

Case Report

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Acute aortic thrombosis as a presenting feature of COVID-19: case report and review of literature

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ABSTRACT

Acute aortic thrombosis is an uncommon vascular emergency that can present with neurologic symptoms like acute paraplegia due to spinal cord ischemia. It frequently causes mortality unless appropriate diagnosis is followed immediately by proper management. Individuals with COVID-19 have coagulopathy with hyper inflammatory response which predisposes to both venous and arterial thrombotic events, especially in severe patients. We report a rare case of 63-year-old COVID-19 patient presenting as acute flaccid paraplegia with hematuria secondary to acute extensive aortic thrombus. CT scan showed bilateral COVID-19 pneumonia inspite of absence of respiratory symptoms. All patients with thrombotic events should be investigated for COVID-19 pneumonia as patients may not have typical respiratory symptoms.

Keywords: Aortic thrombosis, COVID-19, Paraplegia, D-dimer

INTRODUCTION

Coronavirus disease (COVID-19) emerged in China in December 2019 and rapidly spread to the world to become the largest pandemic since the 1918 influenza. This severe acute respiratory syndrome caused by a novel coronavirus-2 (SARS-CoV2) causes significant mortality especially in high risk individuals and causes disability post illness in the form of lung fibrosis and cardiac complications.¹ The disease presents with varied atypical symptoms and also a variety of atypical complications.

Individuals with COVID-19 have a number of coagulation abnormalities which contributes to mortality in these patients. COVID-19 could predispose to both venous and arterial thrombotic events, especially in severe patients. The secondary involvement of many organs with thromboembolic events has been reported with COVID-19, including central and peripheral nervous system, and cardiac or vascular systems.²⁻⁴ Currently, a large number of publications report pulmonary embolism in a significant

number of cases of COVID-19-positive patients; however, there are few publications on cases with arterial thrombosis.^{4,5} Thrombotic arterial events have been previously documented in other viral infections, especially in HIV, but no acute aortic events have been reported.⁶ Here, we present a case of severe COVID-19 without any classical respiratory symptoms presenting with acute paraplegia secondary to acute extensive aortic thrombosis.

CASE REPORT

A 63-year-old male who was a known case of ischemic heart disease on dual antiplatelet therapy presented to the emergency department with history of abrupt onset paraplegia after getting up from the bed. There was no history of prior trauma. Patient had mild asthenia and loss of appetite for three days prior to paraplegia. There was no history of fever, cough, weight loss, lifting of heavy weights, chest pain prior to the illness. On the initial physical examination, the Emergency Department physician detected complete flaccid paraplegia. The

patient had loss of pain and temperature sensation and the absence of tendon reflexes below the L1 level with extensor plantar reflexes. Patient also had associated urinary retention for which he was catheterized. CT spine did not reveal any vertebral compression. He developed sinus tachycardia, acute onset of hematuria with severe back pain within 2 hours of admission. On examination, patient had feeble dorsalis pedis and femoral pulses and cold extremities. ECG showed sinus tachycardia with old q waves in anterior leads. He was taken for urgent CT aortogram which showed thrombosis of infrarenal aorta, bilateral common iliac arteries, and bilateral internal and external iliac arteries. The common femoral artery was refilled with opacification of contrast with no thrombosis in this artery (Figure 1 and 2).

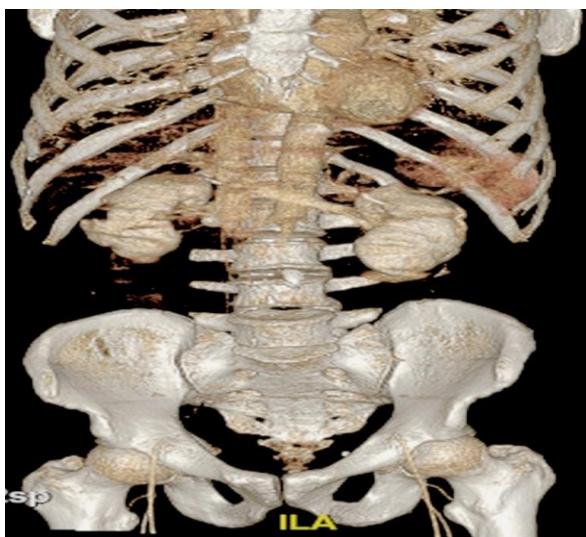


Figure 1: Thrombosis of infra renal aorta, bilateral common iliac arteries, and bilateral internal and external iliac arteries. The common femoral artery is refilled with opacification of contrast with no thrombosis.



Figure 2: Extensive thrombosis of infra renal aorta. The scan also shows small thrombus measuring 5×6 mm in the suprarenal abdominal aorta along the posterior wall.



Figure 3: CT abdomen showing non enhancing areas in both kidneys suggestive of renal infarcts.

