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Left Ventricular Thrombus of Unknown Etiology in a Patient With COVID-19 Disease With No Significant Medical History

Joseph Russell, Michael Wagoner, James DuPont, Douglas Myers, Krishnakumar Muthu, Sudhir Thotakura

Abstract

The incidence of left ventricular thrombus is relatively low. Ventricular thrombi typically manifest in patients with reduced ejection fraction and post myocardial infarction [1]. The impact of COVID-19's hypercoagulability state is presented here.

A 44 year old male who contracted COVID-19, progressed to moderate disease requiring inpatient treatment with supplemental oxygen. During the course of the hospital stay, while receiving National Institutes of Health guideline directed thromboembolism prophylaxis for COVID-19 infected patients [2], the patient developed a left ventricular thrombus which consequently embolized and occluded the left anterior descending and left circumflex coronary arteries requiring rheolytic thrombectomy.

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1. Introduction

The incidence of left ventricular thrombus in the common population is relatively low, with incidence most commonly occurring in a subset of the population with reduced ejection fraction or post myocardial infarction. McCarthy, et al. screened 140,636 echocardiograms and identified 128 (0.091%) total left ventricular thrombi. Of the 128 identified in the study, 68.5% had heart failure and 25.9% were post-acute myocardial infarction. The remaining 5.6% were occurrences from other etiologies [1]. In a select study of patients with anterior wall STEMI, ventricular thrombi were noted 3–12 days post myocardial infarction in 15% [3]. These processes may be further complicated by the hypercoagulable impact of COVID-19. One study identified an increased risk of arterial thromboembolism and mortality amongst COVID-19 patients hospitalized in the New York City Hospital System [4]. We present a case of COVID-19's hypercoagulability.

2. Case presentation

A 44 year old male with no significant past medical history, infected with COVID-19, who during his hospital stay developed a left ventricular thrombus despite receiving guideline directed thromboembolism prophylaxis for moderated-severe COVID-19 disease per National Institutes of Health recommendations at the time [2].

In August 2021, a patient who had tested positive COVID-19 was admitted to Texoma Medical Center for moderate COVID disease and acute respiratory failure with hypoxia after failed outpatient treatment. He was not a candidate for neither monoclonal antibodies nor remdesivir due to his presentation being greater than ten and twelve days post infection respectively. Patient was treated for moderate COVID disease with high dose dexamethasone and supportive care including supplemental oxygen. At time of admission, the patient did not have evidence of left ventricle thrombus per computed tomography angiography and was placed on 40 mg enoxaparin subcutaneously.

On seventh day of admission at 0101, he was noted to have ST-segment depression on telemetry monitoring. The patient was found to be asleep and resting comfortably on 5 L/min of supplemental oxygen. He denied chest pain or discomfort of any sort. A 12-lead ECG was performed and confirmed 3–4 mm of ST-segment elevations in the anterolateral leads with reciprocal ST-segment depression in the inferior leads. Laboratory evaluation revealed a troponin level of 14,109 ng/L and a repeat troponin of 20,384. Acute coronary syndrome protocol was initiated. He was given aspirin, atorvastatin, metoprolol, and started on an unfractionated heparin drip per protocol. Given the patient's complete absence of symptoms, the patient did not undergo...
emergent invasive treatment. Subsequent electrocardiograms at 0144 and 0532 were unchanged and patient remained asymptomatic. In the morning, transthoracic echocardiography showed normal left ventricular function with a very large and mobile left ventricular thrombus. Urgent coronary angiography demonstrated a 100% acute thrombotic occlusion of the ostial left anterior descending coronary artery with a portion of the thrombus extending into the left circumflex artery. Intracoronary thrombolysis was attempted using tissue plasminogen activator. An initial 6 mg dose was administered through the guide catheter, and subsequently a 10 mg dose was administered through a coronary micro catheter. Integrilin was initiated, but the patient developed oral bleeding, and thus discontinued. The patient was taken off of the table to discuss the findings with him and his family. He was brought back to the cardiac catheterization lab in the afternoon to attempt thrombectomy of the extensive coronary thrombus in the LAD artery using an AngioJet Spiroflex device. Given the potential for complete heart block with this device, we were worried that significant ectopy might serve as a trigger for embolization of the large left ventricular thrombus, particularly to the brain. Though off-label, we decided to insert a cerebral embolic protection device prior to the thrombectomy. After thoracic aortography was performed, a Sentinel device was implanted in standard fashion, involving the deployment of one filter in the brachiocephalic artery and another in the left common carotid artery. A temporary pacemaker wire was also placed in the right ventricle apex. With the use of an AngioJet Spiroflex VG rheolytic thrombectomy catheter, multiple passes were performed in the distal left main, left anterior descending artery and the second diagonal branch. This resulted in restoration of TIMI-3 flow in left anterior descending artery. Finally, the Sentinel device was retrieved with no evidence of clot noted, and the patient recovered post procedure in intensive care unit. After three days, he was transferred to a step down unit in stable condition with continued anticoagulation with intravenous unfractionated heparin for the treatment of his left ventricular thrombus. Repeat echocardiogram four days later showed significant decrease in the size of the left ventricular thrombus. On hospital day fifteen, the patient was discharged to home on coumadin for anticoagulation (Fig. 1).

3. Discussion

Studies have shown that COVID-19 has predisposed patients to venous and arterial thromboembolic disease [5]. A study by McGonagle, et al., identified that infected patients are at risk of developing cytokine storm, leading to hyperinflammation, endothelial disruption, platelet activation and coagulopathy, which contribute to thrombotic complications [6]. Early reports have also suggested that prophylactic doses of anticoagulation were inadequate for thrombosis prevention in hospitalized patients [7]. The ACTIV-4a trial identified superiority of therapeutic-dose anticoagulation in noncritically ill patients with COVID-19 as published in August 2021 [8]. In January 2022, the National Institute of Health updated their guidelines to therapeutic-dose heparin...
for noncritically patients who have a D-dimer above the upper limit of normal, require low-flow oxygen, and have no increased bleeding risk. Further stating that low molecular weight heparin is preferred over unfractionated heparin and recommend against the use of therapeutic dose oral anticoagulation [9].

Other studies have identified increased risk of thromboembolism in patients with respiratory infections. Smilowitz, et al., have identified an elevated risk of myocardial infection and thromboembolism in patients with viral respiratory illnesses was highest in the first 30 days after discharge [10]. These processes are similar to those of the hypercoagulability state presented by COVID-19 (Fig. 2).

Since the time when the SARS-COV-2 virus was unleashed on this world, multiple studies have been performed to identify the disease processes, risk factors and treatments. Through revolutionary scientific research, in a short matter of time, the world went from a newly released pandemic plaguing a society to effective measures in treatment and prevention. As a result of the relentless research from our scientific community, this case and many similar cases, could either be prevented, or have better outcomes due to the ever changing, updated guidelines in medical therapy. Further investigations may be warranted in prevention of thrombotic events in other respiratory illnesses.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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