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CASE REPORT

Where is the Thrombus ? Mural Ascending Aortic Thrombus with Embolic Events in a Patient with Recent COVID 19 Infection

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Abstract

The emergence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has triggered a global health pandemic that led to substantial morbidity and mortality worldwide. The virus has been known to predominantly cause severe hypoxemic respiratory failure but there have been multiple reports of extra-pulmonary manifestations. Additionally, there has been increasing evidence of COVID-19 hyper-coagulability. Herein, we present a case of a 49-year-old male with a past medical history of diet controlled type II diabetes mellitus and recently diagnosed COVID-19 who presented to the emergency department with a chief complaint of nausea and vomiting. Our patient was found to have a thrombus-like appearing 1.9 cm × 1.2 cm well-circumscribed mass, attached to the greater curvature of the ascending aorta, superior to the right coronary cusp of the aortic valve almost three weeks after his initial diagnosis of COVID-19 virus.

Keywords: Aortic Thrombus, COVID 19, Embolic stroke, Embolisation to coronaries

1. Introduction

In COVID-19 patients, coagulation abnormalities and thrombosis are frequently found despite prophylactic anticoagulation.[1] While most individuals infected with severe acute respiratory syndrome coronavirus 2 (SARS CoV 2) only experience mild to moderate symptoms, some suffer a more severe or fatal course of the infection. The main thrombotic complications are deep vein thrombosis, microvascular thrombosis, and pulmonary embolism.[1] The incidence of venous thromboembolism in COVID-19 patients admitted to the ICU during COVID-19 infection is reported to be as high as 27%. Arterial vascular events are seen in up to 3.7% of cases.[2] COVID-19 has been recently linked to large vessel stroke in young adults.

2. Case presentation

Our patient is a 49-year-old male with a past medical history of diet-controlled type II diabetes Mellitus who presented to the emergency department with a chief complaint of nausea and vomiting. He reported a one-day history of multiple non-bilious, non-bloody emesis; however, he denied any abdominal pain, constipation, diarrhea, fever, or chills. Symptoms were associated with generalized weakness without any numbness or focal deficits. Furthermore, the patient also endorsed polyuria and polydipsia for two weeks. He denied any chest pain, palpitations, shortness of breath, cough, or sore throat.

Moreover, the patient reported testing positive for SARS-COV-2 three weeks before his presentation to the emergency department and was experiencing fatigue and a sore throat.

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Upon presentation to the emergency department, the patient was awake, alert, and fully orienteto time, place, and person. He was afebrile and hemodynamically stable but tachycardic with a heart rate of 110–120 beats per minute and O2 saturation of 94% on room air.

Lungs were clear to auscultation bilaterally, no wheezes or crackles were heard. The chest was non-tender to palpation. JVP was non-distended, and no bruits were heard over the neck. On cardiac exam, I heard normal S1 and S2. No murmur, rubs, gallops, S3 or S4 were appreciated. The abdomen was soft, non-distended, and non-tender to palpation with normoactive bowel sounds. The skin was warm and dry with no rash. Extremities had no edema, calf tenderness, or erythema. Left-sided dysmetria with an abnormal finger-to-nose test was found on Neuro examination with normal sensory, motor, and reflex system.

Labs revealed blood glucose of 443 mg/dl (74–104), with small amounts of acetone, bicarbonate of 18 mmol/l (22–26), and an anion gap of 15 mmol/l (8–10). Troponin was 3.84 ng/ml (<0,05), brain natriuretic peptide: 761 pg/ml (<100). A white blood cell count of 16.1 K/UL (4.8–10) and platelet count of 441K/UL (140–400) was noted. The coagulation profile was aPTT: 22.7 s (23–38 s) and PT 15.1 s (12.6–12.6). Inflammatory markers were CRP 37.1 ng/ml (<1), ESR: 60 mm/h (0–20), D-dimer: 810 ng/ml (0–230). EKG showed an atrial flutter with the 4:1 atrioventricular conduction, and CT Head revealed an acute left superior cerebellar stroke.

The patient was admitted to the intensive care unit (ICU) for management of diabetic ketoacidosis (DKA) and non-ST segment elevation myocardial infarction (NSTEMI) associated with a new-onset superior cerebellar ischemic stroke. Trans-thoracic echocardiography was performed, which revealed a left ventricular ejection fraction of 20–25% with severe hypokinesis of the heart's basal, mid inferior, and lateral walls.

A thrombus-like appearing 1.9 cm × 1.2 cm well-circumscribed mass, attached to the greater curvature of the ascending aorta, superior to the right coronary cusp of the aortic valve, was also noted (Image 1). Treatment with therapeutic enoxaparin and dual-antiplatelet therapy along with goal-directed medical hearty for heart failure was started.

CT angiography and trans-esophageal echocardiography (Image 2) done three days later showed the absence of the aortic mass but a total occlusion of the mid-right coronary artery secondary to an embolus versus thrombus.

