Chronic Eosinophilic Pneumonia : A Case Report

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Abstract

Chronic eosinophilic pneumonia (CEP) is a disorder, characterized by a history of pneumonia (> 2 months) and eosinophilic pulmonary infiltration without any organic causes. We describe a 28-year-old woman who presented with cough, dyspnea and fever for 2 months. She was diagnosed with mild asthma and allergic rhinitis 2 years before being diagnosed with CEP. For a period of 9 months she took no medication. Her chest roentgenogram at this admission revealed patchy infiltration in both upper lung fields. Laboratory data revealed blood eosinophilia (4,284 /mm^3), and her serum IgE was mildly elevated (245.8 IU/ml). A computerized tomography of the chest did not show bronchiectasis. CEP was diagnosed from significant eosinophilia in bronchoalveolar larvage fluid and transbronchial biopsy revealed eosinophilic infiltration without any demonstrable infectious agent. The patient was treated with prednisolone 45 mg/day. Her symptoms disappeared and her chest roentgenogram showed nearly complete resolution in 2 and 4 days, consecutively.

Key word : Eosinophils, Pneumonia

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Chronic eosinophilic pneumonia(CEP) is a rare disorder in Thailand. We should consider this disease, when the patient presents with chronic pneumonia and peripheral blood eosinophilia without organic causes.[1] We report a patient with CEP who had a typical clinical presentation.

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CASE REPORT

A 28-year-old woman was admitted to Srinakarind Khon Kaen University Hospital because of chronic cough, dyspnea, and fever for two months. Two months previously she noticed a progressive non productive cough, sometimes a change in the color of the sputum, and low-grade fever. The patient had been treated at the district hospital but the symptoms did not improve. Thereafter, the patient was admitted to find the cause of her symptoms. Upon her first admission, the laboratory test revealed peripheral blood eosinophilia. Sputum examination was negative for acid-fast bacilli, but the culture grew numerous Klebsiella pneumoniae. The patient received cotrimoxazole as initial treatment. Fifteen days after treatment, however, the patient complained of cough and dyspnea, which lead to a further admission. Two years previously she has been diagnosed with a mild case of asthma and allergic rhinitis, and was treated with an inhaler and nasal spray corticorsteroid. For a period of 9 months, she took no medication. She had no history of smoking.

Physical examination revealed a young woman with mild tachypnea. Her temperature was 38°C. Her blood pressure was 120/80 mmHg, pulse 100 beats/min, and respiratory rate 40/min. The ear, nose and throat examination was normal. Her lymph node could not be palpated. Chest examination revealed dullness on percussion in left upper lung fields with a decrease in breath sound and vocal resonance. Auscultation of the lungs revealed fine crepitation in the left upper lung fields. The cardiac, abdominal, and neurological examinations were within normal limits. There was no skin rash, digital clubbing, cyanosis, or edema.

Laboratory tests revealed, hemoglobin 13.7 g/dl, white blood cell count 12,600 /mm^3 with 45 per cent neutrophils, 18 per cent lymphocytes, 34 per cent eosinophils, 2 per cent monocytes, 1 per cent basophils ; platelet count was slightly increased. Urinary examination and serum chemistries were within normal limits. In the stool examination, there were no parasites present. The erythrocyte sedimentation rate was 35mm/hour. Serum IgE levels were mildly elevated (245.8 IU/ml). Serology test for autoimmune was negative. Chest radiograph demonstrated bilateral patch infiltration, more on the left upper lung fields. (Fig. 1A) Sputum examination revealed numerous eosinophils without any demonstrable infectious agent. Fiberoptic bronchoscopy was performed, which revealed a normal tracheobronchial tree. Bronchoalveolar larvage (BAL) yielded a cloudy fluid with a total cell count of $0.9x10^6$/L. The differential count was 9 per cent neutrophils, 1 per cent lymphocytes, 5

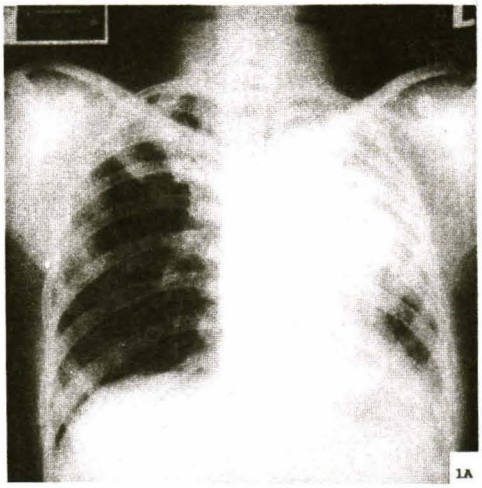

Fig. 1A. The first chest radiography on admission demonstrated bilateral patchy infiltration, more on left upper lung fields.

