A 64 year old man weighing 95 kg was admitted from the emergency department to the intensive care unit in April 2020 with coronavirus disease 2019 (COVID-19). The patient’s D-dimer was greater than 1,000 ng/mL. Platelets and renal function were normal. Deep venous thrombosis (DVT) prophylaxis with subcutaneous enoxaparin 30 mg was given twice daily on admission and empirically increased to 100 mg twice daily on day seven due to worsening hypoxemia. Due to agitation and confusion, the patient underwent a computerized tomography (CT) scan of his head, which showed multiple areas of acute to subacute infarction (Image A). Abdominal distension prompted a CT scan of the patient’s abdomen which showed renal infarction involving the medial and posterior midpole of his left kidney (Image B). He was then switched from enoxaparin to intravenous heparin, which was maintained in therapeutic range (activated partial thromboplastin time, 50–90 seconds) using weight based hospital protocol for dosing. Over the course of several days, the patient lost posterior tibialis and dorsalis pedis pulses on his left side. Arterial ultrasound revealed an acute nonocclusive arterial thrombus in the left tibioperoneal trunk (Image C). Unfortunately, life support for this patient was withdrawn after 38 days in the hospital due to worsening multiorgan failure.

COVID-19 has been associated with both arterial and venous thrombosis in several cohort studies [1–3]. Proposed mechanisms for hypercoagulability in COVID-19 patients include direct endothelial injury leading to a
thromboinflammatory feedback loop and ultimately formation of thrombi [4]. This case illustrates the importance of clinical examination of peripheral pulses in COVID-19 patients, timely imaging when clinically indicated, and the potential need for escalation of anticoagulant therapy when clots are found. Somatic dysfunction in thoracic and lumbar regions may also be seen in association with DVT [5].

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**References**