atypical chest pain; a cardiac stress test at that time was negative. Two years prior to presentation the patient underwent a routine calcium scoring exam which showed a very high calcium score (Fig. 1) but was otherwise asymptomatic. He does not have a documented history of a prior acute coronary event or intervention and no history of diabetes mellitus, hyperlipidemia, or renal disease. He denied smoking, alcohol intake, or substance abuse.

4. Investigations

Initial laboratory studies showed a normal white cell count, low hemoglobin (12.2 g/dL) and normal platelet count. Chemistry panel demonstrated a creatinine of 1.41 mg/dL (reference <1.21 mg/dL) and mildly elevated LFTs. The coagulation profile was mildly deranged (INR 1.6). Chest X-ray demonstrated new multifocal bilateral infiltrates. A CT of the chest was obtained which demonstrated extensive peripheral basal lung predominant consolidations and ground glass opacities (Fig. 2A), typical for COVID-19 pneumonia. The extent of disease is better demonstrated on 3D volume rendered image (Fig. 2B). SARS-CoV-2 infection was confirmed with reverse transcriptase polymerase chain reaction testing (RT-PCR). The patient was also tested for influenza and
RSV, which were negative.

5. Hospital course

The patient was admitted under respiratory isolation and started on ceftriaxone and azithromycin to cover for possible superimposed community acquired pneumonia. On admission day 3, he developed retrosternal chest pain radiating to the shoulders as well as increased oxygen requirements. Laboratory studies demonstrated mild troponinemia (troponin I 0.04 ng/mL). A stat electro-cardiogram (ECG) was obtained which showed findings consistent with true posterior wall STEMI, including large R waves in leads V2-V3 and marked ST depression in leads V2-V3. The patient also developed profound hypotension and hypoxemia requiring intubation and transfer to the ICU. Upon stabilization, he was taken emergently for cardiac catheterization. During the procedure, a complete occlusion of the left circumflex artery was found proximal to four large branches (Fig. 3A). This was successfully treated with drug eluting stent with excellent angiographic results, 0% residual stenosis, and no complications (Fig. 3B). There was also non-obstructive atherosclerosis of the left anterior descending artery visualized with up to 40% stenosis. The patient was hypoxic throughout and was transferred back to the ICU post procedure on mechanical ventilation. Next day echocardiogram demonstrated normal left ventricular function and wall motion with estimated ejection of 63%.

On ICU day 6, he developed further respiratory decompensation and ventricular fibrillation arrest requiring cardiopulmonary resuscitation with defibrillation and subsequent veno-venous ECMO cannulation. Post resuscitation, the patient developed paroxysmal atrial fibrillation and was started on an amiodarone drip. He was decannulated on day 9 and remained intubated with aggressive ventilation. During this time he continued to demonstrate paroxysmal atrial fibrillation and episodes of asystole for up to 20 s, which required pacemaker placement. He was ultimately extubated after 22 days to nasal cannula, and was discharged to rehabilitation 51 days after initial presentation. Of note, during this hospitalization, he was also found to have right limb ischemia from a superficial femoral artery thrombus which was managed with a heparin drip.

6. Discussion

In December of 2019, a new viral pneumonia was first described in the city of Wuhan, China, soon to contribute to considerable morbidity and mortality worldwide. It was found that this illness was caused by a novel strain of coronavirus not previously described in humans, termed Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) by the International Committee on Taxonomy of Viruses. The World Health Organization named the illness resulting from this new strain of coronavirus the “Coronavirus disease of 2019” (COVID-19) and subsequently declared it a global pandemic.

COVID-19 infection often presents with fever, dry cough, and shortness of breath. Less commonly, patients may experience muscle pain, anorexia, malaise, sore throat, nasal congestion, dyspnea, and headache. Symptoms usually manifest 2 to 14 days after the initial exposure. Most patients do not present with typical cardiac symptomatology, although nonspecific chest pain may be a presenting symptom in a minority of patients.
Multiple recent studies have demonstrated an association between cardiovascular risk factors, such as hypertension, diabetes, coronary artery disease, and higher severity of COVID-19 illness. Incidence of associated cardiac risk factors like hypertension and diabetes was two times higher in ICU/severely ill COVID-19 patients than in non-ICU patients. Further, in patients requiring ICU level care, the incidence of acute cardiac injury was 13 times higher than in non-ICU COVID-19 patients. Acute myocardial injury is associated with higher mortality in COVID-19 patients. A report by Guo et al. that looked at 187 patients with COVID-19 and found that more than one-fourth (27.8%) of the examined patients had an elevated troponin level during hospitalization. Also, in-hospital mortality of patients with troponemia was 59.6%, compared to 8.9% in patients without troponemia. Moreover, patients who had underlying coronary artery disease and elevated troponin T levels had the highest in-hospital mortality of 69.4%. It should be noted that troponin elevation may also be seen with myocarditis, which has also been described in cases of COVID-19 infection.

The mechanism of COVID-19-associated cardiovascular injury is not well understood. However, it is postulated that SARS-CoV-2 may directly and indirectly affect the cardiovascular system and particularly the heart. More commonly, the myocardial injury may be due to an increased myocardial demand from the stress of the illness in the context of supply-demand mismatch, as can be seen with type 2 myocardial infarctions. Although less likely, myocardial injury may result from dislodgement of pre-existing atherosclerotic plaque secondary to increased inflammatory burden related to the acute infection, resulting in type 1 myocardial infarction, as was the case in our patient who was found to have 100% occlusion of the left circumflex artery. Additionally, cases of presumed in situ thrombosis have been described in patients with COVID-19 infection without pre-existing evidence of atheroma or embolic source, which may be secondary to local inflammatory changes and cytokine release.

Type 2 infarction is treated conservatively, with the main focus being management of concurrent medical problems, mainly conservative management of COVID infection.

Type 1 infarction in COVID-19 patients is treated based on the patients’ clinical context. In COVID-19 patients who are not critically ill, reperfusion is indicated, with primary percutaneous coronary intervention (PCI) being preferred over fibrinolysis. In patients who are critically ill due to COVID-19 infection, the decision to reperfuse is based on an individual account after considering the primary competing illness, any associated harm from the procedure, likelihood of a poor outcome and risk of infection to the healthcare personnel. Additionally, all ST elevation myocardial infarction patients are treated with aspirin, P2Y12 inhibitor, anticoagulation and high-dose statin.

It is important to understand the implications of COVID-19 infection on the cardiovascular system, so that timely and effective treatment can be initiated and mortality can be reduced. The present case highlights how myocardial infarction can complicate the COVID 19 systemic illness.

Declaration of competing interest

All authors have reported that they have no relationships with industry relevant to the contents of this paper to disclose.

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