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Pulmonary, Cerebral, and Renal Thromboembolic Disease Associated with COVID-19 Infection

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An 84-year-old man with a past medical history of hypertension was brought to the emergency department in respiratory distress after being found at home with oxygen saturation of 40%. The patient reported fever, shortness of breath, cough, and abdominal pain for 2 weeks leading up to presentation. On examination, he was found to have a nonreactive, pinpoint left pupil and new onset atrial fibrillation with rapid ventricular response. Laboratory analysis revealed lymphopenia of $0.29 \times 10^3/\mu\text{L}$, procalcitonin of 0.25 ng/mL, elevated D-dimer of 21.6 mcg/mL, and elevated troponin T of 35 ng/mL. Reverse transcription polymerase chain reaction (RT-PCR) COVID-19 testing was positive. As his respiratory status deteriorated, the patient was expeditiously intubated.

CT of the chest performed on the night of admission revealed diffuse ground-glass opacities and bibasilar consolidations compatible with severe COVID-19 pneumonia, as described in a recent consensus statement (1). In addition, chest CT demonstrated bilateral lobar pulmonary emboli (Fig 1). The patient was started on a low-molecular-weight heparin infusion. CT of the abdomen and pelvis, performed concurrently with the chest CT, demonstrated a new left renal infarct (Fig 2).

Given the patient's unequal pupils, CT of the head and CT angiography of the head and neck were obtained on the night of admission, revealing occlusion of the distal basilar artery, which extended into the proximal posterior cerebral arteries (Fig 3). There was a small thrombus in the aortic arch (Fig 2). The patient was taken emergently for catheter angiography, followed by mechanical thrombectomy with subsequent restoration of blood flow (Fig 3). Despite all treatment measures, the patient rapidly decompensated and succumbed to his illness the next day.

This case illustrates severe coagulopathy in a patient with COVID-19 pneumonia. Possible explanations of his multifocal thromboembolic disease involving the pulmonary, cerebral, and renal circulations include coagulopathy due to COVID-19 versus cardioembolic etiology in the setting of atrial fibrillation. Given that the patient had no known hypercoagulable conditions and no prior history of atrial fibrillation, the latter etiology was felt to be less likely.

There are emerging global reports of coagulopathy in the setting of COVID-19, including pulmonary emboli, cerebral infarcts, and limb ischemia (2-4). A recent publication identified antiphospholipid antibodies in a COVID-19 patient with significant coagulopathy (5). It has also been proposed that coagulopathy may portend a poor prognosis in COVID-19 patients and may require early intervention (6-7). Our case supports the growing body of data by demonstrating a poor outcome in a patient with multifocal thromboembolic disease in the setting of COVID-19.

Figures

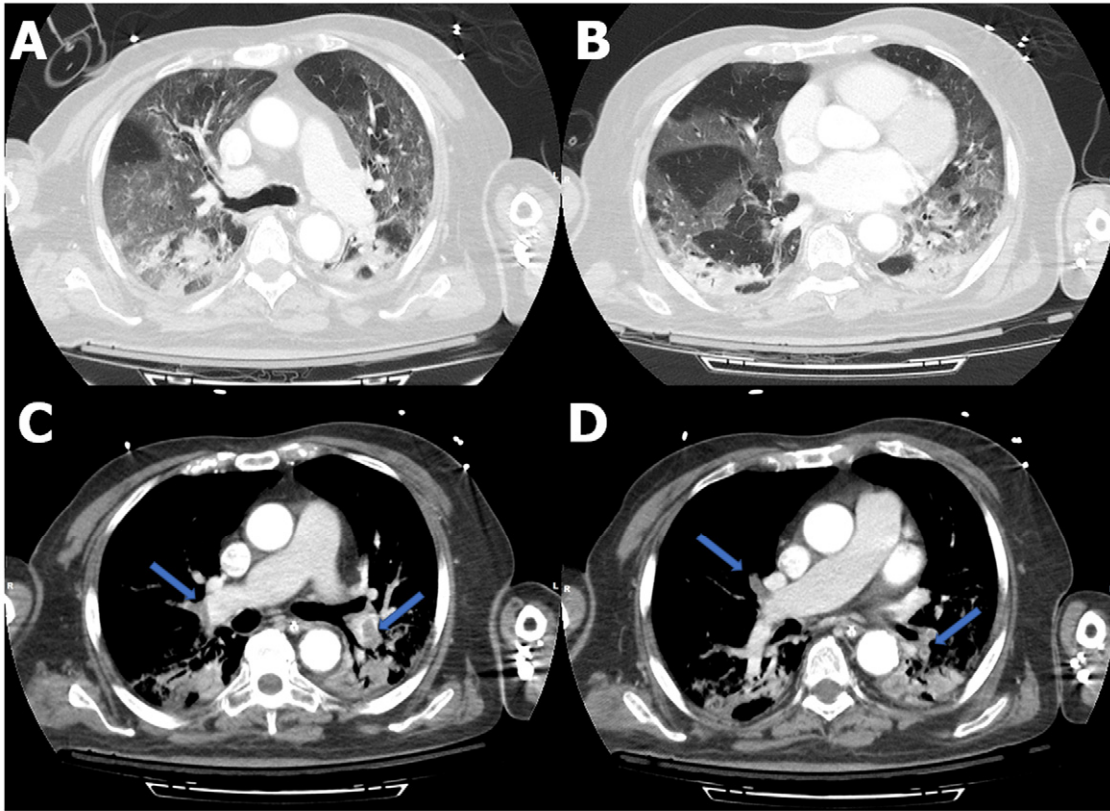


Figure 1. Axial contrast-enhanced CT of the chest. *A, B*, Diffuse bilateral ground-glass opacities and small bibasilar consolidations compatible with COVID-19 pneumonia. *C, D*, Filling defects consistent with pulmonary emboli within the right upper lobe, right middle lobe, right lower lobe, and left lower lobe pulmonary arteries (arrows).