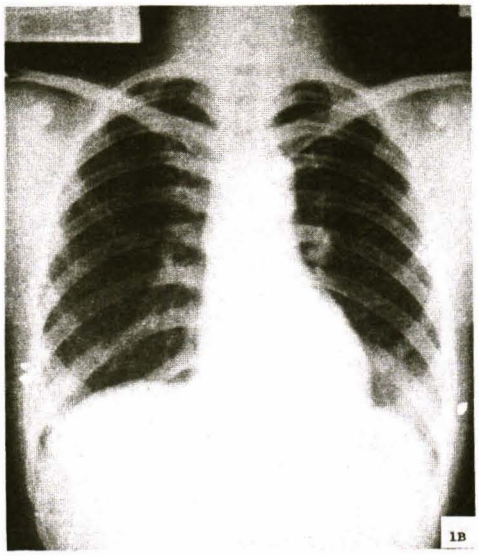

Fig. 1B. On the fourth hospital day after treatment, the follow-up chest radiography was essentially normal.

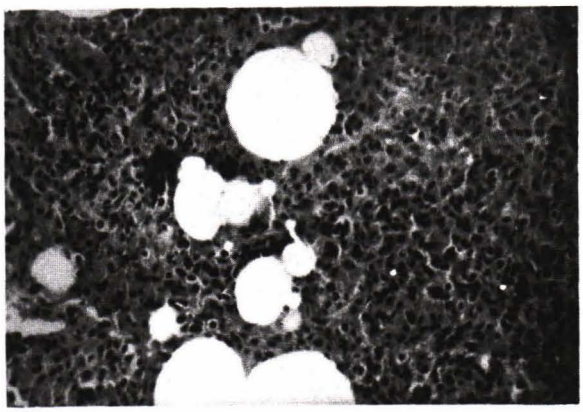

Fig. 2. Microscopic examination of transbronchial lung biopsy specimens show infiltration of eosinophils into the bronchi and alveoli.

per cent macrophages, and 85 per cent eosinophils. There were no parasites found in a fresh smear of the BAL fluid. BAL fluid stains and cultures were negative for bacteria, mycobacterium and fungi.

Microscopic examination of transbronchial lung biopsy specimens showed infiltration of eosinophils into the bronchial and alveoli. (Fig. 2) After clinicopathologic diagnosis, the patient received prednisolone (45 mg/d), and two days later the symptoms had disappeared. On the fourth hospital day, the chest roentgenography was essentially normal (Fig. 1B), and she was discharged. She was apparently stable clinically, when prednisolone was gradually reduced to a low-dose regimen.

DISCUSSION

Eosinophilic pneumonia has been defined as pulmonary infiltration of the lung by eosinophils that may be accompanied by an excess of these cells in the peripheral blood[2]. The classic presenting features having been first described by Carrington et al in 1969[1]. Recently, its criteria for diagnosis based on clinico-pathohistologic findings have been revised. Umeki in 1992,[3] proposed CEP following this diagnostic criteria : Major criteria (essential) (1) history of cough, fever, and dyspnea, (2) history (>2months) of symptoms prior to diagnosis, (3) peripheral infiltration (patchy, nodular, nonsegmental, shifting, or recurrent) on chest roentgenograms, which sometimes resemble photonegative pulmonary edema, (4) a prolonged clinical course, and (5) occasional recurrence. In addition, minor criteria (not essential) for diagnosis of CEP include the following findings : (1) blood eosinophilia greater than 0.5×10^6 / L , (2) BAL eosinophils greater than 15 per cent, (3) absence of organic disorder (e.g., helminthic, mycotic, bacterial, and other infections, collagen disease, allergic bronchopulmonary aspergillosis, sarcoidosis, Hodgkin's disease), and (4) corticosteroid responsiveness, (5) associated with asthma.

