

Fatal Large Intracardiac in Situ Thrombus in a Mildly Symptomatic COVID-19 Patient: A Case Report

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Background: The coronavirus disease 2019 (COVID-19) has caused a global pandemic with a significant negative impact on health worldwide. Although it primarily involves the respiratory tract system, cardiovascular complications, particularly thrombotic events are increasingly reported and are associated with adverse outcomes.

Case Summary: We describe a 30-year-old male who presented with exaggerated dyspnea and hypoxia with mild constitutional symptoms along with a positive PCR test for SARS-CoV-2. He was a known case of bronchiectasis and large bullae in the left lung. Transthoracic echocardiography showed a large right ventricle (RV) mass entrapped within RV papillary muscles and apex. Cardiac MRI was suggestive of RV thrombosis. Regarding diffuse underlying lung disease and improving respiratory symptoms with COVID-19 negative PCR test, we decided to prescribe anticoagulation with heparin infusion and then oral anticoagulant with Rivaroxaban 20 mg daily and follow up after 30 days. Unfortunately, the patient died after 2 weeks in his hometown.

Conclusion: Thrombotic complications are frequently encountered in COVID-19 patients. We report a fatal case of large intracardiac thrombus associated with SARS-CoV-2 infection managed with anticoagulant therapy with a catastrophic outcome.

Keywords: Intracardiac thrombus; Thrombosis COVID-19

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a systemic infectious disease caused by the new severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Its spread caused a global pandemic with more than 140 million infected and more than 3 million deaths worldwide as of 20 April 2021 (1). Although SARS-CoV-2 infection mainly affects the lower respiratory tract system, numerous reports suggest serious cardiovascular complications including acute myocardial injury and myocarditis, cardiomyopathy, arrhythmia, acute coronary syndrome, and massive pulmonary embolism.

Furthermore, the hyperinflammatory state due to cytokine storm, hypoxia, and immobilization in COVID-19 patients imposes a high risk of venous thromboembolism (VTE) (2,3). We report a case of a large right heart thrombus in the apex of the right ventricle (RV), entrapped within RV papillary muscles. It led to a fatal outcome in a 30-year-old male with mildly symptomatic SARS-CoV-2 infection and with underlying chronic lung disease.

CASE SUMMARIES

A 30-year-old male with a previous history of bronchiectasis presented to the SARS-COVID-2 triage with

a 1-week history of progressive dyspnea, fever, malaise, and myalgia. On initial examination, he was hypoxic and tachycardic with O₂ saturation of 82% in room air and heart rate =140 bpm. ECG showed sinus tachycardia, P pulmonale pattern with tall peaked P wave in II, III, and aVF leads.

Laboratory workup was pertinent for anemia with Hb=9.7 gr/dl, leukocyte counts 10600 with 76% PMN and normal platelet count. ESR was elevated up to 110 mm/h and C- reactive protein level (CRP) was elevated at 100 mg/lit. He had normal cardiac biomarkers CK-MB and troponin levels, normal ferritin level, and normal biochemistry results.

Seum Vitamin-D3 was at the range of insufficient level (20 ng/ml). The polymerase chain reaction test (PCR) of the nasopharyngeal swab was positive for SARS-CoV2 on the first day of admission.

Chest CT scan showed hyperaerated lungs and emphysematous bulla in the left upper lobe (LUL), cystic bronchiectasis, and bronchiectasis in both lungs with air-fluid level and scattered nodules in both lungs.

Transthoracic echocardiography (TTE) was done due to hypoxia that showed a large not highly mobile mass entrapped in the RV, apex, and within RV papillary muscles with the size of 25x15 mm mostly suggestive of well-organized RV thrombus. The RV was mildly enlarged with mild systolic dysfunction and systolic pulmonary artery pressure (PAP)=45 mmHg. Left ventricle ejection fraction (LVEF) was preserved at 50% (Figure 1, 2).

The patient was clinically stable with no respiratory distress or hypotension. After 5 days of supportive therapy, systemic corticosteroid therapy, and supplemental nasal oxygen, O₂ saturation reached 97% and the PCR test for COVID-19 became negative.

A cardiac MRI was performed that also was suggestive of RV thrombus (Figure 3). Our team decided to prescribe a full dose of heparin infusion. After 10 days, there was no significant change in the size of the RV thrombus. The patient was discharged with oral anticoagulant therapy on the 15th day of admission with Rivaroxaban 20 mg daily. As his hometown was far away from our center and because of the pandemic situation, we recommended a follow-up after 4 weeks of anticoagulation.

