



Case report

Coexisting portal vein thrombosis and aortic thrombosis in a patient with COVID-19: A case report and literature review



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ABSTRACT

The Coronavirus Disease 2019 (COVID-19) pandemic has rapidly progressed, resulting in significant global morbidity and mortality. Predominantly affecting the respiratory tract, it has been found to be associated with extrapulmonary manifestations such as coagulopathies. We hereby report a case of an elderly man with no predisposing risk factors or history of hypercoagulable disorder who presented with acute onset abdominal pain and was diagnosed with portal vein thrombosis and splenic infarct two weeks following mild COVID-19. Incidentally, the patient was also noted to have aortic thrombosis. The patient was treated with therapeutic anticoagulation with complete resolution in his symptoms. Our case highlights a high risk of coagulopathy following infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Introduction

Since the index case in 2019, COVID-19 has rapidly spread worldwide, becoming a global pandemic. Although pulmonary involvement is more common with COVID-19 infection, many extrapulmonary manifestations have been reported, including an increased risk of arterial and venous thrombosis. Most cases of vascular thrombosis happen among patients with severe COVID-19 infection admitted in the intensive care unit (ICU), but cases of thrombotic events have also been reported with mild cases of COVID-19. The pathogenesis of hypercoagulability in patients with COVID-19 is still not well established. Virchow's triad could be contributing to venous thrombosis which includes endothelial injury, stasis, and hypercoagulable state. On contrary, the arterial thrombosis in COVID-19 happen over atherosclerotic plaques in the setting of endothelial injury [1]. Several mechanisms have been proposed that could be contributing to endothelial injury in COVID-19 and includes dysregulated renin-angiotensin system, oxidative stress, and dysregulated innate immune response [1]. The direct invasion of endothelial cells by the SARS-CoV-2 virus, associated systemic cytokine release, and

complement activation leads to endothelial injury. Immobilization related stasis and hypercoagulable state due to increased circulating thrombotic factors like factor VIII, fibrinogen, circulating prothrombotic microparticles, and neutrophil extracellular traps also contribute to the hypercoagulable state [2,3]. These processes ultimately cause platelet activation, fibrin deposition, and thrombin production, leading to micro- and macro-thrombosis. Although venous thromboembolism (VTE) is more commonly seen in patients with COVID-19, arterial thrombotic events are also reported. Cerebral and coronary arterial thrombosis are more commonly reported arterial thrombotic events, while lower extremity deep venous thrombosis (DVT) and pulmonary embolism (PE) are the common VTE [4–6]. The occurrence of concurrent venous and arterial thrombosis can be associated with increased morbidity and is primarily seen with severe COVID-19. Rare arterial thrombotic events like aortic thrombus formation usually occur in patients with a history of vasculitis, coagulopathy, or aortic diseases, and atypical VTE like acute portal vein thrombosis (PVT) is usually associated with coagulopathy, myeloproliferative neoplasms or underlying liver or pancreatic disease [7]. We present a case of an elderly male who

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presented with abdominal pain secondary to PVT and was found to have incidental aortic thrombosis in the absence of any other predisposing risk factors following mild COVID-19.

Case presentation

A 71-year-old-White man with a past medical history of hypertension, type II diabetes mellitus, chronic obstructive pulmonary disease (COPD) presented with a chief complaint of abdominal pain for one-day in the month of December 2020. His pain started abruptly after waking up in the morning, located in the right upper quadrant, radiating to the umbilical area. He denied fevers, chills, or dyspnea. Seventeen days before his presentation, he developed anosmia, ageusia, and mild shortness of breath and was diagnosed with COVID-19 infection. He had a mild course of COVID-19, and did not require medications or supplemental oxygen. He did not receive monoclonal antibody therapy. The patient did not get vaccinated against COVID-19.

