Massive Biventricular Thrombi Complicating New-Onset Heart Failure in a Patient With Suspected COVID-19

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Background

The novel severe acute respiratory syndrome coronavirus-2 (SARS–CoV-2), first detected in December 2019, has spread to almost every country globally, causing extensive multiorgan failure and mortality. The World Health Organization nominated SARS–CoV-2 as the cause of the Coronavirus Disease 2019 (COVID-19) and has been deemed a pandemic. To date, little is known about the virus's pathologic features and causes of death. One of the more interesting aspects of COVID-19 infection is the unprecedented hypercoagulable state that it renders upon patients and is thought to be a fundamental aspect of mortality and morbidity. Recent clinical data showed that COVID-19 is associated with a hypercoagulable state and predisposes thrombotic complications, both in the venous and arterial circulations. Notably, distinct coagulation activation is correlated with multiorgan failure and increases mortality. Herein, we report extensive biventricular thrombi along with the new-onset severe systolic dysfunction as an unusual catastrophic presentation of COVID-19.

Case Description

The patient is a 58-year-old African-American male with no known past medical history. He presented to the emergency room complaining of shortness of breath. He had been at home taking care of his parents, who were both COVID-19 positive, for the past four weeks. He reported that he has been feeling unwell since then. The initial vital signs revealed; blood pressure 108/64 mmHg, respiratory rate 32/min, heart rate 122 beats/min, fever 98.1 F, oxygen saturation 60% on room air. Lung examination revealed bilaterally diminished breath sounds. Significant swelling and tenderness in the right lower extremity were found. The rest of the physical examination was unremarkable. Complete blood count showed lymphopenia. The D-dimer level was > 20 ug/mL. A chest X-ray showed extensive patchy bilateral alveolar findings compatible with viral pneumonia. The BNP level was 34824 pg/mL. Due to acute hypoxic respiratory failure the patient was intubated and admitted to the critical care unit. Initial SARS–CoV-2 PCR testing was negative in nasopharyngeal swab; however, given the strong suspicion of COVID-19 pneumonia, a repeat test was sent, and the patient remained isolated.

- ✓ **The hypercoagulable state of COVID-19, along with cardiac injury, can lead to an extensive intracardiac thrombus and severe systolic dysfunction even in young patients who don't have previous cardiovascular comorbidities.**
- ✓ **We urge awareness of severe and potentially fatal extensive thrombosis and cardiac failure as the initial clinical presentation of COVID-19.**

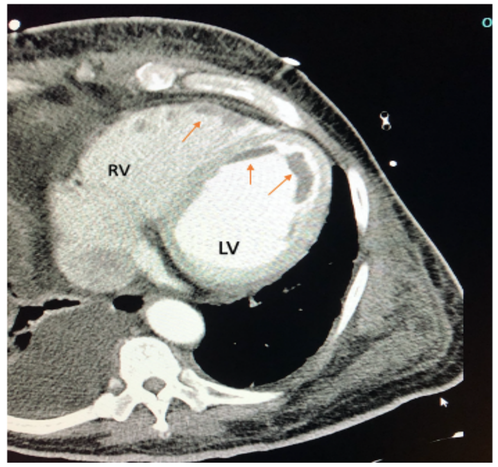

Figure 1: CT Angiogram imaging of the chest showing filling defects along right ventricle (RV) and left ventricle (LV) representing thrombus (arrows)

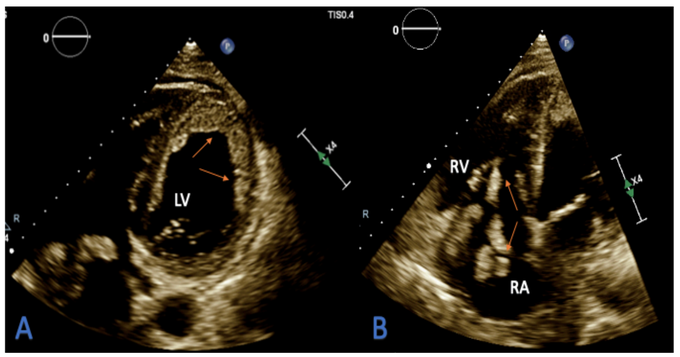

Figure 2A-B. Transthoracic echocardiography (TTE) showing; **A:** An extensive mural thrombus (arrows) seen along all walls of the left ventricle (LV). **B:** Mobile right atrial (RA) thrombus (arrows) extending across the tricuspid valve and extending into the right ventricle (RV).

Case Description cont'd

He was found to have extensive right leg deep vein thrombosis in the right common femoral vein, right popliteal vein, right posterior tibial vein, right peroneal vein, right gastrocnemius, and right small saphenous vein. The CT Angiography of the chest showed bilateral pneumonia with large right pleural effusion, and biventricular thrombi but negative for actual pulmonary embolism **(Figure 1)**. The transthoracic echocardiography showed an ejection fraction of 10% to 15% with biventricular failure and severe global hypokinesis. There was an extensive mural thrombus seen along all walls of the left ventricle. Also, there was a mobile worm-like thrombus in the right atrium with an extension across the tricuspid valve into the right ventricle **(Figure 2A-B)**. The hypercoagulability panel was within normal limits. Anticoagulative treatment was initiated. Unfortunately, he had a cardiac arrest due to persistent ventricular fibrillation and was unable to be resuscitated. He expired within 24 hours of admission.

Discussion

COVID-19 is an emerging threat due to the risk of microvascular, venous, and arterial thrombosis, thereby exacerbating organ injury and mortality. Although the exact mechanism of extensive thromboembolism and myocardial injury caused by SARS-CoV-2 is not illuminated, it is clear that COVID-19 related hypercoagulation increasing the fatality of the disease. In particular, COVID-19 infection may trigger endothelial dysfunction, systemic inflammation, and an increased pro-coagulatory state by tissue factor pathway activation.

In addition to VTE, information on cardiac injury in patients with COVID-19 is limited. There have been various purported mechanisms of cardiac dysfunction in COVID 19. Myocardial injury is a common thread and is evident from elevated troponin, which is ubiquitously seen but not specific for etiology. The various causative mechanisms identified are demand ischemia from acute hemodynamic stresses commonly seen in infections such as fever, tachycardia and adrenergic surge, and hypotension. Acute coronary syndrome, stress cardiomyopathy, and fulminant myocarditis have also been described.