THE CASE
A 16-year-old boy presented to our hospital with a 2-day history of acute onset of cough and dyspnea. He denied chest pain, sputum, fever and chills. Past medical history was unremarkable. He reported no history of allergy, medications, inhalational of volatile liquids or gasses, illicit drugs, or smoking.

On examination, blood pressure was 120/60 mmHg, pulse of 142 bpm, respiratory rate of 24 per minute, and body temperature of 38.5 degrees Celsius. Late-inspiratory fine crackles were audible on bilateral lung fields. Heart sounds were regular and there were no murmur, gallops, or rubs.

Oxygen saturation on breathing room air revealed 93%. Chest roentgenogram and computed tomographic scan showed ground-glass infiltrates in peripheral lung zones mainly in the right upper lobe of the lung (Figures A, B, C). Laboratory data showed leukocytosis without eosinophilia (Table 1). Both urine Pneumococcal and Legionella antigens were negative. Serum antibodies against Mycoplasma pneumoniae and Chlamydophila pneumoniae were sent.

Based on the findings of the imaging study, acute eosinophilic pneumonia (AEP) was suspected and

Figure 1. Chest X-ray (A) and computed tomographic scan (B and C) show infiltrates in the peripheral lung zones of the lung’s right upper lobe.
bronchoscopy was conducted for collecting bronchoalveolar lavage (BAL) fluid. The BAL analysis revealed a high eosinophil count (neutrophil 4%, lymphocyte 16%, eosinophil 72%, and histiocyte 8%). The diagnosis was confirmed as AEP. Because of his severe tachycardia (about 140 bpm) and tachypnea (about 25 per minute), progression into respiratory failure was anticipated and thus steroid therapy was initiated. Subsequent laboratory reports eventually came back and showed that serum antibodies against Mycoplasma pneumoniae and Chlamydia pneumoniae were not elevated.

During further history taking, the patient confessed that he started smoking a week before the appearance of the symptoms. He commented that he had a desire to smoke out of curiosity. His condition rapidly improved after a brief course of prednisone (initially 30 mg/day, followed by rapid tapering). He also received sessions of motivational interviewing for smoking cessation. At follow-up clinics after discharge from the hospital, he reported that he had successfully quit smoking.

**DISCUSSION**

The importance of history taking was confirmed in this case. AEP should be considered when patients present with acute respiratory illness and ground glass infiltrates in peripheral lung zones, following cigarette smoking.1,2 As teenagers may want to hide the fact that they have smoked, it may be necessary to conduct detailed history taking to determine whether they have smoked or not. It is also important since patients may not know their condition is related to their smoking. Smoking cessation may lead to prevention of AEP relapse. Moreover, smoking cessation education can be strongly advised using this causation association.

Cigarette smoking was considered the cause of AEP in this patient. Uchiyama et al reported that patients usually develop the symptoms of AEP associate with tobacco smoking from several days to 1-2 months after initiating smoking or after increasing the amount of tobacco smoking.3 There have not been any cases reported of AEP associated with passive smoking. 

Patients with AEP might have undertaken a variety of activities within the days before onset of the disease, such as cave exploration, plant repotting, woodpile moving, smoke-house cleaning, motocross racing in dusty conditions, indoor renovation work, tank cleaning, exposure to tear gas, or respiratory dust exposure.4 The patient denied any of these exposures before developing his symptoms. Furthermore, smoking has been considered as a major cause of AEP in young adults.
In terms of evaluating diagnostic criteria (5 items) for idiopathic acute eosinophilic pneumonia this patient fulfilled the following 4 items: 1) acute onset of febrile respiratory manifestations (≤1 month, and especially ≤7 days duration before medical examination); 2) bilateral diffuse infiltrates on chest radiograph; 3) lung eosinophilia, with >25% eosinophils on BAL differential cell count (or eosinophilic pneumonia at lung biopsy); and, 4) absence of determined cause of acute eosinophilic pneumonia (including infection or exposure to drugs known to induce pulmonary eosinophilia). Although the patient did not show hypoxia with oxygen saturation on room air of less than 90%, his oxygen saturation on breathing room air revealed 93%, which indicated impending hypoxia based on his young age.

Regarding a diagnosis of AEP, a high index of suspicion should be required. Although there was no eosinophilia in peripheral blood in this patient, increased eosinophils in BAL fluid can establish correct diagnosis of AEP. Thus, if there is a possibility of AEP, it is advisable to perform BAL even in the absence of peripheral eosinophilia.

References