He did not have a history of previous thrombotic events or coronary artery disease. Home medications included albuterol and tiotropium inhalers, metformin, atorvastatin, and hydrochlorothiazide, and did not have any recent change in medications. On presentation, vital signs were normal: temperature 99.0°F, respiratory rate 17 per minute, blood pressure 136/83 mmHg, pulse rate 93 beats per minute, and oxygen saturation 97% on room air. Body mass index was 30 kg/m². Abdominal examination revealed a soft abdomen with tenderness to palpation in the right upper quadrant, epigastric region, and periumbilical region without guarding, rigidity, or rebound tenderness. He had normal bowel sounds on auscultation.

Laboratory work revealed elevated levels of white blood cells (WBCs), D-dimer, and lactate dehydrogenase (LDH). Although glucose was mildly elevated, bicarbonate was normal, and there was no anion gap. The rest of the laboratory examination was unremarkable (Table 1). Contrast-enhanced CT abdomen and pelvis revealed partial PVT (Fig. 1 A) with thrombus within branches of the right portal vein and splenic vein with a wedge-shaped area of hypoattenuation in the spleen, consistent with splenic infarct (Fig. 1 B and C). A CT scan of chest with pulmonary angiogram protocol was negative for pulmonary embolus but showed multiple areas of irregular mural thrombus in the aorta, including at the arch (Fig. 2 A) and in the descending aorta (Fig. 2 B and C). Compared to a CT abdomen and pelvis two weeks prior, these were new findings. The patient was admitted to the regular medical unit. Hereditary thrombophilia workup was not done given the patient's age,

Table 1
Laboratory workup on admission.

Laboratory test	Results	Reference range
White blood cells (WBCs)	11.9 H	3.0–11.0 K/mm ³
Hemoglobin (Hb)	14.3	13.0–18.0 g/dL
Hematocrit (HCT)	44.1	39.0–52.0%
Platelets	446	120–450 K/mm ³
Troponin I	0.01	0.00–0.05 ng/mL
Prothrombin time (PT)	14.1	11.4–14.2 s
Activated partial thromboplastin time (APTT)	25.4	24.0–35.6 s
International normalized ratio (INR)	1.05	0.86–1.14
D-dimer	0.93 H	0–0.52 ug/mL FEU
Blood urea nitrogen (BUN)	24	6–20 mg/dL
Creatinine	1.05	0.57–1.10 mg/dL
Estimated glomerular filtration rate (EGFR)	69.6	> 59 mL/min
Sodium	135	133–144 mmol/L
Potassium	3.7	3.2–5.0 mmol/L
Chloride	102	96–106 mmol/L
Calcium	7.9 L	8.6–10.3 mg/dL
Bicarbonate	22	22–32 mmol/L
Glucose	231 H	65–99 mg/dL
Aspartate aminotransferase (AST)	29	15–41 U/L
Alanine aminotransferase (ALT)	42	17–63 U/L
Alkaline phosphatase (ALP)	86	38–126 U/L
Lipase	53 H	22–51 U/L
Lactate dehydrogenase (LDH)	294 H	98–192 U/L

and the absence of prior thromboembolic event and review of laboratory workup did not suggest the need for myeloproliferative neoplasm testing. After consulting with vascular surgery, medical management was chosen, and the patient was started on intravenous anticoagulation with unfractionated heparin drip. He had complete resolution of abdominal pain a day after initiation of anticoagulation. On day 3, the patient was discharged home after anticoagulation was transitioned to oral apixaban, with plans to repeat abdominal imaging as an outpatient in 3 months.

Discussion

COVID-19 is associated with acute inflammatory changes and a hypercoagulable state, increasing the risk of thrombosis. The highest risk for thromboembolism is among patients with severe COVID-19, receiving treatment in the intensive care unit, and often occur despite prophylactic-dose anticoagulation [4]. High incidence of venous thromboembolism (up to 25%) and arterial vascular events (up to 4%) have been reported with COVID-19 [8]. Myocardial infarction, acute limb ischemia, mesenteric artery thrombosis, and pulmonary embolism are commonly reported thrombotic events with COVID-19 [9]. Typical findings associated with the thromboembolic phenomenon in COVID-19 include elevated levels of D-dimer that correlate with covid illness severity. An increase in von Willebrand factor (VWF) antigen and activity and factor VIII activity and fibrinogen levels are also commonly observed [9]. In patients with COVID-19 with no clinical improvement or rapid clinical deterioration and/or persistently high D-dimer levels, imaging with Doppler ultrasound or CT angiogram may help diagnose systemic thrombosis. We presented a case of concurrent PVT and aortic mural thrombosis in a patient with mild SARS-CoV-2 infection. Although COVID-19 associated PVT has been well described and reviewed in the literature, the data on aortic thrombosis is limited. Hence, we have done a systematic review of cases with aortic thrombosis related to COVID-19.

