CASE REPORT

Acute Eosinophilic Pneumonia Associated with Smoke from Fireworks

Kazuya Hirai, Yoshitaka Yamazaki, Kazuyoshi Okada, Seiichi Furuta and Keishi Kubo*

Abstract

We report a case of acute eosinophilic pneumonia (AEP). Although the patient had been a habitual cigarette smoker for over 4 months, he had had not any respiratory distress. After he inhaled smoke from fireworks for 3 consecutive nights, the patient began to complain of cough, fever and dyspnea. He showed leukocytosis of 16,200/μl and hypoxemia of 58.1 torr. Chest radiograph showed bilateral infiltrates with Kerley A and B lines. The bronchoalveolar lavage fluid revealed 38.5% eosinophils. He was diagnosed as AEP. In this patient, inhaling of smoke from fireworks was clinically suspected to be associated with the induction of AEP.

Key words: bronchoalveolar lavage fluid, leukocytosis, interleukin (IL)-5, granulocyte macrophage colony stimulating factor (GM-CSF)

Introduction

Acute eosinophilic pneumonia (AEP) is generally characterized by an acute febrile illness, severe hypoxemia, diffuse pulmonary infiltrates, an increased number of eosinophils in bronchoalveolar lavage fluid (BALF), and an absence of infection and previous atopic illness (1). The pathophysiology of AEP remains unclear. Drugs, fungus and environmental agents have been implicated in the causes of AEP (2-4). Recently, several papers reported a relationship of cigarette smoking and the development of AEP (5, 6). Here, we report a case of AEP, which might have been associated with inhaling of smoke from fireworks.

Circulating neutrophilia during an early phase is usually seen in AEP (7-10), but the mechanism remains unclear. Cytokines such as interleukin (IL)-5 and granulocyte macrophage colony stimulating factor (GM-CSF) have been implicated in the pathophysiology of AEP (2, 3). To investigate the mechanism of this neutrophilia, we measured IL-5, IL-8, granulocyte colony stimulating factor (G-CSF) and GM-CSF in serum during the early and recovery phases in the present case of AEP.

Case Report

A 16-year-old Japanese man was admitted to our hospital complaining of high fever and dry cough on August 17, 1998. When he had gone camping on August 11 through 14, and he had ignited fireworks with his friends. He had inhaled smoke from fireworks for 3 consecutive nights. He felt cold on the third night of camping and returned home on August 14. The next day, he developed a high fever of 40°C and dry cough. On August 16, he consulted a physician. Circulating leukocytosis of 20,600/μl was detected and bilateral infiltrates were demonstrated on chest radiograph. Therefore, he was referred to our hospital. Although he had started smoking (approximately 5 cigarettes per day) 4 months before the illness, he had remained well. There was no sign of atopic illness.

On admission, the patient was 170 cm of height and weighed 49 kg. Temperature was 39.4°C, pulse rate 109 beats/min, and blood pressure 128/56 mmHg. Cardiac examination was entirely normal and auscultation of the lungs did not reveal any crackles or wheezes. There were no abnormal findings of the abdomen, extremities or nervous system. Laboratory examinations on admission showed the following results: hemoglobin (Hb) 15.4 g/dl, red blood cell (RBC) 529x10^4/μl, white blood cell (WBC) 16,200/μl, neutrophils 91.1%, lymphocytes 3.8%, monocytes 2.2%, basophils 1.7%, and eosinophils 1.2%, platelet count 22.7x10^4/μl, C-reactive protein (CRP) 8.1 mg/dl, IgE 529 U/ml. Arterial blood gas analysis under room air revealed; PaO2 58.1 torr, PaCO2 30.1 torr, pH 7.49. His chest radiograph showed bilateral infiltrates, mainly in peripheral lung zones associated with Kerley A and B lines (Fig. 1). Chest CT scans revealed patchy non-segmental shadows with predominant outer-zone involvement and right pleural effusion. In addition, high-resolution CT scan revealed interlobular septal thickening (Fig. 2). He underwent bronchoscopic examina-
Hirai et al

Figure 1. Chest radiograph reveals diffuse infiltrates with Kerley A and B lines.

