A 67-year-old retired marine engineer presented with a 3-week history of increasing tiredness and dyspnea without cough, sputum, or chest pain. Three months previously, he had been diagnosed as having an inoperable glioma of the left temporoparietal area for which he had received palliative treatment with radiotherapy and dexamethasone. He had had Addison’s disease for 40 years, insulin-dependent diabetes mellitus for 12 years, and bilateral femoral vein thrombi 2 years previously. He had never smoked.

He was apyrexial with a heart rate of 88 beats per minute; BP, 110/70 mm Hg; respiratory rate, 18 breaths per minute; and oxygen saturation, 96% on oximetry. Jugular venous pressure was normal and there were no abnormal signs on chest examination. He had moderate expressive dysphasia, right hemiparesis, and perioral herpes simplex infection.

A chest radiograph (Fig 1) showed a large air-filled cavity in the upper lobe of the right lung. The WBC count was 14.8×10^9/L (92% neutrophils); glucose level, 12 mmol/L; and ECG, normal. Examination of a mouth swab by electron microscopy identified herpesvirus particles. Bronchoscopy did not show any endobronchial lesion, and tests of bronchoalveolar lavage fluid for cytology, bacteria, mycobacteria, fungi, viruses, and *Pneumocystis carinii* were negative. Serial chest radiographs at weekly intervals showed enlargement of the upper lobe cavity of the right lung followed by the emergence of new similar cavitating lesions in the lower lobes of both lungs (Fig 2) and the development of a right pneumothorax.

**What is the diagnosis?**

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Diagnosis: Multiple cavitating pulmonary infarcts

The CT pulmonary angiography is best performed on a spiral CT machine. A large volume of data, up to 24 cm in length, can be acquired during a single breath-hold of 20 s during infusion of intravenous contrast (80 mL) Iopamidol 200 at 2 mL/s). The data can then be reconstructed at any given slice width and increment (usually overlapping 2.5-mm width with 1-mm increment). Using this technique, the three cavitating lesions with thin nonenhancing walls were identified along with thrombus in the pulmonary arteries on the right and left (Fig 3).

Cavitary lung disease can result from a wide range of etiologies and pathogenic mechanisms. Bronchiolar check valves with air trapping are thought to be the main mechanism responsible for the formation of the bullae and larger pneumatoceles found with generalized emphysema and may be important in the evolution of other cavitary lung diseases. Infection-induced necrosis of lung parenchyma with cavity formation can result from septic emboli, localized endarteritis and thrombosis, or from the local production of proteolytic enzymes. Lung abscesses are most commonly due to Staphylococcus aureus, anaerobic organisms, and occasionally Streptococcus pneumoniae. Our patient had diabetes, was receiving corticosteroids, and had malignant disease, all of which are risk factors for tuberculosis, an important cause of lung cavitation. Invasive aspergillosis usually is associated with profound immunosuppression; however, rapid radiologic progression to cavitation has been reported even in patients receiving low-dose corticosteroids.1 More recently, Pneumocystis carinii has been implicated as a cause of lung cavitation in patients with AIDS.2 Primary bronchial carcinomas, metastatic tumors, and, very rarely, lymphomas may cavitate. Other nodular lesions of varied etiology may occasionally cavitate, eg, granulomatous vasculitides and pneumocinotic and rheumatoid nodules.

Cavitating pulmonary infarcts account for about 1.5% of all cavitating pulmonary lesions.3 Lung infarction occurs in 15% of cases of pulmonary thromboembolism, and in only 7% of these does the infarction process progress to cavitation either by aseptic necrosis or from secondary infection. Cavitating pulmonary infarcts arising by aseptic necrosis usually are single (75%), right-sided (69%), in the upper lobe (39%), and have a high mortality rate (41%). The median time to cavitation has been reported as 14 days, with a range of 2 to 63 days.4 Infected cavitating pulmonary infarcts occur more frequently in the lower lobes, evolve faster, and have a higher mortality rate.5

The diagnosis of cavitary pulmonary infarcts is difficult and often missed. In one recent series, 40% of cases were not diagnosed until postmortem examination.5 Although pulmonary angiography remains the definitive investigation for pulmonary thromboembolism, CT angiography has the advantage of being (1) able to image the whole lung, which may indicate alternative diagnoses; (2) noninvasive; and (3) reported to have a high sensitivity and specificity for detecting pulmonary emboli in the second to fourth division pulmonary arteries.6

References

Figure 3. CT pulmonary angiogram showing clot in the main artery (C) of the right lung and pulmonary artery (R) of lower lobe of left lung.