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CASE REPORT

A Case of COVID-19 Induced Descending Aortic Thrombus and Splenic Infarctions

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Abstract

Thromboembolic manifestations like pulmonary embolism and deep venous thrombosis are often reported and contribute to a significant mortality from acute and chronic COVID-19 infections. These phenomena are a result of the activation of the coagulation cascade by the COVID-19 induced inflammatory state.

Majority of the thrombotic incidences are reported as a venous thrombosis but extremely rarely, arterial thrombi can be a manifestation of acute COVID-19 infection.

The patient in our case report was an unvaccinated 47-year-old female who presented with fever, nausea, abdominal pain and vomiting. The imaging confirmed the presence of a non-occlusive thrombus in the descending aorta, multiple splenic infarctions and paralytic ileus. She was treated with systemic anti-coagulation. A hyper-coagulable workup was performed on the patient and no other risk factors that could contribute to a thrombus was identified.

Keywords: Covid-19, Aortic thrombus, Splenic infarction, Ileus

1. Introduction

Among the many other typical and atypical presentations of the novel SARS-CoV-2 (COVID-19) infection, it frequently results in markedly dysregulated coagulation cascade, leading to heightened risk of thrombotic events and significant morbidity and mortality.[1–6] These thrombotic complications range from myocardial infarction, venous thromboembolic diseases to acute cerebrovascular attacks.[7] Although its prothrombotic pathophysiology is still not completely understood, the COVID-19 associated coagulopathy is thought to be a consequence of up-regulation of several inflammatory and immune-mediated pathways that are inter-connected and auto-amplifying.[7] The microvascular and macro-vascular thrombosis can effect the vasculature of many organs including lungs, heart, brain and spleen.[3] While pulmonary embolism and deep venous thrombosis account for a majority of thromboembolic manifestations of COVID-19, there have been reported cases of thrombosis in the aorta as well as other major vessels.[4]

This case report illustrates a case of a large acute thrombus in the descending aorta as a complication of COVID-19. The presentation was further complicated by formation of splenic infarctions, paralytic ileus and resultant bacteremia. The case report illustrates the challenges in it's diagnoses as a result of non-specific features, and provides a literature review of the thrombotic complications associated with COVID-19.

2. Case presentation

The patient was a 47-year-old obese caucasian female with an underlying history of untreated obstructive sleep apnea. She was a non-smoker and had no personal history of asthma, COPD, diabetes, hypertension, or cardiac disease.

She tested positive for Covid-19 in September, 2021. She was unvaccinated at that time. Her sick

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contact was her husband who also was positive for COVID-19 during that period. She described loss of sense of taste and smell, periodic nausea with episodes of vomiting, diarrhea, fatigue, fevers and chills. Later she developed a cough, associated with shortness of breath with minimal exertion. With the worsening of shortness of breath, she presented to the emergency room ten days after being positive for COVID-19.

She was febrile in the emergency room with a temperature of 101.1 F. Chest x-ray showed minor bi-basilar infiltrates [Fig. 1]. She was hypoxic with an oxygen saturation of 88% on room air. Rest of the blood work was unremarkable except for an elevation in the inflammatory markers. At the time of admission, her white cell count was 10,900/uL (reference range 4500 to 11,000/uL), CRP level was 76.1 mg/L (reference range 0–8 mg/L), interleukin-6 level 13.9 pg/ml (reference rage 0–7 pg/ml), D-dimer level 193 ng/ml (normal high <244 ng/ml), LDH level was normal at 187 units/L (reference range 140–280 units/L).

She was started on supplemental oxygen through nasal cannula and high-dose intravenous steroids. Intravenous remdesivir was not considered since it had already been ten days since she tested positive. After a 3-day uncomplicated stay in the hospital, the patient was weaned off of oxygen and was discharged home on a steroid taper. During the hospital course, she also endorsed some right-sided leg cramping which resolved spontaneously with as needed doses of acetaminophen. She received standard subcutaneous anticoagulation DVT prophylaxis with subcutaneous heparin.

Three days later, the patient presented again to the emergency department with intractable epigastric pain of one day duration. She stated that the pain was constant but got worse with any attempt to eat. She had severe nausea with the pain and few episodes of emesis that were non-bloody and non-bilious. She denied any diarrhea, sick contacts or food exposure that might have led to gastroenteritis. She denied any recurrence of her fevers, chills, cough, chest pain or shortness of breath.

