

Aortic Thrombosis in A Patient with SARS-Cov2 Pneumonia

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Abstract

In the outbreak of novel coronavirus infection in Wuhan, significantly abnormal coagulation parameters in severe SARS-CoV2 pneumonia cases were a concern.

The literature on thromboembolic manifestations is limited. There is a case report of a patient who presented with dyspnea and fever and was diagnosed with multiple arterials thromboses.

Introduction

Coronavirus Disease of 2019 (COVID-19) is a novel coronavirus declared a pandemic by the World Health Organization in March 2020. Like other viruses (HIV, CVM...) and bacteria (*Staphylococcus aureus*), COVID-19 has increased the risk of venous and arterial thromboembolic events [1,2].

Arterial and venous thromboses are associated with severe complications and a high mortality risk.

We report a case of COVID-19 infection complicated by multiple arterial thromboses.

Case Presentation

A 58-year-old patient without medical history presented to the emergency department with a fever, asthenia, and shortness of breath that had started two weeks ago. Vital signs were as follows:

- Glasgow coma scale 15/15
- temperature 37 C°
- heart rate 99 beats/min
- blood pressure 122/64 mmHg
- oxygen saturation 80 % on room air and 96 % on supplemental oxygen (16 liter/min of oxygen by an NRB mask)

His nasopharyngeal swap was positive for COVID-19 infection. The electrocardiography was standard. Initial laboratory results were: hemoglobin, 13.6g/dl (13-16,5 g/dl); white blood cells, 10700 /mm³ (4000-10000/mm³); lymphocytes, 510 /mm³ (1500-4000/mm³); platelets, 181000/mm³ (150000-400000), glucose, 17 mmol/l(3.9-6mmol/l); creatinine, 84 μM/l (80-110 μM/l); blood urea nitrogen, 11.4 mmol/l (2,5-7,6 mmol/L); D-dimer, 635 ng/mL(< 500 ng/mL); prothrombin time, 91 % (> 70 %); aspartate transaminase, 27 mmol/l (8-30 mmol/l); alanine transaminase, 30 mmol/l (8-35 mmol/l); and C-reactive protein, 195 mg/l (< 6 mg/l).

The patient has been treated with dexamethasone 12 mg per day, low molecular weight heparin (LMWH) 0,4 mg twice per day, cefotaxime 3g per day, and vitamins like vitamin C and vitamin D. He also received insulin for his newly diagnosed diabetes. Two days after, he was also dependent on oxygen (16 liter/min), but he had painful lower extremities, and the pedal pulse was present without edema. After a few hours, his feet were so aching. On examination, his bilateral lower extremities were mottled and cold, and his femoral, posterior tibial, and pedal pulse was not palpable. A bilateral sensory-motor deficit was found. Computed tomography angiography of the aorta and iliofemoral arteries showed complete thrombosis of the abdominal aorta with extension to both common iliac arteries (**figure 1, sectiona, b**), total occlusion of the right popliteal artery (**figure 1 section c**), the left tibiofibular trunk, and the left anterior tibial artery. A splenic infarction was also present. The patient was treated with an unfractionated heparin drip (UFH). One day later, the patient had respiratory distress complicated by cardiac arrest and died.

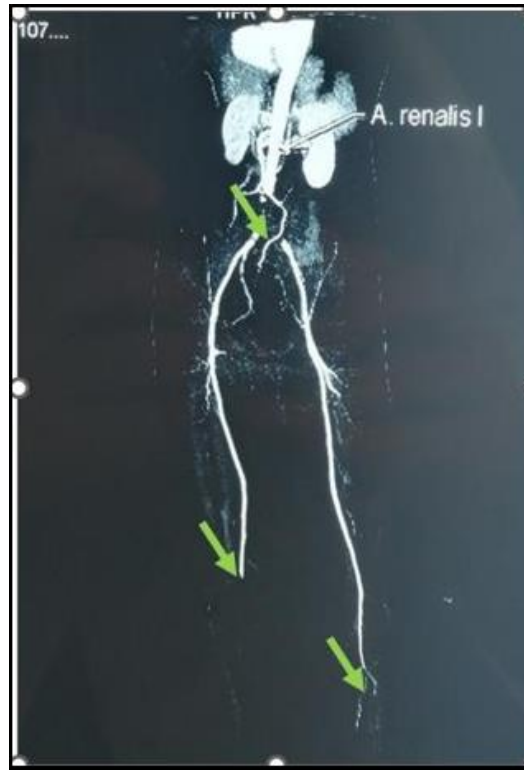


Figure 1a: CT angiography of the aorta and iliofemoral arteries showed:

- Complete thrombosis of the abdominal aorta with extension to both common iliac arteries
- Complete occlusion of the right popliteal artery, the left tibiofibular trunk, and the left anterior tibial artery



