We describe a 68-year-old male patient with diffuse large B-cell lymphoma (DLBCL) who suffered from relapse in bilateral upper lobes of the lung. The patient presented with cough, weakness and fever. A bronchovascular-lymphangitis-like shadow was detected in the bilateral upper lobes on a chest roentgenogram. Although cytological and cytofluorometric examinations revealed no malignant cells in the bronchoalveolar lavage fluid, trans-bronchial lung biopsy (TBLB) showed involvement of DLBCL in the bronchial mucosa. Recurrent lymphoma tends to extend along the bronchovascular bundles, resulting in granulomatous consolidation that may mimic tuberculosis and is likely to involve the lower lobes. Thus, TBLB proved to be essential for the diagnosis of the lung involvement of non-Hodgkin’s lymphoma (NHL).

Key words: bronchovascular-lymphangitis-like shadow, lymphoma, upper lung, trans-bronchial lung biopsy

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months, in March 2005, the patient had attained complete remission (CR) confirmed by (18) F-2-deoxy-2-fluoro-D-glucose positron emission tomography (FDG-PET).

In May 2005, the patient complained of dry cough, general malaise, and fever. Physical examination detected a coarse crackle in the bilateral upper lung, and oxyhemoglobin saturation was reduced at the level of 88% in room air. A chest roentgenogram showed a bronchovascular-lymphangitis-like shadow (Fig. 1). CT-scan detected the multiple small nodules in the bilateral upper lobe (Fig. 2). However, ultrasonography detected neither lymphadenopathy nor any relapse of the adrenal lesion. Blood cell count was normal. Serum C-reactive protein (CRP), lactate dehydrogenase (LDH) and serum interleukin-2 receptor was 16.2 mg/dl, 423 IU/l, 1,180 U/ml, respectively. The blood and sputum cultures were negative for viruses, bacteria, and fungi. Since there was no improvement in the symptoms following antibiotics therapy, the patient subsequently underwent a trans-bronchial lung biopsy (TBLB). Bronchoscopic evaluation of the upper airways showed diffuse redness. Although bronchoalveolar lavage (BAL) specimens were collected, neither cytological examination nor flow-cytometry analysis of the BAL fluid showed malignant cells. Histopathologic examination of the TBLB specimens showed abnormal lymphomatous infiltration of the bronchial mucosa (Fig. 3a). Immunostaining of leukocyte common antigens, CD20 and CD79a showed positivity for tumor cells, leading to a diagnosis of recurrent DLBCL (Fig. 3b). After two cycles of salvage therapy of ESHAP therapy (40 mg/m$^2$ of etoposide for 4 days; 500 mg/m$^2$ of methylprednisolone for 4 days; 25 mg/m$^2$ of CDDP for 4 days; 2000 mg/m$^2$ of Ara-C for 1 day), complete regression of the pulmonary infiltration was achieved. However, 24 days later, lung infiltration accompanied by mediastinal adenopathy was detected on a chest roentgenogram. Serum LDH and CRP levels increased to 1028 mg/dl and 21.6 mg/dl, respectively. In spite of additional administration of DeVIC therapy (40 mg/m$^2$ of dexamethasone for 3 days; 100 mg/m$^2$ of etoposide for 3 days;
1500 mg/m² of ifosfamide for 1 day and 300 mg/m² for carboplatin for 1 day), the patient died due to multi-organ failure on September 1, 2005.

**Discussion**

We describe a patient with relapse of NHL in bilateral upper lobes of the lung, showing a typical bronchovascular-lymphangitis-like shadow on radiographic examination. Pulmonary parenchymal involvement with lymphoma has been reported in 3.7% of extranodal NHL and particularly in 25% of recurrent disease (4). Pulmonary lymphoma may show various patterns on chest roentgenogram including bronchovascular or lymphangitis-like patterns with thickening of bronchovascular bundles and interlobular septae (41%), discrete pulmonary nodular (39%) patterns, and pneumonic or alveolar patterns with areas of consolidation (14%). The least common is miliary or hematogenous pattern with disseminated micro nodules (6%) (3, 4).

In the current patient, TBLB successfully differentiated pulmonary relapse of lymphoma from infection. The differentiation of pulmonary lymphoma from other diseases can be difficult only based on radiographic findings. Since new pulmonary parenchymal disease is often treated empirically as an infection in patients with an immunosuppressive condition secondary to chemotherapy, pulmonary disease which fails to respond to antibiotic therapy deserves consideration of biopsy for definitive tissue diagnosis. In the case of NHL, the diagnostic accuracy rates of TBLB and BAL were 67.5% and 36.3%, respectively (5). TBLB is a useful tool for the final diagnosis (5, 6).

The present patient showed lymphoma lesion in bilateral upper lobes of the lung, an unusual site of relapse, because pulmonary relapse of NHL usually occurs in the lower lobes (2). The morphology and surface phenotype of lymphoma cells did not differ from those of patients who have relapsed with pulmonary NHL. No correlation between histological and radiographic findings of NHL based on the WHO classification has been reported. It has been previously reported that nine of 13 patients with bronchovascular lymphangitis-like shadow was associated with “histiocytic lymphoma” according to the modified Rappaport classification (7). The histological findings in the current patient are not compatible with “histiocytic lymphoma”. Since the clinical symptoms or chest radiograph findings are usually nonspecific, TBLB could be a useful procedure for reliable diagnosis of pulmonary relapse of NHL.

**References**


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