COVID-19 and Aortic Thrombosis: A Case Report

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Abstract

Coronavirus disease 2019 (COVID-19) is an infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). It is known to cause a myriad of symptoms ranging from mild respiratory illness to severe pneumonia and acute respiratory distress. Since its discovery in late 2019 in Wuhan, China, the virus has caused a devastating worldwide pandemic. Although COVID-19 most commonly causes respiratory symptoms, complications such as hypercoagulability are now known to occur in some patients. In this case report, we present a COVID-19 patient that suffered a stroke and was found to have an aortic thrombus. In this case report, we discussed hypercoagulability, venous and arterial thrombosis in COVID-19 patients. We hope to highlight the importance of monitoring laboratory markers of hypercoagulability and thromboembolism symptoms in COVID-19 patients and encourage appropriate prophylaxis and treatment with anticoagulants when necessary. It is unclear whether or not a causal relationship exists given the nature of the syndrome. However, given the growing number of reported cases physicians should maintain awareness of this possible complication when evaluating COVID-19 patients.

Keywords: Covid-19, Complications of COVID-19, Aortic thrombus

1. Introduction

As of July 21, 2021, there have been 191,148,056 confirmed cases and 4,109,303 deaths from COVID-19 globally, out of which 33,828,878 cases and 604,252 deaths were in the United States alone. Thousands of new cases continue to be reported daily, even with the widespread vaccination campaign administered over 55 million doses in the United States. In addition to receiving the vaccination, other preventive techniques continue to be highly recommended. These include frequent handwashing, avoiding touching of the eyes, nose, mouth, social distancing of six feet between people not from the same household, avoiding contact with sick individuals, wearing a mask around others, and daily cleaning of frequently touched surfaces. Over the last one and a half years since the emergence of the virus in December 2019, clinicians continue to discover new things about the natural course of COVID-19. One of the complications that COVID-19 is now known to cause is a hypercoagulable state with an increased risk of thromboembolism. Therefore, clinicians must keep an eye out for this
complication while managing a COVID-19 patient, especially if they are at high risk for thrombosis or when new physical exam findings or symptoms arise.

2. Patient presentation

An 83-year-old female with a past medical history of hypertension, type two diabetes mellitus, hyperlipidemia, and stroke presented to the emergency department with altered mental status. She was conversational and alert but oriented only to herself. Her son reported that she had been acting confused before admission. On initial examination, she was found to have a temperature of 39.3 °C, heart rate of 107, respiratory rate of 27, BP 156/97, and oxygen saturation of 91% at room air. In the Emergency Room (E.R.), the patient was noted to be in atrial fibrillation on the cardiac monitor. On physical examination, the patient appears to be non-toxic and in no acute distress. Alert and oriented x1 but pleasant and conversational. Cardiovascular examination showed Irregular rhythm and tachycardic. S1 and S2 appreciated. No jugular venous dilation. On respiratory examination scattered crackles were noted. On neurological examination motor strength and sensation symmetric throughout. Refer to Table 1 for a summary of laboratory results upon admission. Computed tomography (C.T.) chest without contrast revealed bilateral multifocal opacities with imaging features typical for COVID-19 infection. Polymerase chain reaction (PCR) test confirmed the COVID-19 infection. She was started on Lovenox 90 mg subcutaneous injections twice a day due to the paroxysmal atrial fibrillation. Her home medications included aspirin 81 mg, atorvastatin 80 mg, carvedilol, amiodipine, famotidine, and insulin lispro sliding scale. On her second day of hospitalization, she was alert, oriented to time, place, and person, and answered questions coherently (Table 2). She had returned to her baseline mentation, confirmed by her son via phone call. During the morning of day three of hospitalization, she was found to be in sinus rhythm, so her Lovenox was discontinued, and Eliquis 5 mg twice a day was started. On day seven, the patient started requiring oxygen therapy via nasal cannula, and a chest x-ray revealed worsening infiltrates. Due to concerns of hospital-acquired pneumonia, she was started on vancomycin and Zosyn. On day nine, the patient’s mental status drastically changed; she was significantly more confused and lethargic but arousable. She was nonverbal and unable to follow commands or vocalize understanding. Neurological examination was positive for subtle right facial asymmetry and droop, right arm flaccidity, with sensation to pain upon deep stimulation. She could not overcome gravity with her right upper or lower extremities but did withdraw to pain. A stroke alert was called. Repeat D-dimer was 1337 ng/mL DDU (normal <255 ng/mL DDU). CT scan of the brain without contrast showed no evidence of intracranial hemorrhage or acute pathology. Severe small vessels ischemic change in the deep white matter supratentorial and cortical atrophy no other significant interval change from previous C.T. scan of the brain done upon admission. She had a National Institutes of Health Stroke Scale of 18. C.T. angiogram of the head showed no evidence of large vessel intracranial occlusion, but it showed irregularity and

