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Recurrent Transient Ischemic Attack in a Young Patient with COVID-19

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Dear Editor,

The novel coronavirus SARS-CoV-2 (which results in COVID-19) was recently discovered in Wuhan, China. This virus has a predominant tropism for the respiratory system. Increasing evidence for the concomitant involvement of the central nervous system has emerged from reports of numerous cases of neurological manifestations during COVID-19, including acute cerebrovascular diseases, impaired consciousness, and skeletal muscle injury.[1] SARS-CoV-2 has been found in the cerebrospinal fluid, suggesting that the virus can cause encephalitis.[2] The exact correlation between cerebrovascular manifestations and virus infection is currently unclear, although hypoxia together with respiratory and metabolic acidosis induced by severe pneumonia could play a role.[3]

We describe the case of a 47-year-old male admitted to hospital with a 10-day history of fever and the subsequent appearance of dyspnea. He had a medical history of allergic asthma. Chest CT detected multiple areas of focal parenchymal thickening with irregular margins, with a partial ground-glass appearance in both lungs, especially in the lower lobes. Arterial gas analysis showed 91% oxygen saturation. The D-dimer level was 1,084 ng/mL (normal range <500 ng/mL), and the reactive C-protein level was 4.12 mg/dL (normal range <1 mg/dL). A nasopharyngeal swab was positive for SARS-CoV-2 in a real-time reverse transcriptase–polymerase chain reaction assay. Therapy with opinavir/ritonavir (100/25 mg daily), hydroxychloroquine (200 mg twice daily), azithromycin (500 mg daily), and ceftriaxone (2 g daily) was started, which resulted in the resolution of respiratory symptoms.

Five days after hospitalization, the patient experienced a transient episode of paresthesia in the left hand and loss of vision (amaurosis fugax), which lasted 5 minutes. The next day he experienced two similar episodes of longer durations (15 and 30 minutes), with the last one including weakness of the left hand. A neurological examination revealed weakness and hypesthesia in the left upper limb with ataxic oscillations. The findings of brain CT were normal. Brain CT angiography revealed the presence of a hypodensity compatible with thrombotic formation at the origin of the right internal carotid artery. The intracranial arteries were normal. Carotid echo color Doppler sonography (ECD) revealed 65% stenosis of the right carotid artery (peak systolic velocity of 188 cm/sec) caused by a mixed plaque. Considering the presence of concomitant pneumonia, surgery was deferred. A diagnosis of transient ischemic attack (TIA) was made, and therapy was started with acetylsalicylic acid (300 mg daily) and enoxaparin (4,000 UI daily). Statins were not introduced due to their interactions with antiviral drugs. The patient did not present with new symptoms over the following several days.

After 2 weeks, control carotid ECD showed bilateral calcification causing 30% stenosis of the carotid artery with a smooth margin, and the thrombotic component in the right carotid artery was no longer evident. The patient was discharged on acetylsalicylic acid (100 mg daily) and atorvastatin (40 mg daily).

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Stroke and other thrombotic events such as acute myocardial infarction and disseminated intravascular coagulation have previously been reported in association with severe acute respiratory syndrome as a consequence of a hypercoagulable state.[4] It is reported that coagulation parameters are altered in patients with COVID-19, so that routine hemostasis tests may be additional useful tools for improving the ability to perform early diagnoses.[5] Severe infection may be a trigger of acute ischemic stroke via the involvement of intravascular events as well as hypoxia.

It was particularly interesting that, unlike previously described stroke patients, our case did not present risk factors for stroke. It is likely that the inflammation cascade is responsible for the thrombotic apposition in a small calcific stenosis of the carotid artery, causing neurological manifestations.

Prophylactic anticoagulant treatment is increasingly being prescribed in COVID-19 patients due to concerns about frequent and severe venous thromboembolic events.[6] In the present patient, antithrombotic therapy (which is effective against arterial thrombotic disease in both the coronary and cerebral districts) paralleled the reduction in the borderline-significant carotid stenosis, thereby avoiding the risks inherent in surgery when severe pneumonia is present. We emphasize that a nonaggressive approach with medical therapy alone may be an appropriate choice in the presence of COVID-19 pneumonia and TIA due to carotid atherothrombotic disease. Performing an ECD follow-up after 2–4 weeks is important, which in the present case revealed rapid modification of the atherosclerotic plaque.

Author Contributions

Conceptualization: Vittorio Mantero. Data curation: Vittorio Mantero, Paola Basilico, Gisella Costantino, Ugo Pozzetti, Andrea Rigamonti. Writing—original draft: all authors. Writing—review & editing: Andrea Salmaggi.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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