A 44-year-old man presented to the emergency department with complaints of neck swelling, bothersome cough, and difficulty in breathing. His breathing difficulty began 3 days earlier and progressively worsened.

His medical history was significant for stage IV non-Hodgkin lymphoma, which had been diagnosed 5 months previously, and hypothyroidism. The patient received rituximab therapy at initial diagnosis and a chemotherapy regimen of cyclophosphamide, doxorubicin, vincristine, and prednisone (CHOP), and he had a significant response.

Two months after diagnosis, he had shortness of breath. A CT scan of the chest revealed a mediastinal mass that identified recurrent disease. He began radiation therapy directed at his chest; at presentation, he had completed 13 of 16 treatments and was not currently on any chemotherapy regimen.

His medications on admission were dexamethasone 2 mg twice daily, levothyroxine 150 µg daily, temazepam 15 mg at bedtime as needed, prochlorperazine 10 mg as needed, and hydrocodone 7.5 mg/acetaminophen 500 mg every 4 hours as needed.

His vital signs on admission were temperature of 37.5ºC (99.5ºF), blood pressure of 146/92 mm Hg, pulse rate of 136 beats per minute, respiration rate of 16 breaths per minute, and oxygen saturation of 96% on 2 L/min via nasal cannula.

On admission, he was alert, oriented, and sitting up in bed. He had bilateral cervical, anterior, and posterior lymphadenopathy and supraclavicular adenopathy. Auscultation of the lungs revealed bilateral posterior crackles. He was tachycardic with a regular rhythm. He had some right flank pain just lateral to the transverse process at the L2 level.

A comprehensive metabolic profile, complete blood cell count, measurement of thyroid-stimulating hormone and troponin levels, urinalysis, blood culture, sputum culture, and chest radiograph were ordered on admission. Results were unremarkable except for a lactate dehydrogenase level of 576 U/L (normal, 100 to 200 U/L), a thyroid-stimulating hormone level of 4.54 µIU/mL (normal, 0.35 to 4.1 µIU/mL), and a hemoglobin level of 11.4 g/dL (normal, 12.4 to 17 g/dL).

Figures 1 and 2 are the patient's chest radiograph and CT scan of the chest, respectively.

What is the likely diagnosis?
A. *Pneumocystis jiroveci* pneumonia
B. *Legionella pneumophila* pneumonia
C. Lymphomatous infiltrate
D. Pulmonary edema
E. Drug-induced lung injury
F. Radiation-induced lung injury

Answer on next page.

A man with difficulty in breathing and right lung consolidation: The chest radiograph on admission demonstrated a densely consolidated band of opacity in the right upper and lower peripheral lung zones, a left midlung zone opacity, and a widened mediastinum with compression of the trachea. A CT scan of the chest showed significant right lower and upper lobe peripheral consolidation, representing either opportunistic infection or lymphomatous infiltrate. Bronchoscopy was performed. Findings from pathologic examination were consistent with lymphomatous infiltrate. No infectious causes were noted on the bronchoscopy specimens. Discussion

It has been estimated that about 60,000 cases of non-Hodgkin lymphoma are diagnosed annually in the United States. While undergoing therapy, patients with non-Hodgkin lymphoma are at risk for infections with opportunistic pathogens, including *Aspergillus* species, *P jiroveci*, herpes simplex virus, and cytomegalovirus.

In patients with underlying malignancy, clinicians should also consider that cancer can masquerade...
as pneumonia, with similar signs and symptoms.\textsuperscript{3} Tumors and leukemic cells can obstruct lymphatic and vascular drainage, resulting in pulmonary infiltrates.\textsuperscript{2}

Pulmonary infiltrates in a patient with non-Hodgkin lymphoma can result from numerous causes, including infection, treatment toxicity, pulmonary edema, and disease recurrence.\textsuperscript{4} Infiltrates in a patient with disease recurrence can be focal, diffuse, cavitary, or nodular.\textsuperscript{2,4} Pulmonary involvement specific to non-Hodgkin lymphoma has been classified as nodular, bronchovascular-lymphangitic, pneumonic-alveolar, or miliary-hematogenous.\textsuperscript{5}

To evaluate the pulmonary infiltrates observed in our patient, a thorough history of radiation exposure and medication use, including chemotherapy, needed to be obtained. Bronchoscopy definitively yielded the diagnosis. This approach has been determined to be the modality of choice for the diagnosis of pulmonary infiltrates in non-HIV-infected immunocompromised patients.\textsuperscript{2,4,6} In a study by Rano and associates,\textsuperscript{6} bronchial aspirates and bronchoalveolar lavage fluid were determined to have the highest yield and most impact on therapeutic decisions in these patients. In this setting, it is pertinent to evaluate all potential causes of pulmonary infiltrates. In a patient with \textit{P jiroveci} pneumonia, radiographs typically show diffuse, bilateral, interstitial, or alveolar infiltrates.\textsuperscript{7} Chest radiographs from patients with \textit{L pneumophila} pneumonia may demonstrate a consolidative pattern; a urinary antigen test can detect the most common serotype and is very useful in the diagnosis.\textsuperscript{5} Pulmonary edema typically presents as bilateral diffuse infiltrates.\textsuperscript{4} Measurement of serum B-type natriuretic peptide level, echocardiography, and pulmonary artery catheterization are useful diagnostic tools.\textsuperscript{4}

The diagnosis of drug-induced pulmonary toxicity is determined by a history of exposure to drugs that cause pulmonary injury and the absence of other causes of toxicity.\textsuperscript{2} This toxicity may resolve with the discontinuation of the offending agent.\textsuperscript{2}

A diagnosis of radiation-induced lung injury may be made in patients with a history of radiation to the chest and alveolar infiltrates on a chest radiograph. In some of these patients, CT scans demonstrate sharply bounded areas of injury that typically do not follow an anatomic border.\textsuperscript{2,4} The cumulative dose and location of the radiation, together with preexisting lung disease and the use of concomitant drugs that may sensitize the lung to radiation, are risk factors for radiation-induced lung toxicity.\textsuperscript{2}

\textit{Case and photographs courtesy of Jeffrey S. Stroup, PharmD, BCPS, of University of Oklahoma College of Pharmacy, and Montgomery L. Roberts, DO, of Oklahoma State University Center for Health Sciences, Tulsa.}


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