| Laboratory findings                           | Patient’s value (normal values) |
|-----------------------------------------------|---------------------------------|
| White blood cell count                        | 4.7 (4.0–11.0 X10⁹/L)           |
| Lymphocytes absolute (low)                    | 0.8 (1.0–3.5 X10⁹/L)            |
| Potassium (low)                               | 3.4 (3.5–5.0 mmol/L)            |
| Creatinine (high)                             | 1.19 (0.40–1.00 mg/dL)          |
| Glucose (high)                                | 202 (65–99 mg/dL)               |
| Aspartate aminotransferase (high)             | 60 (40–41 U/L)                  |
| Alanine aminotransferase (high)               | 38 (0–31 U/L)                   |
| Procalcitonin (high)                          | 0.13 (<0.05 mg/mL)              |
| D-dimer (high)                                | 1439 (<255 mg/mL DDU)           |
| C-reactive protein (high)                     | 4.6 (0.00–0.744 mg/dL)          |
| Lactate (high)                                | 3.1 (0.4–2.0 mmol/L)            |
| Erythrocyte sedimentation rate (high)         | 73 (0–30 mm/h)                  |
| Ammonia (high)                                | 60 (11–35 umol/L)               |
| Portable troponin                             | 0.07 (0.00–0.09 ng/mL)          |
| B-type natriuretic peptide (high)             | 178 (<100.0 pg/mL)              |
| Myoglobin, serum (high)                       | 107.7 (14.3–65.8 ng/mL)         |
| International normalized ratio                | 1.1 (0.8–1.1)                   |
| Activated partial thromboplastin time (low)   | 24 (26–37 s)                    |
| Body Mass Index                               | 31.85 kg/m²                     |
narrowing of the anterior cerebral artery's distal branches. C.T. angiogram of the chest showed a mural thrombus in the distal aortic arch extending into the descending aorta consistent with prior dissection, as shown in Figs. 1 and 2. The brain's M.R.I. without contrast showed moderate-sized acute/subacute ischemic infarction at the left frontal lobe's medial aspect along with the left anterior cerebral artery distribution. At this time, nurses shared that the nursing aides noticed that the patient had been spitting out her medications, and they were not sure if she had been receiving all her medications. Eliquis was discontinued, and a heparin drip was started. Routine electroencephalogram showed left frontal-temporal dysfunction with evidence of right temporal cortical dysfunction and moderate diffuse encephalopathy; no epileptiform discharges or seizures were seen. She was transitioned to Coumadin as per agreement of vascular surgery and cardiothoracic surgery. On day seventeen of admission, the patient was discharged to a skilled nursing facility on Coumadin with the instructions to follow-up with vascular surgery to evaluate the need for further surgery. On discharge, she answered yes and no questions, but her comprehension and mental status were still impaired. She continued to have aphasia and right-sided weakness.

