

Right Heart Atrioventricular Thrombus in a SARS-CoV-2 Patient without Pulmonary Embolism: A Case Report

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Abstract

SARS-CoV-2, the novel Coronavirus of 2019, and its associated coagulopathy has been reported to have many deleterious complications involving the cardiovascular system. Despite these many observed effects, there is a paucity of reports regarding SARS-CoV-2 and cardiac thrombus formation. In the absence of SARS-CoV-2, thrombi in the heart are typically seen in the left ventricle, with right heart thrombi (RHT) being very uncommon. When present, RHT are usually associated with pulmonary embolism (PE), which can lead to right sided heart failure in some cases. In rare instances, right heart thrombi are present in the absence of PE. In either situation, timely identification of RHT is critical as it carries a high mortality without timely treatment. Here, we present a unique case report detailing the presence of severe right heart failure from right atrioventricular thrombus without PE in a previously healthy patient diagnosed with SARS-CoV-2.

Keywords

Cardiac thrombus, Right heart thrombus, SARS-CoV-2, Transthoracic Echocardiography, Transesophageal Echocardiography, Anticoagulation

Introduction

The novel Coronavirus of 2019 (SARS-CoV-2) quickly became an epidemic, challenging healthcare delivery across the globe. From the abundance of literature reports, the complications of SARS-CoV-2 have been well documented, such as respiratory failure, cardiac complications, thromboembolic complications, secondary infections, and complicated recovery with long-term sequelae. In terms of cardiovascular complications, the medical community has seen a high prevalence of cardiovascular disease in patient with SARS-CoV-2, including cardiomyopathy, arrhythmia and acute myocardial injury. Some reports have suggested that 20-25% of SARS-CoV-2 patients have had some sort of cardiac complication [1]. Of the SARS-CoV-2 effects on the cardiovascular system, very little has been described regarding cardiac thrombus formation. Hypercoagulability has been a predominant feature in the overall clinical picture of SARS-CoV-2, hypothesized to be due to the actions of pro-inflammatory cytokines that alter the homeostatic mechanisms of endothelial cells as well as the possible infiltration of endothelial cells by SARS-CoV-2 viral particles, resulting in cell injury and a pro-thrombotic state [2]. Although some reports suggest a 31% incidence of thrombotic complications in intensive care unit (ICU) patients infected with SARS-CoV-2, the frequency or mechanism of intracardiac thrombus remains unknown [3].

Typically, intracardiac thrombus is seen in the left ventricle (LV), using transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE). On rare occasions, irrespective of SARS-CoV-2 infection, thrombi are found in the right atrium (RA) or right ventricle (RV). This is associated with high early mortality, usually from concomitant pulmonary embolism (PE) [4]. Right heart thrombi (RHT) are thought to originate from mobilized deep vein thromboses that have temporarily become stuck in the RA or RV [5]. Few case reports have portrayed intracardiac thrombus in the setting of SARS-CoV-2. Here we present one of the first case reports documenting a SARS-CoV-2 positive patient found to have a right heart atrioventricular thrombus with depressed RV function in the absence of PE.

Case Report

A 42-year-old male with no medical history presented to the emergency department with ten days of fevers and shortness of breath. On initial evaluation, the patient was mildly tachycardic and tachypneic, with an oxygen saturation of 82% on room air. The patient was placed on 15L simple mask, with improvement in saturations to > 90%. Examination showed an obese, diaphoretic male in mild respiratory distress, with no labored respirations and clear lungs. Laboratory investigations were significant for WBC $6.9 \times 10^9/L$, LDH 779 U/L, CRP 101.0 mg/L, D-dimer 804 ng/mL, ferritin, 502 ng/mL and procalcitonin 0.16 ng/mL. Chest radiograph demonstrated diffuse bilateral hazy opacities suspicious for multifocal pneumonia (Figure 1). SARS-CoV-2 testing was positive. The patient was admitted to the floor and initiated on Decadron, Lovenox prophylactic dosing and received convalescent plasma.



Figure 1: Chest radiograph showing diffuse bilateral hazy pulmonary parenchymal opacities suggestive of multifocal pneumonia with SARS-CoV-2 as a likely etiology. There is no evidence of wedge infarction.

