# Acute aortic thrombosis in COVID-19

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## ABSTRACT

Acute aortic occlusion is an infrequent disease but with significant mortality. The new pandemic of the SARS-CoV-2 coronavirus disease (COVID-19) represents a great challenge for health systems. This contagious disease is generating high infection and mortality rates in several countries. It is speculated that the inflammatory process accompanying the infection is triggered by massive macrophage activation and is associated with the development of coagulopathy. We present three cases of COVID-19 patients, treated in our hospital during a period of 2 weeks, who presented with an acute thrombosis of the infrarenal abdominal aorta. (J Vasc Surg Cases and Innovative Techniques 2020;6:483-6.)

**Keywords:** Acute; Infrarenal; Aortic; Thrombosis; Coronavirus

Acute aortic occlusion (AAO) is a rare disease with catastrophic consequences. Clinical presentation 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. Most of the publications are reviews of cases collected over the years, which provides an idea of how unusual this pathologic process is.[1-5]

The new pandemic of the SARS-CoV-2 coronavirus disease (COVID-19) is generating high infection and mortality rates.[6] One of the most important characteristics for poor prognosis is the development of coagulopathy.[7] In the midst of this crisis, guidelines that define the risks to optimize the appropriate treatment are being drafted.[8] There seems to be some general consensus regarding the use of low-molecular-weight heparins.[9] Currently, a large number of publications report a large number of cases of COVID-19-positive patients diagnosed with pulmonary embolism (PE); however, there are few publications on cases with arterial thrombosis. Specifically, to date, we have no knowledge of publications on AAO in these patients. We present three cases, which occurred in just 2 weeks, of AAO of the infrarenal abdominal aorta in patients who had previously been diagnosed with COVID-19. All three patients agreed to publication of their case details and images.

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## CASE REPORTS

In all three cases, patients had two hospital admissions. Diagnosis of SARS-CoV-2 was confirmed by reverse transcriptase-polymerase chain reaction analysis during the first admission in each patient. The Table summarizes the main laboratory results and thrombophilia study pertinent to the first and second hospital admissions of each patient. Discrete calcified plaques on the aortoiliac axis were observed on every computed tomography study.

**Patient 1.** The patient, a 63-year-old woman, is an ex-smoker with human immunodeficiency virus infection, hepatitis C, dyslipidemia, ischemic stroke with residual cognitive changes, anticoagulated atrial fibrillation, and rheumatic mitral stenosis.

The first admission on March 9, 2020, was for a mitral and tricuspid valve replacement. Postoperatively in the intensive care unit, she had a temperature up to 38.5°C and cough with expectoration. Chest radiography did not detect characteristic patterns. She was discharged without deterioration of respiratory function and antiviral treatment with ritonavir-darunavir, cyclosporin A, hydroxychloroquine, doxycycline, and bemiparin.

The second admission was on April 4, 2020, for sudden-onset pain in the lower limbs of 6 hours' evolution. She presented with lividity, absence of pulses at all levels, and partial loss of sensation and mobility. Computed tomography angiography (CTA) showed extensive thrombosis of the infrarenal aorta with extension through both iliac axes (Fig 1).

Surgical procedure: extra-anatomic axillobifemoral bypass under general anesthesia.

Outcome: bilateral pedal pulse recovery.

Treatment at discharge: acenocoumarol according to usual regimen.

**Patient 2.** The patient is a 69-year-old male former smoker with high blood pressure, diabetes mellitus, dyslipidemia, lumbar canal stenosis, obesity, chronic obstructive pulmonary disease, and intermittent claudication.

The first admission, on March 27, 2020, was due to an 8-day clinical picture with general discomfort, cough, and temperature >38°C. Chest radiography showed signs suggestive of bilateral pneumonia. He was discharged with clinical improvement,