The patient did not develop any new neurovascular complications during his hospital stay and

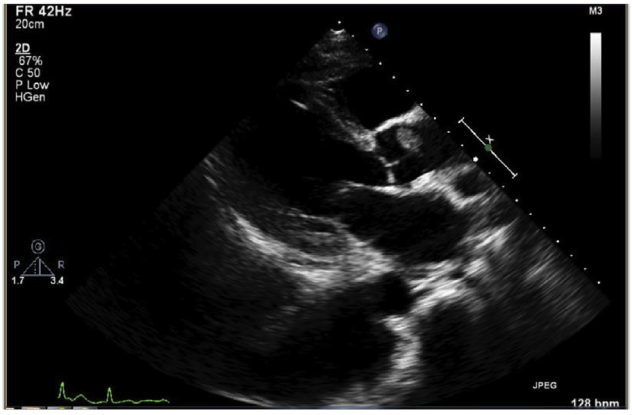

Image 1. Parasternal long axis view on trans-throacic echocardiography showing a thrombus above the aortic valve.

was discharged on 20 mg rivaroxaban. Cardiac Catheterization one and a half months later showed 100% occlusion of the mid-right coronary artery with collaterals from the left anterior descending to the distal right coronary artery. Also, 50% stenosis of the obtuse marginal and 40% stenosis of the mid-left anterior descending artery was noted. The patient denied any anginal symptoms at this time, and anticoagulation with rivaroxaban was continued.

3. Discussion

Studies have shown that COVID-19 infection is frequently complicated by a prothrombotic state and inflammation of the endothelium, which leads to damage and release of cytokines in the vasculature. Although venous thromboembolic events have been well documented in the literature, arterial thromboembolic events are increasingly being reported. Commonly known presentations are stroke, myocardial infarctions, aortic thrombosis, limb ischemia, and mesenteric ischemia.[5] Our patient was found to have an aortic thrombus, which likely

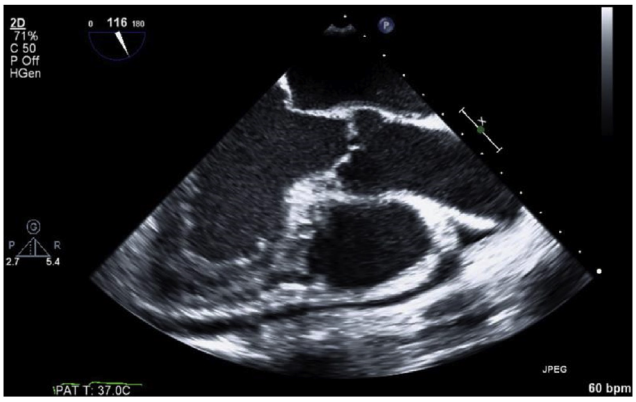

Image 2. Repeat echocardiography three days later shows absence of thrombus, indicating likely embolization.

embolized to the brain and the right coronary artery. However, one can argue that the occlusion in the right coronary artery could be secondary to an acute on chronic thrombus; in the absence of other signs of vasculopathy like calcification of the aorta and young age of the patient, an embolus seems more likely.

Avila et al. have reported that, in similar COVID-19 infection cases, labs are significant for elevated D-dimer and C-reactive protein levels.[5] D-dimer above 1000 ng/mL has been associated with increased mortality.[3] Although initially, it was believed that only medium and small-sized arteries are involved in thromboembolic events, cases large-sized arterial involvement is also being increasing reported.[4] Albeit thrombus in the ascending aorta is rare due to high pressure and blood flow in this region, many such cases are documented. Some known risk factors for the same in patients with COVID-19 are age >50, male gender, smoking history, and obesity.

These thrombi can cause severe complications by embolizing to the viscera and extremities, causing infarcts.

Gomez-Arbelaez et al. report four cases of arterial thrombosis in otherwise healthy patients who presented with normal prothrombin time, activated partial thromboplastin time, and platelet count.[6] Although our patient presented with elevated prothrombin time and activated partial thromboplastin time, the cases described by Gomez-Arbelaez et al. also had no history of hypercoagulable disease or atrial fibrillation similar to our patient. Despite the use of thromboprophylaxis measures, they were found to have an aortic thrombus.[6] Therefore, in patients presenting with embolic events in the absence of other risk factors, it is reasonable to screen for COVID-19. Treatment with therapeutic anticoagulation with close monitoring for embolic events is recommended. Prophylactic anticoagulation is vital to prevent these complications; however, the dose and duration of therapy remain to be investigated.

4. Conclusion

COVID 19 is a disease with a very high burden on society regarding physical health and economics. It is seen to affect both young and old individuals.

Although the disease primarily affects the lungs, it is considered to be a multi-system disorder involving many vital organs. A well-known complication related to the vascular system is thrombosis, both arterial and venous. Aortic thrombus can be deadly given its ability to embolize to vital organs, causing infarcts. The best management strategy and dose of anticoagulation to prevent or treat these thrombi are yet to be determined, and we request our readers to conduct further studies on it.

As COVID 19 is a reasonably new virus with a lot yet to be determined about the disease syndrome it causes, we request our readers to continue publishing data about its atypical features and complications.

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Conflict of interest

No conflict of interest was reported by the authors.

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