Preventing stroke in symptomatic carotid artery disease during the COVID-19 pandemic

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virus transmission was constantly provided to the patients and their relatives.
5. As long as the patient's vital signs were stable, preoperative screening of COVID-19 was enforced; thus, every patient was assessed for potential COVID-19 infection using both serum antibody testing and computed tomography examination. Some typical pulmonary computed tomography features must be carefully ruled out, such as bilateral ground-glass opacity and subsegmental consolidation.[4]
6. Whenever possible, the patients were kept in isolation rooms postoperatively.

Worldwide experience in managing life-threatening surgical emergencies (eg, type A aortic dissection) under the extremely stressful condition during the COVID-19 pandemic is limited. We hope our lessons learned from this small series of four patients can help surgeons to manage the challenges caused by the epidemic.

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Preventing stroke in symptomatic carotid artery disease during the COVID-19 pandemic

As the coronavirus disease 2019 (COVID-19) pandemic is escalating, many countries are struggling to contain the virus and ensure appropriate care. Hospitals and their intensive care units have seen an overwhelming increase in the number of patients, which has had substantial effects on the care for all other patients.

For patients with symptomatic carotid artery disease, carotid endarterectomy (CEA) can prevent major stroke or death.[1,2] These semi-acute treatments are, however, likely to be cancelled or postponed because of the reallocation of resources, such as anesthesiology teams, ventilators, and operation room capacity. However, analyses of the pooled North American Symptomatic Carotid Endarterectomy Trial and European Carotid Surgery Trial data have shown that the benefit of surgery is considerable reduced when patients are treated more than 2 weeks after the presenting symptoms.[3]

To ensure the care for patients with symptomatic carotid artery disease during the COVID-19 outbreak, we decided to temporarily switch our primary form of treatment from CEA to carotid artery stenting (CAS) because CAS does not require anesthetics or intensified care on a standard basis. The use of CAS can achieves long-term benefits similar to those with CEA but has been associated with an increased risk of periprocedural stroke or death.[4] Within our center, a tertiary referral center located in the northern region of The Netherlands, we evaluated the results for CEA and CAS for the past 2 years to assess the safety of CAS in daily practice. The primary endpoint was ischemic or hemorrhagic stroke within 90 days after the procedure.

A total of 155 patients had been treated from January 2018 to December 2019 for symptomatic carotid artery disease. Of the 155 patients, 110 had undergone CEA and 44 had undergone CAS because severe comorbidities, a hostile neck, or a high cervical carotid bifurcation. Within the CAS group, 2 postprocedural hemorrhagic stroke events occurred (4.5%). One patient experienced intracerebral hemorrhage within 90 days of treatment. For that patient, clopidogrel was replaced by ticagrelor, in addition to aspirin, because of poor (0%) platelet aggregation inhibition with clopidogrel, as measured by P2Y12 platelet function testing. The second patient had had symptoms of a transient ischemic attack on the ward, with no new ischemic damage found on a computed tomography scan but an unexpected asymptomatic thalamic hemorrhage under triple anticoagulation regimen. Both patients recovered completely. One patient had developed amaurosis fugax 6 months after CAS because of in stent stenosis, with explantation of the stent performed, followed by formal endarterectomy.

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.