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Management of ST-Elevation Myocardial Infarction in the COVID-19 Era: The Role of Thrombosis and Anticoagulation Strategy

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Abstract Cardiac manifestations of COVID-19 include myocarditis, demand ischemia, myocardial infarction and arrhythmias with prothrombotic state being a major underlying pathogenetic mechanism. In this report we present a case of a 57-year-old, otherwise healthy, woman who presented with chest pain and nausea and was found to have an inferior wall ST-elevation myocardial infarction (STEMI) in the setting of an active COVID-19 infection. Angiography revealed tortuous coronary arteries with a 100% right coronary artery occlusion with high thrombus burden and normal left coronary system. In light of the available literature regarding the pro-thrombotic effects of this novel corona virus, we continued full dose anticoagulation with Enoxaparin after the cardiac catheterization and transitioned to rivaroxaban and we also continued the patient on dual antiplatelet therapy prior to discharge.

Keywords: *STEMI (ST-Elevation Myocardial Infarction), COVID-19, severe acute respiratory syndrome 2 (SARS-Cov-2), Percutaneous Coronary Intervention, Fibrinolysis, Thrombosis, Anticoagulation*

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1. Case Presentation

A 57-year-old Hispanic female notified the Fire Department of New York's EMS services for a 3-hour history of substernal chest pressure, worsened with exertion and associated with nausea. On arrival to our "COVID-only" institution, the patient was noted to have ST segment elevation (STEMI) in the inferior leads II, III and aVF with reciprocal ST depressions and T-wave inversions in leads I, aVL, V1 and V2. In our emergency department, she continued to mention active chest pressure. Vital signs were noted as follows: temperature of 37.5 °C, blood pressure of 130/75 mmHg, respiratory rate of 21 breaths per minute, oxygen saturation at room air was 98% and a heart rate of 88 beats per minute. She denied having fevers, chills, shortness of breath, rhinorrhea, cough, recent travel, or sick contacts.

Past Medical History

Diet-controlled Hyperlipidemia

Medications

None

Differential diagnosis

Acute Coronary Syndrome, COVID-19 Viral Myocarditis, Pulmonary Embolism

Investigations

Initial electrocardiogram (EKG) showed STEMI in the inferior leads II, III, aVF with reciprocal ST depressions and T-wave inversions in the anterolateral leads 1, aVL, V1 and V2 (Figure 1). Laboratory investigation in the emergency room was significant for positive COVID-19 virus (via nasopharyngeal swab), troponin I 0.01 ng/L, Brain Natriuretic Peptide <10 pg/mL, creatinine 0.5 mg/dL, magnesium 1.0 mg/dL, potassium 3.0 mmol/L, lactate dehydrogenase 171 U/L, C-reactive protein < 4 mg/L, ferritin 144.9 ng/mL, and D-dimer 736 ng/mL.

Management

Upon witnessing the aforementioned EKG changes, the cardiac catheterization lab was emergently activated. The patient received loading doses of aspirin (324 mg), clopidogrel (600 mg), and IV unfractionated heparin (5000 units). Cardiac catheterization revealed a 100% proximal right coronary artery (RCA) obstruction (Figure 2). After initial balloon dilatation, angiography revealed significant thrombus burden and clot-in-transit (distal embolization) with normal left coronary system

(Figure 3, Figure 4, Figure 5). Percutaneous coronary intervention (PCI) was successful and a drug-eluting stent was deployed with 1% residual occlusion (Figure 6). The patient was continued on dual antiplatelet therapy (DAPT) with aspirin and clopidogrel and started on rosuvastatin for further ACS management. Post-catheterization echocardiography revealed a preserved left ventricular ejection fraction of 50-55% with severe hypokinesis of the basal-mid inferoseptal and inferior walls. After reviewing the literature of an increased predisposition to venous and arterial thrombotic events in the COVID-19 population, our patient was kept on therapeutic doses of Enoxaparin during the entirety of her hospitalization. Enoxaparin was used to minimize ancillary staff exposure to COVID-19. The patient's enoxaparin was transitioned to Rivaroxaban 2.5 mg twice daily prior to discharge, to be continued for one month (in addition to DAPT).

