Common carotid macrothrombosis causing acute ischaemic stroke in a patient with COVID-19

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DESCRIPTION
A 50-year-old woman with a medical history significant only for well-controlled hypertension and recent SARS-CoV-2-positive test presented as a stroke alert for aphasia and right hemiparesis with a National Institutes of Health Stroke Scale score of 20. She was out of the window for thrombolytic therapy. CT head did not reveal any acute abnormalities, with an ASPECTS (Alberta Stroke Program Early CT Score) of 10. CT angiogram demonstrated a focal area of soft plaque within the left common carotid artery, for which the patient was taken to the angiography suite immediately. A diagnostic cerebral angiogram revealed a non-occlusive non-flow-limiting thrombus within the left common carotid artery measuring approximately 2.5 cm caudal to the left carotid bifurcation (figure 1), with additional findings of a regional perfusion defect within the left M4 cortical branches suspected to be embolic from the carotid thrombus. Thrombectomy was not attempted given concern of clot fragility and the high risk of fragmentation and emboli during manipulation. A non-contrast MRI brain revealed ischaemic infarct in the left distal middle cerebral artery (MCA) territory and external watershed zones of the anterior cerebral artery/MCA and MCA/posterior cerebral artery (figure 2). Standard serological tests and coagulation markers were sent in addition to standard stroke etiological workup, without any radiographic or echocardiographic evidence of a proximal source of thromboembolism, and the aetiology of her stroke was attributed to macrothrombosis secondary to SARS-CoV-2-related endotheliopathy. She was initiated on heparin infusion and later transitioned to apixaban, which she continued on discharge to inpatient rehabilitation. Follow-up in clinic was planned after repeat CT angiography to document thrombus resolution.

Prior studies have shown that the extent of coagulopathy correlates with severity of respiratory illness,1 predominant sequelae of which include venous thromboembolic events, end-organ failure secondary to a microangiopathy believed to be similar to disseminated intravascular coagulation,2 and stroke which was seen in 5.7% of those with critical illness in comparison to 0.8% of those with milder COVID-19 disease.3 Tested for suspected exposure to SARS-CoV-2, our patient had no respiratory or infectious symptoms, suggesting that in addition to the previously characterised venous thrombosis and microangiopathy associated with SARS-CoV-2 infection, there may also be a tendency towards large-vessel arterial thrombosis, which may be secondary to virally mediated disruption of the endothelium causing endotheliitis.4 A case series of three SARS-CoV-2-infected individuals with mild respiratory symptoms and stroke secondary to large-vessel thrombosis without occlusion has been previously reported,5 with the aetiology of coagulopathy attributed to virus-mediated endotheliopathy. Given the significant implications for primary and secondary stroke prevention in patients with COVID-19, further systematic studies are needed to establish the role of anti-inflammatory agents and anticoagulants, if any.

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Learning points

- Mechanisms of coagulopathy in SARS-CoV-2 infection include previously characterised venous thrombosis and microangiopathy, with the extent of coagulopathy correlating to the severity of respiratory symptoms, as demonstrated by stroke inflicting 5.7% of critically ill patients compared with 0.8% with milder COVID-19 disease.
- Coagulopathy in the absence of respiratory symptoms in a patient with COVID-19 suggests a tendency toward large-vessel arterial thrombosis, secondary to virally mediated endotheliitis, as demonstrated in this case.

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