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## Case Report

## Multimodality imaging in COVID19 patient with large mobile RV thrombus protruding into the pulmonary trunk with bilateral pulmonary embolism



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## ABSTRACT

Inflammation related to coronavirus disease (COVID)-19 can promote a pro-thrombotic process and subsequent thrombosis. We report a confirmed COVID-19 case in a 51-year-old patient who presented with chest pain and severe hypoxemia. Although the right heart chambers are unusual locations for a thrombus, an echocardiogram demonstrated a large mobile right ventricular thrombus protruding into the right ventricle outflow tract. A computed tomography angiogram and cardiac magnetic resonance image showed the extension of the thrombus into the pulmonary trunk. A continuous intravenous unfractionated heparin infusion resulted in a dramatic clinical and echocardiographic improvement.

**<Learning objective:** With COVID-19 infection, the thrombus phenomenon is becoming more common. Since thromboembolic complications are common in patients with severe COVID-19 who need intensive care unit care, heparin infusion is recommended for patients who are unlikely to need procedures. Multimodality imaging can be useful in determining the diagnosis, prognosis, and treatment plan.>

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## Introduction

Thrombosis related to coronavirus disease (COVID)-19 has been frequently reported during the disease's active phases and recovery period and can be associated with higher morbidity and mortality. The most reported thromboembolic phenomena associated with COVID-19 are acute coronary syndrome, pulmonary embolism (PE), deep vein thrombosis, superior mesenteric artery thrombosis, renal infarct, and acute ischemic stroke [1,2]. The hypercoagulability that accompanies COVID-19 is caused by systemic inflammation, endothelial damage, immobility, and hypoxia [3].

A right ventricular (RV) thrombus is uncommon. It is typically observed with a coinciding pulmonary embolism and can worsen the prognosis [4]. We present a case report with a large mobile RV

thrombus protruding into the RV outflow tract (RVOT) in a confirmed COVID-19 case.

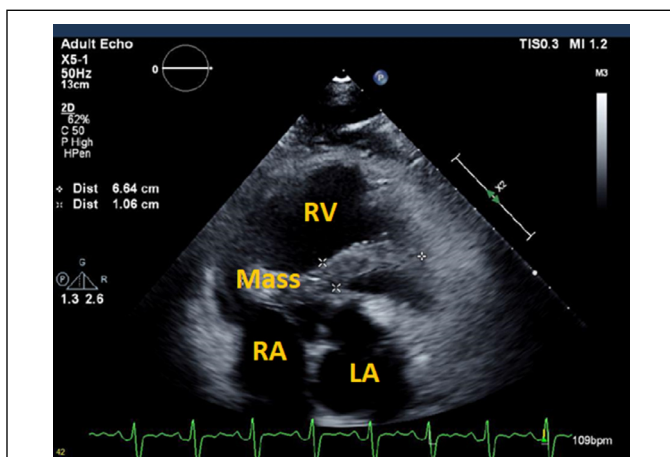
## Case report

A 51-year-old man with no significant previous medical history presented with a fever (38.8° C), diarrhea, shortness of breath, and pleuritic chest pain for four days before admission. On physical examination, his blood pressure was 126/77 mmHg, heart rate was 100 beats/min, oxygen saturation was 88% on room air, and respiratory rate was 25/min. His cardiovascular examination was remarkable for tachycardia and a loud second heart sound. His laboratory tests revealed an elevated troponin level of 0.528 ng/mL (normal range 0–0.05 ng/mL) with a high pro-B-type natriuretic peptide of 3936 pg/mL (normal range 0–125 pg/mL), a D-dimer level of 9.08 mg/L (normal range 0–0.5 mg/L), and a lactate dehydrogenase of 301.32 U/L (normal range 85–227 U/L).

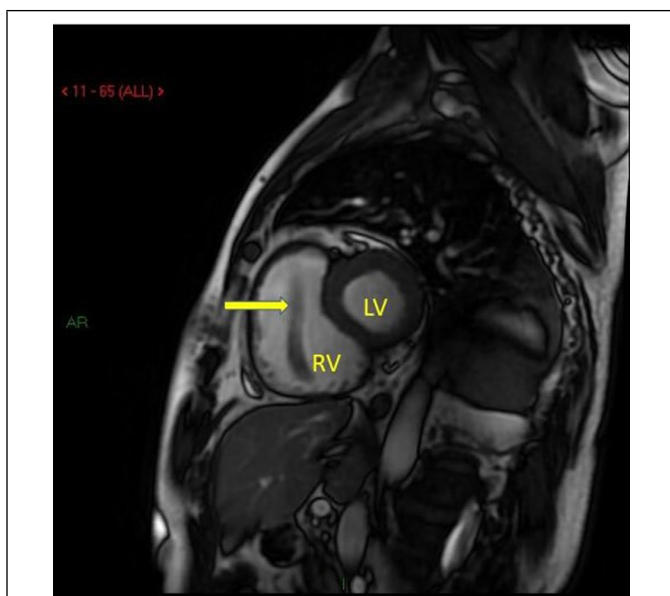
The initial electrocardiogram showed sinus tachycardia with an S1Q3T3 sign (Fig. 1). A chest radiogram demonstrated multiple