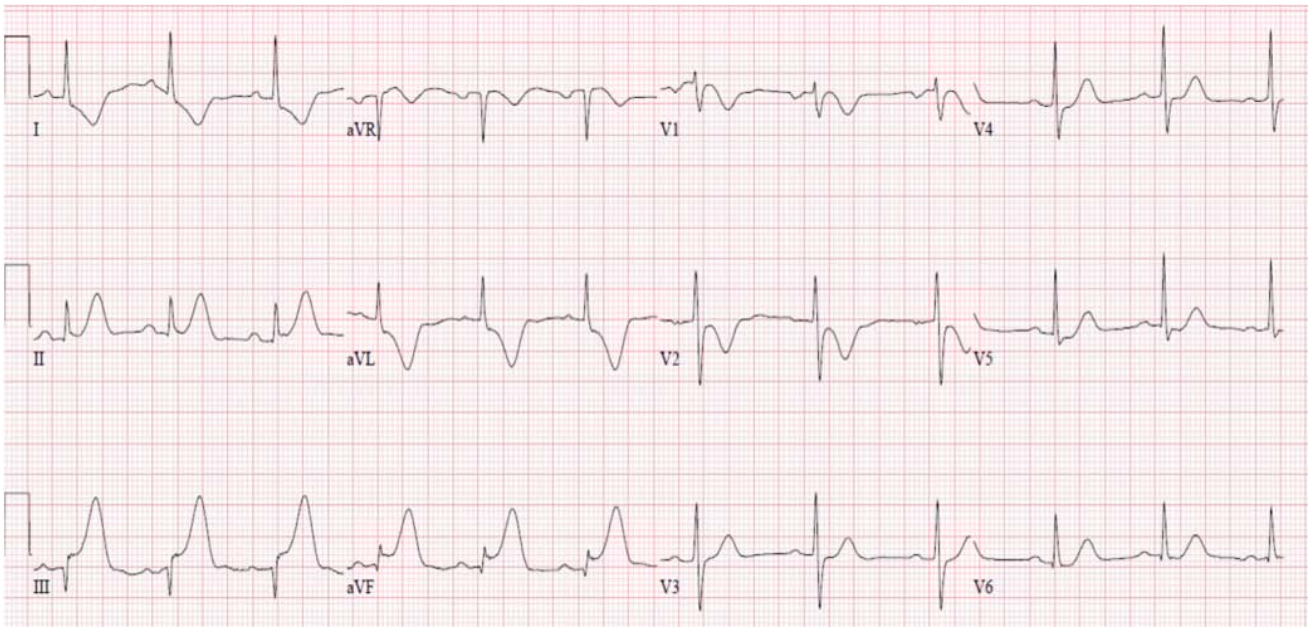

Figure 1. EKG: STEMI in the inferior leads II, III, aVF

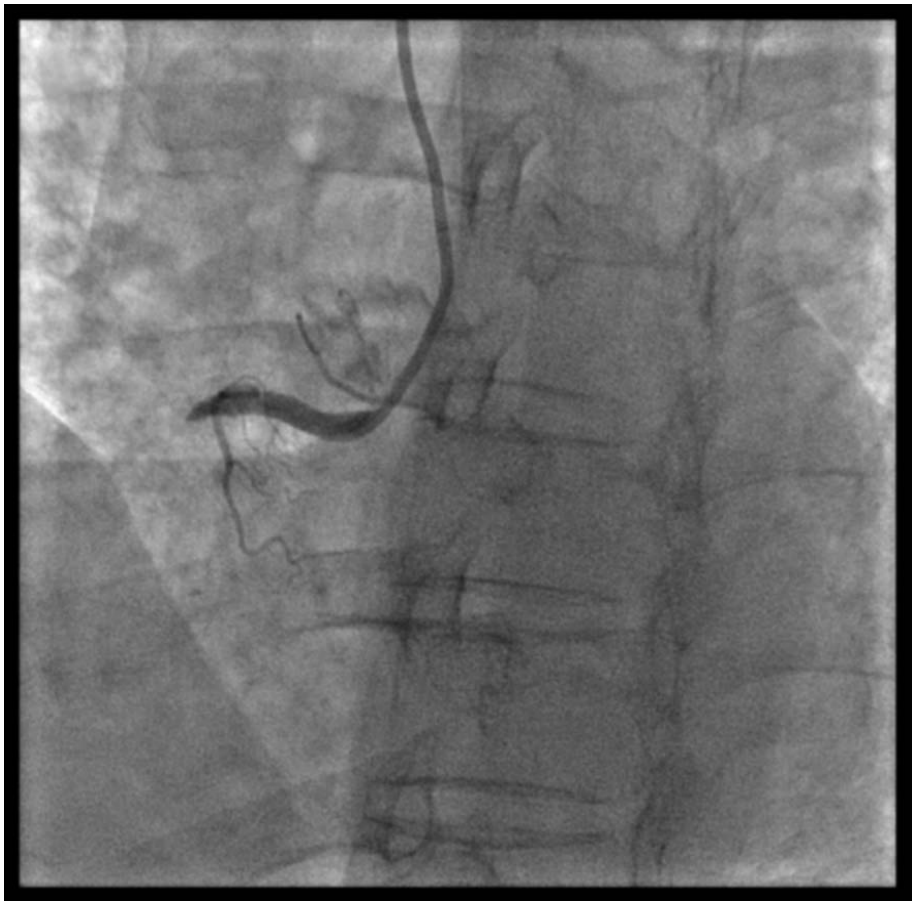

Figure 2. Coronary angiography revealed proximal RCA 100% Occlusion.