While on the simple mask, the patient had rapidly progressing hypoxia, prompting the need for non-invasive mechanical ventilation. After an episode of acute decompensation with hypotension and hypoxia, the patient was emergently intubated, transferred to the ICU, started on vasopressors and, inhaled nitric oxide and referred for extracorporeal membrane oxygenation (ECMO) assessment. TEE pre ECMO showed a D-shaped LV with an ejection

fraction of 45%, no regional wall motion abnormalities, and an enlarged RA and RV, with 2.2 cm thrombus extending from the RA/inferior vena cava junction through the tricuspid valve into the RV (Figures 2a and 2b). There was evidence

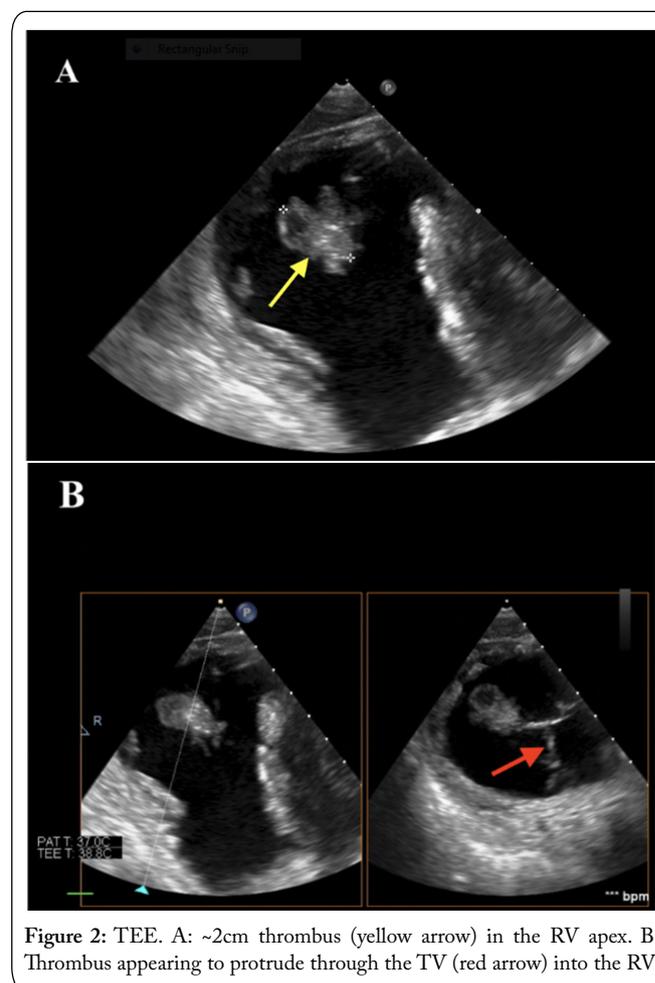


Figure 2: TEE. A: ~2cm thrombus (yellow arrow) in the RV apex. B: Thrombus appearing to protrude through the TV (red arrow) into the RV.

of RV pressure overload but systolic function was grossly normal. No tricuspid regurgitation was visualized. The patient was initiated on heparin infusion. Computed Tomography Angiography (CTA) of the chest was subsequently performed, showing bilateral consolidations consistent with SARS-CoV-2 pneumonia, but no evidence of PEs (Figure 3).

Duplex ultrasound of the lower extremity was negative for deep vein thrombosis (DVT) as well. He was soon placed on veno-venous ECMO (vv-ECMO) for acute hypoxic respiratory failure, acute respiratory distress syndrome and severe pneumonia secondary to SARS-CoV-2. Due to new onset thrombocytopenia while on heparin infusion, there was concern for development of disseminated intravascular coagulation versus heparin induced thrombocytopenia. Thus, the patient was switched to argatroban infusion one month into their ICU stay. While on vv-ECMO, the patient continued to deteriorate, with worsening RV function and subsequent RV failure, refractory to pressors and inotropes. Interval echocardiographic studies demonstrated small decreases in size of RV thrombus while first on heparin and then argatroban, however the patient continued to have a

severely dilated RV and reduced RV systolic function and an akinetic RV free wall. Repeat imaging of the chest showed no evidence of PE. Ultimately, the patient passed away from mixed septic/cardiogenic shock.

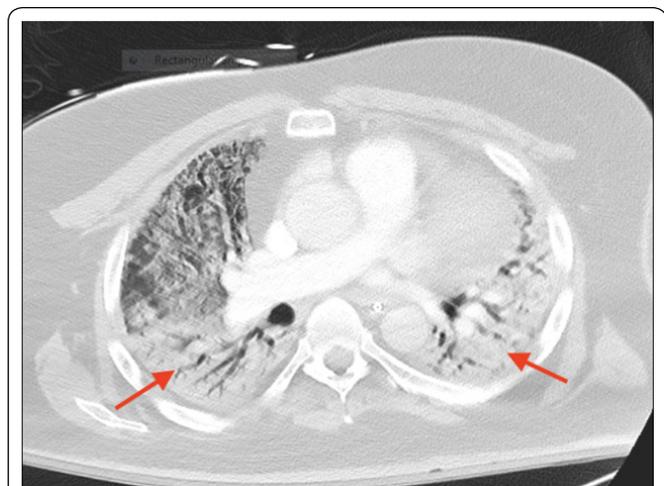


Figure 3: Chest CTA with diffuse, bilateral ground glass opacifications and consolidations (red arrows) predominantly in the posterior lung. There was no evidence of pulmonary embolism.

