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Case Report
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Subclavian Artery Thrombus: A Late Complication of COVID-19

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Abstract

Coronavirus Disease 2019 (COVID-19) is a viral respiratory illness caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). Although, it's main consequences are viral pneumonia and respiratory manifestations, both arterial and venous thromboembolic events have also been recognized as potential complications associated with it. Here, we present a case of arterial thrombosis in a young patient with no significant past medical history who had recovered from COVID-19 several months prior to presentation.

Introduction

Coronavirus Disease 2019 (COVID-19), the latest outbreak of infectious disease, is a viral respiratory illness caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [1]. Since its emergence, it has caused enormous medical challenges and an unprecedented global health crisis. Affected individuals mainly present with upper and lower respiratory symptoms in addition to several digestive symptoms [2]. However, it has also being recognized to include different manifestations including myocardial infarction, seizures, meningitis, and coagulopathy. It has been described to predispose to thrombotic disease in both the venous and arterial circulations, which may lead to serious consequences as duration of anticoagulation in these patients remains unclear [3]. However, unlike venous thromboembolism, no sufficient data are available on arterial thrombosis in SARS-CoV-2 infected patients.

Here, we are describing a case of upper extremity arterial thrombosis for a patient who recovered from COVID-19 two months prior to presentation, during which time he had also developed a stroke and subsequently completed over a month of anticoagulation.

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Material and Methods

By submitting this manuscript, a consent is given for the materials described in the manuscript to be freely available to researchers and scientists who intend to use it for non-commercial purpose, without violating participant's confidentiality.

Case Presentation

We present a 36-year-old Hispanic male with no significant past medical history presented with left hand numbness and shortness of breath. He recovered from COVID-19 two months prior to admission during which time he developed a stroke with no residual deficits and completed a five-week course of apixaban. The etiology of his stroke was thought to be related to his COVID-19 illness as workup of stroke at that time was negative.

On examination, the patient was alert, oriented, and vitally stable. His left hand was warm, with no color changes. Pulses and capillary refill in both hands were equal and symmetrical. Neurologic examination was normal.

Due to suspicion of acute pulmonary embolus given his presentation with shortness of breath in the setting of a recent COVID-19 infection, Computed Tomography Angiography (CTA) of the chest with contrast was performed. No central or segmental filling defect to suggest pulmonary embolus was noted; however, it revealed a non-occlusive filling defect within the origin of the left subclavian artery (Figure 1a, 1b).

D-dimer was elevated at 1004 ng/ml (normal range <500 ng/ml). His thombophilic profile including anti-thrombin III level, factor V Leiden mutation, prothrombin gene mutation, protein C and protein S deficiency were all negative. Electrocardiogram showed normal sinus rhythm.

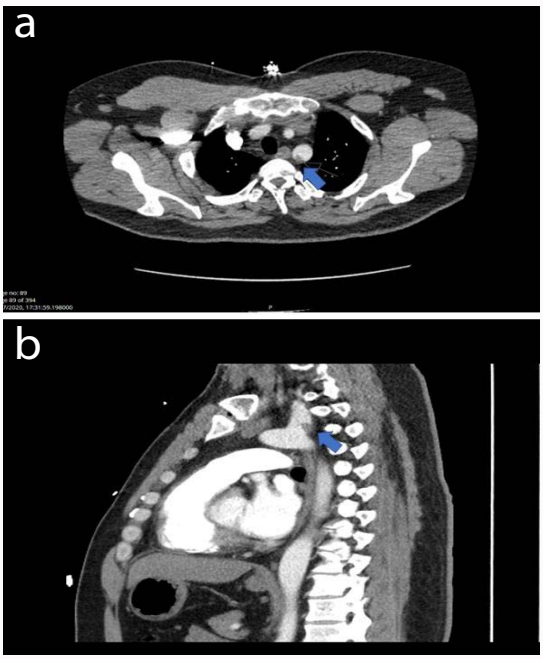

Figure 1a, 1b: CTA of the chest showing non-occlusive filling defect within the origin of the left subclavian artery.

