Case Report

Floating thrombus in the arch of the aorta: A diagnostic dilemma in coronavirus disease 2019

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ABSTRACT

Coronavirus disease 2019 (COVID-19) primarily involves the respiratory system, however, hypercoagulability and thromboembolism are its unique and common association. COVID-19 may predispose patients to an increased risk of thrombotic complications through various pathophysiologic mechanisms, such as inflammation, immobilization, endothelial dysfunction, and a hypercoagulable state. Therefore, a high index of clinical suspicion is needed for diagnosing thrombotic complications such as floating thrombus in the arch of the aorta as described in this case report and prompt anticoagulation should be started to prevent life-threatening complications. We describe a rare presentation of a patient who presented to intensive care unit of our hospital with respiratory symptoms and was being treated for COVID-19 and despite the management as per COVID protocol was difficult to wean off from the non-invasive ventilation. We will focus on how we arrived at this rare diagnosis "floating thrombus in the arch of aorta" and further management of the case.

Key words: Coronavirus Disease 2019, Floating thrombus, Thrombotic complications

Coronavirus disease 2019 (COVID-19) a viral respiratory illness caused by the severe acute respiratory syndrome coronavirus 2 characterized by acute respiratory distress syndrome with thromboembolism being one of its common associations. COVID-19 may predispose patients to an increased risk of thrombotic complications through various pathophysiologic mechanisms, such as inflammation, immobilization, endothelial dysfunction, and a hypercoagulable state [1]. Hence, thromboprophylaxis is recommended for all individuals with moderate-to-severe disease as hypercoagulability is associated with elevated d-dimer levels and fibrinogen levels. Furthermore, in COVID-19 patients, fibrin polymerization could lead to microcirculation thrombosis and even thrombus in the major vessel [2]. Even though antithrombotics are advised as prophylaxis in COVID-19, yet there may remain thrombi in a large vessel, therefore, a high index of suspicion for any thrombus formation is necessary despite antithrombotic management. We report a rare case of floating thrombus in the arch of the aorta of a patient who presented to our hospital with respiratory symptoms and was being managed for COVID-19.

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CASE REPORT

A 61-year-old male presented to the emergency of our hospital with chief complaints of cough, breathlessness, and fever for 3 days. At presentation, the patient was conscious and oriented, febrile with a temperature of 101°F, tachypneic with a respiratory rate of 28/min, and peripheral saturation (SpO_2) of 60% on room air. Arterial blood gas analysis (ABG) on room air showed oxygen saturation of 82%, pH of 7.3, pCO_2 of 32 mmHg, pO_2 of 48 mmHg, lactate 2 mmol/L, and HCO_3^- of 19 mmol/L. His saturation of oxygen in the blood improved to 95% by the use of a partial rebreathing mask.

Electrocardiography (ECG) showed changes suggestive of the right bundle branch block (RBBB). Chest X-ray (CXR) on admission showed patchy infiltrations in bilateral lung regions, involving the basal region predominantly.

He was thus admitted with a provisional diagnosis of bilateral pneumonitis with high suspicion of COVID-19 infection with RBBB shifted to the intensive care unit. The events occurring during the intensive care unit (ICU) stay are given in Table 1. There, he was put on non-invasive ventilation with positive pressure support with FiO2 of 60%. Systemic broad-spectrum antibiotics, dexamethasone, and enoxaparin were started as per

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Table 1: Summary of events during the ICU stay

Days	Mode of O_2	SpO_2 (%)	Respiratory rate	Deranged investigations	Events
1–10	NIV	99	18	TLC-21,500/mm^3 SGPT/SGOT-193/176 IU/l Elevated D dimer-1970 ng/ml	Patient shifted from high-flow mask to NIV. Antibiotics changed Tazopip 4.5 g IV TDS, Targocid 200 mg IV OD started Convalescent plasma given
11–15	NIV	96–97	20	Nil	Multiple failed attempts of weaning, high peak inspiratory pressures
16–21	Shifted from NIV to HFNC	94–98	22	CK-Nac and CK-MB values deranged	The patient complained of chest pain. Provisional diagnosis of acute coronary syndrome/pneumothorax was made Loading dose of aspirin given CXR showed right-sided pneumothorax – ICD inserted
21–30	Shifted from HFNC intermittently to non-breathing mask	92–94	24	Nil	CECT chest done –showed finding of the right-sided floating thrombus. CT Angiography chest – floating thrombus in the arch of aorta measuring approx. 3 cm×0.7 cm. Repeat RTPCR sample sent patient came out to be COVID-19 negative on the 24th day of ICU stay – ICD removed. Enoxaparin dose increased from 40 mg OD to 60 mg BD. Patient weaned from HFNC to non-rebreathing mask