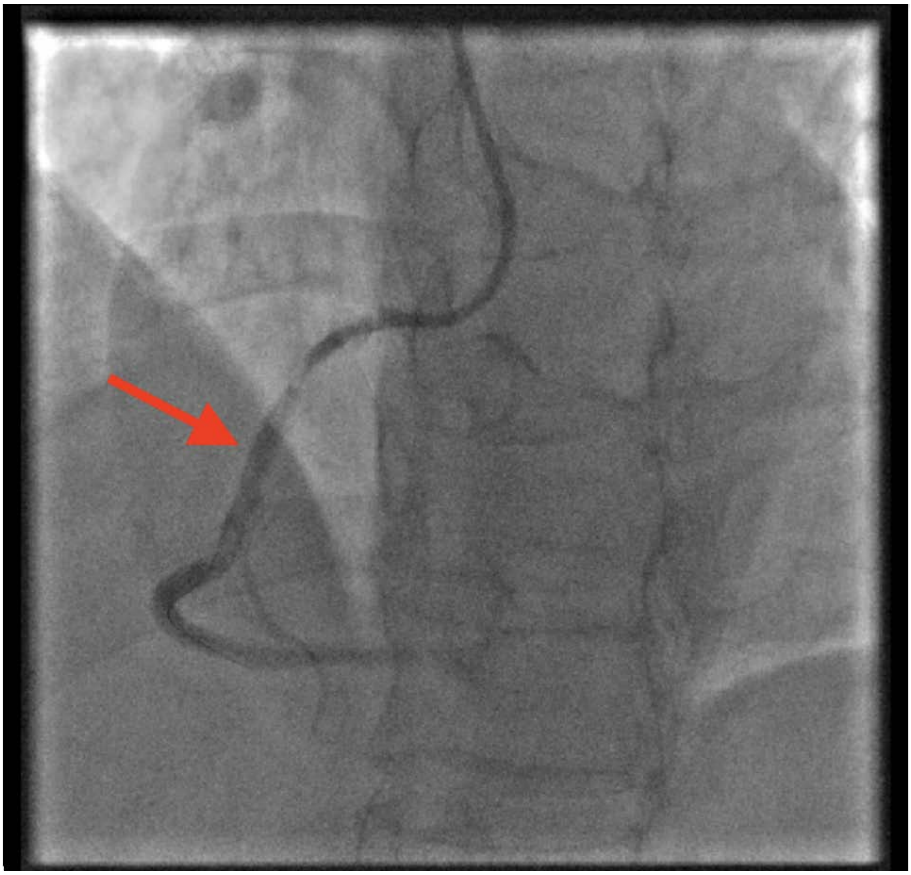

Figure 3. Coronary angiography revealed significant thrombus burden

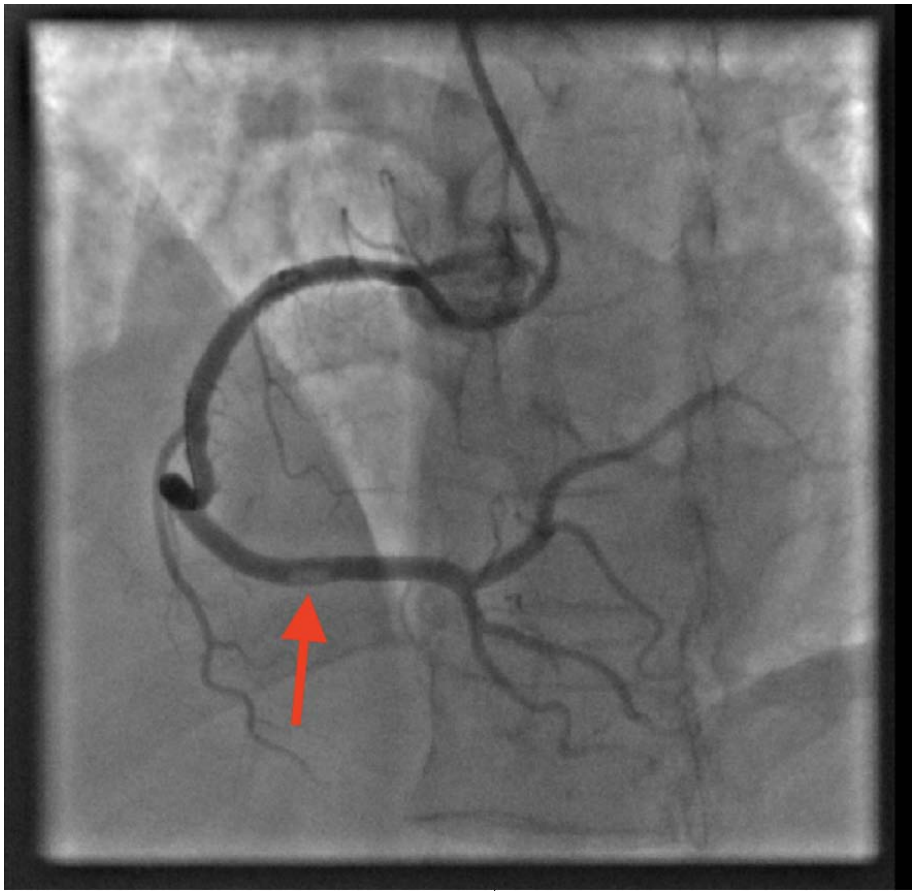