Portal vein thrombosis (PVT) with COVID-19

PVT is a thromboembolic disease that mainly occurs in the setting of cirrhosis, myeloproliferative disorders, abdominal infection, inherited thrombophilia, hepatocellular or pancreatic cancers. However, PVT is considered a relatively rare thrombotic complication in COVID-19, with around 30 cases reported from our literature review [10]. The prevalence of PVT following COVID-19 was more remarkable in males and the most common clinical presentation was abdominal pain [11]. The patients belonged to a wide age range anywhere between 27 and 79 years, with the median age around 43 years. Most cases develop within two weeks from COVID-19 onset. However, a fatal case of extensive gastrointestinal necrosis due to portal and mesenteric vein thrombosis approximately six weeks after the onset of critical COVID-19 has also been reported [12]. As evident from our report and some previously published case reports, it is crucial to note that such thrombotic complications are reported even in asymptomatic/mild cases of COVID-19 illness [13]. In our current healthcare climate, the finding of PVT should prompt an investigation for COVID-19 in addition to work-up for hypercoagulability [13]. Clinicians should strongly consider abdominal imaging in patients presenting with abdominal pain and or new-onset ascites in the setting of recent COVID-19 to rule out portal vein thrombosis.

In patients with acute PVT, liver function tests are typically normal because hepatic arterial blood flow compensates for decreased portal inflow, although a transient, moderate increase in serum aminotransferases is seen in some patients. In rare cases, it can lead to acute liver failure. Most common laboratory findings include elevated D-dimer, C-reactive protein (CRP), and fibrinogen levels. Most of these patients treated with full-dose anticoagulation had resolution of symptoms, and in a few cases, repeat imaging showed complete or partial resolution of