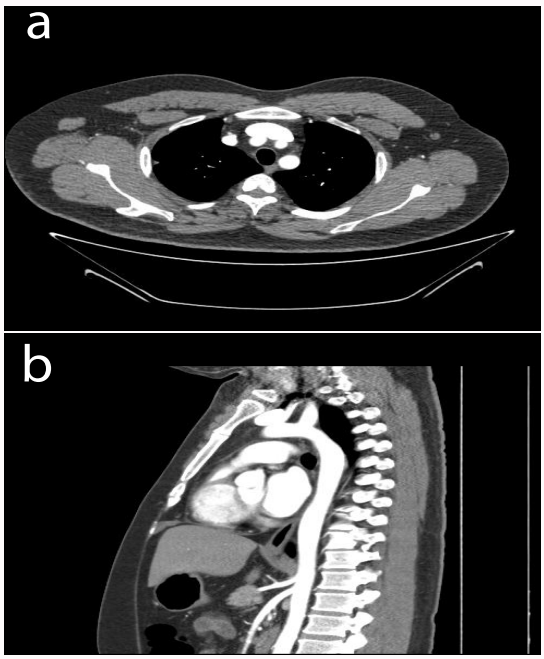

Figure 2a, 2b: CTA of the chest showing resolution of the previously seen filling defect within the origin of the left subclavian artery.

During hospitalization no arrhythmia such as atrial fibrillation was detected on telemetry monitoring. On transthoracic echocardiogram, he did not have any evidence of intra-cardiac thrombus or any intra-cardiac shunt. The patient was started on apixaban and aspirin with significant improvement in his symptoms.

On follow up 4 weeks later, a repeat Computed Tomography Angiogram (CTA) of chest with and without contrast showed interval resolution of the previously seen filling defect within the proximal left subclavian artery (Figure 2a, 2b). The etiology of the subclavian thrombus was thought to be related to his COVID 19 infection that he had recovered from two months prior to admission.

Discussion

Recently, the association between COVID-19 and coagulopathy has gained more interest, mainly due to the increase in number of cases afflicted by it and the fact that hypercoagulability adversely impact prognosis. The reported incidence of various thrombotic events in patients with COVID-19 had a range of 7.7% to 49%, which is significantly higher than the incidence in patients without COVID, with the incidence of venous thrombosis being much higher in comparison to arterial thrombosis [4,5]. There is a substantial pool of evidence of venous thrombotic events that has been well described; however, data on arterial thrombosis in these patients is still limited.

One of the studies reported that arterial thrombosis develops in approximately 4.4% of severe COVID-19 patients [6]. Observed risk factors include older age, male sex, Hispanic ethnicity, history of coronary artery disease and elevated D-dimer levels on presentation [6].

The exact underlying pathophysiology of thromboembolic events in patients with COVID-19 is incompletely understood. However, several contributing factors including vascular endothelial dysfunction, platelet activation, excessive inflammation, hypoxia, immobilization and diffuse intravascular coagulation are thought to play a vital role in their development [7]. Historically, SARS-CoV-1 and MERS-CoV also exhibited similar prothrombotic complications, as well as thrombocytopenia [8]. This theory is backed by the reports of elevated inflammatory and coagulation markers; and a correlation between elevated D-dimers, prothrombin, Interleukin (IL)-6 and fibrinogen levels [7]. Other emerging evidence suggests that COVID-19 is associated with endothelial inflammation which is characterized histologically by diffuse endothelial damage and infiltration by inflammatory cells [9]. Damage to the endothelium could be as a result of direct viral infection, which is facilitated by the overexpression of Angiotensin-Converting Enzyme Receptor 2 (ACE 2), the receptor for cell entry of SARS-CoV-2, in endothelial cells [7,9].

The main dilemma is how to optimally approach management in such high-risk hospitalized patients with COVID-19 in the absence of well conducted trials. Current strategies are influenced by observational reports, case series and empirical institutional protocols [5,7,10].

The current case is an interesting one that demonstrates the occurrence of left subclavian artery thrombus as a late sequela of COVID-19 despite completing 5 weeks of anticoagulation. However, after giving the anticoagulant for another 4 weeks, the thrombus has resolved completely as confirmed by the repeat of CTA. More data is needed to improve the prevention, diagnosis, and treatment of thrombotic complications in these patients.

Conclusion

Thromboembolic complications, specifically arterial thrombosis can be seen months after recovering from COVID-19 infection despite over a month of anticoagulation. Further studies with a focus on arterial thrombotic complications are needed to characterize arterial consequences of coronavirus and to evaluate for a longer-term post discharge thromboprophylaxis for high-risk hospitalized patients with COVID-19 who have a low risk of bleeding.

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