

Arterial Thrombosis in an Ambulatory COVID-19 Patient in Recovery

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Abstract

Arterial and venous thrombotic events have increasingly been reported in critically ill patients with COVID-19, however these events are not known to occur in patients who have since recovered from active infection. This is a case report of an ambulatory patient with a two-week history of COVID-19 infection with resolved respiratory symptoms who presents with acute right lower extremity ischemia secondary to occlusive thrombus in the popliteal artery. During his hospitalization, CT angiography, transthoracic echocardiogram, telemetry and hypercoagulable work-up excluded all other thrombosis resources and his previous COVID-19 infection was noted to be the only possible predisposing factor for this arterial thrombosis event.

Keywords: COVID-19; Arterial thrombosis

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Case Report

We describe an ambulatory patient with a two-week history of COVID-19 infection with resolved respiratory symptoms who presents with acute right lower extremity ischemia secondary to occlusive thrombus in the popliteal artery. The patient is a 52-year-old male with a past medical history of prediabetes and hypercholesterolemia who presented to the emergency department complaining of right lower extremity numbness, tingling, pain and coolness to the touch for the past five days with difficulty walking. He denied any recent trauma, history of circulatory issues, smoking history or prior thrombotic events. He had absence of any respiratory or gastrointestinal symptoms. Physical examination demonstrated a palpable right lower extremity femoral pulse and absence of doppler detected signals in the dorsalispedis, posterior tibial and popliteal regions. His motor and sensory function were fully intact.

Right lower extremity arterial duplex at the time of presentation displayed completely occlusive acute thrombus present in the popliteal artery with minimal collateral flow distally. Unfractionated intravenous heparin was immediately administered and the patient was brought to the angiography suite for emergent angiography and catheter-directed thrombolysis.

After securing contralateral percutaneous femoral artery access, aortography with run-off was obtained demonstrating absence of proximal atherosclerotic or thrombotic disease. From the level of the above knee popliteal artery down to the distal tibial vessels

there was occlusive thrombus present with minimal collateral flow. Catheter-directed thrombolysis with tissue plasminogen activator (tPA) was initiated after insertion of an ultrasound-accelerated thrombolytic catheter within the popliteal artery.

His foot pain and temperature gradually improved and he returned to the angiography suite the next day for follow-up angiography, which demonstrated the popliteal artery had less than twenty percent residual thrombus. The proximal tibial vessels were now patent, however significant distal tibial residual thrombus was present. Given the patient's high fibrinogen levels, as well as stability of his neurovascular exam, it was felt he would benefit from an additional duration of catheter-directed thrombolysis and a new thrombolysis catheter was inserted into the popliteal artery level.

On admission day 3, the follow up arteriogram showed widely patent right common femoral, deep femoral, superficial femoral and popliteal arteries. The anterior tibial was widely patent proximally however it had occlusive thrombus present in its mid segment with distal reconstitution the thrombus present in the dorsalispedis. The tibioperoneal trunk and peroneal arteries were widely patent. The posterior tibial artery was widely patent until the level of the ankle and was occluded with thrombus distally.

Table 1. Hypercoagulable laboratory findings.

Labs	Value
White-cell count (x10 ³ /mCL)	7.8
Differential count	
Neutrophils (percentage)	66.7
Lymphocyte (percentage)	20.5
Monocyte (percentage)	11.8
Hemoglobin (g/dL)	15.7
Platelet count (x10 ³ /mCL)	303
Albumin (g/dL)	3.8
Alanine aminotransferase (U/liter)	75
Aspartate aminotransferase (U/liter)	49
Creatinine (mg/dL)	0.97
EGFR (mL/min/1.73 m ²)	81
Blood urea nitrogen (mg/dL)	15
Lactate dehydrogenase (U/liter)	386
D-dimer (mcg/mL)	2.33
Prothrombin time (sec)	14.8
Activated partial-thromboplastin time (sec)	31.7
Fibrinogen (mg/dL)	475
Anti FactorXa (IU/mL)	0.68
Serum ferritin (ng/mL)	503.8
Procalcitonin (ng/mL)	0.09
C-reactive protein (mg/dL)	6.93
Factor V Leiden	negative
Homocysteine (Umol/L)	7.76
ANA titer	<1:80
Antithrombin III (percentage)	62
Activated protein C resistance	2.4
Protein C (percentage)	105
Protein S (percentage)	46
Anticardiolipin antibodies	IgM and IgG elevated

Aspiration and mechanical thrombectomy were performed at the level of anterior tibial, dorsalispedis, distal posterior tibial and plantar arteries. Completion angiography demonstrated complete absence of thrombus with patency of all distal vessels. On admission day 4, his physical examination had improved significantly with a now palpable pedal pulses and resolution of pain and a hypercoagulable work-up was initiated (Table 1). He was discharged home on hospital day 5 on apixaban.

During his hospitalization, the source of the thrombosis was investigated with cardiac and aortic imaging as well as serologic testing. CT angiography of the chest, abdomen, and pelvis demonstrated no evidence of aortic aneurysm, thrombus formation, or active pneumonia. A transthoracic echocardiogram did not reveal thrombus or valvular disease and telemetry did not reveal arrhythmias. His ferritin, c-reactive protein, and lactate dehydrogenase levels were all elevated, however these findings may be explained by infection and an inflammatory response.

Arterial and venous thrombotic events have increasingly been reported in critically ill patients with COVID-19, [1-7] however these events are not known to occur in patients who have since recovered from active infection.

References

- 1 Xie Y, Wang X, Yang P, Zhang S (2020) COVID-19 complicated by acute pulmonary embolism. *Radiol: Cardiothorac Imaging* 2: e200067.
- 2 Klok FA, Kruijff MJ, Van der Meer NJ, Arbous MS, Gommers DA, et al. (2020) Incidence of thrombotic complications in critically ill ICU patients with COVID-19. *Thromb Res* 191: 145-147.
- 3 Chan KH, Slim J, Shaaban HS (2020) Pulmonary embolism and increased levels of d-dimer in patients with coronavirus disease. *Emerg Infect Dis* 26: 2532-2533.
- 4 Griffin DO, Jensen A, Khan M, Chin J, Chin K, et al. (2020) Pulmonary embolism and increased levels of d-dimer in patients with coronavirus disease. *Emerg Infect Dis* 26: 1941-1943.
- 5 Koleilat I, Galen B, Choinski K, Hatch AN, Jones DB, et al. (2020) Clinical characteristics of acute lower extremity deep venous thrombosis diagnosed by duplex in patients hospitalized for coronavirus disease 2019. *J Vasc Surg Venous Lymphat Disord*.
- 6 Soumagne T, Lascarrou JB, Hraiech S, Horlait G, Higny J, et al. (2020) Factors associated with pulmonary embolism among coronavirus disease 2019 acute respiratory distress syndrome: a multicenter study among 375 patients. *Crit Care Explor* 2: e0166.
- 7 Thachil J, Srivastava A (2020) SARS-2 coronavirus-associated hemostatic lung abnormality in covid-19: is it pulmonary thrombosis or pulmonary embolism? *Semin Thromb Hemost* 46: 777-780.