A 29-year-old homosexual man with AIDS and a history of *Pneumocystis carinii* pneumonia, intestinal *Mycobacterium avium-intracellulare*, meningococcal meningitis, and anal herpes was admitted to the hospital with a 1-month duration of fever, chills, sweats, and a cough productive of scant white sputum. A chest roentgenogram performed 1 month prior to hospital admission had shown a “possible right lower lobe infiltrate.” The patient denied exertional dyspnea, paroxysmal nocturnal dyspnea, or weight change. He had two pet cockatiels, but both were healthy. He smoked one pack of cigarettes per day. Previous antiviral therapy had included zidovudine (azidothymidine) and didanosine (2',3'-dideoxyinosine). Medications at the time of admission were acyclovir and fluconazole.

Significant physical findings included fever of 38.4°C, cachexia, mild diaphoresis, absence of skin lesions, and posterior cervical adenopathy. Chest examination revealed dullness to percussion and rales at the right base, without egophony. The chest roentgenogram (Figs 1 and 2) revealed a right lower lobe infiltrate, a subtle patchy left lower lobe infiltrate, bilateral small pleural effusions, and prominent Kerley B lines. A nipple ring was also noted.

The patient was initially treated with erythromycin for presumed community-acquired pneumonia. Sputum smears were negative for acid-fast bacilli and *P carinii*. Fiberoptic bronchoscopy revealed diffuse erythema along the right lower bronchial tree. Brushings and bronchoalveolar lavage from the right lower lobe were nondiagnostic and as the patient’s condition was not improving with erythromycin, he was referred for thoracoscopic lung biopsy.
Diagnosis: Pulmonary Kaposi’s Sarcoma

The lung biopsy specimen revealed a patchy spindle cell infiltrate predominantly involving the lung interstitium, septa, and especially notable in the subpleural area. Tumor surrounded larger blood vessels. Focal areas of alveolar edema were present.

Kaposi’s sarcoma (KS) was a rare malignancy before the AIDS epidemic, with an annual US incidence of 0.02 to 0.06 cases per 100,000.\(^2\) It now occurs in about 35% of patients with AIDS,\(^3\) predominantly affecting homosexual men,\(^4\) for unclear reasons. About 20% of patients with AIDS-related KS develop clinically apparent pulmonary involvement.\(^5\)

Almost all patients with pulmonary KS will have evidence of disease elsewhere before pulmonary involvement is noted. Garay et al\(^5\) found that 18 of 19 patients diagnosed as having pulmonary KS had developed skin lesions. In patients such as ours, who have no other evidence of KS, the diagnosis can be difficult, as both the clinical presentation and the roentgenographic findings are nonspecific.

The clinical presentation of pulmonary KS is difficult to differentiate from opportunistic infection.\(^5\) Most patients complain of fever, cough, and dyspnea, and as many as 50% of patients have a pulmonary opportunistic infection at the time of diagnosis,\(^6\) compounding the difficulty.

Both the roentgenographic and computed tomographic (CT) findings associated with pulmonary KS have been reported recently.\(^3,5-8\) Roentgenographic findings are variable and nonspecific. Garay et al\(^5\) found nodular infiltrates in 26% of patients with pulmonary KS, bilateral alveolar-interstitial infiltrates in 58%, unilateral alveolar infiltrates in 5%, and absence of infiltrates in 11%. Concomitant pulmonary infections frequently increase the difficulty with roentgenographic interpretation.\(^4\) Pleural effusions are noted in approximately 60% of cases, and are usually bilateral and moderate in size.\(^9\) They are thought to be due to direct involvement of the visceral pleura with tumor as well as pulmonary lymphatic invasion and obstruction by tumor.\(^10\) Hilar, mediastinal adenopathy, or both are reported to occur in up to 50% of patients, but are usually not bulky.\(^4,10\) Cavitary lesions\(^11\) and normal chest roentgenograms\(^5\) are unusual. To our knowledge, there have been no prior reports of pulmonary KS causing the finding of Kerley B lines.

The CT findings associated with pulmonary KS have been reported by Naidich et al\(^7\) and Wolff et al.\(^8\) The typical findings are multiple flame-shaped or nodular lesions with ill-defined margins, usually most predominant near the hilum. There often is a pattern of radiation along bronchovascular bundles.

At bronchoscopy, endobronchial KS appears as multiple slightly raised lesions, often at segmental orifice. The lesions are characteristic and therefore the diagnosis is often made clinically as endobronchial biopsy usually does not yield enough tissue for a definitive diagnosis. In one report, of 17 patients with pulmonary KS who had bronchoscopic biopsies, only 4 specimens were diagnostic (24%), 2 by endobronchial and 2 by transbronchial biopsy.\(^5\)

Analysis of pleural fluid does not help establish a diagnosis of pleural KS. The fluid is almost always a serosanguineous exudate, but cytologic findings are never diagnostic. Also, because only the visceral pleura is involved, closed pleural biopsy is not helpful.\(^10\)

Kerley B lines represent thickened interlobular septa usually due to edema of the peripheral and subpleural interstitium.\(^12\) The location of the spindle cell infiltrate in our patient and the evidence of alveolar edema suggest that the Kerley B lines were visible due to subpleural lymphatic invasion or by more central lymphatic obstruction. In another reported case, however, Kerley B lines were not noted.\(^9\)

This roentgenogram adds the variable of Kerley B lines to the protean nature of the roentgenographic features of KS. The finding of Kerley B lines in a patient with AIDS should suggest a limited number of diagnoses: congestive heart failure or lymphatic obstruction from lymphoma, bronchogenic cancer, or KS.

REFERENCES