Case Report
Cavitary Pulmonary Infarct in Immunocompromised Hosts

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Pulmonary disease in immunocompromised patients is common, but cavitary lung disease is less common and is usually associated with a fungal or mycobacterial infection. Pulmonary embolism is a noninfectious cause of a cavitary pulmonary process. Pulmonary embolism causes infarction in fewer than 15% of cases, and only about 5% of infarctions cavitate. Herein we describe two cases of cavitary infarcts in immunocompromised patients and review the clinical aspects of pulmonary infaracts and cavitation. Cavitary pulmonary infarction has been reported only rarely in immunocompromised patients. It is a dangerous but treatable pulmonary disease that must be considered in the differential diagnosis of immunocompromised patients with lung disease.

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Pulmonary infiltrates in immunocompromised hosts present a challenging problem of considerable urgency to clinicians. A wide range of pulmonary diseases may be responsible.¹ Establishing the specific causative process, if possible, is important to ensure that therapy will be optimal and toxicity minimal.² Chest radiographic abnormalities in immunocompromised hosts are either diffuse or focal; cavitary lesions are less frequently encountered. Herein we describe two immunocompromised patients with cavitary lung disease caused by pulmonary infarction.

REPORT OF CASES
Case 1.—A 70-year-old woman was admitted to her local hospital 2 months after dermatomyositis had been diagnosed. For the preceding 2 months, she had been taking prednisone, 60 mg daily; during that time, her weight had increased, and peripheral edema and glucose intolerance had developed. One week before admission, an infiltrate was noted in the left midlung area on a chest roentgenogram obtained during an examination in the emergency department for polydipsia and polyuria (Fig. 1). Subsequent computed tomography of the chest revealed a 2.5-cm cavitary lesion located peripherally in the superior segment of the lower lobe of the left lung (Fig. 2). No associated mediastinal adenopathy was evident. The patient was referred to the Mayo Clinic for diagnostic evaluation. Specifically, she denied having respiratory symptoms or chest pain. She had quit smoking 25 years earlier. She lived in the Upper Midwest and had traveled to the southwestern part of the United States approximately 6 years previously.

On examination, the patient had a cushingoid appearance and proximal muscle weakness. Her vital signs and temperature were normal. Skin changes and neurologic findings were consistent with dermatomyositis. Results of an examination of the chest were normal. Findings on an examination of the cardiovascular system were unremarkable, except for bilateral pitting edema of the lower extremities.

The hemoglobin concentration was 13.3 g/dL, leukocyte count was 12.9 x 10⁹/L, platelet count was 239 x 10⁹/L, and creatinine value was 0.9 mg/dL. The erythrocyte sedimentation rate was 18 mm in 1 hour. The alkaline phosphatase, aspartate aminotransferase, lactate dehydrogenase, and creatine kinase values were all within normal limits.

Infectious diseases, including fungal or tuberculous cavities, nocardiosis, or actinomycosis, were the chief concern. Secondary considerations were neoplastic conditions. Bronchoscopy demonstrated only mild, diffuse, inflammatory changes of bronchitis. Results of washings for cytologic and microbiologic analyses were negative. The lesion was not clearly visible by fluoroscopy; a transbronchoscopic lung biopsy was not performed. The patient underwent a transthoracic needle aspiration, which also was diagnostically unproductive. An open-lung biopsy was planned, but the patient was found unresponsive and pulseless on the floor after walking in a hallway. Resuscitative efforts were unsuccessful. An autopsy showed that the patient died of a recurrent pulmonary thromboembolism. Multiple pulmonary infarcts, some of which were cavitary, were evident; the lesion noted on computed tomography was a cavitary pulmonary infarct. Venous thrombosis of the lower extremities was present.

Case 2.—A 64-year-old morbidly obese woman was admitted to the Mayo Clinic because of infected skin ulcers under her breasts and in the deep folds under her pannus. During the 3 weeks before admission, a nonproductive cough and chills had developed. After an episode of pleuritic chest pain, she was admitted to her local hospital for
Fig. 1 (case 1). Chest roentgenogram of 70-year-old woman, demonstrating spheric infiltrate in left midlung area.

Intravenously administered antibiotic therapy. After dismissal, she continued to feel poorly and came to our emergency department, where a chest roentgenogram disclosed an infiltrate in the lower lobe of the right lung. Her medical history was remarkable for hypertension, a 10-pack-year history of smoking, adjustment disorder, and Cushing's syndrome that had been induced by self-medication with a preparation from Mexico containing corticosteroids. After consultation with infectious disease specialists and plastic surgeons, fluconazole and a combination of imipenem and cilastatin were administered. Computed tomography of the chest revealed consolidation with associated volume loss and pleural fluid in the lower lobe of the right lung posteriorly. A pulmonary disease specialist was consulted.

