A 31-year-old HIV-positive Hispanic man with a history of intravenous drug abuse was admitted with sudden onset, sharp, bilateral chest pain with shortness of breath. The pain was predominantly in the posterior thorax and was not pleuritic in nature. The patient also reported fevers to 39.0°C for two days prior to admission. In the more distant past, the patient described a cough productive of yellow sputum and a 40.82 kg (90 pound) weight loss over the eight months prior to admission. Physical examination revealed a temperature of 39.3°C, decreased breath sounds at both lung bases, and oral thrush. Laboratory data demonstrated a WBC of 4.1K. Arterial blood gas values on room air showed a pH of 7.45, Pco2, 31; Po2, 83; and a 93 percent O2 saturation.

A PA view of the chest demonstrates cystic parenchymal changes at the lung bases, greater on the right than left. In addition, bilateral apical pneumothoraces are present (Fig 1 and 2). There are no superimposed infiltrates or masses in the lung fields. There is no evidence of hilar or mediastinal adenopathy or pleural effusions.

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**Diagnosis: Atypical Pneumocystis carinii pneumonia**

Pulmonary abnormalities, both infectious and neoplastic, represent a major source of morbidity and mortality in AIDS patients. More than 50 percent of patients with AIDS develop pulmonary manifestations at some time in the course of their disease. *Pneumocystis carinii* pneumonia (PCP) is the most common life-threatening infection in this population, occurring at least once in the course of the disease in approximately 60 percent of AIDS patients.1

Typically, PCP presents as bilateral perihilar or basilar reticular or reticulonodular infiltrates that rapidly progress in three to five days to diffuse bilateral air-space consolidation. However, a spectrum of atypical patterns has been reported for PCP. These include a normal chest x-ray film, predominant upper lobe involvement simulating pulmonary tuberculosis, and focal unilateral infiltrates. In addition, when the disease is bilateral, the severity of involvement may be asymmetric. Hilar and mediastinal lymphadenopathy and pleural effusions, although unusual, have also been reported in PCP. Rare manifestations of the pneumonia include a miliary pattern or a solitary cavitating or noncavitating pulmonary nodule.2,4

Lastly, as demonstrated by our case, PCP has also been reported to present as a pattern characterized by air-filled, cystic parenchymal changes. In a series reported by DeLorenzo et al.,4 cystic parenchymal changes were noted to occur roentgenographically in seven of 104 (6.7 percent) documented cases of PCP. Another series recently reported by Sandhu and Goodman5 described cystic parenchymal changes in ten of 100 (10 percent) cases of documented PCP. The cysts are typically thin-walled with no intracystic material and no predilection for a particular area of the lung. The cysts may be solitary or multiple, unilateral or bilateral. They range in size from 1 cm to greater than 8 cm. These cysts pose a significant potential risk for rupture and thereby, the development of spontaneous pneumothorax. The natural history of the cysts has not yet been established. Early reports, however, suggest that the majority of the cysts resolve following treatment of the pneumonia, usually within seven months.5

The mechanism by which these cysts develop is not completely understood. It has been proposed that the cystic changes may result from a check-value mechanism caused by airway inflammation and resultant partial airway obstruction. Alternatively, these changes may be due to a disordered parenchymal architecture secondary to chronic infection and inflammation.6

The PCP may present in a variety of roentgenographic patterns. It is, however, important to recognize that cystic parenchymal changes seen in patients with AIDS are most likely secondary to PCP. Such cysts pose a significant risk for rupture, and thus, the development of spontaneous pneumothorax.

**References**