Discussion

Coagulopathy is a frequent finding in patients diagnosed with SARS-CoV-2, with multiple reports describing both arterial and venous thromboembolism, the latter with a frequency between 25 - 49% [6, 7]. RA/RV thrombus is an infrequent condition, which characteristically has been seen in association with pulmonary embolism, at a rate of about 4% [8]. Thrombi in the right heart are associated with considerably increased mortality, with some estimations suggesting a mortality of 27-45% despite treatment and nearing 100% if untreated [8]. According to a 1989 study done by the European Working Group on Echocardiography, RHT can be organized into three morphological types, Types A, B and C. Type A thrombi are serpiginous, elongated and highly mobile clots, commonly seen in association with DVT and pulmonary embolism. Type B thrombi are mostly non-mobile, ovoid in appearance and assumed to develop in situ in association with underlying cardiac anomalies. Type C thrombi, the rarest of the three, are highly mobile and structurally resemble cardiac myxomas [9]. Our patient likely had a Type A RV thrombus. RHT seldomly occur and their true incidence remains unreported, especially in SARS-CoV-2 patients.

Imaging diagnosis and following progression of RV thrombosis is normally achieved using either TTE or TEE. However, suboptimal echocardiographic images can occur in 15% of patients, for which the American Society of Echocardiography recommends contrast images, which have been shown to improve diagnostic accuracy of intracardiac thrombi [10, 11]. In a study of 100 SARS-CoV-2 positive patients who underwent echocardiography, no patients were found to have RV thrombosis, further suggesting its low frequency. However, 20% of patients had significant RV dysfunction and failure on sequential echocardiograms [1]. In

42% of these patients, DVT was found, but due to increased risk for disease transmissibility during CTA scanning, diagnostic evaluation was not performed to assess for PE [1]. Typically, RV dysfunction is characterized by RV/LV end diastolic diameter > 1 in the apical four chamber view, RV end diastolic diameter ratio > 30 mm and paradoxical RV septal motion [12]. A distinct echocardiographic pattern of RV dysfunction, described as akinesia of the RV free wall with normal motion at apex, has been shown to occur in acute PE and has been termed “McConnell’s sign” [13]. Our patient was found to have an RV end diastolic diameter > 30 mm as well as the finding of reduced RV systolic function with akinetic RV free wall (possibly suggestive of McConnell’s sign), in the absence of PE. Identification of RV dysfunction is critical, as studies have shown patients with both RHT and RV dysfunction have an increased mortality compared to those with RV dysfunction without RHT [14].

Presently, no guidelines exist on how to manage patients with RHT. Similarly, no expert recommendations are universally accepted regarding how to treat coagulopathy in SARS-CoV-2. Currently, management of coagulopathy in SARS-CoV-2 is centered around the D-dimer value and presence or absence of thrombosis, which guides selection of prophylactic or therapeutic anticoagulation. Treatment of right heart thrombi has typically followed practice guidelines for DVT or PE, with intravenous or oral anticoagulation and possibly thrombolysis or thrombectomy in certain cases. In patients with RHT associated with acute PE, one report has determined no difference in mortality and major bleeding outcomes between patients receiving anticoagulation alone or combined anticoagulation plus systemic thrombolysis [15]. However, it remains unknown the best way to manage isolated RHT in the absence of PE. Comparatively, studies have been done on anticoagulation in LV thrombus, with direct oral anticoagulants (DOACs) demonstrating promising results compared to vitamin-K antagonists [16, 17]. It would seem that treatments for LV thrombus would be similarly efficacious in RV thrombus, however, no evidence exists for this claim. Regardless, further research is needed to further characterize the adverse effects and complications of DOACs in RV thrombus management. As data on the efficacy of anticoagulation in SARS-CoV-2 emerges, the management of coagulopathic complications such as RHT will be better understood and more effectively treated.

Conclusion

This is one of the first reports of RHT, a cardiac complication, in a patient diagnosed with SARS-CoV-2. It would appear logical that RHT in patients with SARS-CoV-2 is associated with PE, however as this case shows, that may not always be the case. Nevertheless, resultant RV dysfunction and ultimate RV failure from RHT can lead to worsening cardiorespiratory hemodynamics, as seen in our patient. Early use of echocardiography in patients with cardiac dysfunction to identify RHT is vital, as it allows for immediate anticoagulant treatment. Although there are no consensus guidelines for

treatment, appropriate anticoagulation is necessary, as RHT with or without PE carries a high morbidity and mortality.

Conflict of Interest

The authors declare no conflict of interest.

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