On examination, the patient was 145 cm tall and weighed 101.2 kg. Her blood pressure and heart rate were normal. The respiratory rate was 20/min, and her temperature was 38.0°C. The skin had ecchymoses and hyperpigmentation consistent with Cushing's syndrome. On auscultation of the chest, crackles were detected in both lung bases, but they were louder on the right than on the left. No clubbing or adenopathy was evident. Findings on a cardiac examination were unremarkable. Approximately 12 deep, moist ulcers, some up to 3 to 4 cm in diameter with sinus tracks up to 3 cm deep, were found under the pronounced pannus. They were erythematous at the base, but no diffuse erythema was noted. The hemoglobin concentration was 12.3 g/dL, leukocyte count was 15.3 x 10^9/L, and platelet count was 227 x 10^9/L. The erythrocyte sedimentation rate was 50 mm in 1 hour. The differential count included 82% polymorphonuclear leukocytes, 11% immature types, and 4% lymphocytes. Results of sputum and fungal serologic studies were normal. On bronchoscopy, no abnormalities were noted. Bronchoalveolar lavage of the posterior basilar segment of the lower lobe of the right lung was performed, and brushings were sent for cytologic and microbiologic analyses. The lavage specimen was negative on Gram stain and negative for Legionella on direct immunofluorescence microscopy. Tests for Pneumocystis, acid-fast bacillus, and cytomegalovirus were negative, as were bacterial cultures. Fungal cultures grew Candida albicans.

Five days after admission, computed tomography of the abdomen and lower chest, done to search for deeper soft tissue involvement, showed cavitation of the lesion in the lower lobe of the right lung. While the patient was initiating ambulation, she experienced tachypnea and then apnea; she was intubated on an emergency basis and transferred to the intensive-care unit, where her condition rapidly deteriorated because of progressive hypotension. A pulmonary artery catheter was inserted; the pulmonary artery pressure was 40 mm Hg, and the systemic systolic pressure was 80 mm Hg. Emergency echocardiography showed adequate left ventricular function and a substantially dilated right ventricle. Because of continued deterioration and a presumptive diagnosis of acute pulmonary embolus, the patient was given 250,000 U of urokinase directly into the pulmonary artery catheter, but she died. An autopsy revealed multiple pulmonary emboli involving the bilateral and lower lobe pulmonary arteries, a hemorrhagic pulmonary infarct, and a sterile abscess involving the lower lobe of the right lung, corresponding to the cavitory lung lesion evident on computed tomography. No pulmonary infection was present.

Fig. 2 (case 1). Computed tomogram, showing that the rounded infiltrate noted on chest roentgenography was a cavitory parenchymal lesion in superior segment of lower lobe of left lung.
CAVITARY PULMONARY INFARCT IN IMMUNOCOMPROMISED HOST

DISCUSSION

Cavitary lung disease results from various causes. In our immunocompromised patients, infectious causes were certainly considered foremost; mycobacterial and fungal (Histoplasma, Cryptococcus, Coccidioides, and Aspergillus) causes, Actinomyces, Nocardia, gram-negative necrotizing infection, Staphylococcus, and anaerobic organisms were sought. P. carinii has been reported to cause cavitary lesions. Noninfectious causes such as primary or metastatic neoplasms, granulomatous vasculitis (Wegener's and Churg-Strauss), pulmonary sequestrations, and pneumoconiosis must also be considered; however, in immunocompromised hosts, they would be of lower probability. Septic pulmonary emboli deserve special consideration when multiple cavities are present, especially if the cavities contain a radiopaque density. Cavitation caused by bland pulmonary infarction is an important diagnosis that is often not considered but, if correctly diagnosed, has specific therapy.

Cavitary pulmonary infarction is rare. In a series of 92 patients with nontuberculous cavitary disease, infarct accounted for only one cavity; in another such series of 90 patients, no thromboembolic infarcts were observed. In a series of seven masslike lesions in immunocompromised patients, three were cavitary (one chronic abscess, one necrotizing gram-negative abscess, and one vasculitic), and none were infarcts. Only about 10 to 15% of pulmonary emboli cause infarction. Infarction is rare because the lung has two blood supplies, through the bronchial and pulmonary arteries. It is most common in patients with congestive heart failure, mitral valve disease, and severe pulmonary disease, probably because of impaired circulation to lung parenchyma.

After infarction occurs, cavitation can follow as a result of either aseptic liquefaction or secondary infection, which occurs by direct infection with bronchial organisms, aspiration of orally administered fluid, or hematogenous spread. Cavitation complicates infarction in 2.7% to 7% of cases; in a recent literature review, it was infective almost as often as aseptic. Cavitation occurred at a mean of 5 days after the first chest radiographic appearance of an infiltrate and was in the upper lobes of the lungs in 10 to 24% of cases. Symptoms and signs of cavitary infarct are nonspecific, and the correct antemortem diagnosis is overlooked in more than half of the cases. Purulent sputum and fever are considered common in patients with superinfected infarcts, but the clinical picture mimics many other causes of cavitary lung disease. Because of diagnostic difficulty, cavitary infarcts have been resected as suspected bronchogenic carcinomas, and they have been confused with tuberculosis, complicated bacterial infection, and anaerobic abscess. Correct antemortem diagnosis has been confirmed by ventilation-perfusion lung scanning, pulmonary angiography, and biopsy (a less desirable method). Pulmonary embolism is a common cause of in-hospital mortality, often occurring in patients who can least tolerate it because of serious comorbidity, and it is responsible for 50,000 deaths annually. In patients with immunocompromised lung disease, clinicians are confronted with a challenging diagnostic problem. Recent reviews have emphasized a thorough assessment of risk factors (such as type of immune deficit) for narrowing the differential diagnosis and targeting the diagnostic evaluation. Although infections, drug reaction, and immunologic and neoplastic processes predominate, almost all immunocompromised patients may be at risk for thromboembolic disease. Venous thromboembolic disease is a dangerous but rare cause of cavitary lung disease; it can be present in immunocompromised patients and has specific and efficacious therapy. Thus, clinicians must consider this diagnosis when cavitary disease is evaluated in immunocompromised hosts.

REFERENCES