She received a contrast enhanced CT scan of the abdomen and pelvis that showed [Fig. 2] non-occlusive filling defect in the descending thoracic aorta measuring 28 × 11 × 11 mm, likely representing a thrombus and mild fat stranding along the common origin of celiac trunk and SMA which is nonspecific, however may be seen with vasculitis. No other abnormality in the arterial and venous vasculature, or acute process in the abdomen and pelvis.

The CRP level was 16.4 mg/L (reference range 0–8 mg/L). The renal function panel, electrolyte panel and hepatic panel were completely within normal limits. She had a leukocytosis of 15,300 cells/mm3 which was predominantly neutrophilic, this was considered secondary to the use of systemic steroids that she was discharged with in the previous admission.

She was admitted, started on anticoagulation with low molecular weight heparin, 1 mg/kg body weight twice a day with an intention to transition to direct oral anticoagulation, once she was able to tolerate oral diet and medications. The etiology of the

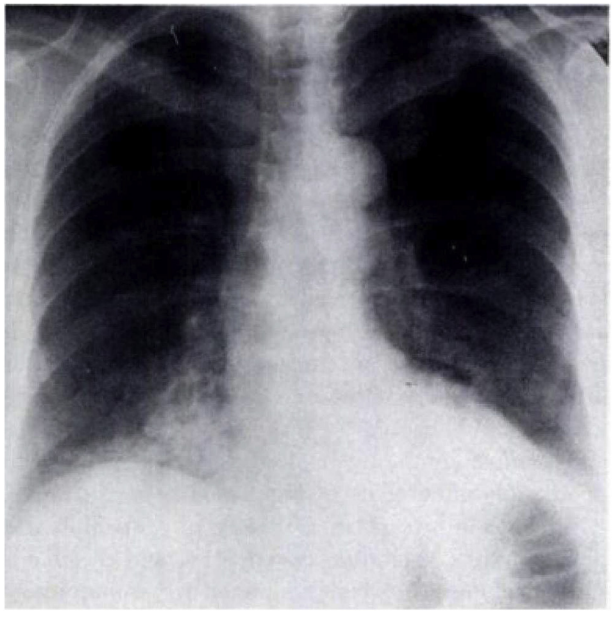

Fig. 1. Bibasilar infiltrates from COVID-19 infection.

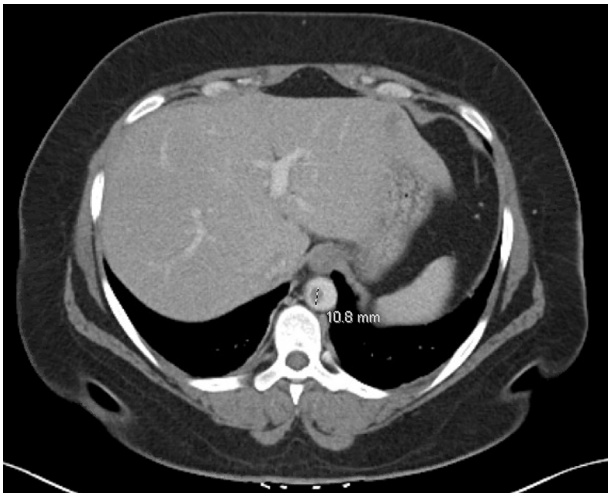

Fig. 2. Non-occlusive thrombus in the descending thoracic aorta (28 × 11 × 11 mm).

abdominal pain was quite unclear at the time of admission and multiple differential diagnosis were considered. COVID-19 induced abdominal pain and gastroenteritis was a potential diagnosis and the systemic steroids were continued. Symptomatic management was offered with pantoprazole and ondansetron intravenously. The other differential was occlusion of the superior mesenteric or celiac arteries by the thrombus, thus leading to post-prandial abdominal pain. A Doppler study of the mesenteric arteries was requested but the study was inconclusive because of the patient's obese body habitus. A bilateral venous Doppler excluded deep venous thrombosis in the lower extremities.

On day 2 of the hospitalization, the patient's abdominal pain worsened and she had multiple bouts of emesis. The emesis was bilious and non-bloody. She also developed a low-grade fever. Lactic acid level was elevated at 3.4 mmol/L. She had a repeat CT angiogram of the abdomen and the celiac artery, superior mesenteric artery and rest of the vasculature were unremarkable. However, there were several small to moderate sized splenic infarctions. The largest infarction was 2.4 cm [Fig. 3].