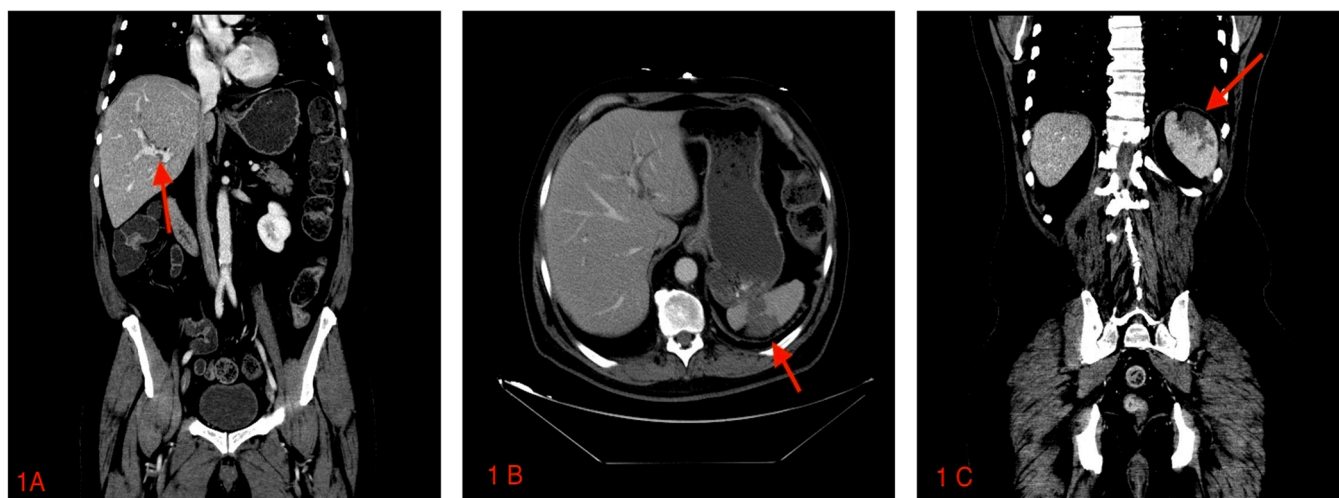


Fig. 1. Contrast-enhanced CT abdomen and pelvis showing portal vein thrombus on coronal view (Fig. 1 A) and splenic infarct on axial view (Fig. 1 B) and coronal view (Fig. 1 C), respectively.

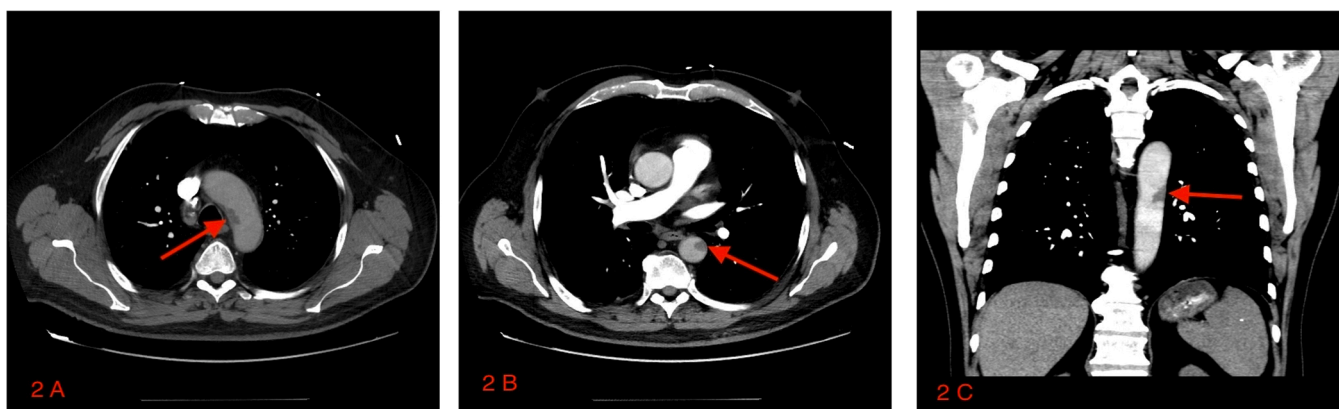


Fig. 2. Contrast-enhanced CT chest showing aortic mural thrombus on axial view (Fig. 2 A) and thrombus in descending thoracic aorta on axial (Fig. 2 B) and coronal view (Fig. 2 C), respectively.

the thrombus as well [14,15]. In one patient, follow-up abdominal ultrasound showed cavernous transformation [16]. A single case of gastric ischemia with gastric pneumatosis in the setting of partial PVT is reported in COVID-19 infection [14]. Early diagnosis and treatment of PVT can prevent complications, such as portal hypertension and intestinal infarctions.

The goal of treating acute PVT is to achieve the patency of the vein, thus preventing bowel infarction, liver injury, and late complications of portal hypertension. The timing for starting an anticoagulant therapy is crucial to avoid potentially life-threatening gastrointestinal bleeding. Evidence on which anticoagulant therapy should be used in patients with PVT is limited, and choice is based mainly on clinical experience. Commonly intravenous Unfractionated heparin and subcutaneous enoxaparin are being used. Potential malabsorption in case of intestinal ischemia should always be considered a potential risk of lack of efficacy for oral therapy [17]. When anticoagulation in acute PVT is initiated early and continued for six months, recanalization can occur in more than 60% in the first week but in less than 20% if started after that [18].