3. Discussion
This patient had many risk factors for stroke upon admission. In addition to the presence of atrial fibrillation, her history of stroke, type two diabetes mellitus, and hypertension with her age and gender make her CHA₂DS₂-Vasc score 7. This score put her at moderate-high risk for stroke and made her a candidate for anticoagulation. In addition to this risk, our patient had a COVID-19 infection, which is known to cause hypercoagulability. The hypercoagulable state mechanism in a patient with COVID-19 is thought to be due to endothelial damage via direct invasion, cytokine release, and the activation of complement pathways; stasis; and hypercoagulable

| Day of Admission | Patient's Mental Status |
|------------------|------------------------|
| 1 (At presentation) | Conversational but alert and oriented only to herself, she had been acting confused per her son. |
| 2 | Alert, oriented to time, place, and person, and answered questions properly. She had returned to her baseline mentation, confirmed by her son via phone call. |
| 9 | The patient's mental status drastically changed; she was significantly more confused and lethargic but arousable. She was nonverbal and unable to follow commands or vocalize understanding. Neurological examination was positive for subtle right facial asymmetry and droop, right arm flaccidity with sensation to pain upon deep stimulation. She could not overcome gravity with her right upper or lower extremities but did withdraw to pain. National Institutes of Health Stroke Scale (NIHSS) of 18. |
| 17 (Discharge) | Answered yes and no questions, but her comprehension and mental status were still impaired. She continued to have aphasia and right-sided weakness. |
A report published by Panigada et al. provides evidence that supports the theory of increased prothrombotic factors in patients with COVID-19. They observed a series of patients admitted to the intensive care unit whose lab parameters were consistent with a state of hypercoagulability. These patients were found to have normal or increased platelet counts, along with an increase in fibrinogen, D-dimer, C-reactive protein, factor VIII, von Willebrand factor, and protein C marginally decreased antithrombin. As a result of this hypercoagulable state, the incidence of venous and arterial thromboembolism increases during COVID-19, further increasing the mortality in these patients. The presence of thrombosis and microangiopathy in the lungs’ small vessels and capillaries has been reported in multiple autopsies performed on patients who died of COVID-19.

A study involving 1114 COVID-19 patients at Mass General Brigham integrated health network analyzed the incidence of arterial and venous thromboembolism and their effects on patients managed in intensive care (n = 170), hospitalized non-intensive care (n = 229), and outpatient (n = 715) settings. They found major arterial or venous thromboembolism, major cardiovascular adverse events, and symptomatic venous thromboembolism were highest in the intensive care group (35.3%, 45.9%, 27.0%, respectively). Whereas hospitalized non-intensive care group had 2.6%, 6.1%, and 2.2%, respectively, and 0% occurrence in the outpatient group. Prophylactic anticoagulation was given to 89.4% of the intensive care group and 84.7% of the hospitalized non-intensive care group, suggesting that coagulopathy is common among COVID-19 patients with more severe infections.

Arterial thromboembolism in patients with COVID-19 is not as well documented in literature as venous thromboembolism. Arterial thrombosis most commonly causes myocardial infarction, stroke, and limb ischemia. In a review performed by Cheruiyot et al., they assessed 27 studies (5 cohort studies, 5 case series, and 17 case reports) that reported arterial thrombotic events in patients with COVID-19. Arterial thromboembolism in critically ill patients treated in intensive care units was 4.4% in the five cohort studies. These events were symptomatic in over 95% of those patients and involved multiple arteries in 18%. The most commonly involved arteries were limb arteries (39%), cerebral arteries (24%), great vessels including the aorta, common iliac, common carotid, and brachiocephalic trunk (19%), coronary arteries (9%), and the superior mesenteric artery (8%). A mortality rate of approximately 20% was reported in these patients.

Table 3 gives a brief account of similar case reports that chronicle the occurrences of arterial thromboembolism in patients with COVID-19.

