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Case Report

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A Case with the Left Common Carotid Artery Involvement as Large Vessel Vasculitis after COVID-19

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ABSTRACT

It is known that there are many other organ involvements in the coronavirus disease of 2019 (COVID-19) apart from respiratory tract involvement, and the disease may occur in different clinical spectrums in different patients. One of the most important structures affected by the virus is the vascular structure. It creates a tendency to thrombosis, especially by causing damage to the vascular endothelium and activating the coagulation system and may lead to important complications. It is known that the virus, which especially affects venous and small-sized arterial vessels, also affects medium-sized vessels. However, large-sized arterial vascular involvement is very rare. In this case report, it was aimed to present the left common carotid artery vasculitis, together with radiological appearence and clinical features, in a patient who presented with the complaint of left anterior neck pain in 4 weeks after COVID-19 infection.

Keywords: Carotid artery, COVID-19, vasculitis

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INTRODUCTION

Coronavirus disease of 2019 (COVID-19) disease, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which entered our lives in late 2019, involves many different systems and organs with different mechanisms.[1,2] Although the underlying mechanisms are not fully known yet, cytotoxic damages, autoimmune reactions, and vascular pathologies caused by the virus are generally held responsible for most of the pathologies. In addition, it is known that the virus causes thrombotic events by activating coagulation mechanisms.[2,3] Deep vein thrombosis in the lower extremities, which it is frequently seen as a result of coagulation activation during COVID-19 infection, is an important cause of morbidity encountered in some patients.[3] It may also cause mortality by affecting the pulmonary vessels causing pulmonary thromboembolism; myocardial infarction, and cardiomyopathies as a result of coronary vasculitis; or neurological manifestations as a result of direct neurotoxic and vasculitic damage in the brain vessels.[2] It is an accepted view that some autoimmune and rheumatological events are triggered as a result of intense immune reactions caused by the virus.[4] It has been shown that the incidence of autoimmune diseases such as subacute thyroiditis, Guillain-Barré syndrome, and systemic lupus erythematosus increases after COVID-19 infection. It has also been reported that the frequency of giant cell arteritis increases after COVID-19.[5] Although it usually affects small and medium-sized vessels, in this case report, it was aimed to share the ultrasonography and clinical findings of a case with the involvement of the left common carotid artery (CCA) as a large vessel involvement COVID-19.

CASE REPORT

A 44-year-old male patient with no accompanying comorbid disease was admitted to the internal medicine outpatient clinic of our hospital due to anterior left neck pain unrelated to the movement for the past 3 days. In his clinical history, it was learned that the patient had COVID-19 pneumonia with pneumonia with predominantly cough, weakness, and fever symptoms 1 month ago. Low-dose non-contrast thorax computed tomography revealed infiltrates in the form of ground-glass densities and crazy-paving patterns with a predominantly peripheralobasal distribution in both lungs (Fig. 1). The patient, whose complaints completely regressed with medical treatment and whose last polymerase chain reaction turned negative 2 weeks ago, had newly developed severe anterior neck pain for the past 3 days. On physical examination, no significant pathological finding was detected in the neck region. All laboratory parameters of the patient, except erythrocyte sedimentation rate (ESR), were within the normal reference range (Table 1). Neck ultrasonography was performed with the preliminary diagnosis of possible space-occupying pathologies, cervical lymphadenopathy (LAP), and subacute thyroiditis in terms of pain etiology. Thyroid and salivary glands were normal in ultrasonography, and no parenchymal disease or demarking lesion was detected. No pathological LAP was detected in both cervical chains. However, diffuse wall thickening reaching 4.5 mm was noted on the anterior wall of the left CCA just before the bifurcation (Fig. 2). The neck ultrasonography of the case previously performed for check-up was completely normal, and this finding was thought to have developed newly. In the case with isolated left neck pain, the cause of the pain was evaluated as large-vessel vasculitis affecting the left CCA after COVID-19. Dexamethasone 4 mg and rivaroxaban 20 mg were started as treatment. Within a few days, the patient's complaints regressed rapidly, and ESR returned to normal in control laboratory tests. In the control ultrasonography examination performed 2 weeks later, the left CCA wall thickness was within normal limits (<1 mm).