Figure 4. Coronary angiography revealed clot in transit (distal embolization).

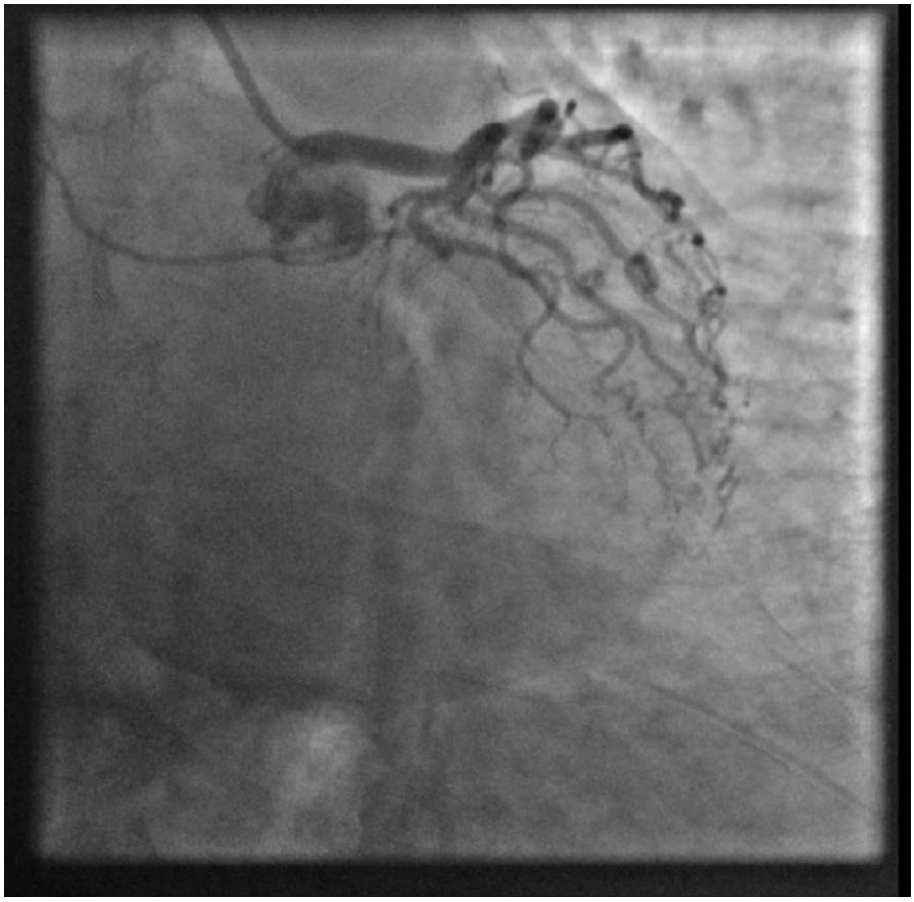

Figure 5. Coronary angiography revealed patent nondominant left system.