The scan also showed small thrombus measuring 5×6 mm in the suprarenal abdominal aorta along the posterior wall. Thrombosis of inferior mesenteric artery was also noted. Non enhancing areas were seen in both kidneys suggestive of renal infarcts (Figure 3). Lungs on HRCT thorax showed peripheral patchy ground glass opacities in all lobes typical of moderate COVID-19 pneumonia (Figure 4). His blood reports showed markedly raised inflammatory markers i.e., LDH was >1300 U/l, ferritin was 1500 mcg/l, D-dimer was >1000 ng/ml, ESR 90 mm/hr, CRP 60 mg/l. Patient had mild leucocytosis and mildly raised liver enzymes. His COVID-19 RT-PCR was positive with very low cycle threshold value. Echocardiography revealed poor left ventricular function of 15% with akinetic anterior wall. He was anticoagulated and immediately referred for emergency revascularization. Patient was taken for emergency embolectomy but died during surgery.



Figure 4: HRCT thorax showing peripheral subpleural patchy ground glass opacities in all lobes typical of moderate COVID-19 pneumonia.

DISCUSSION

Acute aortic occlusion is an uncommon vascular emergency that can present with predominantly neurologic symptoms due to spinal cord ischemia. Most of the

existing literatures are case reports of catastrophic events with 75% mortality and 20–50% even after revascularization.^{7,8} Two primary causes are identified: embolism (65%) and thrombosis (35%). Acute aortic thrombosis is a rare entity even in common hyper coagulability states such as sepsis, polycythemia, disseminated intravascular coagulation, autoimmune disorders, pregnancy, and cancer. It is also an uncommon cause of peripheral arterial embolization.⁸ This syndrome can be mistaken for other neurologic disorders and is usually missed in up to 50% of cases presenting with paraplegia.⁹ Clinical presentations is sudden and characterized by pain in the lower extremities, paralysis, and lividity. In addition, it can lead to ischemic complications including gastrointestinal malperfusion, renal infarction, and paralysis secondary to spinal cord ischemia

The new pandemic of the SARS-CoV-2 Coronavirus disease (COVID-19) is generating high infection and mortality rates.¹⁰ One of the most important characteristics for poor prognosis is the development of coagulopathy and athero inflammatory response.¹¹ The incidence of venous thromboembolism during COVID-19 infection is reported as high as 25%, and arterial vascular events in up to 4%.¹² Kashi et al reported seven cases of severe arterial thrombosis, including two COVID-19 patients with asymptomatic floating thoracic aortic thrombi.¹³ Our patient presented to the hospital with descending aortic thrombosis instead of the classical respiratory symptoms of COVID-19. This implies that all thrombotic cases should be screened for COVID-19 pneumonia as patients may not have typical clinical features.

Blood coagulation derangements including elevated D-dimer, fibrinogen, and fibrin degradation products, as well as low antithrombin levels are reported in COVID-19 patients.¹² D-dimer elevations more than 1000 ng/mL have been associated with high COVID-19 mortality.¹⁴ Although there are no set cut-off values for active thromboembolic disease in patients with COVID-19, a single-centre retrospective study suggested that a D-dimer cut off of >3000 ng/mL predicted venous thromboembolism with a sensitivity, specificity, and negative predictive value of 76.9%, 94.9%, and 92.5%, respectively.¹⁵ Recently, there have been publications on positive antiphospholipid antibodies in COVID-19 patients with ischemic limb thrombosis.¹⁶

Patients with suspected hypercoagulable states in COVID-19 should be aggressively anticoagulated and referred to endovascular services. The use of heparin in patients with COVID-19 infection has been shown to decrease mortality, especially in patients with Pulmonary Embolism and high D-dimer levels.¹⁷

The vascular events in COVID-19 could be associated with several factors including hyper-inflammatory processes, hypoxia, diffuse intravascular coagulation, and immobilization. A recent publication found evidence of

the presence of virus in endothelial cells.¹⁸ The angiotensin-converting enzyme (ACE-2) receptor that the virus uses to infect cells is widely expressed in endothelial cells. This causes endotheliitis, which could explain the reason behind venous and arterial thrombosis. Direct endothelial injury induces apoptosis and inflammation which trigger the activation of macrophages and granulocytes synthesizing pro-inflammatory cytokines, generating cytokine storm.¹⁹ If the infection is not controlled, the inflammation progresses leading to a procoagulant state, characterized by massive thrombin production.¹⁹ The hypercoagulable state may be further enhanced by hypoxemia and acute respiratory distress syndrome (ARDS). Hypoxia-inducible transcription factors (HIFs) may directly activate platelets and coagulation factors and inhibit the endogenous anticoagulant protein contributing to worsening hypercoagulability.²⁰

COVID-19 has been recently linked to a large vessel stroke in young adults and peripheral arterial thrombosis. The contribution of the aortic thrombus to these arterial cerebral events, and other territories embolisms is underestimated and requires diagnosis with proper imaging techniques. In some specific patients with no clinical improvement, high D-dimer values, and no other exclusion diagnosis, CT angiogram may be useful to diagnose these aortic events.

CONCLUSION

Coagulopathy in COVID-19 patients could predispose to both venous and arterial thrombotic events, especially in severe patients. Acute aortic thrombus is an uncommon but important thrombotic complication of COVID-19 which can contribute to peripheral thrombotic events and mortality if not diagnosed in time. A very high clinical suspicion is required to diagnose aortic thrombus in severe COVID-19 patients with no clinical improvement, high D-dimer values, sudden worsening and in acute paraplegia for timely endovascular management. Further research is required on the actual contribution of aortic thrombi to peripheral thrombotic events and strokes encountered in COVID-19 patients.

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