both the supine and right lateral postures: the PEF and maximal inspiratory flow rates were reduced, the MEFV curve developed a small, but clear flow plateau, the MIFV curve flattened, and the UAO ratios generally increased; in particular, the FEV1/PEF ratio in the right lateral posture and the FEV50/FIF50 ratio in the supine position. (Table 3, Fig 2). The patient refused surgery and was treated with sodium levotheroxine.

**DISCUSSION**

In this report, we show that FVLs performed with patients in recumbent positions were able to bring out physiologic evidence of UAO, which was suspected from the past medical history but not detected by the FVL performed when the patient was in the upright sitting position.

Generally, the upright FVL is considered a sensitive test for the detection and localization of upper airway disorders,\(^1\) such as goiter, even when these do not produce respiratory symptoms.\(^3,4,6\) Because of its dynamic nature, the upright FVL is even more sensitive and in any case a much simpler and less expensive test than static radiologic techniques, including the CT scan performed at various lung volumes.\(^4,6\)

Here, we show in two patients that the recumbent FVL was an even more sensitive indication of upper airway flow limitation than the traditional upright FVL. This finding was clinically important, because before its detection both patients' recumbent symptoms (dyspnea) were attributed to either nocturnal asthma and congestive heart failure, respectively, and were managed by reinforcement of anti-asthma and cardiac treatment. These methods were without benefit. After thyroid surgery, symptoms and posture-related FVL changes disappeared in patient 1; this confirmed the cause-and-effect relationship between the goiter and the respiratory symptoms. Why these patients developed symptoms and physiologic evidence of UAO in the recumbent as opposed to the upright posture is not clear, but probably can be explained by posture- and gravity-induced changes in the anatomic relationship between the goiter and the trachea. Such posture- and gravity-induced changes in upper airway dimension do exist in normal subjects\(^7,8\) and awake patients with obstructive sleep apnea,\(^9\) as shown by acoustic reflection techniques or fast-CT scanning and appear independent of posture-related changes in lung volume. In general, recumbent postures reduce cross-sectional areas or wall-to-wall distances in the upper airway, either of the pharyngeal or the subglottic areas. Patients with position-dependent obstructive sleep apnea, it was shown that body position while the patient is awake affects the lateral rather than the anteroposterior dimension of the upper airway.\(^9\) Hence, it is not surprising to find posture-related changes in airflow (and upper airway dimension) in patients with an extrinsic mass, such as a goiter, around or near to the upper airway. We speculate that due to its weight, the goiter in both of our patients exerted extrinsic tracheal compression more marked in one or in both of the recumbent postures than in the upright sitting posture. The individual spatial relationship between the goiter, its shape, and its mass may then determine in which of the various recumbent postures airflow limitation occurs in the upper airway. The patient in case 1 had a semicircular extrathoracic goiter, and changes occurred on the MIFV curve in all three recumbent postures. The patient in case 2 had a large left-sided intrathoracic goiter and developed inspiratory and small expiratory changes in the right lateral posture. Therefore, in patients with a goiter and respiratory symptoms while recumbent, the FVL should be performed with the patient not only upright but also in the various recumbent positions in order to bring out physiologic evidence of upper airway flow limitation.

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**A Case of Acute Eosinophilic Pneumonia**

**Evidence for Hypersensitivity-Like Pulmonary Reaction**

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We examined an 86-year-old man with acute respiratory failure. A chest roentgenogram showed diffuse reticular shadows. Transbronchial biopsy revealed thickening of the alveolar septa accompanied by moderate eosinophil infiltration. After admission to the hospital, the patient's symptoms immediately improved

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without any medication. Clinical course and pathologic findings suggested acute eosinophilic pneumonia caused by a hypersensitivity reaction.

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Key words: acute respiratory failure; elderly; natural resolution; pulmonary eosinophilia

Abbreviations: AEP=acute eosinophilic pneumonia; TBBx=transbronchial lung biopsy

Most cases of eosinophilic pneumonia have been reported with a chronic course. However, in 1989 Badesch et al first described acute eosinophilic pneumonia (AEP) as acute in onset caused by a hypersensitivity reaction. In this report, we presented a case of AEP with acute respiratory failure in an 86-year-old man; this case of AEP was thought to be caused by a hypersensitivity reaction.

CASE REPORT

An 86-year-old man, an ex-smoker, was admitted to our hospital in mid-September 1992 because of high fever, dyspnea, wheezing, and dry cough of several days in duration. No medication had been taken before and after the onset of the symptoms. He had been in a very damp wooden house and had no history of contact with the animals or dry grass. A humidifier had not been used. There were no unusual findings in his past medical and family histories.

On admission, the results of the physical examination were as follows: body temperature, 38.2°C; BP, 126/80 mm Hg; pulse rate, 76 beats per minute; respiratory rate, 21 breaths per minute. End-expiratory wheezing was audible in the upper field of the right lung. Neither lymphadenopathy nor peripheral edema was noticed. The arterial blood gas analysis with the patient breathing room air showed acute respiratory failure with a pH value of 7.484, PaO₂ of 34.6 mm Hg, and PaCO₂ of 58.9 mm Hg. The peripheral blood examination showed a WBC count of 7,874/uL (normal range, 4,000 to 10,000/uL), with a differential cell count of 74.3% neutrophils (normal range, 41 to 64%), 12.6% lymphocytes (normal range, 20 to 46%), 3.6% eosinophils (normal range, 1 to 5%), 0.4% basophils (normal range, 0 to 2%), and 10.1% monocytes (normal range, 4 to 10%). Hemoglobin level was 12.6 g/dL (normal range, 14 to 18 g/dL). C-reactive protein was 7.7 mg/dL (normal range, <0.3 mg/dL) and IgE level was 2,700 U/mL (normal range, <250 U/mL).

