Acute Eosinophilic Pneumonia Caused by Cigarette Smoking

We read a case report by Shintani et al (1), entitled “Acute eosinophilic pneumonia caused by cigarette smoking.” They also reported a similar case in Chest (2). In both reports, they cited our case report of cigarette smoke-induced eosinophilic pneumonia (CS-AEP), which appeared in abstract form in Jpn J Thorac Dis in 1997 (3). Because there seems to be a misunderstanding related to our report in their discussion, we would like to clarify the following facts.

They stated that no fever, significant chest radiological changes, or abnormal inflammatory data such as those of CRP and fibrinogen were observed in our case. However, our patient indeed manifested high fever (39°C) and severe dyspnea, and had abnormal WBC count, PaO₂, and pulmonary function test results after a provocation test with cigarette smoking (three cigarettes). These facts were detailed and clearly shown in Radiology in 1998 (4). Fifteen hours after the provocation test, the WBC count abruptly increased to 9,600/μm³ from 5,700/μm³, PaO₂ decreased to 56 mmHg from 93 mmHg, Dl,co (diffusing capacity of the lung for CO) decreased to 59% from 97%, and FEV₁% (expiratory volume in 1 second/forced vital capacity) decreased to 61% from 83% (Fig. 1) (3). We had to stop the provocation test at this point, because the provocation test induced obvious signs, symptoms, and laboratory abnormalities of pulmonary function, and possible serious complications were a major concern. After steroid treatment with methylprednisolone, these abnormal levels quickly returned to normal. Seriously abnormal serum data or the appearance of severely abnormal shadows on chest X-ray are not necessary to prove the connection of cigarette smoke to AEP. It should be realized that some patients with CS-AEP may experience severe respiratory failure and that a provocation test may culminate in a second episode of respiratory failure or even death.

At any rate, their report was not the first to present direct evidence of CS-induced AEP. Furthermore, it is apparent that their description of our case was inaccurate and that their literature search was insufficient. In 1997, other investigators and we (5) also reported cases of cigarette smoke-induced AEP in which the possibility of tolerance was suspected and discussed.

We agree with the issue that they raised on the importance of history taking. History of cigarette smoking should be sought in any patients with AEP, particularly in young patients. Moreover, it should be described in any case report, not only in those of AEP, but also in any other patients with respiratory symptoms. In CS-AEP, it is essential to know when the patient started smoking.

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Figure 1. Graphs of data obtained during a provocation test in cigarette smoke-induced acute eosinophilic pneumonia. Top: white blood cell (○) and eosinophil (●) count (in count x10⁹ per microliter) versus time. Middle: Arterial partial pressure of oxygen (○) and arterial partial pressure carbon monoxide (●). Bottom: Diffusion capacity of lung for carbon monoxide (▲) and forced expiratory volume in 1 second/forced vital capacity (●) versus time. Methyl PSL: methylprednisolone sodium succinate, and h: hour or hours.
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References