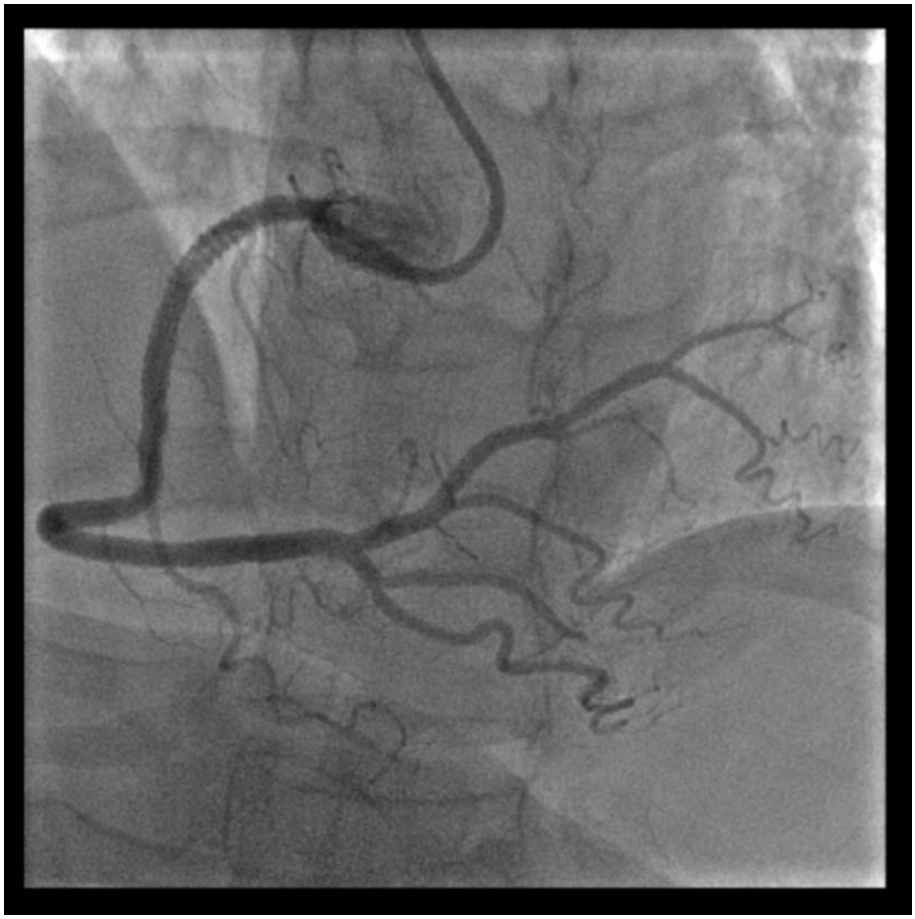

Figure 6. Coronary angiography revealed post PCI open RCA

2. Discussion

The coronavirus disease of 2019 (COVID-19) is a viral infection caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) and was announced a global pandemic by the World Health Organization in 2020. It is affecting all nations over the globe. COVID-19 is associated with higher morbidity and mortality in patients with preexisting cardiovascular disease. [1] While the global mortality of COVID-19 is approximately 2.5%, patients with cardiovascular disease exhibit a mortality of nearly 10.5%. These percent estimates continue to rise if there is evidence of cardiac injury on arrival (i.e. troponin elevation). [2] Early studies have shown that infection occurs as a result of binding of the viral spike protein to the human angiotensin-converting enzyme 2 (ACE2). These receptors are predominantly found in the type 2 pneumocyte in the lungs. The ACE2 binding sites can also be found in the blood vessels, oral mucosa and peri-infarct regions of myocardium. [3]

