A 33-year-old, previously healthy woman was referred to our hospital with a 3-week history of progressive shortness of breath and a nonproductive cough. Physical examination on admission revealed a well nourished, tachypneic white woman with a temperature of 37.6°C, blood pressure 121/73 mm Hg, and pulse rate of 96. Shotty right axillary adenopathy and decreased breath sounds at the lung bases were noted. The remainder of the physical examination was unremarkable.

Laboratory findings were notable for a WBC count of 73,500 mm$^3$, hemoglobin 8.5 g/dL, and platelet count of 66,000 mm$^3$. The peripheral smear showed leukocytosis with blasts, atypical eosinophilic myelocytes, and thrombocytopenia. Electrolytes and coagulation parameters were normal. Arterial blood gas on 3 L oxygen revealed a pH of 7.39, $P_{CO_2}$ 46, $P_O_2$ 85, and $SaO_2$ 96%. Bone marrow biopsy was diagnostic for acute myelogenous leukemia.

Chest radiograph showed bilateral airspace disease greatest in the mid- and lower-lung zones (Fig 1). High-resolution CT scan was performed. A representative image at the level of the right middle lobe bronchus revealed bilateral poorly defined air space radiopacities with sparing of the subpleural lung (Fig 2). Bronchoscopy with bronchoalveolar lavage was performed.

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Diagnosis: Leukemic infiltration of the lung

Up to 80% of patients with acute leukemia develop pulmonary complications in the course of their disease. These complications, despite more efficacious chemotherapeutic regimens, remain a major cause of morbidity and mortality.1

Isolated leukemic cell infiltrates are found at autopsy in 31 to 66% of patients who die from their disease.2 Symptomatic pulmonary disease, however, due to leukemic infiltrates, however, is uncommon. In one study, only 10% of the patients with documented leukemic infiltrates at autopsy had complained of respiratory symptoms before death.3 Leukemic infiltrates are extravascular collections of blast cells most often located in the interstitium of the lung within the perivascular and peribronchial tissue. As in our case, a high percentage of blast cells (blast count above 40%) is a common feature.4 Subpleural and more nodular-appearing infiltrates have been described. Morphologically, leukemic infiltrates should be distinguished from an entity known as leukostasis where intravascular aggregates of leukemic blast cells are shown.

Chest roentgenograms may be normal or may show a diffusely bilateral interstitial or airspace pattern.5 Cases of focal leukemic pulmonary infiltration, however, more suggestive of bacterial pneumonia, have been described.6 A case of cavitation in a proven leukemic infiltrate has also been presented recently.6

The differential diagnosis of bilateral pulmonary infiltrates in patients with leukemia at presentation includes pulmonary hemorrhage, edema, and vascular leukostasis. Infection, while a major cause of morbidity and mortality after treatment, is a less common cause at the time of initial diagnosis. Pulmonary hemorrhage is seen when the platelet count is less than 50,000/mL.7 Radiographically, a diffuse interstitial, alveolar, or mixed pattern is typically seen on plain radiographs and computed tomography. Hemorrhage, however, may result in more focal parenchymal radiopacities;8 a pattern frequently associated with fungal infection or pulmonary infarcts (see below).

Diffuse pulmonary edema may be either cardiogenic or noncardiogenic in origin. Noncardiogenic edema may result from iatrogenic fluid overload. Cardiac decompensation, though most often related to cumulative doses of cardiotoxic chemotherapeutic agents, may occur when the hemoglobin concentration falls below 5 mg. There is no definite correlation between congestive heart failure and myocardial leukemic infiltration.9 In patients with leukocyte counts greater than 200,000 mm3, leukostasis must also be considered.3

Once therapy has been instituted, the leading cause of bilateral pulmonary infiltrates is infection. Included in the differential diagnosis are the aforementioned, drug reactions, hyaline membrane disease of oxygen toxicity,8 and pulmonary complications unique to leukemia (leukemic cell lysis pneumopathy and hyperleukocytic reaction).3 There is an association between pulmonary alveolar proteinosis, hematologic malignancies, and opportunistic infections.2

Infectious causes have a wide spectrum of radiographic presentations. Common bacterial pathogens often result in patchy airspace consolidation while many opportunistic organisms are characterized by a diffuse interstitial or alveolar pattern.5 Opportunistic infection, however, may present as focal, mass-like parenchymal radiopacities on chest radiographs and computed tomography. For example, the CT appearance of invasive aspergillosis early in the course of treatment is quite distinctive with a zone or halo of lower attenuation surrounding a more solid mass. After chemotherapy and bone marrow recovery, frank cavitation and crescent formation may be seen.10

In summary, this is an unusual case of a patient with leukemia presenting with shortness of breath secondary to leukemic infiltration of the lung.

REFERENCES