Systematic review of aortic thrombosis with COVID-19

Similarly, it is important to diagnose and treat aortic thrombus promptly to prevent embolization which could be fatal. This may require urgent medical, endovascular, or surgical treatment. However, the optimal treatment modality for free floating thrombus, occurring in the

setting of COVID-19 remains unknown. On conducting a PubMed database search using keywords (COVID-19) AND ("Aortic thrombus") on January 31, 2022, we found 17 publications of which 15 articles were pertinent (Table 2). The median age of the patients was 58 years (range 50–78 year) with majority of the patients being male (15 male and 6 females). Most patients presented with regular COVID-19 symptoms including fever, cough, dyspnea while some patients were asymptomatic, and some progressed to respiratory failure requiring oxygen supplementation. Majority of the patients (12 out of 21) had severe COVID-19 and required mechanical ventilation while 4 had mild and 6 had moderate COVID-19. D-dimer was elevated in all the cases. Besides D-dimer, other prominent laboratory abnormalities found in these patients included elevated levels of CRP, ferritin, LDH, and fibrinogen however these were not consistently reported. Median time to diagnosis of aortic thrombosis in relation to diagnosis of COVID-19 was 9 days (range 0–28 days). Aortic thrombi were mostly located in aortic arch (10 out of 21) and descending aorta (10 out of 21) followed by ascending aorta (5 out of 21) and abdominal aorta (2 out of 21).

In few cases, a sudden decline in clinical course coupled with significant elevation in D-dimer levels led to additional imaging which revealed aortic thrombus. Therefore, we recommend considering CT angiogram in patients with persistently elevated or acutely elevated D-dimer, to rule out aortic thrombosis [19]. Aortic thrombus may require urgent medical, endovascular, or surgical treatment with a multi-disciplinary approach. Most of the patients were treated medically

Table 2
Literature review on cases of Aortic thrombosis among patients with COVID-19.

S. No.	Authors, Year of Publication	Age/Gender/Race	PMH	Presentation	COVID-19 Severity	D-Dimer	Other labs	Time to thrombus	Location of thrombus	Treatment	Outcome	Miscellaneous		
1	Gandotra et. al.2020	53/F/ND	None	F, SOB, Cough	Severe	Elevated at 8180 ng/mL (<230 ng/mL)	Elevated ferritin and CRP	10 days	Aortic Arch	UFH, alteplase and argatroban	Partial resolution of thrombus on follow-up CT	–		
2	Siddiq et. al.2021	62/M/ND	COPD	SOB	Moderate	Elevated at 4800 ng/mL (<500 ng/mL)	None	0 days	Aortic Arch	Therapeutic anticoagulation followed by discharge on apixaban	Medically managed	CT scan four months later showed complete resolution of aortic thrombus	–	Complicated by right tibial artery occlusion requiring PCL.
3	Kashi et. al 2021	58/F/ND	HTN, T2DM	Incidental diagnosis	Moderate	Elevated at 1200 ng/mL (<500 ng/mL)	Elevated fibrinogen, thrombocytosis	0 days	Descending Aorta	Medically managed	ND	ND	–	–
4	Kashi et. al 2021	69/M/ND	Stroke, HTN, Thrombocytopenia	Incidental diagnosis	Severe	Elevated at 3700 ng/mL (<500 ng/mL)	Elevated fibrinogen	14 days	Arch and Descending Aorta	Medically managed	ND	ND	–	Developed thrombus despite ASA and prophylactic anticoagulation
5	Dagar et.al. 2021	61/M/ND	ND	Chest pain, respiratory symptoms	Moderate	Elevated at 1970 ng/mL (<500 ng/mL)	ND	21 days	Aortic Arch	Enoxaparin followed by warfarin	CTA at 4 weeks showed improved size of thrombus	–	–	
6	Dao et.al. 2021	61/M/ND	HTN	SOB and cough	Severe	Elevated at 6840 ng/mL (<500 ng/mL)	Elevated CRP, LDH, ferritin	9 days	Aortic Arch and Descending Aorta	Aortic mechanical thrombectomy, UFH followed by apixaban	Improved oxygenation following mechanical thrombectomy	–	–	
7	Masana et.al 2021	67/F/ND	T2DM, CKD, HLP, anemia	SOB, cough	Severe	Elevated at 2136 ng/mL (<500 ng/mL)	Elevated CRP	0 days	Descending Aorta	Enoxaparin	Discharged on AC after 20 days. Repeat CT at one month showed complete resolution of the thrombus	–	B/L PE and right ventricular clot	
8	Mukherjee et. al. 2020	71/M/ND	None	F, cough, SOB	Moderate	Elevated at 1113 ng/mL (<211 ng/mL)	Elevated CRP, ferritin, LDH	9 days	Ascending Aorta	Enoxaparin followed by apixaban	Discharged on Day 14	–	Left superior renal artery thrombus	
9	de Carranza et. al. 2020 (Case 1)	78/M/ND	HLP, bladder cancer	F	Severe	Elevated at 3570 ug/L (<211 ug/L)	Elevated CRP, ferritin, LDH	9 days	Aortic Arch, Descending Aorta	Enoxaparin	Died on 18th day	–	PE	
10	de Carranza et. al. 2020 (Case 2)	76/M/ND	HTN, HLP, T2DM, BPH	F, hypoxia, tachycardia	Severe	Elevated at 1340 ug/L (211 ug/L)	Elevated CRP, ferritin, LDH	26 days	Ascending Aorta	Enoxaparin	Stroke resulting in hemiplegia and global aphasia. Discharged to long term care	–	Left middle cerebral artery stroke	
11	de Carranza et. al. 2020 (Case 3)	64/M/ND	HTN, COPD, Hep B	F, cough, SOB	Severe	Elevated at 4640 ug/L (211 ug/L)	Elevated CRP, fibrinogen, LDH	11 days	Descending Aorta	Enoxaparin	Resolution of thrombus on repeat CT 17 days later	–	–	
12	Mullan et.al 2020	62/M/ND	HLP	hypoxia and diarrhea	Severe	Elevated at 14.87 mg/L (<0.57 mg/L)	Elevated fibrinogen	3 days	Ascending Aorta and Descending Aorta	UFH	ND	–	Large right parietal stroke	
13					Severe			9 days		UFH		–	B/L renal infarcts	