ICU: Intensive care unit, NIV: Non-invasive ventilation, TLC: Total leukocyte count, SGPT: Serum glutamic pyruvic transaminase, SGOT: Serum glutamic-oxaloacetic transaminase, CK-MB: Creatine kinase myocardial band, CXR: Chest X-ray, CECT: Contrast-enhanced computed tomography, ICD: Intercostal drainage tube, HFNC: High-flow nasal cannula

institutional protocol. The reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was positive. His initial investigations showed elevated total leukocyte count (TLC) of 21,500/mm^3, prothrombin time of 18.2 s, INR of 1.7, D-dimers of 1970 ng/ml, and platelet count of 370 × 10^9/L which was normal. His alanine and aspartate transferase levels increased to 600 IU/L by the 6th day of admission. Because of deranged liver enzymes, remdesavir was avoided. His condition did not improve and the patient required non-invasive ventilation despite improvement in arterial oxygen pressures and oxygen saturation on ABG analysis.

On day 16 from admission, the patient complained of chest pain on the right side. The patient was evaluated with a CXR, ECG, and serum levels of cardiac enzymes. A provisional diagnosis of acute coronary syndrome/pneumothorax was made. His cardiac enzymes came out to be elevated. A loading dose of aspirin, clopidogrel 300 mg, and 5 mg morphine iv was given. Pneumothorax involving the right lung was seen on a CXR. The surgeon's opinion was sought in view of same, and intercostal drain was inserted and serial CXR showed expansion of involved segments, however, the patient showed marginal improvement. The patient was put on high-flow nasal cannula (HFNC) but remained tachypneic and required non-invasive ventilator support. Hence, a contrast-enhanced computed tomography (CECT) of the chest was performed to rule out any respiratory cause for tachypnea including pulmonary embolism as one of the differential diagnoses, it revealed a thrombus in the arch of the aorta (Fig. 1a).

An opinion was sought from the cardiothoracic vascular surgeon who suggested a CT angiogram to rule out aneurysm or dissection of the aorta. The CT angiography of the chest confirmed the CECT findings and the thrombus was found to be attached to the aortic wall. Since no aneurysm or dissection of the aorta was seen, medical management was initiated. The enoxaparin dose was increased from 40 mg to 60 mg b.d subcutaneous.

He progressively improved and was put on HFNC and then to non-rebreathing mask and Venturi mask. Subsequently, the patient improved and could maintain his oxygen saturation on room air after 60 days from the date of admission. The patient came out COVID-negative 24 days after the admission through RT-PCR done on nasopharyngeal/oropharyngeal swab. Further, the X-ray showed near-complete right-sided lung expansion. After 26 days, implantable cardioverter-defibrillator (ICD) was removed and the patient vitals remained stable. CXR was done which showed right consolidation but no pneumothorax (Fig. 2). During the stay, no neurological deficit or signs and symptoms of systemic embolization were observed.

Repeat CT angiography done after 2 months showed decreased size of floating thrombus (Fig. 1b). The patient was discharged after 86 days of hospital stay and followed telephonically and has been advised to follow-up with the cardiovascular surgeons for further management.

DISCUSSION

The incidence of thrombosis in major vessels is underestimated, even though major thrombotic events and myocardial infarction are also reported [3]. The more commonly reported events are in relation to deep vein thrombosis and pulmonary embolism [4]. However, the evidence about arterial thrombosis in patients with COVID-19 is limited [5].

Endothelial cell dysfunction induced by infection results in excess thrombin generation and fibrinolysis shutdown, which indicates a hypercoagulable state in a patient with infection [6]. Factors may favor the COVID-19-related thrombotic complications,

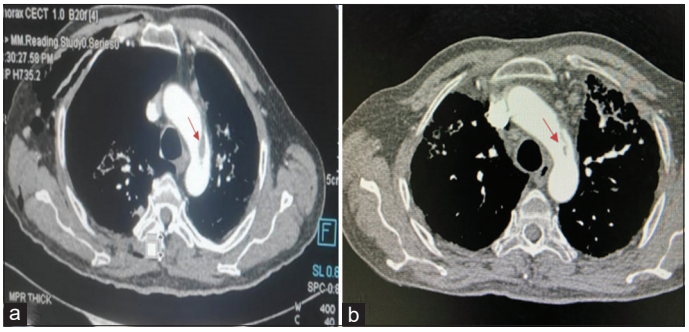

Figure 1: (a) CECT chest showing floating thrombus (arrow) in arch of aorta size 3 cm × 0.7 cm, massive right-sided pneumothorax with intercostal drainage tube *in situ*; (b) computed tomography angiography showing floating thrombus (arrow) in the arch of the aorta which was thinner, wavier as compared to the previous study. Fibrotic changes with bronchiectasis in bilateral lungs with resolving consolidation in the right basal segments sign of post-coronavirus disease fibrosis

