Case Report

COVID-19 and Lung Cavitation: A Clue to Pathogenesis?

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Abstract

Description
Lung cavitation as a complication of COVID-19 is rare. A 56-year-old male presented with lung cavitation, small volume hemoptysis, and violaceous discoloration of the right great toe, 5 weeks after diagnosis with COVID-19 pneumonia. The digital changes were consistent with previously described microvascular changes called "COVID toe." CT angiography of the chest was negative for pulmonary embolism but showed a 2.5 x 3.1 x 2.2 cm cavitation within the right lung. Extensive evaluation for commonly implicated infectious and autoimmune causes was negative. We concluded that the cavitary lung lesions were likely a complication of COVID-19 pneumonia and may implicate microangiopathy as an important component of pathogenesis. This case highlights a rare complication of COVID-19 of which clinicians should be aware.

Keywords
lung cavitation; lung diseases; SARS-CoV-2; COVID-19/complications; COVID-19/diagnosis; viral pneumonia; thoracic radiography; male

Introduction
A pulmonary cavity is defined as "a gas-filled space, seen as a lucency or low-attenuation area, within pulmonary consolidation, a mass, or a nodule."1 The differential diagnosis of cavitary lung lesions is broad.2,3 Typical infectious causes include bacterial and fungal etiologies as well as septic pulmonary emboli. Non-infectious causes include pulmonary infarction, rheumatologic disease, and malignancy.2,3 Lung cavitation is not commonly seen in viral pneumonia. Pulmonary cavitation is a rare finding in the course of COVID-19. We present a case of cavitary lung lesions that were likely a complication of COVID-19 pneumonia and may implicate microangiopathy as an important component of pathogenesis.

Case Description
A 56-year-old male, who never smoked, had an onset of fatigue, diarrhea, fever, and headache 5 weeks prior to admission. He was seen in an outpatient clinic where nasal swab PCR testing was positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The following week, he developed a dry cough and had a chest X-ray (CXR) consistent with novel coronavirus disease 2019 (COVID-19) pneumonia (Figure 1). He was started on prednisone 40 mg daily for 5 days. One week later, he presented to the emergency room with lower extremity pain and swelling. A lower extremity duplex ultrasound showed bilateral distal lower extremity deep venous thromboses (DVTs). He was treated with apixaban and discharged home. Three weeks later, he experienced small volume hemoptysis without respiratory distress and was admitted to the hospital. His oxygen saturation was 93% on room air. His pulmonary auscultation revealed vesicular breath sounds without wheezes, rales, or rhonchi. He was noted to have erythema and edema on the dorsal surface of the right great toe with
only minimal associated discomfort (Figure 2), onset of which he reported within 1 week prior to presentation, or approximately 4 weeks after initial symptom onset. It was determined that this symptom was consistent with "COVID toe," a previously-described, pernio-like lesion presenting on the digits and distal extremities.

The CXR showed improvement in the prior pattern of peripheral pulmonary infiltrates (Figure 1). A computed tomographic angiography (CTA) of the chest demonstrated patchy peripheral ground glass opacities, an area of cavitation measuring 2.5 x 3.1 x 2.2 cm abutting the right major fissure (Figures 3A-3C) but no evidence of pulmonary emboli. The patient’s laboratory findings were significant for a C-reactive protein (CRP) of 0.67 mg/dL (normal range <0.29 mg/dL) and an erythrocyte sedimentation rate (ESR) of 20 mm/hr (reference range 0-15 mm/hr). Negative studies included: fungal markers (serum galactomannan and beta-d-glucan); interferon gamma release assay for latent tuberculosis; anti-nuclear antibody (ANA) and anti-neutrophil cytoplasmic antibody (ANCA); induced sputum for bacterial culture, gram stain, and acid-fast bacillus (AFB) smear and culture; human immunodeficiency virus (HIV) antibody; and transthoracic echocardiogram. Following a brief admission, the patient demonstrated clinical improvement and was discharged with a course of amoxicillin-clavulanate. A CT scan was obtained 3 months later, which showed a decrease in the size of the cavitation to 1.1 x 1.4 cm with resolution of ground glass opacities. He reported his right toe symptoms resolved approximately 6 to 8 weeks after discharge.

Figure 1. AP portable CXR 5 weeks after diagnosis with COVID-19 demonstrates bilateral peripheral pulmonary infiltrates.

Figure 2. Right great toe shows dorsal erythema and edema, consistent with “COVID toe”.

Figure 3. A computed tomographic angiography (CTA) of the chest demonstrates patchy peripheral ground glass opacities, an area of cavitation measuring 2.5 x 3.1 x 2.2 cm abutting the right major fissure (Figures 3A-3C) but no evidence of pulmonary emboli.
Discussion

A pulmonary cavity is defined as "a gas-filled space, seen as a lucency or low-attenuation area, within pulmonary consolidation, a mass, or a nodule."1 The differential diagnosis of cavitary lung lesions is broad.2,3 Typical infectious causes include bacterial and fungal etiologies as well as septic pulmonary emboli. Non-infectious causes include pulmonary infarction, rheumatologic disease, and malignancy.2,3

Lung cavitation is not commonly seen in viral pneumonia. Other human coronaviruses, including SARS-CoV and MERS-CoV, are not known to cause cavitary lesions.4-6 Cavitary lesions may be seen with other viral etiologies such as influenza. However, they are typically due to superimposed bacterial pneumonia.7 Pulmonary cavitation is a rare finding in the course of COVID-19, but select cases have been documented. Selveraj et al. describe a patient diagnosed with COVID-19 who developed pulmonary cavities on a chest CT 3 weeks later.8 This case is unique from ours since their patient had a high titer positive ANA, though positive ANA have been described in the setting of severe COVID.9 One systematic review including 4410 COVID-19 patients estimated a pooled incidence of cavitary lesions to be 0.1%.10 A review by Zoumot et al. included 689 patients and found a higher incidence of cavitary lesions, comprising 1.7% of all patients admitted with a diagnosis of COVID-19 and 11% of those who were admitted to the intensive care unit.11 The patients with cavitary lesions in this study differ from our patient in that they all received tocilizumab, were mechanically ventilated, and were more severely ill.

Various mechanisms have been proposed to describe how SARS-CoV-2 may uniquely cause cavitary lesions, including cystic degeneration of consolidations, lung parenchyma caused primarily by the SARS-CoV-2 virus, bacterial or fungal coinfection, and small-vessel vasculopathy.12-14 An autopsy study by Ackerman et al. described widespread microangiopathy with 9 times the prevalence of alveolar capillary microthrombi in SARS-CoV-2 infected lungs when compared to lungs infected with influenza. This study also noted intussusceptive angiogenesis and severe endothelial injury, suggesting a pathogenesis unique to SARS-CoV-2 among other viral pneumonias.15

Conclusion

As the SARS-CoV-2 pandemic evolves, so too must the clinician’s understanding of its clinical presentation. We aim to inform providers of this uncommon manifestation and to elicit further discussion and investigation to guide clinical decision-making.
Conflicts of Interest
The authors declare they have no conflicts of interest.

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