

case report

Acute infrarenal abdominal aortic occlusion in a patient with COVID-19

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A high rate of thrombotic complications have been observed in patients infected with COVID-19. These complications are related to increased blood hypercoagulability, which can cause both venous and arterial thrombosis. We report a case of a 60-year-old man with COVID-19 pneumonia and thrombotic occlusion of the infrarenal abdominal aorta at the time of admission to the hospital. A CT scan showed a crazy-paving pattern in the lungs, consistent with COVID-19. A clinical suspicion of aortic thrombosis was confirmed by CT angiography. Embolectomy was undertaken a few hours later. At the end of the procedure, the patient was taken to the intensive care unit while intubated. The patient then worsened, developing severe renal failure, and died on day 1 after admission to the hospital. A CT scan, which is necessary for diagnosis of COVID-19, and a CT angiography, can be used to diagnose thrombotic events. It should be kept in mind that arterial thrombosis can be present not only in hospitalized COVID-19 patients but also at the time of admission.

SIMILAR CASES PUBLISHED: 1

SARS-CoV 2 infection (COVID-19) is a major worldwide health problem that can cause blood hypercoagulation, which can lead to venous and arterial thrombosis.¹ We report a case of a 60-year-old male patient with COVID-19 pneumonia and thrombotic occlusion of the infrarenal abdominal aorta.

CASE

A 60-year-old man without any known systemic disease, peripheral arterial disease, cardiovascular comorbidity or other atherosclerotic risk factors except smoking presented to the emergency service with the complaint of respiratory distress lasting for two days. Laboratory tests showed an increase in ferritin (>2000 ng/mL), C-reactive protein (98 mg/L), and leukocytes (11.200 K/ μ L). Unfortunately, the D-dimer level was not measured. Creatinine and blood urea nitrogen (BUN) levels were elevated at 3.79 mg/dL and 177 mg/dL, respectively. Hyponatremia (165 mmol/L) and hyperkalemia (6.9 mmol/L) were also present. Decompensated metabolic acidosis (pH 7.13) and hyperlactatemia (17.3 mmol/L) were observed in the blood gas analysis. Tachypnea (25 cycles per minute) with low blood oxygen saturation (86%), low blood pressure (90/45 mmHg), and tachycardia (114 bpm) were detected on physical examination. Pallor, pulselessness, and hypothermia of both legs were seen. Both legs were ischemic, with loss of mo-

tor and sensory function. Ischemia was present at the time of presentation and was thought to have been ongoing for several hours. The electrocardiogram was in sinus rhythm. On echocardiographic examination, the global ejection fraction was 50%, the right heart chambers were large, and there was no valve pathology. A CT scan showed a crazy-paving pattern in the lungs (**Figure 1**). An oropharyngeal swab test result was positive for SARS-CoV-2. CT angiography confirmed an aortic thrombosis that was suspected clinically (**Figure**

2). An occlusive thrombus was observed in the infrarenal abdominal aorta (**Figure 3**). The thrombus did not involve the renal arteries (**Figure 4**). A thrombectomy of all iliac arteries and embolectomy was performed with femoral cutdowns and transverse arteriotomy. A Fogarty embolectomy catheter was advanced approximately 25 cm proximally and then approximately 70 cm distally, and a large amount of acute thrombus was removed. Embolectomies were performed just proximal to the common femoral artery bifurcation. If there

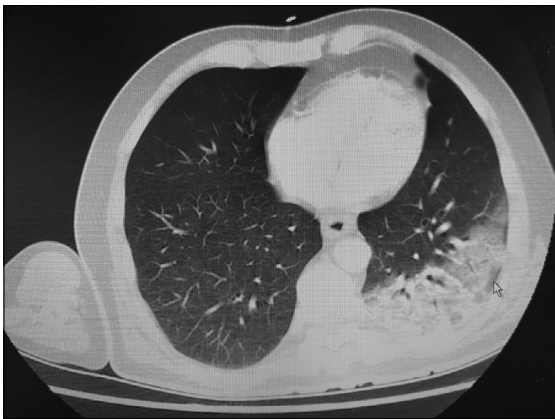


Figure 1. CT showing crazy-paving pattern.