DISCUSSION

In this case report, a case of COVID-19 infection, which was considered to be compatible with isolated unilateral large vessel vasculitis after a latent period, is presented together with its radiological and clinical features. SARS-CoV-2 is a virus with high vascular affinity. It has the potential to affect both arterial and venous vessels. Venous pathologies are mostly encountered in the form of endothelial damage as a result of intense immune reactions that occur in the

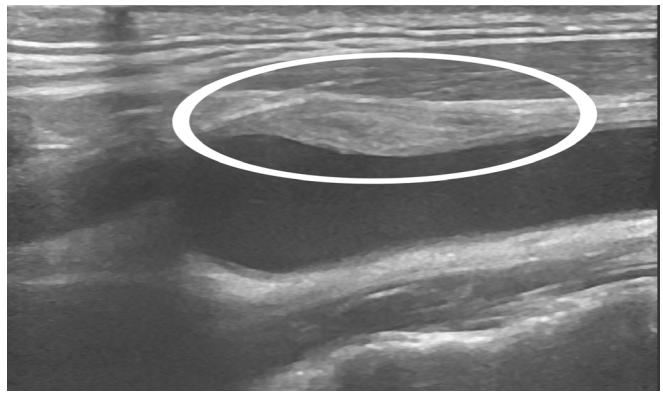

Figure 1. Regular anterior wall in the left CCA distal extending to the level of the bulb just before the bifurcation and reaching 4.5 mm at its thickest level.

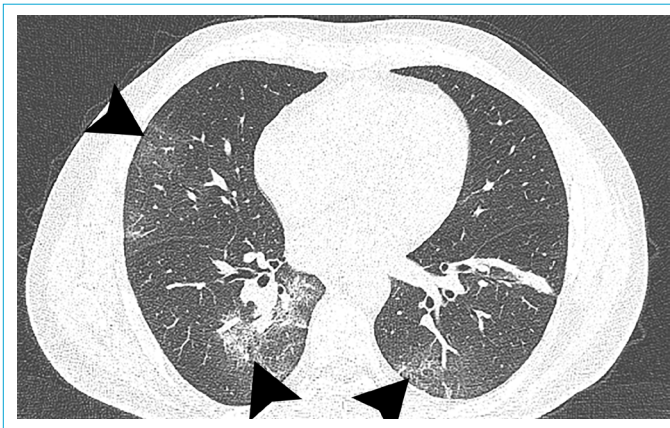

Figure 2. Bilateral peripherobasal dominantly infiltrations in the form of ground-glass and crazy-paving pattern in the low-dose thoracic CT scan without contrast, taken during the patient's COVID-19 pneumonia.

Table 1. Laboratory parameters of the patient

	Patient's Values	Reference Range
WBC (×10³/uL)	6	4–10
Hemoglobin (g/dL)	12.5	12–15
Platelets (×10³/uL)	323	150–450
Neutrophil (%)	63	40–70
Lymphocyte (%)	33	20–50
CRP (mg/dL)	0.33	<0.5
ESR (mm/h)	24	<20
TSH (uIU/mL)	0.897	0.27–4.2
Anti-TPO (IU/mL)	24	0–60

CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; TPO: Thyroid peroxidase; TSH: Thyroid stimulating hormone; WBC: White blood cells.

disease and predisposition to thrombosis in the early period when the virus activates the coagulation system.[3,6-8] On the other hand, arterial pathologies are thought to occur through immune mechanisms, usually triggering autoimmune reactions after a latent period. It has been reported that there is an increase in the frequency of giant cell arteritis during the COVID-19 pandemic, which mostly affects small and medium-sized arterial structures.[8,9] In these cases, the clinical outcomes of both giant cell arteritis and COVID-19 releated arteritis are similar.[10] Headache, vision problems, and serious neurological complications can be encountered in both cases.[8,10] Therefore, for the fast and accurate diagnosis of these two entities, which can cause significant morbidity and mortality, these two clinical conditions must be considered, and their differential diagnosis must be made correctly. Although there are not many reports on large vessel involvement due to the virus, a recent study showed that the SARS-CoV-2 virus infects the carotid arteries, and a large amount of SARS-CoV-2 RNA was isolated in the histopathological examination of the carotid arteries in the autopsy series of 32 COVID-19 patients.[11] The most important feature of inflammatory and autoimmune vasculopathy is the formation of wall thickening as a result of the accumulation of immunocomplexes in the subendothelial area and vessel wall without atherosclerotic changes.[12] In our case, only diffuse wall thickening was detected without any signs of atherosclerotic intimal irregularity in other arterial vascular structures and without accompanying plaque formation. Our major limitation is the lack of histopathological diagnosis in this case. However, in the patient whose sedimentation levels are high during the active complaint period, the rapid response to steroid treatment and the complete normalization of the carotid artery in the control ultrasonography strongly support our diagnosis. In addition, obtaining the SARS-CoV-2 virus loaded in the carotid artery walls in the studies of Pfefferle et al. is an important study supporting our case. The most important feature that distinguishes this case report from other similar reports is that unilateral isolated large artery involvement that responds rapidly to steroid treatment.[11]

CONCLUSION

It is important to always keep in mind the possibility of arterial vascular involvement in patients presenting with focal pain during or after COVID-19 infection to prevent serious morbidity and mortality.

Disclosures

Informed Consent: Written informed consent was obtained from the patient for his anonymized information to be published in this article.

Conflicts of Interest: The author declares that there are no conflicts of interest.

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