Fig. 2. Sagital view of the chest CT showing extension of the aortic thrombus.
In our patient, we see an example of arterial thrombosis in the aortic arch extending into the descending aorta. Although the aorta is not a typical location to experience thrombotic disease, due to the hypercoagulability of COVID-19, it is fairly common to find thrombi in atypical locations. We reviewed eight articles that described 19 cases involving aortic thrombosis in COVID-19 patients. Table 4 summarizes the locations of thrombosis within the aorta as reported in these case studies.14

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Table 3. Locations of arterial thromboembolic events in four COVID-19 case reports.

| Study              | Number of Patient Cases | Arterial Locations of Thromboembolic Event                                      |
|--------------------|-------------------------|--------------------------------------------------------------------------------|
| Levolger et al.11  | 4                       | Common iliac artery, tibial-fibular trunk, subclavian artery, internal carotid, and superior mesenteric artery |
| Singh et al.12     | 3                       | Tibial artery, aorta, popliteal artery, common iliac artery, and left ventricle apex |
| Lushina et al.13   | 1                       | Distal basilar artery and renal artery                                           |
| Kashi et al.14     | 7                       | Aorta, popliteal artery, common femoral artery, iliac artery, deep femoral artery, and femoropopliteal bypass |

Table 4. Locations of aortic thrombosis in eight case reports.

| Study                      | Number of Patient Cases | Aortic Location of Thrombosis (number of patients)a |
|----------------------------|-------------------------|------------------------------------------------------|
| Kashi et al.14             | 2                       | Descending aorta (2), aortic arch (1)                |
| de Carranza et al.15       | 3                       | Aortic arch (1), ascending aorta (1), descending aorta (2) |
| Baeza et al.16             | 3                       | Abdominal aorta with extension to both iliac arteries (3) |
| Mukherjee et al.17         | 1                       | Ascending aorta (1)                                 |
| Gomez-Arbelaez et al.18    | 4                       | Aortoiliac (2), aortic arch (1), descending thoracic aorta (1) |
| Woehl et al.19             | 4                       | Descending aorta (1), abdominal aorta (1), abdominal aortic with extension to both iliac arteries (1), abdominal aorta with extension to the right iliac artery (1) |
| Katchanov et al.20         | 1                       | Lower thoracic aorta with extension to the abdominal aorta and both iliac arteries (1) |
| Wickham et al.21           | 1                       | Lower thoracic aorta (1)                            |

a Some patients had a thrombosis in multiple locations.

4. Conclusion

Reflecting on our case report and the information we gathered, we know that a delayed diagnosis makes arterial thrombosis a lethal complication in COVID-19 patients. Since vigilant monitoring of symptoms and procoagulant labs, and timely intervention prevents thromboembolic complications in patients, it is important to keep an eye out for these to reduce morbidity and mortality. Since imaging has a potential role in screening and early diagnosis of thromboembolism in COVID-19 patients, imaging frequency should be individualized for the patients based on a risk-benefit analysis. Additionally, more research needs to be conducted to assess the best level and type of anticoagulation used in COVID-19 patients based on the risk factors and severity of the disease. It is important to note an increased incidence of confusion associated with the COVID-19 patient population, especially in those with increased isolation status. Hence, it would be useful to use anticoagulants whose serum levels can be monitored, such as warfarin instead of Eliquis or Xarelto. COVID-19 is still a relatively new disease and continues to be elusive in its presentation and course. Newer complications and difficulties with management emerge every day. Persistent observation, research, and reflection from all of us would help improve patient care.

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Informed consent

Obtained.

Author contributions

M.B., VMK, SN, SA, VS and G.P.M. assisted in conceptualization and data. M.B., H.S. and M.D.
helped in curation, analysis, writing, revision, and editing; M.P., MB, SSHR, G.P.M. assisted in investigation, writing, revision, and editing; M.B. assisted in data curation, writing, editing, revisions, supporting, and supervision.

Data availability

From the patient’s chart and PUB MED, google scholar.

Conflict of interest

None.

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