(continued on next page)

Table 2 (continued)

S. No.	Authors, Year of Publication	Age/Gender/Race	PMH	Presentation	COVID-19 Severity	D-Dimer	Other labs	Time to thrombus	Location of thrombus	Treatment	Outcome	Miscellaneous
	Mullan et.al 2020	57/M/ND	HTN, HLP, T2DM, TIA	F, myalgia, Abdominal pain		Elevated at 29.97 mg/L	Elevated lactic acid		Descending Aorta		Resolution of abdominal pain and return of distal pulses	
14	Cora et. al. 2021	74/F/ND	ND	Fatigue, cough, abdominal and bilateral leg pain	Severe	ND	ND	0 days	Abdominal Aorta	–	Cardiac arrest and death	–
15	Cora et. al. 2021	53/M/ND	T2DM	F and SOB	Severe	ND	ND	Unclear	Aortic Arch	Enoxaparin, Embolectomy and pentoxifylline	Septic shock and death	–
16	Schmidt et. al. 2021	64/M/ND	HTN, smoking, Recent PE	AMS, LUE weakness, RUE pain and loss of pulse	Mild	ND	ND	0 days	Ascending Aorta	UFH, Open thrombectomy of the AA, and Right MCA endovascular thrombectomy	Resolution of LUE weakness with return of pulse, Discharged home on apixaban	Right renal infarct, Developed thrombus on bridging therapy (enoxaparin/warfarin)
17	Buikema et. al. 2021	72/M/W	None	Hypoxia, shock	Severe	ND	Elevated CRP	21 days later	Aortic Arch and Descending Aorta	UFH followed Acenocoumarol	Repeat CT one week later showed improvement in thrombus.	Needed digital amputation for blue toe syndrome
18	Al-Mashdali et. al. 2021	66/F/ND	HTN, T2DM	F, cough, slurred speech, right sided weakness	Mild	Elevated at 0.68 mg/L (<0.46 mg/L)	Elevated CRP and LDH	0 days	Aortic Arch	Enoxaparin followed by Rivaroxaban	Repeat CT on Day 10 showed resolution of aortic thrombus	Left middle cerebral artery stroke
19	Spreadbury et. al. 2021	50/M/ND	HLP, CAD	BLE pain, pallor, coldness, and reduced sensation	Mild	Elevated at 14.8 mg/L (< 0.57 mg/L)	Elevated fibrinogen	28 days	Aortic Arch	Dalteparin followed by warfarin	Improved perfusion in BLE. Discharged on Day 9.	B/L popliteal arteries thromboembolectomy
20	Udongwo et. al. 2021	63/F/ND	Breast cancer S/P mastectomy, COPD	Severe right foot pain, chest tightness and SOB	Moderate	Elevated at 5559 ng/mL (<500 ng/mL)	Elevated CRP and LDH	11 days	Ascending Aorta and Abdominal Aorta extending to right common iliac artery	HMWH followed by warfarin with goal INR 2.5–3.5	Resolution of symptoms by Day 9. Repeat CT 5 months later showed complete resolution of aortic thrombus	Developed aortic thrombus while on rivaroxaban
21	Akella et. al. 2022	71/M/W	HTN, T2DM, COPD	Abd pain	Mild	Elevated at 0.93 (0–0.52 ug/mL)	Elevated LDH	17 days	Aortic Arch and Descending Aorta	UFH followed by apixaban	ND	Associated with PVT

