

Extensive Arterial, Venous and Intracardiac Thrombosis in a Patient After Recovery From COVID-19

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Dear Editor,

Thrombosis is now well-known to be associated with acute COVID-19 infection, however, little is understood about development of thrombosis after recovery from COVID-19. A 61-year-old male with a history of recent COVID-19 infection presented with progressively worsening shortness of breath of one week duration. Two weeks prior to the presentation, the patient tested positive for COVID-19 by nasopharyngeal polymerase chain reaction (PCR). He was asymptomatic at the time and therefore did not meet the criteria for pharmacotherapy for COVID-19 including thromboprophylaxis. On presentation, he was hypoxic requiring ~10 L of supplemental oxygen, tachycardic with heart rate between 110 to 120 bpm, and had low normal blood pressures with systolic blood pressure ranging between 90 to 100 mm Hg. Physical exam was notable for increased work of breathing. COVID-19 PCR returned negative. Laboratory workup was notable for an elevated D-dimer > 30 mg/L (normal: 0-0.5 mg/L).

A CT angiogram (CTA) of the chest showed extensive sub-segmental pulmonary emboli involving the right upper, right lower, and left lower lobes. Sequela of recent COVID-19 infection was notable on lung parenchyma. His CTA also revealed partially adherent aortic thrombus in the distal descending thoracic aorta at risk for distal embolization. A transthoracic echocardiogram (TTE) showed a large, mobile thrombus in the right ventricle without any evidence of right heart strain. The patient was started on full-dose anticoagulation with intravenous heparin.

The patient had no other cardiovascular risk factors other than class 2 obesity (BMI 35.29 kg/m²). Standard thrombophilia and vasculitis workup were unremarkable. Given the current guidelines recommending using an oral vitamin K antagonist for anticoagulation in the presence of intra-cardiac thrombus, we initiated warfarin while on bridging therapy with heparin for a target INR of 2 to 3.¹ The patient was subsequently discharged with close outpatient follow-up. A repeat

TTE and CTA two months later showed complete resolution of right ventricular and aortic thrombus, respectively.

Our case highlights the development of delayed venous, arterial, and endocardial thrombosis in a patient with recent asymptomatic COVID-19 infection without significant cardiovascular or other hypercoagulable risk factors. Recovery from COVID-19 in our patient is supported by a negative COVID-19 PCR and clinical improvement with therapeutic anticoagulation without the need for COVID-19 pharmacotherapy. The patient was successfully treated with warfarin and resolution of thrombosis was confirmed on imaging two months later. Given the thrombosis has successfully resolved and the median age of development of delayed thrombosis appears to be around 80 days from initial COVID-19 infection per few published cases in the medical literature,² we plan to continue anticoagulation for a total of 4 to 6 months for the provoked nature of thrombosis.

Thrombosis is not yet recognized as a sequela of COVID-19 by the Centers for Disease Control and Prevention (CDC) and National Institute for Health (NIH). Continuous research involving pathophysiology, diagnosis, and management is warranted.

Meanwhile, maintaining a high suspicion of a thrombotic process in patients with history of COVID-19 is the key to early diagnosis and improving patient outcomes.

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