**Common Carotid Artery Thrombus in the Setting of COVID-19 Vasculopathy**

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**Introduction**

Since the outbreak of the global pandemic of coronavirus disease 2019 (COVID-19), there have been increasing numbers of case reports of large cerebral vessel thrombosis in patients with COVID-19.[1-4]Angiotensin-converting enzyme 2 receptor-mediated endothelial damage, rupture of atherosclerotic plaques, inflammation, or hypercoagulability have been described as the possible mechanisms of COVID 19 related thrombosis and strokes.[5-7] Here, we present a case of common carotid artery thrombus and embolic strokes in a young COVID-19 patient without significant stroke risk factors.

**Case Report**

The patient is a 58-year-old male with no significant past medical history. He initially presented with holocephalic headaches, cough, fever, and general malaise. Chest x-ray showed bilateral patchy infiltrates. Real-time reverse transcription polymerase chain reaction (RT-PCR) was positive for coronavirus 2 RNA. Blood test was significant for elevated C reactive protein (7.35), ferritin (602), and d-dimer (590). He was treated with acetaminophen, dexamethasone, remdesivir, and supplemental oxygen via nasal cannula. He was also started on prophylactic doses of Lovenox to prevent deep venous thrombosis. On day 10 of hospitalization, the patient woke up with acute onset of left sided weakness and numbness. Initial National Institutes of Health Stroke Scale (NIHSS) score was 2. Neurological examination showed left arm pronator drift and decreased sensation in the left upper and lower extremities. He denied recent trauma or chiropractic neck manipulation. A computed tomography (CT) of the head did not show any hemorrhage or early ischemic signs. Given last-known-well time at least five hours ago and mild neurologic deficits, tissue plasminogen activator (tPA) was not indicated. Stat Magnetic Resonance Imaging (MRI) of the brain showed scattered acute ischemic stroke in the right centrum semiovale and right posterior parietal/occipital lobe (Figure, A). Stroke workup showed total cholesterol 130, high density lipoprotein 26, low density lipoprotein 84, glycosylated hemoglobin 6.4%. Electrocardiogram (ECG) and telemetry showed normal sinus rhythm. A transesophageal echocardiogram showed a left ventricular ejection fraction of 61% with a negative bubble study.

Approximately 8 hours after the symptom onset, his left arm muscle strength deteriorated to 3/5 along with decreased sensation in his left face, arm, and leg. A repeat MRI of the brain showed interval worsening of infarction in the right posterior parietal/occipital lobes (Figure, B). A CT Angiography of the head and neck revealed an intraluminal eccentric filling defect in the proximal/mid right common carotid artery, consistent with an acute thrombus (Figure, C & D). No arterial dissection or atherosclerotic disease was noted. Given no large vessel occlusion and relatively mild deficit, endovascular thrombectomy or carotid endarterectomy was not indicated. The patient was started on anticoagulation with Apixaban 5mg twice a day for stroke prevention and Atorvastatin 20 mg daily for mild dyslipidemia. He received physical and occupational therapy and improved significantly with only had mild left arm pronator drift and paresthesia in his left face and arm at hospital discharge.

 The patient had clinic follow-up 2 weeks after stroke. His left arm weakness resolved completely but still felt numb in left face and arm. At 4-month follow-up, he only had residual left arm numbness. He denied recurrent symptoms or signs of stroke. His private neurologist changed his Apixaban to Aspirin 81 mg daily.

**Discussion**

The patient developed embolic strokes from common carotid artery thrombus after COVID 19 infection. He had no history of trauma, chiropractic neck manipulation, or vascular risk factors. CTA showed no arterial dissection or underlying atherosclerotic stenosis. The intraluminal thrombus was in common carotid artery, not a typical location for atherosclerotic disease. Blood test was significant for elevated levels of COVID-19 related inflammatory biomarkers and di-dimer. Cardiac workup was negative. Therefore, the etiology of the common carotid thrombosis and stroke was likely COVID-19 related inflammation and hypercoagulable state.

 In a case series of 6 patients with COVID-19 and embolic stroke from intraluminal thrombus in the internal carotid artery, all was found to have vascular risk factors and elevated inflammatory biomarkers.[3] COVID-19 related inflammation may result in the rupture of vulnerable atherosclerotic plaques, resulting in thrombosis and acute ischemic stroke in these patients.[3,5] A large meta-analysis found that 7.39% of COVID-related strokes were atherothrombotic in etiology.[7]

 Currently, there is no evidence-based proven therapy for COVID 19 related thrombosis and stroke. There have been case reports of treatment with intravenous tPA, thrombectomy, or endarterectomy followed by antiplatelet or anticoagulation.[1-4]Large thrombus is often associated with higher risk of complete artery occlusion or distal embolization in middle cerebral artery. Compared to other reported cases with carotid thrombus and severe stroke (NIHSS 13 to 23),[1-3]our patient had substantially lower NIHSS score and was outside the time window for iv tPA. He was therefore only treated with oral anticoagulation for stroke prevention. In the seminar case report published in New England Journal of Medicine, there was complete resolution of the thrombus following 10 day's anticoagulation therapy in the 5 patients with COVID 19 related large vessel thrombosis.[1] Since the thrombosis was likely provoked by COVID-19 infection, it is therefore reasonable to consider 3-6 months of anticoagulation therapy for stroke prevention.

**Conclusion**

Our case report suggests that COVID 19 induced inflammation cascade and hypercoagulable state may cause common carotid artery thrombus and embolic stroke. For patients with a minor stroke who are not eligible for intravenous thrombolysis, oral anticoagulation may be a reasonable treatment of choice for long-term stroke prevention.

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