On day 3 and day 4 of the hospitalization, the lactic acid level normalized with intravenous hydration but there was no improvement in her nausea and vomiting. An obstruction series was done that showed gaseous distention of the small bowel loops, up to 4.6 cm in the mid abdomen. The findings were concerning for small bowel obstruction and were new compared to the CT scan from 2 days prior [Fig. 4]. A nasogastric tube was inserted to relieve the obstruction and was managed with complete bowel rest and intermittent suction. Patient was also noted to be febrile with a temperature

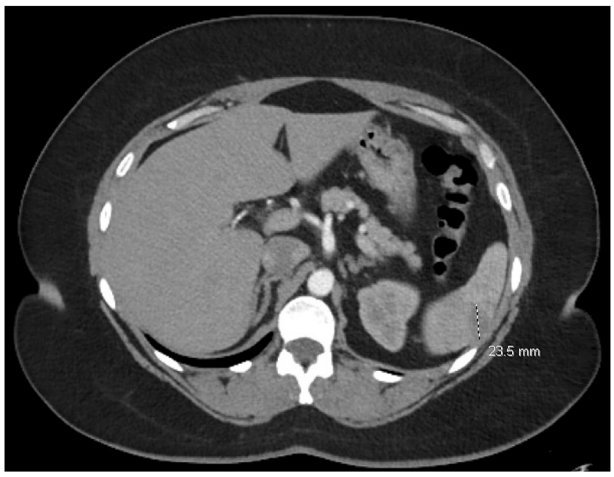

Fig. 3. 2.4 cm splenic infarction noted on day 2 hospitalization from the aortic thrombus.

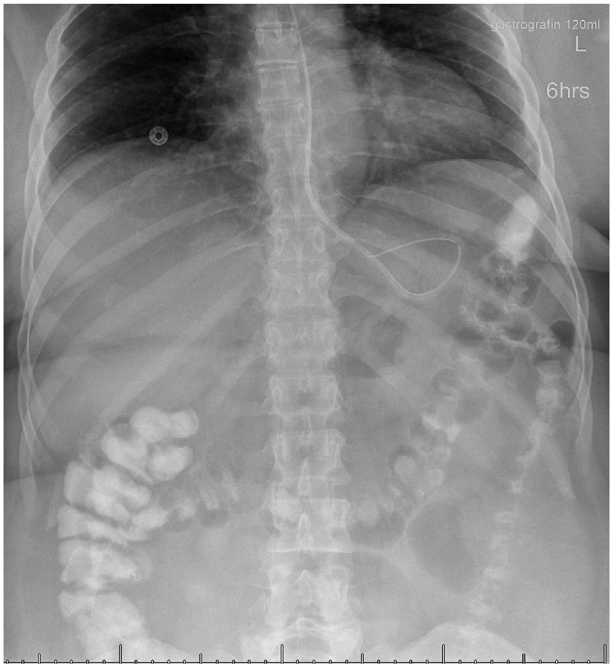

Fig. 4. Distal aortic thrombus leading to small bowel ileus (4.6 cm gaseous distention of the small bowels).

of 101.2 F. The leukocytosis also worsened to 20,600 cells/mm3. Blood cultures were obtained and she was empirically started on intravenous Piperacillin/Tazobactam.

The nausea, vomiting and abdominal pain gradually improved. A small bowel follow through obstruction series performed two days later ruled out a complete small bowel obstruction. Blood cultures from admission grew pan-sensitive *Klebsiella pneumoniae* which likely originated from the bowel. Antibiotic coverage was downgraded to intravenous Cefazolin while inpatient and discharged on a 2-week course of oral cefuroxime. She was also transitioned to Apixaban on discharge. A complete hyper-coagulable work up could not be attempted because of the active thrombus but genetic mutations like prothrombin gene mutation, factor 5 Leiden mutation, anti-cardiolipin antibodies and lupus anti-coagulant were negative. Follow up CT scan of chest, abdomen and pelvis two weeks after discharge revealed decreased size of the thrombus in the descending aorta [Fig. 5].

3. Discussion

Isolated descending thoracic aortic thrombus, to our knowledge, is an uncommon finding associated with COVID-19 infection. Although its exact mechanism is unclear, the SARS-CoV-2 infection creates pro-thrombotic environment by altering vessel barrier integrity, promoting pro-coagulative state,

