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Case Report

Acute Upper Limb Ischemia in a Patient with COVID-19 Pneumonia

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Abstract

Coronavirus disease 19 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). COVID-19 is primarily apparent with respiratory symptoms [1], but there are increasing reports of cardiovascular [2], hematological [3] and thromboembolic complications [4-8]. We are presenting an uncommon presentation of a 29-yearold previously healthy Bangladeshi male patient who presented with COVID-19 viral pneumonia and also had acute limb ischemia in the left upper extremity which required open thromboembolectomy with endarterectomy.

Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which is an enveloped RNA beta coronavirus. It is a novel coronavirus responsible for the current global pandemic and poses a critical threat to global health worldwide [1]. While SARS-CoV-2 is known to cause significant pulmonary disease, including pneumonia and acute respiratory distress syndrome (ARDS), clinicians have observed many extrapulmonary manifestations of COVID-19 particularly thrombosis [3]. In May 2020, Xiong and colleagues reported meta-analysis that showed prothrombin time and D-dimer levels were significantly higher in patients with severe COVID-19 than in those with the mild disease [4]. Acute limb ischemia is one of the most potentially overwhelming but treatable diseases challenged vascular surgeon. It is crucial to diagnose the ischemic process early, as the outcomes of early intervention can lead to limb saving. However late recognition places the patient at risk for limb loss and potential mortality.

Case Presentation

A 29-year-old Bangladeshi male with negative past medical history admitted to the hospital because of shortness of breath and hypoxemia during the pandemic of coronavirus disease 2019 (Covid-19). The patient had been well until 6 days before this admission, when he started to have fever and cough. In the emergency department, the temperature was 38.8 °C, the heart rate 110 beats per minute, the blood pressure 120/80 mmHg, the respiratory rate 30 breaths per minute, and the oxygen saturation 94% while the patient was receiving supplemental oxygen through a nasal cannula at a rate of 4 liters per minute. On examination, the patient appeared ill and was lethargic. On initial laboratory evaluation, the following values were noted: Hemoglobin 14.1 g/dL (13-17 g/dL), total leukocyte count 5.64 × 10^3 /µL (4-11 × 10^3/µL), absolute neutrophil count 4.51 × 10^3/µL (2-7 × 10^3/µL), absolute lymphocyte count 0.718 × 10^3/µL (1.5-4 × 10^3/µL), platelets 145 × 10^3/µL (150-450 × 10^3/µL), blood urea nitrogen 13.74 mg/dL (8.9-20.6 mg/dL), serum creatinine 0.75 mg/dL (0.7-1.3 mg/dL), prothrombin time 12.40 sec (11.5-15 sec), international normalized ratio 0.92 (0.9- 1.1), partial thromboplatin time 40.80 sec (26-40 sec), D-dimer 0.47 ug/mL (0-0.5 ug/mL), creatine kinase 83 U/L (30-200 U/L), lactate dehydrogenase 383 U/L (125- 243 U/L), ferritin level 1073.20 ng/mL (21.81-274 ng/ mL), C-reactive protein 9.260 mg/dL (< 1).

Nucleic acid testing of a nasopharyngeal swab was negative for influenza A and B viruses and respiratory syncytial virus but was positive for SARS-CoV-2. Chest X-ray showed bilateral patchy airspace opacities. Electrocardiogram showed sinus tachycardia of 115 beats/minute. Acetaminophen and empirical ceftriaxone, azithromycin, and hydroxychloroquine were administered. On the fourth hospital day, the patient reported sudden onset of severe pain in the left arm with cyanosis. Pulsation not felt on Radial/Brachial artery. Heparin infusion started. Diagnosis of acute upper limb ischemia was made and patient was taken for emergency Embolectomy by vascular surgery. One day later, patient

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had intact radioulnar pulse on left side with clips removed and started oral anticoagulants (Warfarin). Echocardiogram done showed - normal LV systolic function with EF 55% - normal LV internal dimensional, no RSWMA - normal mitral valve morphology, no MR - no MS normal aortic valve and mild TR - normal RVSP - no masses, effusion or thrombi. Ten days later, patient discharged from hospital on oral anticoagulants.

Discussion

Coronavirus disease 2019 (COVID-19) can present with a spectrum of clinical manifestations including severe acute respiratory distress, Gastrointestinal symptoms and coagulation dysfunction [3]. COVID-19 predispose to both venous and arterial thromboembolism due to excessive inflammation, hypoxia, and diffuse intravascular coagulation [7]. The incidence of VTE in patients with severe novel coronavirus pneumonia is 25%, which may be related to poor prognosis [5].

Han and colleagues described changes in blood coagulation during severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection with increased values of D-dimer, fibrin or fibrinogen degradation products, and fibrinogen; decreased anti-thrombin values, prothrombin time activity, and thrombin time [6]. Systemic proinflammatory cytokine response is a mediator of atherosclerosis by inducing the expression of procoagulant factors, local inflammation, and hemodynamic alterations and the receptor for SARS-CoV-2 (angiotensin-converting enzyme 2) is expressed on the membrane of vascular muscle and endothelial cells [5,6].

A recently published cohort study from Lombardy, Italy, reported a higher-than-expected incidence of Acute Limb Ischemia (ALI) due to arterial thrombosis in COVID-19 pneumonia patients [2]. Remarkably, mean d-dimer level in that cohort was only 2,200 ng/mL, which is much lower than in this patient. ALI is a limb-threatening thromboembolic event that is considered a surgical emergency. The most common etiology of ALI is cardiac embolization, particularly in patientswithout preexisting peripheral arterial disease [3]. However, a previous retrospective analysis of patients whohad undergone lower extremity revascularization procedures for ALI revealed that 40% had some evidence of a hypercoagulable condition [4].

With increasingly reported severe vascular complications during SARS-CoV-2 infection in young healthy patients, a prospective registry should be established to support an understanding of the prevalence and risk factors of acute limb ischemia with the intention of defining prophylactic and therapeutic protocols in those patients.

In conclusion, we report a COVID-19 patient who developed arterial thrombosis leading to acute ischemia in the left upper extremity. Health care providers should be aware of life-threatening thromboembolic events associated with COVID-19 therefore prompt and appropriate intervention should be undertaken.

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