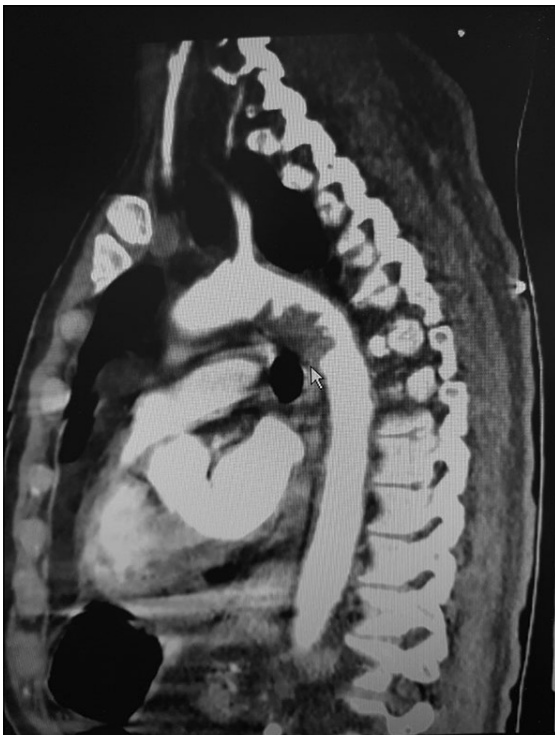


Figure 2. CT angiography (sagittal) showing thrombus and narrowed aortic lumen.



Figure 3. CT angiography showing a coronal total occlusive thrombus in the infrarenal abdominal aorta.



Figure 4. CT angiography (cross-section) at level of the renal artery.

were thrombi in the renal arteries, it would not be possible to clean the renal arteries with this technique. Surgery was conducted under general anesthesia with inotropic support provided throughout the operation. There was no distal perfusion in either leg before the operation. Intraoperative imaging was not performed. After the thrombectomy, pulsatile flow was present and the legs were viable after the thrombectomy. During the follow-up, compartment pressure did not increase and fasciotomy was not performed. At the end of the procedure, the patient was taken to the intensive care unit and intubated. Postoperative arterial blood gas results still showed decompensated metabolic acidosis (pH 7.12, PCO₂ 29 mmHg, PaO₂ 149 mmHg, hemoglobin 10.6 g/dL, SaO₂ 97.4%, HCO₃ 9.6 and lactate 7.0 mmol/L). Creatinine, blood urea nitrogen and creatine kinase levels were 2.96 mg/dL, 161 mg/dL and 183 010 U/L. There was also hyperkalemia (6.2 mmol/L). Hemodialysis could not be performed due to persistent hypotension despite inotropic support. Although the thrombus did not involve the renal arteries the patient worsened, developing severe renal failure due to prolonged hypotension and co-existing rhabdomyolysis, and died on day 1 from admission to the hospital.

DISCUSSION

In early 2020, the novel coronavirus was declared the cause of the COVID-19 outbreak by the World Health Organization. Although the main symptom with COVID-19 is respiratory distress, thromboembolic events are also reported.² The incidence of abdominal aortic occlusion is 2.7 to 5.0 cases per 1 million person-years.³ In recent reports mortality rates ranging from 25% to 75% and morbidity rates between 30% and 74% have been reported.^{3,4}

Thromboembolic complications are associated with inflammation, stasis, and hypercoagulable states.^{5,6} Hypoxia is a strong trigger of thrombosis,^{5,7} which results from the severe inflammatory response induced by COVID-19 with subsequent endothelial dysfunction and a procoagulant effect.⁸ A recent study sug-

gests COVID-19 infection may be responsible for the sequelae caused by impairment of endothelial function and microvascular circulation in multiple organs.⁹ Unfortunately, our patient's thrombus was not sent for histological or microbiological examination. Our patient had respiratory distress, thrombosis, and renal failure concurrently. We suspected extensive endothelial damage because of the severity of the disease. COVID-19 infection is more severe in patients with endothelial damage due to hypertension, smoking and diabetes. Although hypertension and other comorbidities are reported as present in aortic thrombosis cases, our patient did not have known cardiovascular disease or a history of thrombosis.¹⁰⁻¹⁵ COVID-19 disease itself creates a hypercoagulant state, independently of concomitant diseases or predisposing conditions for the formation of large-vessel thrombosis.

The use of low molecular weight heparin (LMWH) is recommended for COVID-19 treatment, but many cases of thrombosis are reported despite anticoagulation prophylaxis.¹⁰ Cases of successful treatment with unfractionated heparin (UFH) infusion have been reported.^{12,14} Preoperatively, our patient received 6000 IU of LMWH once, intraoperatively 5000 IU of UFH once; postoperatively 6000 IU of LMWH and 5000 IU of UFH were given twice daily. Thromboembolic events such as aortic thrombosis occur more frequently after the second week of hospitalization.^{10,12} Our patient had aortic thrombosis at the time of admission to the hospital, suggesting that he might have been hesitant to go to the hospital due to the COVID-19 outbreak. Large-vessel thrombosis should be kept in mind in COVID-19 patients admitted to the emergency department for other symptoms other than respiratory distress, as in our case, and examination and imaging methods should be undertaken to assess these conditions. A chest CT scan is necessary for the diagnosis and treatment of COVID-19 along with CT angiography in the case of pulmonary embolism or arterial occlusion. Since acute aortic thrombosis is a fatal condition, it must be quickly diagnosed and treated.

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