Figure 2. Chest CT scan shows patchy and non-segmental shadows with a predominant outer zone and interlobular septal thickening.

Table 1. Laboratory Data during the Early and Recovery Phases in a Case of Acute Eosinophilic Pneumonia

<table>
<thead>
<tr>
<th></th>
<th>Early phase</th>
<th>Recovery phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (/μl)</td>
<td>16,200</td>
<td>4,100</td>
</tr>
<tr>
<td>Neutrophil (%)</td>
<td>91.1</td>
<td>43.8</td>
</tr>
<tr>
<td>Lymphocyte (%)</td>
<td>3.8</td>
<td>35.9</td>
</tr>
<tr>
<td>Eosinophil (%)</td>
<td>1.2</td>
<td>10.1</td>
</tr>
<tr>
<td>Serum levels of;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL-5 (pg/ml)</td>
<td>490</td>
<td>&lt;7.8</td>
</tr>
<tr>
<td>IL-8 (pg/ml)</td>
<td>&lt;10</td>
<td>&lt;10</td>
</tr>
<tr>
<td>GM-CSF (pg/ml)</td>
<td>4.18</td>
<td>&lt;2</td>
</tr>
<tr>
<td>G-CSF (pg/ml)</td>
<td>&lt;5</td>
<td>&lt;5</td>
</tr>
</tbody>
</table>

WBC: circulating white blood cells, IL-5 and IL-8: interleukin-5 and -8, respectively, GM-CSF and G-CSF: granulocyte macrophage- and granulocyte-colony stimulating factor, respectively.

Discussion

Although drugs, inhalation of fungus and environmental agents (2-4) have been proposed as causes of AEP, the pathogenesis of AEP remains unclear. Recently, several papers reported that, especially in young people, AEP developed 2 days to 2 months after they began cigarette smoking (5, 6). However, only a few smokers develop AEP, so there remains room for discussion on the cause of AEP. In this patient, although he had started smoking 4 months before the current illness, he had remained well, and he had smoked only one cigarette on the first day of camping. He had inhaled smoke from fireworks for 3 consecutive nights, and felt cold on the third night of camping. Thereafter, he developed AEP. In addition, when he smoke...
resumed cigarette smoking after discharge, there was no recurrence of AEP. From this evidence, inhaling of the smoke from fireworks was clinically thought to have been the cause of AEP.

Taniguchi et al (10) reported that an elevated level of activated CD4+ lymphocytes in the lungs may play an important role in eosinophil accumulation through IL-5 production in AEP. IL-5 and GM-CSF are potent TH-2 cytokines, which regulate both recruitment and activation of eosinophils. It seems that generation of eosinophil-activating cytokines in the lung, such as IL-5 and GM-CSF, is central to the pathophysiology of AEP.

During the early phase of AEP, circulating neutrophilia was seen in most cases reported to date (7–10). The mechanism, however, is not clear. We evaluated IL-8, G-CSF and GM-CSF levels in serum during the early and recovery phases. IL-8, G-CSF and GM-CSF are potent cytokines, which attract and activate neutrophils in the peripheral blood. Although G-CSF and IL-8 in serum were within the normal range during both phases, GM-CSF increased remarkably during the early phase. Furthermore, the GM-CSF concentration in BALF collected on the day of admission was also elevated. Therefore, it is speculated that GM-CSF, which was produced by macrophages and/or lymphocytes in the lungs, induced the proliferation and differentiation of hematopoietic progenitor cells, and probably led to leukocytosis.

In summary, a 16 year-old man presented with AEP after inhaling smoke from fireworks for 3 consecutive nights. IL-5 and GM-CSF were remarkably elevated in peripheral blood as well as BALF. It seems likely that GM-CSF plays an important role in circulating neutrophilia during the early phase of AEP.

References