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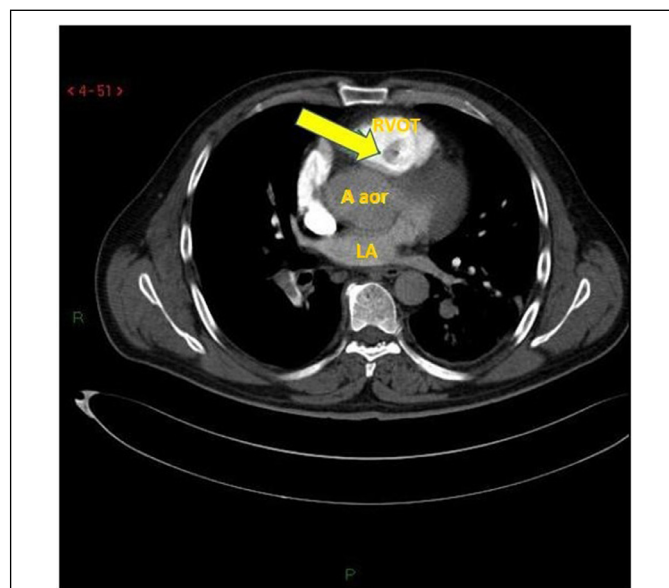
E-mail address: [emancardiology@yahoo.com](mailto:emancardiology@yahoo.com) (E. Elsheikh).



**Fig. 1.** 2D parasternal short-axis view showing long mass in right ventricular outflow tract. LA, left atrium; RA, right atrium; RV, right ventricle.



**Fig. 2.** Cardiac magnetic resonance imaging short-axis image demonstrates elongated filling defect (yellow arrow) in the right ventricle with enlargement of the right ventricle. RV, right ventricle; LV, left ventricle.



**Fig. 3.** Axial computed tomography (CT) scan of the CT pulmonary angiogram at the level of RVOT showing extension of the right ventricular thrombus to the pulmonary trunk (yellow arrow). RVOT, right ventricular outflow tract; A aor, ascending aorta; LA, left atrium.

left-sided opacities. Nasal swab polymerase chain reaction testing was positive for COVID-19. A transthoracic echocardiogram (TTE) demonstrated a large, freely mobile mass in the RV cavity extending towards the RVOT and reaching the pulmonary valve with a positive McConnell’s sign (regional RV dysfunction with mid-free wall akinesia but normal movement at the apex; **Fig. 2**, **Movie 1**, **Movie 2**, **Movie 4**, and **Movie 5**). Intravenous unfractionated heparin infusion was started at once with initial bolus dose 80 units/kg IV followed by heparin infusion 18 units/kg/hour with activated partial thromboplastin time follow up until reaching therapeutic range of 60–80 sec. Computed tomographic angiography and cardiac magnetic resonance imaging showed a bilateral submassive PE, dilated RV, an RV thrombus extending to the RVOT and the pulmonary trunk, and left lower lobe consolidation, likely lung infarction (**Fig. 3**, **Online Figs 1–3**, **Movie 3**, and **Movie 6**). Besides PE management, the protocol for patients with confirmed COVID-19 is using favipiravir: 1800 mg/dose twice a day on the first day followed by 800 mg/dose twice a day for 7–10 days.

Given his hemodynamic profile, the cardiology and cardiothoracic surgery team discussed various therapeutic approaches, such as systemic thrombolysis and surgical embolectomy [5]. However, as a COVID-19 outbreak was a major concern, the management plan was to commence intravenous unfractionated heparin while awaiting coagulation profile results and excluding any sources of embolization. Regardless, thrombolytic therapy would be considered if the hemodynamic profile deteriorated. A lower limb duplex revealed no evidence of deep vein thrombosis. A coagulation profile showed normal prothrombin time, partial thromboplastin time, protein C, and protein S levels, and negative anti-cardiolipin IgG and B2 glycoprotein 1 (IgG) antibodies. The patient’s vital signs showed gradual improvement. Moreover, a serial TTE confirmed regression of the thrombus. As the pre-discharge TTE showed a normal RV function with no remnant clot, the patient was discharged home on a therapeutic dose of warfarin.

### Discussion

We reported on a COVID-19 patient presenting with a submassive PE as an extension of a large RV thrombus. In this case report, we used multimodal imaging to confirm the diagnosis. There are multiple mechanisms of thrombosis in COVID-19, such as increased blood viscosity, hypoxia-inducible transcription factor-dependent signaling cascade [6], hypoxia, direct infection symptoms, and disseminated intravascular coagulation [7]. Similar to this case, Athappan et al. and Kaplan et al. reported right heart mural thrombi in COVID-19 patients [8, 9]. Evolving literature has demonstrated the importance of multimodal imaging, including echocardiography, computed tomographic pulmonary angiography, and cardiac magnetic resonance imaging in a diagnosis and management plan [1–3]. The treatment choices for RV thrombosis include anticoagulants, thrombolytic therapy, and surgical thrombectomy. Studies suggest a survival benefit with either thrombolysis or surgery [10], while active thrombolytic therapy has been reported in unstable COVID-19 patients complicated with right heart thrombi [10]. The patient’s stable presentation led us to begin with a conservative approach. Despite the acuity of his illness, his rapid im-

provement within 24 hours convinced us to postpone thrombolysis or thrombectomy. We do not yet have adequate answers to questions emerging from the complexities and management problems of severe COVID-19 cases, as the global scientific community is still studying COVID-19 complications. However, in patients with serious COVID-19 requiring intensive care unit treatment, thromboembolic complications are common, and heparin infusion may be recommended for patients who are unlikely to require procedures.

### Conclusion

We have detailed a case of a critical RV thrombus in a COVID-19 patient. COVID-19 likely contributes to a higher risk of developing thromboembolic disorders. Clinically diagnosing and treating thromboembolic consequences in this condition requires a high level of attentiveness. Finally, multimodal imaging plays a key role in the diagnosis, prognosis, and treatment approach.

### Declaration of Competing Interest

The authors declare that there is no conflict of interest.

### Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:[10.1016/j.jccase.2021.08.008](https://doi.org/10.1016/j.jccase.2021.08.008).

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