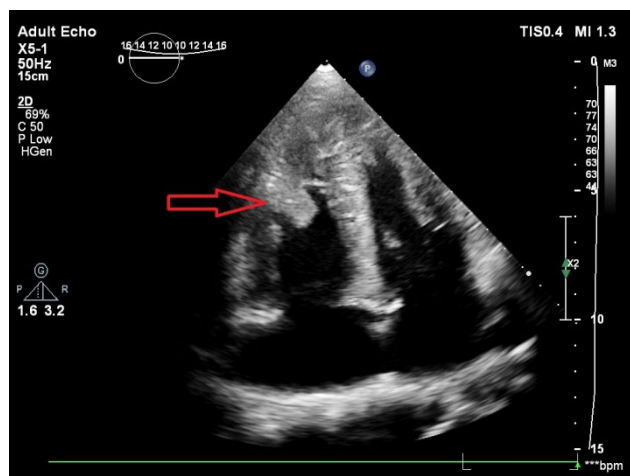


Figure 1. Apical 4-chamber view of transthoracic echocardiogram (TTE) showing a large mass (red arrow) in right ventricle (RV) entrapped within papillary muscles

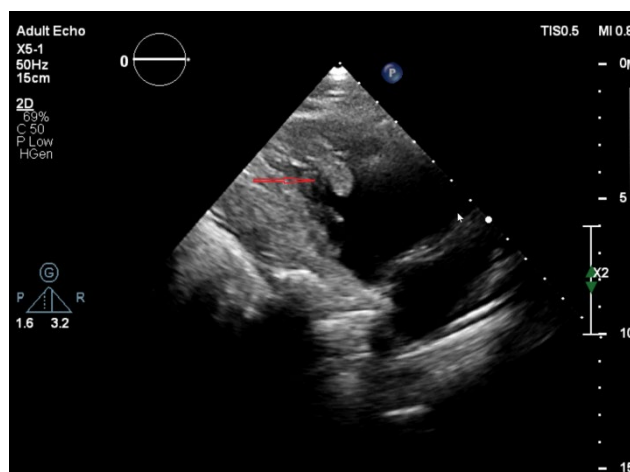


Figure 2. Transthoracic off-axis RV-inflow view showing entrapment of mass (red arrow) within RV papillary muscles and apex

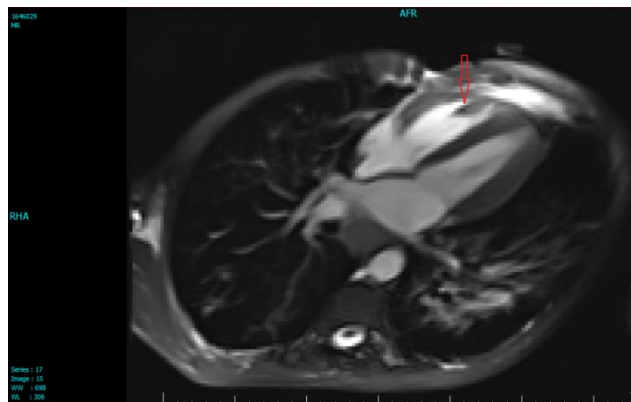


Figure 3. Cardiac MRI showed RV mass suggestive for RV thrombus

Unfortunately, the patient died suddenly in his home after 2 weeks of discharge due to undefined etiology.

DISCUSSION

Patients with COVID-19 and cardiovascular complications or underlying heart disease have worse outcomes. In situ and intracardiac thrombosis is occasionally encountered in these patients. Thrombotic complications are commonly seen in COVID-19 patients (4). In a multicenter study from the Netherlands, the incidence of VTE was 31% among COVID-19 patients admitted to the ICU ward. In another study conducted in Wuhan, China, VTE incidence was 25% (5).

The underlying mechanisms of accelerated thrombosis are hypercoagulability induced by hyperinflammatory state, endothelial dysfunction, and stasis. Several laboratory parameters suggestive of this hypothesis are characterized by elevation in fibrinogen level and D-dimer level (6).

In some patients with severe COVID-19 infection, thrombosis is the result of disseminated intravascular coagulation (DIC). Interleukin-6 (IL-6) levels were also correlated with disease severity and procoagulant profile (7).

Large vessel or intracardiac thrombus formation have been reported rarely in COVID-19 patients case reports (4). Our patient had a large right RV thrombosis. He was clinically stable with no acute respiratory failure; thus, cardiac MRI was done for a better evaluation of RV mass which was highly suggestive for RV thrombus. We decided to prescribe anticoagulant therapy for RV thrombus. The percutaneous thrombectomy or Angio-Vac system was reported successfully in hemodynamic instability or cardiogenic shock as a result of massive pulmonary emboli from RV thrombus. However, this device was not available in our country in addition to the fact that our patient was clinically stable with no hemodynamic instability (8). There have been case reports of successful thrombolytic therapy for right heart thrombus in transit or massive pulmonary emboli in patients with hemodynamic compromise. For

clinically stable patients, isolated anticoagulation therapy is appropriate (9,10).

In our case, surgical thrombectomy or thrombolytic therapy was not performed due to high surgical and bleeding risk, as a result of underlying lung disease and diffuse bronchiectasis, despite clinical stability. So, we discharged the patient after 15 days on Rivaroxaban 20 mg daily but unfortunately, he died after 2 weeks with unspecified cause.

Herein, we report a case of large intracardiac and in situ thrombus of the right ventricle associated with SARS-Co V-2 infection with grave outcome. Our case highlights the need for a more invasive approach in addition to anticoagulant therapy, such as Angiovac or surgical thrombectomy, for patients with poor prognosis.

Consent

The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient compatible with Ethical Committee of Shahid Beheshti University of Medical Sciences (Committee's reference number: IR.SBMU.NRITLD.REC.1396.398).

Acknowledgments

None

Conflict of interest

The authors declare that there is no conflict of interest.

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