**Table.** Main laboratory results and thrombophilia study pertinent to first and second hospital admissions of each patient

| Test | Patient 1 | | Patient 2 | | Patient 3 | | Reference value |
|---|---|---|---|---|---|---|---|
| | First admission (COVID-19 diagnosis) | Second admission (AAO) | First admission (COVID-19 diagnosis) | Second admission (AAO) | First admission (COVID-19 diagnosis) | Second admission (AAO) | |
| Blood cell count | | | | | | | |
| Leukocytes, × 103 μL | 4.48 | 22.1 | 8.61 | 15.72 | 9.32 | 16.74 | 3.5-12.0 |
| Lymphocytes, × 103 μL | 0.6 | 11 | 0.7 | 16 | 16 | 12 | 1.2-5.0 |
| Lymphocytes, % | 12.3 | 5.6 | 9.7 | 11.1 | 17.5 | 8.5 | 20-45 |
| Red blood cells, × 106 μL | 3.8 | 3.4 | 5.2 | 4.7 | 4.6 | 4.8 | 3.5-5.8 |
| Hemoglobin, g/dL | 11.3 | 10.2 | 13.7 | 12.7 | 13.9 | 14.8 | 12-5 |
| Hematocrit, % | 33.5 | 30.8 | 42.4 | 38.0 | 39.7 | 43.4 | 36-43 |
| Platelets, × 103 μL | 370 | 112 | 243 | 295 | 244 | 174 | 150-450 |
| Basic profiles | | | | | | | |
| D-dimer, μg/L | 4298 | 7097 | 1385 | 6624 | 664 | 1517 | 68-494 |
| Fibrinogen, mg/dL | — | 636 | — | 627 | — | 1036 | 200-400 |
| Proteins | | | | | | | |
| C-reactive protein, mg/dL | 12.6 | 5 | 30.8 | 1.2 | 34.41 | 6.1 | <0.5 |
| Ferritin, ng/mL | 973 | 579 | 518 | 374 | 1472 | 813 | 13-150 |
| Immunologic studies | | | | | | | |
| Interleukin 6, pg/mL | 30.3 | — | 136 | 1.55 | — | 29.5 | <7 |
| KL-6, U/mL | — | 715 | — | 725 | — | 550 | <650 |
| Enzymes | | | | | | | |
| Creatine kinase, IU/L | — | 5280 | — | 5588 | — | 71 | <190 |
| Thrombophilia screening | | | | | | | |
| Protein C, % | — | 139 | — | 124 | — | 110 | 70-130 |
| Protein S, % | — | 55.8 | — | 55.3 | — | 74.3 | 58-123 |
| Lupus anticoagulant | — | Positive | — | Positive | — | Positive | |
| Antiphospholipid antibodies | — | Negative | — | Negative | — | Negative | |

*AAO*, Acute aortic occlusion.

receiving treatment with lopinavir-ritonavir, prednisone, hydroxychloroquine, and doxycycline but no anticoagulant.

The second admission, on April 5, 2020, was for severe pain in the lower left limb of 4 hours' evolution, with impaired mobility and lack of sensitivity. Pulses were not palpable at any level. He reported onset of numbness in the right limb, in which he had decreased pulses with normal coloration of the limb. CTA showed occlusion of the distal abdominal aorta and the origin of both common iliacs, which extended through the left iliac axis to the femoral bifurcation, with a patent right iliac axis (Fig 2).

Surgical procedure: surgical thrombectomy with Fogarty balloon through bilateral femoral access under regional anesthesia.

Outcome: bilateral pedal pulse recovery.

Treatment at discharge: anticoagulant treatment with bemiparin 10,000 IU/24 h.

**Patient 3.** The patient is an 85-year-old woman with high blood pressure and atrial fibrillation anticoagulated with dabigatran. She has no vascular clinical history.

The first admission, on March 25, 2020, was due to an 8-day history of clinical symptoms with asthenia, cough, general malaise, and low temperature >37.7°C. Chest radiography showed signs of bilateral pneumonia. She was discharged with clinical improvement, receiving treatment with azithromycin, ceftriaxone, prednisone, hydroxychloroquine, and dabigatran at usual doses.

The second admission, on April 18, 2020, was for gluteal pain radiating to both lower limbs and numbness of 12 hours' evolution with pale and cold lower limbs with lividity to the abdomen, impaired mobility, and absence of sensitivity without pulses. CTA showed an occlusive filling defect in the aortic bifurcation and both common iliac arteries affecting internal and external iliacs that were repermeabilized distally (Fig 3).

Surgical procedure: surgical thrombectomy with Fogarty balloon through bilateral femoral access under regional anesthesia.

Outcome: bilateral pedal pulse recovery.

Treatment at discharge: anticoagulant treatment with enoxaparin 6000 IU/12 h.