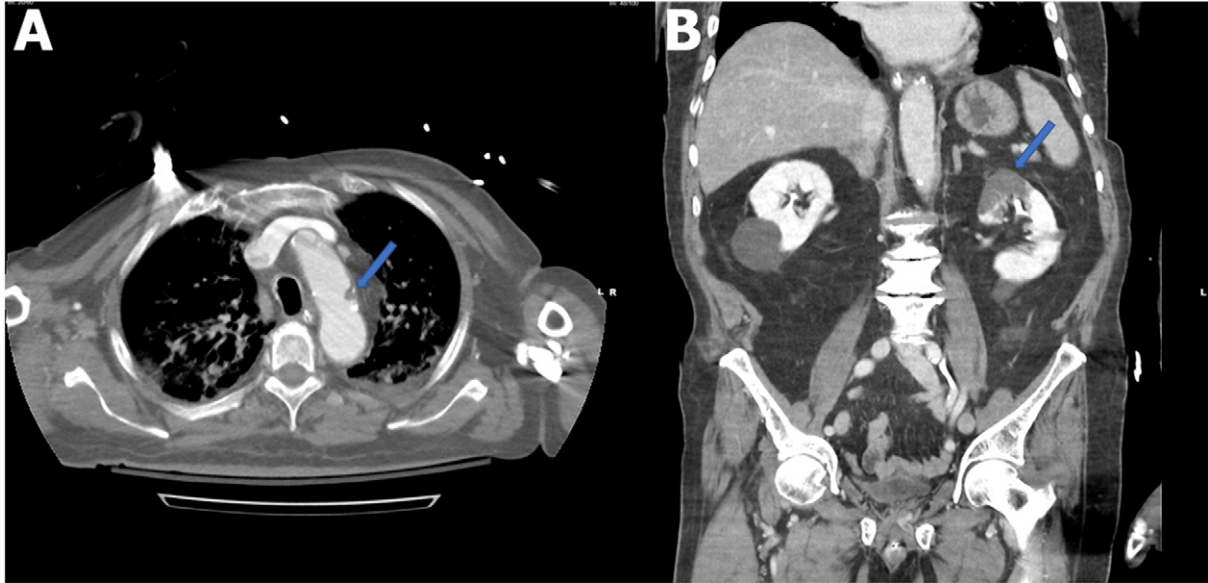


Figure 2. *A*, Axial contrast-enhanced CT of the chest demonstrates a filling defect in the aortic arch (arrow) consistent with thrombus. *B*, Coronal contrast-enhanced CT of the abdomen and pelvis demonstrates a wedge-shaped low-attenuation region in the superior pole of the left kidney (arrow) consistent with renal infarct.

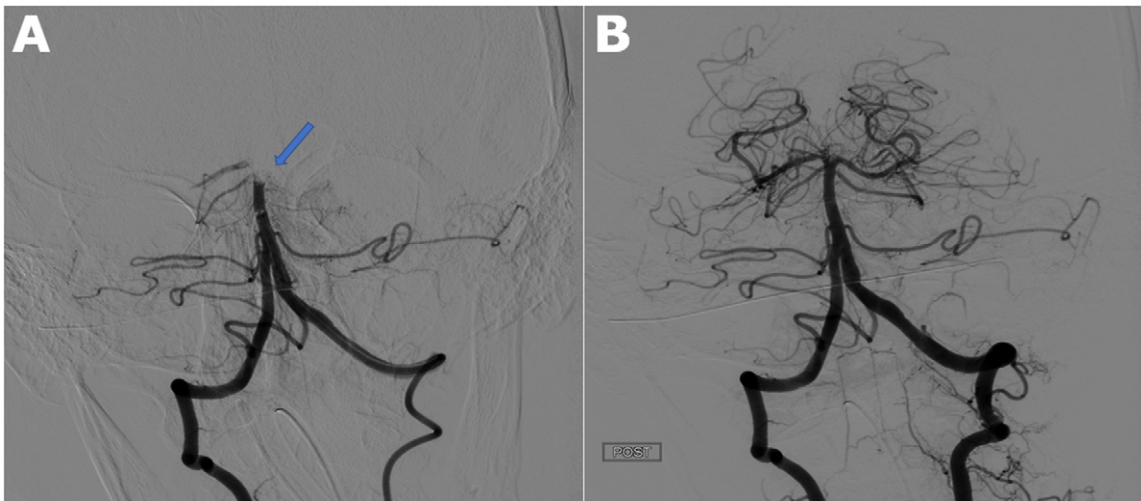


Figure 3. Catheter directed cerebral angiography. *A*, Pre-thrombectomy angiogram demonstrates an occluded distal basilar artery (arrow). *B*, Postthrombectomy angiogram demonstrates successful restoration of the posterior circulation.

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