The majority of CEP cases occur in women (2:1 ratio). The peak incidence is in the fifth decade but not uncommon in young adults[1]. From our case and those of Umeki et al, CEP can occur in young adults[4]. Before making a diagnosis in this case, we made a search for evidence of the causation, as mentioned by the criteria above[5]. The accurate cause of CEP is unknown, and assumed to represent some alteration in immunologic response or allergic reaction[3]. Prin et al 1986, demonstrated that eosinophils in patients with CEP are activated and then release eosiniphilic protein (EP)[6,7]. The macrophage uptakes EP and distributes it to it's different compartments,which protects the alveolar walls by preventing a high extracellular concentration of eosinophil protease, which is necessary for these proteins to exert their cytotoxic effect on lung parenchyma[8]. Despite this progress, there is incomplete knowledge about the protective and deleterious effects of eosinophils in the lung.

Blood eosinophilia is found in 80 per cent of cases[9], and do not provide direct evidence of eosinophilic infiltration into the lung. In the present study, BAL was the important tool in the diagnosis of CEP. Eosinophils in BAL fluid are reported to increase in CEP range 30-40 per cent[10], and the value in our case was higher than these earlier reports. Chest roentgenography provides the initial provisional diagnosis of CEP. In particular, the presence of a photographic negative pulmonary edema pattern is considered highly diagnostic of CEP, although the frequency of its appearance is only 25 per cent[11]. The other radiographic findings are pulmonary nodules with or without cavitation (20%) and pleural effusion (<10%)[10]. Mediastinal lymph node enlargement can be seen[12]. Histologic examination of CEP reveals an interstitial and alveolar infiltration with eosinophils, macrophage, and multinucleated giant cells[1,12]. Corticosteriod dosage equivalent to prednisolone 20 to 60 mg daily, will result in rapid resolution of symptoms

within 1 week and radiological improvement within 3 weeks[13]. The striking feature of CEP is the relapse of the disease. So, after remission has been induced, the patient should be treated with low dose corticosteroid therapy[1]. From the study of Naughton at al 1993, the mean duration of treatment was 10.2 years (range 1 to 13 years), and maintenance dosage of prednisolone was 2.5 to 10 mg[13]. Although the majority of patients will require treatment for many years, in some cases, corticosteroid treatment can be successfully tapered off in a few years. Nowadays, there is no predictor to determine subgroups of patients, who can be tapered off corticosteriod treatment in a few years. Jacderlinic et al recommended at least 6 months of corticosteroid therapy,begining with a high-dosage tappering to a low-alternate day dosage[14].

SUMMARY

We report a patient with CEP who had a typical clinical presentation. To our knowledge, this may be the first report in the Thai population. Diagnosis of CEP is not difficult. The typical clinical picture and blood eosinophilia, together with pulmonary infiltration, are usually sufficient to make the diagnosis[15].

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ปอดอักเสบเรื้อรังจากอีโอซิโนฟิล : รายงานผู้ป่วย

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ผู้ป่วยหญิงอายุ 28 ปี มาโรงพยาบาลด้วยอาการไข้ ไอ หายใจหอบมา 2 เดือน ผู้ป่วยเคยได้รับการวินิจฉัยว่าเป็นโรคภูมิแพ้และหอบหืดได้รับการรักษาด้วยยาพ่นสูดชนิดสเตียรอยด์จนมีอาการเป็นปกติโดยไม่ได้รับยาใดๆเลยเป็นเวลา 9 เดือน ภาพรังสีทรวงอกแรกรับพบฝ้าขาวที่ปอดกลีบบนทั้งสองข้าง ตรวจเลือดพบเม็ดเลือดขาวอีโอซิโนฟิลสูง, ตรวจเสมหะ, น้ำล้างหลอดลมและถุงลมปอดพบเม็ดเลือดขาวอีโอซิโนฟิลสูงโดยไม่พบเชื้อก่อโรค ผลการตัดชิ้นเนื้อปอดผ่านหลอดลมพบการอักเสบจากอีโอซิโนฟิล ระดับค่า IgE ในเลือดสูงเล็กน้อย (245.8 IU/ml) ภาพรังสีคอมพิวเตอร์ทรวงอกไม่พบหลอดลมโป่งพอง จึงให้การวินิจฉัยว่าเป็นปอดอักเสบเรื้อรังจากอีโอซิโนฟิล ผู้ป่วยตอบสนองดีมากต่อการรักษาด้วยคอร์ติโคสเตียรอยด์ขนาด 45 มก.ต่อวัน โดยมีภาพรังสีทรวงอกใกล้เคียงปกติภายในวันที่ 4

คำสำคัญ : อีโอซิโนฟิล, ปอดอักเสบ

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