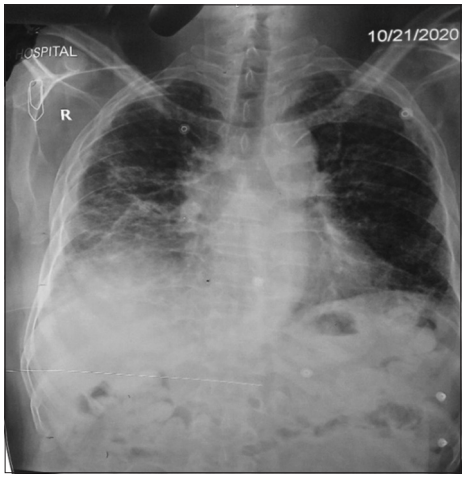

Figure 2: Chest X-ray showing ill-defined homogenous opacities noted in the right mid and lower zone, silhouetting of the right heart border and right consolidation; however, no pneumothorax was seen

the pathogenesis of hypercoagulability in this disease has yet to be fully elucidated. The endothelial dysfunction could be due to direct invasion of endothelial cells by the virus or mediated by the presence of cytokines (e.g., interleukin-6) and other acute-phase reactants [7]. These inflammatory markers were markedly elevated in this patient as well. Hypoxia-inducible transcription factors may directly activate platelets and coagulation factors, increasing tissue factor expression, and increasing plasminogen activator inhibitor-1 could also attribute to the hypercoagulability by inhibition of anticoagulant protein [8].

Although there is a risk of thrombosis, the thrombosis in the arch of the aorta is rare. There have been very few case reports wherein the association of floating thrombus in the arch of the aorta in association with COVID-19 has been described in the literature [5]. The mean time for the appearance of thrombus has been 12–17 days [9]. Possibly, the same time would have elapsed in this patient as the patient showed signs of clinical improvement but tachypnea persisted and the patient continued to require non-invasive ventilation.

A stepwise diagnostic approach helped us arrive at this rare diagnosis and thereafter by further increasing the dose of enoxaparin alongside antiplatelet medications, the patient was progressively weaned from the non-invasive ventilator to oxygen by Venturi mask, shifted to the ward on room air, and eventually discharged after 86 days of hospital stay. The investigation of choice for diagnosis is CT angiography to determine the site, morphology size, as well as characteristics of the aortic wall [10]. As the CT angiography showed a hypodense filling defect in the arch of the aorta tethered at one end and floating at the other, however, there was no evidence of aneurysm or dissection, so the cardiovascular thoracic surgeon suggested proceeding with the medical management in the form of anticoagulants.

Low-molecular-weight heparin (LMWH) is used for the management of COVID-19 as an anticoagulant therapy. LMWH improves the prognosis in COVID-19 [7] and has anti-inflammatory properties [11]. LMWH is the mainstay treatment for floating thrombus as well, in this patient also, who was already on enoxaparin the dose was increased to 60 mg subcutaneous per day, and on discharge, there was a decrease in size of thrombus and also a significant improvement in the clinical condition of the patient. It is advocated to give full-dose anticoagulant for the management and to continue anticoagulants for up to 3 months post-discharge for these patients [5]. The surgical management of the thrombus has been suggested by few authors [12]. Considering the potential hazard for emboli yet in all the case reports of the thrombus in COVID-19, medical management has been used. Soyer *et al*. have suggested that there could be recurrence despite surgical thrombectomy while the complete resolution has been reported following anticoagulant therapy [13,14].

CONCLUSION

In severe COVID-19 patients, a differential diagnosis of thrombosis in a major vessel should also be considered if the other causes of tachypnea have been ruled out. Moreover, therapeutic intervention with antithrombotic agents like heparin/LMHW should always be kept at the back of the mind for managing thrombotic, bleeding, and hemorrhagic event in COVID-19 patients. We still need more information and prime quality data to understand how COVID-19 and floating aortic thrombus interact. In conclusion, aortic thrombus may be a precedent/coincidental factor in patients with COVID-19.

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