STEMI management amongst COVID-19 positive patients or patients under investigation (PUI) is a debated topic with multiple schools-of-thought. Despite a 38% reduction in STEMI activation after COVID-19 [4], cardiac catheterization teams still need to act in a timely fashion. Initial data from the Peking Union Medical College Hospital prioritized timing of symptom onset, with early presentation patients receiving thrombolytic therapy followed by medical management. [5] Institutional protocols have primarily driven ST-elevation MI's in COVID-19 patients in the United States. However, the American College of Cardiology (ACC), Society for Cardiovascular Angiography and Interventions (SCAI) and the American College of Emergency Physicians (ACEP) released a joint statement that PCI should remain the standard of care for STEMI patients at PCI-capable hospitals when therapy can be provided in a timely fashion. [6] In managing COVID-19 patients presenting with STEMI, it is prudent to ensure all cardiac catheterization staff have access to proper personal protective equipment (PPE) and the lab is ventilated with positive pressure. Finally, if the COVID-19 patient presents with a STEMI complicated by acute respiratory failure, it is advised to have the patient intubated in the emergency room prior to transport to the cardiac catheterization lab. [6] Because of the numerous delayed-presentation STEMI's in the COVID-19 era, clinicians have to be very keen on future complications in the upcoming months, including wall motion abnormalities, ventricular septal defects and evidence of left ventricular thrombi.

In a large retrospective study, [1] amongst 416 patients who tested positive for COVID-19, myocardial injury was found in 19.7% evident by elevation in high sensitivity troponin I levels. Patients with myocardial injury were found to have elevated in-hospital mortality of 51.2% vs 4.5% of those without evidence of myocardial injury. On the other hand, a study of *Tang et al.* [7] reported that in patients with severe COVID-19 with either elevated sepsis-induced coagulopathy (SIC) score or D-dimer results who received one of heparin derivates for 7 days or longer showed lower 28-day mortality in heparin users than nonusers, in patients with SIC score ≥4 (40.0% vs 64.2%) or D-dimer (32.8% vs 52.4%). While D-dimer levels can be mildly elevated in patients with and acute STEMI, severely ill COVID-19 patients have D-dimer levels well beyond the upper limit of normal. These results hypothesize the pro-thrombotic nature of COVID-19.

Preliminary autopsy pathologies of COVID-19 patients have revealed multiple small vessel thrombi resulting in alveolar hemorrhage. [8] Several studies reported an increased incidence of not only venous, but also arterial thromboembolism in severe COVID-19 patients. The theory behind this hypothesis might be related to cytokine-induced inflammation, hypoxemia, sepsis, diffuse intravascular coagulation (DIC), and in hospitalized and critical care (ICU) patients, immobilization. *Klok et al.* [9] reported 31% of COVID-19 patients admitted to ICU was found to have evidence of thrombosis. With this, several hospitals have begun starting anticoagulation in severe COVID-19 with elevated SIC or/and D-dimer with the recognition that early anticoagulation is needed to prevent propagation of microthrombi.

Our patient presented with a STEMI in the setting of COVID-19 and was found to have high burden of thrombosis in her right coronary artery. While coronary thrombi are common in STEMI, it is unknown if our patient's thrombus was primarily from plaque rupture and true acute coronary syndrome or from endothelial damage from the concomitant COVID-19 infection. With this hypothesis, it is reasonable to presume that many STEMI presentations in COVID-19 patients with minimal risk factors for cardiovascular disease (as in our patient) can be from the pro-thrombotic effects of the COVID-19 virus. This poses a challenge for anticoagulation while the STEMI patient with thrombus recovers from COVID-19. In order to treat our patient, we extrapolated data from the ATLAS ACS 2-TIMI trial which showed that in patients with recent acute coronary syndrome, addition of low dose rivaroxaban at 2.5 mg twice daily reduced overall and cardiovascular mortality without a significant increase in the rate of fatal bleeding. [10] Even though participants in this trial did not have COVID-19, we felt the benefit of anticoagulation (in addition to DAPT) would outweigh the risks of bleeding in our patient. At this time, further studies are needed to determine if the thrombus burden in a STEMI can be from a COVID-19 etiology. If so, the decision of optimal anticoagulation and platelet therapy in these patients need to be addressed, in order to minimize the risk of future thromboembolic events, while considering the elevated risk of bleeding.

3. Conclusion

Myocardial infarction is a potentially lethal complication of COVID-19 infection due to the prothrombotic nature of the viral disease. While the ideal long-term management of these patients with anticoagulation and DAPT is not well established, our case highlights the importance of early recognition of myocardial infarction in patients with COVID-19 infection and the identification of individuals at increased risk of thrombotic complications as well as considering the long-term management of anticoagulation and DAPT

in STEMI patients in this very high-risk, COVID-19 patients.

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