COVID myocardial involvement presenting as left ventricular aneurysm and clot associated with normal coronary anatomy, deep vein thrombosis, and abnormal brachial artery flow-mediated dilatation

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ABSTRACT

A 40-year-old non-diabetic, non-hypertensive male patient presented with complaints of dyspnea of a few days duration and coronavirus-19 disease (COVID) pneumonia. The electrocardiography (ECG) revealed sinus tachycardia with T inversion in V1 only. The ECG revealed a left ventricular aneurysm with a clot and severe left ventricular dysfunction. He had deep vein thrombosis involving the left lower leg. The cardiac magnetic resonance imaging revealed a left ventricular posterodorsal aneurysm with a large clot. Computed tomography angiography revealed normal coronaries and no evidence of pulmonary embolism or aortitis. The d-dimer was raised. A brachial artery Doppler revealed severe impairment of flow-mediated dilatation, suggesting endothelial dysfunction. He was stabilized with anti-platelets and anticoagulants, and diuretics.

Keywords: COVID-19, deep vein thrombosis, hypercoagulable state, left ventricular aneurysm

Introduction

In patients with the COVID-19 disease without any risk factors for atherosclerosis, an assessment of hypercoagulability and endothelial dysfunction may point to a non-atherosclerotic mechanism of vascular complications. We describe a case of severe myocardial dysfunction with posterodorsal left ventricular aneurysm formation and clot without any typical angina and electrocardiographic changes of myocardial infarction.

Case Description

A 40-year-old non-diabetic and non-hypertensive male presented to the emergency room with a history of dyspnea at rest for a few days. On admission, he was afebrile, the blood pressure was 90/60 mmHg, heart rate of 98 beats per min, SpO2 was 90% on 4 L of oxygen. The nasopharyngeal swab was positive for severe acute respiratory syndrome coronavirus 2 (SARS CoV2) by reverse transcription–polymerase chain reaction (RT-PCR). High-resolution computed tomography (HRCT) of the thorax revealed peripheral bilateral ground-glass haziness and consolidation [Figure 1a] and cystic aneurysm from left ventricle with hyperintense contents [Figure 1b]. Computed tomography angiography revealed normal coronaries and no evidence of pulmonary embolism or aortitis. The d-dimer was raised. A brachial artery Doppler revealed severe impairment of flow-mediated dilatation, suggesting endothelial dysfunction. He was stabilized with anti-platelets and anticoagulants, and diuretics.
left ventricular dysfunction, and posterobasal left ventricular aneurysm with clot and moderate mitral regurgitation [Figure 2].

Computed tomography coronary angiography revealed normal coronaries [Figure 1d], no pulmonary embolism, or aortitis. There was deep vein thrombosis of the left lower leg and elevated D-dimer. Brachial arterial Doppler showed impaired flow-mediated dilatation percentage (2.4%) suggestive of endothelial dysfunction. The heart's magnetic resonance imaging revealed a posterobasal aneurysm with a clot [Figure 1c] with severe left ventricular dysfunction. Tc 99 methoxyisobutyl isonitrile (MIBI) myocardial perfusion scan revealed evidence of scarred myocardium in the lateral wall of the left ventricle and hypo-refused myocardium in the anterolateral and inferolateral walls and normal perfusion in the apex, anteroseptal, and inferoseptal walls and septum with severe left ventricular dysfunction and ejection fraction of around 15% [Figure 3].

The patient received oxygen supplementation, steroids, diuretics, acetylsalicylic acid, and clopidogrel, and low-molecular-weight heparin. Because of the coexisting deep vein thrombosis and left ventricular clot, acitrom was started, and the international normalized ratio (INR) was targeted between 2 and 3. He recovered to New York heart association (NYHA) class II and was discharged.

**Discussion**

A patient with COVID-19 pneumonia with the requirement of supplemental oxygen and hypotension should undergo ECG

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**Figure 1:** (a) HRCT of the thorax in a COVID-proven adult male showing peripheral bilateral ground-glass haziness and consolidation (curved arrow). (b) Non-contrast CT-scan in mediastinal window settings showed hypodense cystic area (curved arrow) continuous with the wall of the left ventricle with hyperdense contents. (c) T2-weighted sequence on cardiac MRI demonstrated focal outpouching from the left ventricular wall and hypointense contents within showing susceptibility artifacts, MR diagnosis of a left ventricular aneurysm with a blood clot within was given. (d) Volume rendering is done after CT coronary angiography showing normal right and left coronary systems.

**Figure 2:** Electrocardiography showing the absence of significant changes suggestive of ST-elevation myocardial infarction and echocardiography showing dilated left ventricle, apex, and posterobasal area aneurysmal with a large clot.

**Figure 3:** Tc 99 MIBI myocardial perfusion scan revealed the evidence of scarred/non-viable myocardium in the lateral wall of left ventricle and hypo-refused myocardium in the anterolateral and inferolateral walls and normal perfusion in the apex, anteroseptal, and inferoseptal walls and septum with severe left ventricular dysfunction and ejection fraction of around 15%.
screening to rule out the underlying myocardial involvement, which may occur in the absence of typical angina and ECG changes suggestive of myocardial infarction. We considered COVID-related myocardial involvement and ischemic cardiomyopathy as differential diagnoses. The patient also had no risk factors for atherosclerotic heart disease or a family history of premature atherosclerosis. The presence of deep vein thrombosis and elevated D-dimer level >5000 ng/mL and left ventricular clot suggested a hypercoagulable state. There may be a possibility of coronary occlusion and recanalization or microvascular clogging and myocardial necrosis and scarring. Prompt anticoagulation seems to be key.

Szekely et al. reported in a case series of COVID-19 patients that 32% had a normal echocardiogram, right ventricular dilatation, and dysfunction was observed in 39% of the patients, followed by left ventricular diastolic dysfunction in 16% and left ventricular systolic dysfunction in 10% but did not report any left ventricular aneurysm with clot. Rare case reports of fulminant myocardial involvement, myocarditis, myopericarditis with tamponade have been described. The COVID infection has been reported to have raised fibrinogen levels and hypercoagulable state in a recent study.

**Conclusion**

COVID coagulopathy may lead to myocardial damage with minimal electrocardiographic changes. Family physicians and primary care providers should look for evidence of venous, pulmonary, or microvascular thrombosis and D-dimer elevation, and abnormal endothelial function may be clues to a generalized hypercoagulable state and non-atherosclerotic cause of myocardial involvement.

**Key points**

1. The COVID infection is associated with a hypercoagulable state, endothelial dysfunction, and may result in coronary, pulmonary, peripheral arterial and venous thrombosis.

2. The absence of atherosclerotic risk factors suggests a hypercoagulable state and may be associated with normal coronaries but can lead to permanent myocardial damage.

**Patient consent and ethical Clearance**

Written informed consent of the patient was taken. The study was approved by the institutional ethics committee via letter number—AIIMS/IEC/20/255, dated 09/05/2020, and is a part of COVID Cardiovascular Registry: Clinical Trial Registry of India via letter number CTRI/2020/05/025216 [Registered on: 16/05/2020].

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**Conflicts of interest**

There are no conflicts of interest.

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