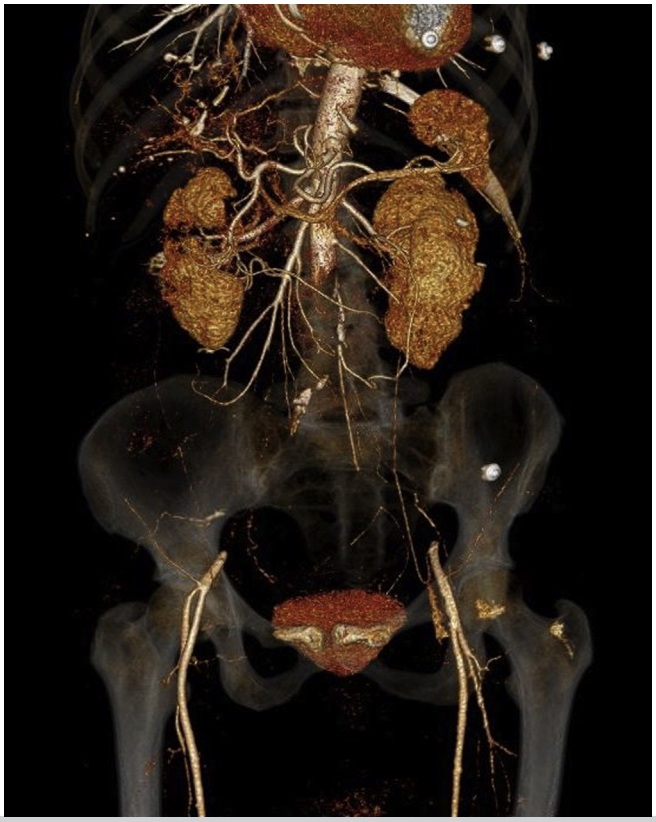

**Fig 1.** Computed tomography angiography (CTA) three-dimensional image shows extensive thrombosis of the infrarenal aorta with extension through both iliac axes.

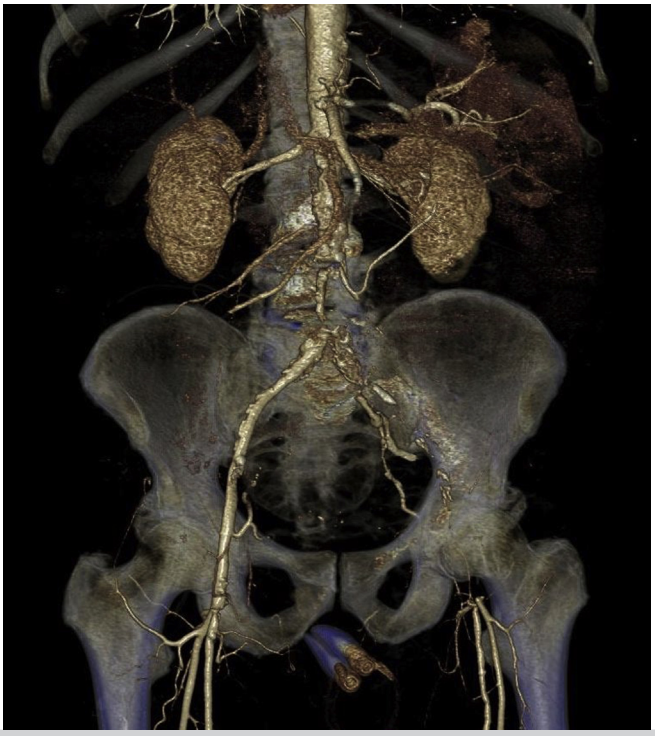

**Fig 2.** Computed tomography angiography (CTA) three-dimensional image shows occlusion of the distal abdominal aorta and the origin of both common iliac arteries, extended through the left iliac axis to the femoral bifurcation, with a patent right iliac axis.