Blood chemistry analysis revealed only an increase in lactate dehydrogenase (626 IU/L; normal range 180 to 460 IU/L). The angiotensin-converting enzyme and the total serum complement activity, CH50, were within normal limits. Trichosporon cutaneum and viral antibody tests, bacterial examination, and all of the precipitin tests were carried out extensively to rule out sensitivity to fungi, and antinuclear antibody tests were all negative. Pulmonary function tests disclosed the obstructive pattern, with 2.31 L (81.6%) for vital capacity, 0.66 L (50.0%) for FEV₁, 18.9% for percent maximal forced expiratory volume and 8.32 mL/M/mg Hg for the diffusing capacity of carbon monoxide.

At the time of admission, a chest roentgenogram showed diffuse interstitial reticular shadows, predominantly in the right lung (Fig 1, top), and a chest CT scan clearly demonstrated diffuse interstitial infiltrates (Fig 1, B). On the 3rd hospital day, BAL performed from right segmental bronchus 3 (B3) of anterior segment (S3) where interstitial reticular shadows were shown on the chest x-ray film and CT scan demonstrated 33.4% lymphocytes and 66.6% neutrophils, without an increase in eosinophils. BAL fluid was also cultured for bacteria, fungi, mycobacteria, viruses, and Legionella organisms; the cultures were negative for all these organisms.

After admission, a diagnosis of Japanese summer-type hypersensitivity pneumonitis was made based upon the chest roentgenogram, CT scan, and BAL fluid analysis findings; we then observed the patient free from medication except for antifebrile agents. Fever and cough resolved, and the inflammatory reaction was spontaneously diminished within a week. With the patient breathing room air, the arterial blood gas analysis improved; the pH value was 7.433; PaO₂, 37.3 mm Hg; and PaCO₂, 93.3 mm Hg. Peripheral eosinophil count, that was not increased on admission, showed the transient increase to 550/uL 5 days after admission. Four weeks later, the IgE level decreased to 1,200 U/mL and lactate dehydrogenase level was within normal limits. Almost all of the abnormal shadow on the chest roentgenogram and CT scan disappeared spontaneously.

Four of the specimens obtained by transbronchial lung biopsy (TBBx) from right B3a of S3 performed on the same day as BAL showed the thickening of the alveolar septa accompanied by the
infiltration of eosinophils and lymphocytes. There was no fibrosis or granuloma formation, or both, noticed in the biopsy specimen (Fig 2). In addition, returning home was performed as a challenge test immediately after curing the disease, and was negative. These findings allowed us to make a final diagnosis of AEP rather than HP. He had been followed up at the outpatient clinic and was in good health without any respiratory symptoms. A 1-year follow-up examination showed neither interstitial infiltration shadow on a chest CT scan nor an obstructive pattern as evidenced by the pulmonary function test (FEV1, 83.1%).

**Discussion**

Our case showed acute onset of high fever, hypoxia, diffuse pulmonary infiltrates, and immediate complete resolution without any medication in a week. BAL on admission showed no increase in eosinophils; however, TBBx clearly demonstrated alveolar septal thickening with moderate eosinophil infiltration. The granuloma formation was not detected in the specimen obtained by TBBx. Furthermore, both the challenge test and the serum *T cutaneum* antibody test were negative. These findings allowed us to make a diagnosis of AEP, not of Japanese summer-type hypersensitivity pneumonitis.2-5

The classification of pulmonary eosinophilia is still confusing because it depends on both etiology and clinical manifestations. AEP is recognized to be distinct from chronic eosinophilic pneumonia, and no predisposing cause or asthma.2,3 Our case showed no predisposing factors, such as drug use or extrinsic allergen exposure. Furthermore, there was no past medical history of bronchial asthma, and his pulmonary function test showed no abnormality without any steroid administration 1 year later.

At the onset of AEP, almost all of the cases showed no increase in eosinophils in the periphery, but the increase in eosinophils was seen in a few days after the onset of the disease. It is important to note that eosinophil dynamics may play a major role in this disease. Badesch et al2 suggested that a hypersensitivity-like pulmonary reaction manifested by a marked eosinophilic response occurred in the lung. They also reported that eosinophils could usually be detected in BAL fluid specimens about a week after the onset of the symptoms. In our case, bronchoscopic examination was performed only 3 days after the onset of the symptoms. We found both alveolar septal edema and eosinophil infiltrations in the TBBx and no eosinophils in the BAL fluid specimens. Therefore, we assumed that bronchoscopic examination was done in the early phase of this disease. Our findings might support the hypothesis of hypersensitivity-like reaction proposed by Badesch et al.2 The increase in IgE was also reported in some cases2 as well as in our case, suggesting that a unique inhaled antigen could produce this hypersensitivity reaction. However, its clinical role is yet to be elucidated. Results of a serum *T cutaneum* antibody test were negative in this case, indicating that AEP would be different from hypersensitivity pneumonia.6

In this case, local wheezes were found in the upper field of the right lung. However, none were detected in the five patients of Umeki with AEP,7 the four patients of Allen et

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**Figure 2.** TBBx obtained from right B3a of S3, showing alveolar septal thickening accompanied by moderate eosinophil infiltration (hematoxylin-eosin, original ×100).
al, or the one patient of Badesch et al. In this case, FEV₁ spontaneously improved from 50.0% on admission to 83.1% at the 1-year follow-up, suggesting that this patient had bronchiolitis obliterans in the acute phase of this disease.

AEP causes acute and severe respiratory failure. However, it is reported that the steroid therapy was very effective in almost all of the cases, and some of them as well as our case improved without any medications. In our case, there is also a possibility that this patient might be free from unknown antigen by the admission in the early phase of this disease based upon the results of TBBx and BAL fluid analysis.

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