Figure 1b: CT angiography of the aorta and iliofemoral arteries showed: Thrombosis of common left iliac artery



Figure 1c: CT angiography of the aorta and iliofemoral arteries showed: Complete occlusion of the right popliteal artery

Discussion

Multiple arterial thromboses are a vascular surgery emergency. They can lead to severe morbidity and mortality. Many reviews suggest that COVID-19 patients are predisposed to arterial thrombosis. Tang et al. found that 74 % of patients who died of COVID-19 had disseminated intravascular coagulopathy [3]. Cheruiyot et al. reported that arterial thrombotic events can occur in up to 4 % of critical patients with COVID-19, sometimes affecting multiple territories [4]. In a few patients, arterial thromboembolic complications have been observed with increasing frequency in the vasculature of the brain, lungs, spleen, and limbs [5].

In Emilio Ribas Institute of Infectious Diseases in Brazil, from March to July 2020, approximately 350 patients with COVID-19 were admitted. Only 0,3 % of them were diagnosed with spleen infarction. An apparent increase in infarction prevalence was noticed, with other manifestations of ischemic events involving the abdominal viscera [6]. Most cases of arterial thrombosis in SARS-CoV2 positive patients were elderly and had chronic diseases. A recent review by Putko M. et al. noticed that most patients had cardiovascular comorbidities and diabetes mellitus [7]. Our patient was a 58-year-old man without medical history. However, diabetes was newly diagnosed on admission.

The mechanism of thrombotic events may be multifactorial. It associates cytokine storm, increased procoagulant factors, stasis, hypoxia, and endothelial dysfunction.

Our patient presented with complete thrombosis of the abdominal aorta with extension to both common iliac arteries, a spleen infarction, occlusion of the right popliteal artery, the left tibiofibular trunk, and the left anterior tibial artery. This topography suggested that the emboli originated from a thrombus in the aorta, detached from the aorta, and caused multiple arterial thromboses.

In the literature, several cases of aortic thrombosis are reported in patients with severe COVID-19 [8,9]. Gomez-Arbelaiz D et al. has reported four cases of aortic thrombosis and ischemic complications in patients with severe SARS-CoV2 infection despite the antithrombotic prophylaxis: Two patients had an aortic occlusion, and the others had an aortic floating thrombus. All the patients had other ischemic complications like acute limb ischemia, acute mesenteric ischemia, and stroke [10].

Conclusion

Despite prophylactic measures, SARS-CoV2 infection can be associated with multiple arterial thrombotic events. These complications are significantly more common in severe forms.

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In the literature, multiple ischemias usually affect the lower limbs. In the Putko M et al. review, 47 patients with COVID-19 were diagnosed with lower limb ischemia, and 6 patients had bilateral ischemia [11]. Monteiro L et al. reported a case of a 61-year-old man with a medical history of diabetes mellitus and dyslipidemia who had severe SARS-CoV2 pneumonia. A few hours after admission, he complained about intense pain in the lower limbs. The CT angiography revealed an intramural aortic thrombosis with more than 50 % stenosis of the descending thoracic aorta, complete occlusion of the lower abdominal aorta, and extension to both common iliac arteries. Left renal infarction was also found. Despite adequate medical treatment, the patient died [12].

The most important indicators of the cytokine storm are D-dimer, platelets, ferritin, LDH, prothrombin time, fibrinogen, and IL-6. [13]. D-dimer was frequently elevated. In China, a study of 1099 patients showed that 46,4 % of patients had a high level of D-dimer ($\geq 500 \mu\text{g/l}$), and 60% of them had severe forms of COVID-19 infection [14]. Tang et al. showed that abnormal coagulation results, significantly elevated D-dimer and fibrinogen, are common in deaths with COVID-19 infection [15].

Because of the hemostatic derangements in these patients, some clinicians use a variety of prophylactic, intermediate, or therapeutic doses of heparin for routine care of patients with COVID-19 pneumonia [16].

Many authors suggest using heparins at therapeutic doses in patients with COVID-19 from admission [17]. The timing of thrombosis diagnosis and treatment is intriguing [17]. In a retrospective study of COVID-19 patients with limb vascular ischemia, the median time from the onset at the clinic consistent with coronavirus infection to the development of limb ischemia was 19 days [18].

In the three cases presented by Baeza C, surgical treatment associated with curative anticoagulation has allowed the rescue of patients with extensive aortic thrombosis [18]. So, this case aims to reinforce the difficulty of managing multiple thromboses despite a therapeutic dose of LMWH or UFH. But, prophylaxis, prompt diagnosis, and initiation of medical therapy or even surgery are the mainstay of managing these complications [19].

Prompt diagnosis and initiation of directed therapy are essential to avoid morbidity and mortality.

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