## DISCUSSION

AAO is an atypical disease with morbidity between 30% and 74% and an overall mortality of 34% to 62%.[1,2] The incidence reported by the few publications in this regard is low and is collected during a long time.[1,3-5]

COVID-19 is a disease with a high mortality rate in critical patients.[10] Around 20% of patients present with complications, including coagulation disorders.[11,12] Inflammatory and thrombotic processes are closely related.[13] There is some general consensus that the inflammatory process developing after viral infection is triggered by a massive activation of macrophages that generate a "cytokine storm."[14] Despite the fact that PE is indicated as the most frequent thrombotic disease in these patients, arterial thrombosis could be even more important,[15] and its presentation as AAO is suggestive of this. Coagulopathy leads to high mortality, and laboratory abnormalities, such as elevated D-dimer, are a particularly important marker.[16-18]

Antiphospholipid syndrome, an autoimmune condition that generates hypercoagulability in the blood, is caused by antibodies directed against phospholipid-binding proteins in cell membranes. Some viral infections are known to cause antiphospholipid syndrome.[19,20] Recently, case series have been published of positive COVID-19 patients with ischemic limb thrombosis and in whose serologic study antiphospholipid antibodies were detected.[15,17] In our three cases, acute aortic thrombosis, accompanied by a positive lupus anticoagulant, could be consistent with an antiphospholipid syndrome.[20] However, this positivity could also be accessory to the viral infection itself or secondary to organic endothelial damage, so its presence should be confirmed within a few weeks.

The use of heparins in patients with COVID-19 infection has been shown to decrease mortality, especially in patients with PE and high D-dimer levels.[9] The correct dose of low-molecular-weight heparin is a matter of immediate interest, and many authors suggest use of heparins at therapeutic doses in patients with COVID-19 from the moment of admission.[21]

Timing of thrombosis presentation is interesting. In a retrospective analysis of COVID-19 patients with limb vascular ischemia, the median time from onset at the clinic consistent with coronavirus infection to the development of limb ischemia was 19 (11-23) days.[15] In the three cases presented, 20 days, 7 days, and 24 days, respectively, elapsed from the time SARS-CoV-2 was confirmed until the presentation of the ischemic symptoms. In addition, it is of clinical interest that the three cases had lymphopenia and marked elevation of

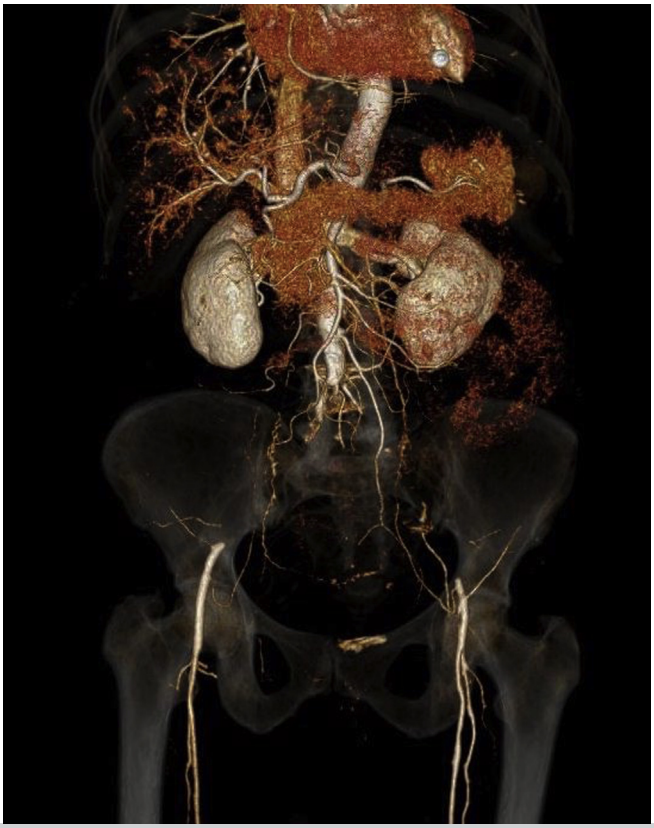

**Fig 3.** Computed tomography angiography (CTA) three-dimensional image shows an occlusive filling defect in the aortic bifurcation and both common iliac arteries extended through internal and external iliac arteries.

D-dimer, interleukin 6, and ferritin, all of which are markers of poor prognosis and that highlight the need to maintain this type of patient with anticoagulant treatment, although the appropriate dose still has to be defined.

## CONCLUSIONS

Despite the pre-existence of risk factors in these patients, our study observed that there is likely to be an association between COVID-19 infection and the development of an intensely prothrombotic state that can lead to significant arterial-type complications, which should be avoided as much as possible with the use of the correct anticoagulant therapy.

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