Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Within the CEA cohort, 1 patient experienced a transient ischemic attack (0.9%), and 4 patients developed a postoperative neck hematoma for which repeat intervention was needed (3.6%). During follow-up, 3 patients (2.7%) developed symptomatic ipsilateral restenosis of the carotid artery, 2 of which within 90 days of CAS. These patients were all successfully treated with CAS. Complications such as myocardial infarction or cerebral hyperperfusion syndrome were not reported in the CAS and CEA groups.

With these results, we believe that a temporary CAS first approach within our center is a safe and reasonable approach. Primary treatment with CAS could reduce the burden of care within hospitals and ensure adequate and timely care for this patient group during a time of limited capacity.

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Risk of peripheral arterial thrombosis in COVID-19

Since the report of the first COVID-19 cases in Wuhan (China) on December 31, 2019, several thrombotic complications associated with this disease have been described.[1,2] These have mainly included venous thromboembolic events[3] and myocardial infarction.[4] However, we have noted a rapidly increasing occurrence of a not previously described vascular complication in critically ill patients: acute peripheral arterial thrombosis.

To date, in our institution (Hospital Clinic, Barcelona, Spain; a reference center for COVID-19 treatment), we have diagnosed acute limb ischemia in four patients infected with COVID-19 that was attributed to the secondary hypercoagulable state. Of the four patients, three had presented with infrapopliteal arterial thrombosis of all distal vessels in one or both legs (one and two patients, respectively; Fig). The fourth patient had presented with femoral-popliteal and radial-ulnar arterial thrombosis. All four patients had associated distal cutaneous microembolism of the toes or fingers, with progressive distal clinical onset of symptoms: toe or finger dysesthesia and paresis, without muscular infarct. The mean patient age was 71 years. The four patients (three men) had had no previous severe comorbidities or known cardiovascular disease that could have caused the arterial embolisms. Acute ischemia had appeared on average 15 days after the onset of respiratory COVID-19 symptoms. All four patients had previously been admitted to intensive care units because of severe respiratory syndrome, with high oxygen and dedicated treatment requirement (lopinavir/ritonavir, hydroxychloroquine, corticosteroids, azithromycin, anticoagulation, and, eventually, tocilizumab or plasma exchange). Only one case had presented with coexisting venous thromboembolism and splenic infarct. The blood samples revealed an average high D-dimer (>10,000 ng/mL), lactate dehydrogenase (823 U/L), and ferritin (2473 ng/mL) levels, with moderate elevation of C-reactive-protein, platelets, and leukocytosis, and decreased mean coagulation times.

Fig. Angiography computed tomography three-dimensional reconstruction of three cases of infrapopliteal arterial thrombosis.

Of the four cases, two underwent surgery. One patient had undergone open bilateral popliteal direct thrombectomy of all distal vessels and one had undergone open femoral-popliteal and radial-ulnar thrombectomy. Both patients recovered immediate patency of all declotted arteries and experienced improved clinical symptoms, despite the finding of segmental distal asymptomatic rethrombosis at 24 hours after surgery (plantar arch or one of the distal infrapopliteal vessels). The other two patients did not undergo surgery because of their extremely poor respiratory condition and relatively well-tolerated ischemia; these patients have continued to receive follow-up.

The vascular community should be aware of this new thrombotic complication in critically ill patients with COVID-19. It can be due to the hypercoagulable secondary status, and surgery is a valuable treatment option for complicated cases, despite the risk of rethrombosis.

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Intra-arterial thrombosis associated with COVID-19

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a global pandemic. The development of an acquired thrombophilia with activation of the coagulation cascade in response to the inflammatory process has been described.[1] SARS-CoV-2 appears to have an affinity for angiotensin-converting enzyme 2 receptors that seem to be downregulated, which may drive the proinflammatory/prothrombotic consequences.[2]

As this is a novel illness, there has been limited opportunity to understand and to investigate the effect of SARS-CoV-2. In severe disease, there appears to be elevation of D-dimer levels. Prolongation of prothrombin time and activated partial thromboplastin time seems to be associated with an increased risk of acute respiratory distress syndrome and death.[3] The development of disseminated intravascular coagulation appears to be common in nonsurvivors.[4]

There appears to be an increased risk of pulmonary thromboembolic events in ventilated patients with SARS-CoV-2, despite the administration of low-molecular-weight heparin prophylaxis. In a small series of minimally invasive autopsies, a significant proportion of patients were found to have small fibrinous thrombi in pulmonary arterioles.[1] Similar data have been described across Europe with revision of low-molecular-weight heparin prophylaxis dosing. As well as the described pulmonary thrombotic complications, a series of early circuit occlusions has been encountered in patients on hemofiltration as a consequence of multiple organ dysfunction syndrome from SARS-CoV-2.