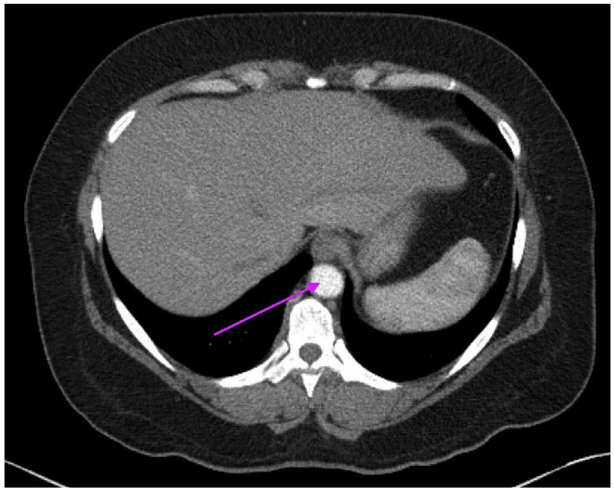

Fig. 5. Resolution of the descending aortic thrombus after 2 weeks of anticoagulation.

inducing vascular inflammation (endotheliitis) and mediating inflammatory cell infiltration.[8]

SARS-Cov-2 infection can directly infect endothelial cells, leading to inflammation, apoptosis and dysfunction.[9] The endothelial cell invasion instigates the recruitment of granulocytes and macrophages, synthesizing pro-inflammatory cytokines. As the inflammatory process progresses, like in seriously ill patients, the end result is COVID-19 associated coagulopathy that manifests as arterial and venous thrombosis. This is a downstream consequence of the host's inflammatory response to the viral insult and activation of innate immunity. The thrombus formation is an end-product of a complex interplay of numerous pathways that involve cytokine storm, macrophage activation, complement activation, platelet activation, and renin-angiotensin system dysregulation.[8] The COVID-19 illness is associated with profound inflammatory cytokine response, that is manifested by an increased expression of IL-1, IL-6, and tumor necrosis factor (TNF)-α. While IL-1 and TNF-α play an important part in the suppression of endogenous anticoagulant pathways, IL-6 triggers the coagulation cascade by inducing tissue factor expression on endothelial cells.[9] This coagulopathy is postulated to be the primary etiology of acute respiratory distress syndrome in COVID-19 patients, as it triggers the formation of fibrin-platelet microthrombi in the pulmonary microcirculation and parenchyma.[10] The findings of increased angiogenesis and micro-angiopathic thrombosis in the pulmonary vasculature of COVID-19 patients who succumbed from ARDS give more credibility to the hypothesis.[11]

The presence of significant thrombus burden in the aorta in the absence of vessel wall damage or massive atherosclerosis is unexpected in viral infections. However, SARS-CoV-2 infection might be an exemption. Studies on SARS-CoV-2 have shown a high specificity for virus binding to the angiotensin-converting enzyme-2 (ACE-2) receptor as a mechanism to invade host cells. The ACE-2 receptors are expressed widely in the human body, including on the vascular endothelium.[12] Hence, it is possible that the aortic thromboembolism in these patients may be caused by ACE-2 mediated SARS-CoV-2 infection of the large vessel wall, resulting in local injury and intravascular activation of the clotting cascade.

Although the exact incidence of aortic thrombotic events in COVID-19 infections is unknown, there is a high likelihood that it would be under-reported and underestimated given the non-specific presentation, and their incidental revelation on imaging studies that are primarily performed for some other reason. The existence of venous and/or arterial thromboembolic events, in general, has been under-recognized in patients suffering from a severe course COVID-19 infection and D-dimer level measurement at admission can be utilized as an early tool to facilitate timely recognition.[13,14] The mainstay of therapy is treatment with heparin for reducing thrombus load in the aorta and for prevention of new thrombi formation. Other treatment options can include thrombolysis, interventional, or surgical thrombectomy.[14]

There have been some reports of thrombotic and more commonly thrombocytopenic complications from the different types of COVID-19 vaccinations.[15–18] However, there is no evidence to report that the incidence of thrombotic complications further increases after the vaccination once the individuals have a thrombotic incident from the acute COVID-19 infection. The patient in our case was encouraged to get the vaccine after recovery from the acute phase.

In light of the increasing incidence of cases, from the COVID-19 variants, it is even more important to encourage everyone to take any of the available vaccines against the virus.[19,20]

4. Conclusion

In this case report, we report a rare case of descending aortic thrombus in the setting of COVID-19 infection. COVID-19 induced arterial or venous coagulopathy is a consequence of the inflammatory pathways amplifying the coagulation cascade. It is a known fact that pulmonary embolism and deep venous thrombosis are quite common and account for a significant mortality from COVID-19.

However, aortic and other major vessel thrombotic complications have been reported rarely. This life-threatening complication reinforces the importance of vaccinations and social distancing in the current pandemic era.

Conflicts of interest

No conflict of interest.

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