STEMI in a Young Patient with COVID-19; Too Great a Burden?
Naman Upadhyay, MD, Gillian Naro, MD, Gregary Marhefka, MD

INTRODUCTION
Myocardial infarcts (MIs) can be especially devastating when their pathogenesis stems from a coronary artery occlusion by an intracoronary thrombus (ICT). ICTs are most frequently a result of an underlying atherosclerotic plaque rupture, however, can also be a result of emboli, vasospasm, hypercoagulable states, among other etiologies. Patients suffering from the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) appear to be in both a hypercoagulable and proinflammatory state resulting in an increased risk of clot formation, endothelial damage, and plaque destabilization, thus increasing the ICT burden in infected patients. The literature does reflect an increase in ICT ST-segment elevation myocardial infarction (STEMI) in already high risk patients with an active SARS-CoV-2 infection. Our case discusses this observed pathology in a young 24 year old patient not typically considered among a high risk population for an ICT STEMI.

CASE PRESENTATION
A 24-year-old male presented to the Emergency Department with a two-week history of intermittent fevers, anosmia, ageusia, and shortness of breath. The patient’s nasopharyngeal swab was positive for SARS-CoV-2 by real-time reverse-transcriptase-polymerase-chain-reaction assay. His vitals on admission were as follows: Blood pressure was 146/88 mm Hg; heart rate was 127 beats/min, and respiratory rate was 24 breaths/min. With five liters of supplemental oxygen therapy his oxygen saturation was 96%. Heart auscultation revealed no murmur. He was in mild respiratory distress, but able to speak in full sentences with no accessory muscle use. Lung auscultation revealed coarse bilateral breath sounds with bilateral rales. He was morbidly obese. Laboratory results were significant for the following: White blood cell count of 6,700/mL, creatinine of 2.16 mg/dL, sodium of 127 mmol/l, aminotransferase of 258 IU/L, alanine aminotransferase of 71 IU/L, creatine kinase of 17,732 IU/L, c-reactive protein of 4.3 mg/dL, and ferritin of 1,224 ng/mL. High-sensitivity troponin T (hs-TnT) was 24 ng/L on initial presentation and 2 hours later was 19 ng/L (normal <19 ng/L). Electrocardiogram on presentation was significant for sinus tachycardia and poor r-wave progression (figure 1).

The patient underwent treatment for SARS-CoV-2 pneumonia and rhabdomyolysis with five days of remdesivir, six days of dexamethasone, and intravenous fluids. On hospital day six the patient no longer required supplemental oxygen therapy but experienced a three second pause on telemetry monitoring. The patient left against-medical-advice and re-presented to the ED within 12 hours complaining of new left-sided chest pain. Laboratory results were significant for an initial hs-TnT of 217 ng/L and repeat of 415 ng/L. Electrocardiogram was significant for sinus arrhythmia and acute anterolateral injury pattern with ST-elevations in leads I, aVL, V3-V6 (figure 2). Chest radiograph was significant for vascular congestion in bilateral lung fields (figure 3). Echocardiography was significant for left ventricular dysfunction with peri-apical akinesis. The patient received 324mg of aspirin, 80mg of atorvastatin and was initiated on intravenous heparin therapy. A transfer was initiated to a PCI-capable facility.

DIFFERENTIAL DIAGNOSIS
The differential diagnosis included acute ST-elevation myocardial infarction, pulmonary embolism and COVID-19 associated myocarditis.

OUTCOME & FOLLOW-UP
The patient was transferred to the cardiac catheterization laboratory for consideration of emergency coronary angioplasty. Coronary angiogram showed mid left anterior descending (LAD) artery thrombus with 50% stenosis and distal LAD thrombus causing 100% occlusion at the apex (figure 4). No intervention was done as the patient had resolution of his chest pain. The patient was loaded with 180mg of ticagrelor and placed on a tirofiban infusion for 24 hours due to thrombus burden. The patient was maintained on full dose enoxaparin until discharge due to intracoronary thrombus burden then subsequently discharged on dual-antiplatelet therapy, high-intensity statin and lisinopril. As of a six week follow up the patient has been chest pain free and doing well.
Figure 1. 12 lead electrocardiogram: baseline sinus tachycardia.

Figure 2. 12 lead electrocardiogram: anterolateral ST-segment elevation.

Figure 3. Portable Chest Radiograph: vascular congestion in bilateral lung fields.

Figure 4. Coronary angiogram: mid left anterior descending (LAD) artery thrombus with 50% stenosis and distal LAD thrombus causing 100% occlusion at the apex.
DISCUSSION

Acute coronary syndrome (ACS) events are pharmaco-logically treated with platelet inhibition using a loading dose of aspirin, a P2Y12 antagonist, as well as a continuous heparin drip, titrated to maintain full anticoagulation. If the institution’s facilities allow, the patient will be emergently taken to the cardiac catheterization lab to assess ICT burden and intervention via thrombectomy and angioplasty. Facility capabilities, clot burden, and the patient’s overall clinical picture will certainly play a role in management decisions and which treatments are most appropriate for each patient. The most appropriate management depends on patient past medical history, thrombus burden, and overall goals of care. Early and effective interventions are associated with better outcomes.

In our case of a young patient positive for SARS-CoV-2 presenting with ACS, the mechanism of how SARS-CoV-2 can affect ICT should be considered as the precipitating factor and how it may impact our clinical management decisions. SARS-CoV-2 infections are known to create a systemic proinflammatory state which appears to also lead to hypercoagulation and increased thrombus burden in infected patients. An increase in production of inflammatory markers and cytokines in turn increases thrombin production and atherosclerotic plaque instability by increasing the conversion of fibrinogen to fibrin in turn activating platelets thereby promoting clot formation. Moreover, in patients that are critically ill with a SARS-CoV-2 infection can experience dysfunction in their coagulant concentrations thereby leading to disordered development of thrombosis and bleeding called disseminated intravascular coagulation. Sepsis, tachycardia and systemic inflammation can also increase coronary blood flow demand precipitating myocardial ischemia and plaque destabilization.

These proposed mechanisms may have led to our 24 year old patient’s STEMI in the setting of a SARS-CoV-2 infection. There has been an increase in case reports in the literature on ACS and ICT in SARS-CoV-2 positive patients. Knowing the possible risks of hypercoagulation in SARS-CoV-2, recent studies including the “IMPROVEDD Score” create risk stratification scores to determine the need for venous thromboembolism prophylaxis. Similarly, further study is required to guide prevention and management techniques in ICT in patients with SARS-CoV-2 infections.

KEY POINTS

Intracoronary thrombus, COVID-19, Anticoagulation Management, STEMI

REFERENCES