

Case Report

Vascular involvement in SARS-CoV-2: A case report

Ilkeli E*

Department of Cardiovascular surgery Duzce State Hospital, Duzce, Turkey

***Corresponding author:** Ekin Ilkeli*, Department of Cardiovascular surgery Duzce State Hospital, Duzce, Turkey**Received:** November 17, 2020**Accepted:** December 09, 2020**Published:** December 10, 2020**Abstract**

It is known that coronavirus disease 2019 (SARS-CoV-2) predisposes to thrombotic disease in both venous and arterial circulation. In a 47-year-old covid patient with no cardiovascular, diabetes, hypertension, coagulation or smoking history, 3 arterial involvement was detected. We present as cases of thrombus formations occurring simultaneously in the aorta, brachial and carotid artery.

Keywords: Viral infection; cardiovascular; public health**Introduction**

Coronavirus disease (SARS-CoV-2) associated arterial thrombosis is quite new and limited in the literature. Acute embolism or thrombosis is always a serious complication that can result in significant morbidity and mortality [1]. Hypercoagulation associated with SARS-CoV-2 occurs for many reasons; it is a chain of mechanisms based on extensive endothelial inflammation and a high inflammatory state associated with cytokine storm [2,3]. Over time, the importance of SARS-CoV-2 coagulation abnormalities becomes more evident. We noticed that 3 different arterial involvements at the same time are rare in the literature. We present as cases of thrombus formations occurring simultaneously in the aorta, brachial and carotid artery.

Case Presentation

A 47-year-old man admitted to emergency service with right upper extremity pain. A 47-year-old male patient was admitted to the emergency department with right upper extremity pain.

He had mild dry cough and fatigue that started 6 days ago. The patient had no history of cardiovascular disease, diabetes, hypertension, and smoking. On examination, right upper extremity did not have brachial, ulnar and radial pulses.

Platelets were 77,000, D-dimer level was 3200 mg/dl, INR 1.02. APTT levels were normal. CRP was 9.2 mg/L and erythrocyte sedimentation rate was 85 mm/hour. The patient had no history of trauma. Echocardiography and electrocardiography were normal. Chest CT and CT angiography was performed.

Plaque/thrombus was visualized in the aortic arch on CT angiography (Figure 1A). CT angiography scans also revealed right common carotid artery thrombosis (Figure 1B), right brachial artery thrombosis (Figure 1C), and bilateral confluent and consolidative pulmonary opacities (Figure 1D), interestingly, the patient had no ischemic cranial symptoms.

Computed tomography showed normal right axillary and subclavian arteries (Figure 2).

Chest CT scanning revealed bilateral multilobes pneumonia, throat and nasopharyngeal swabs were taken and SARS-CoV-2 was detected by reverse transcription polymerase chain reaction (RT-PCR) (BioGerm) test. He underwent urgent brachial artery embolectomy operation under local anesthesia successfully, no further intervention was planned for carotid artery thrombosis. The patient took medications of favipiravir, azithromycin and low molecular weight heparin for Covid 19 for 7 days and discharged with recovery with the same treatment regimen.

Discussion

The mechanism of thromboembolic complications that develop after SARS-CoV-2 disease is not yet fully understood. Cytokine storm and endothelial damage are the most likely causes of coagulation associated with SARS-CoV-2. In this infection, it can cause serious respiratory dysfunction with various complications such as cardiovascular disorders,

kidney dysfunction, cerebral events and shock. Endothelial dysfunction and coagulopathy in SARS-CoV-2 infection are associated with high mortality especially at patients with high D-dimer levels [4]. Vascular thrombotic complications had been reported in critically ill SARS-CoV-2 patients who admitted to intensive care unit [5]. High plasma levels of proinflammatory cytokines as interleukin-2, interleukin-7, tumor necrosis factor α have been observed in SARS-CoV-2 patients. This is consistent with a “cytokine storm” with secondary development of hemophagocytic lymphohistiocytosis [6]. This high cytokine activity may trigger the coagulation system and may be the possible pathophysiology of thromboembolic complication. High D-dimer level is an indication of increased coagulation activation and hyperfibrinolysis. D-dimer are often used to detect active thrombus with high sensitivity but low specificity [7]. Prothrombin time and D-dimer appear to be related to the severity of the disease. Coagulation tests are important in the diagnosis of disseminated intravascular coagulation in SARS-CoV-2 patients. As shown in influenza pneumonias, there is an increased risk of developing pulmonary embolism in SARS-CoV-2 pneumonia, and the presence of significant hypoxemia despite the conserved compliance may be due to the presence of coagums in the pulmonary vessels. There is not enough data about arterial thrombosis in SARS-CoV-2 patients. We detected arterial thrombosis in the extremities. We observed thrombosis on the aortic plaque. Arterial thrombosis has been reported to develop despite adequate antiaggregant and anticoagulant therapy [8,9]. Ischemia caused by thrombosis may be irreversible. We saved the limb from amputation by intervening early.

Therefore, prophylaxis with low molecular weight heparin is known to patients who are being followed up with the diagnosis of SARS-COV-2.

In this paper we presented multiple arterial thrombosis in a patient who is SARS-CoV-2 but not critically ill and was admitted with evident thrombotic findings.

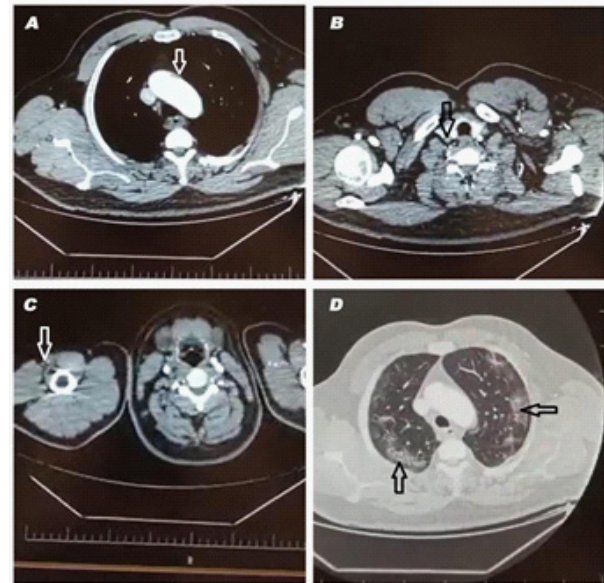


Figure 1: Contrast-enhanced computed tomography (CT) performed on a 48-year-old male. A, Plaque/thrombus at aortic arc (White arrow). B, Right common carotid artery thrombosis (Black arrow). C, Right brachial artery thrombosis (White arrow). D, Bilateral pneumonia as in Covid-19 (White arrows).

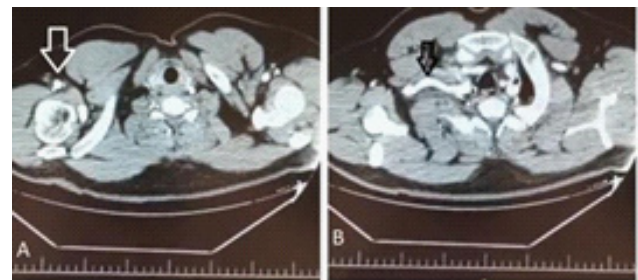


Figure 2: Contrast-enhanced computed tomography (CT). A, Right axillary artery (White arrow). B, right subclavian artery (Black arrow)

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