Abbreviations: CRP = C-reactive protein, F= fever, AMS = altered mental status, T2DM = type 2 diabetes mellitus, HLP = hyperlipidemia, HTN=hypertension, TIA=transient ischemic attack, COPD=chronic obstructive pulmonary disease, UFH=unfractionated heparin, HMWH = high molecular weight heparin, SOB=shortness of breath, PE= pulmonary embolus, B/L= bilateral, CTA= Computed tomography angiography, LUE=left upper extremity, MCA=middle cerebral artery, RUE=right upper extremity, LDH=lactate dehydrogenase, RBA=right brachial artery, ND = not described, PVT=portal vein thrombosis

using either therapeutic dose of enoxaparin (8 out of 21) or unfractionated heparin (UFH) (8 out of 21), with a resolution of thrombus on repeat imaging, suggesting medical therapy alone to be optimal. However, two patients underwent urgent mechanical thrombectomy in addition to full dose anticoagulation, with significant clinical improvement and favorable outcomes, suggesting need for individualized care based on severity and symptoms [20,21]. It is not clear from the current review which patients will benefit from each treatment modality. Complication following medical/surgical therapy such as delayed embolization after a month was seen in one patient despite being on oral anticoagulation [22]. Data on anticoagulation upon discharge, outcome, and follow-up were not consistently reported, and hence a conclusion on the optimum time to follow up imaging and anticoagulation could not be drawn. Despite aggressive treatment, the mortality rate was 10% in our literature review, suggesting increased mortality of patients with SARS-CoV-2 infection with aortic thrombus. Significant morbidity with need for lower extremity digital amputation for ischemia from embolization was also required in one patient indicating the need for early diagnosis and treatment of this condition.

Conclusion

During the COVID-19 pandemic, the presentation of multiple thromboembolic events without an underlying source should raise suspicion for COVID-19 hypercoagulability. Clinicians should strongly consider abdominal imaging in patients presenting with abdominal pain in the setting of recent COVID-19 to rule out portal vein thrombosis. Early diagnosis and treatment of thromboembolic events could prevent severe complications such as stroke, peripheral ischemia, and intestinal infarctions. Laboratory investigations such as D-dimer, fibrinogen, LDH, ferritin and CRP, and imaging studies aid in early diagnosis. At this time, individualized treatment is recommended with medical or surgical management based on the patient's condition.

CRediT authorship contribution statement

RA, RR, LK and AJ equally contributed in conceptualizing, data acquisition, data interpretation, manuscript preparation, and review of the literature. All the authors reviewed and approved the final revised manuscript.

Conflict of interests

The author(s) declare no potential conflicts of interest with respect to the publication of this article.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the

written consent is available for review by the Editor-in-Chief of this journal on request.

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