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CORRESPONDENCE

Reversing rapidly deteriorating lung function in eosinophilic bronchiolitis by pulse steroid and anti-IgE therapy



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Eosinophilic lung diseases are a heterogeneous group of pulmonary disorders that include bronchial asthma, fungal or parasitic infections, drug or toxin reactions, eosinophilic vasculitis, and eosinophilic pneumonia.¹ A new disease entity known as eosinophilic bronchiolitis was first reported in Japan in 2001 with the following characteristics: (1) asthmatic-like symptoms that do not respond to asthma treatment guidelines; (2) diffuse centrilobular nodules in chest computed tomography (CT); and (3) eosinophilic bronchiolar involvement on histopathologic examination.² Until now, only seven cases have been reported in the literature and corticosteroid treatment has had equivocal results.³

A 22-year-old woman was referred to an allergy clinic due to difficult-to-treat severe asthma and rapidly deteriorating lung function. She did not have asthma before but developed dyspnea, asthmatic wheezing, and productive cough once she started working as a cleaner 2 years ago. Occupational asthma was suspected, but her pulmonary

function deteriorated rapidly despite leaving her job and having received regular asthmatic medications according to the Global Initiative For Asthma (GINA) guidelines.

Physical examination revealed intractable subcostal retraction, diffuse wheezing, and difficult breathing. Laboratory examination showed marked eosinophilia (47%) and a high serum immunoglobulin (Ig) E level (5700 IU/ml). Bronchoscopy did not reveal any anatomical anomalies, but instead showed eosinophilia (12%) in the bronchoalveolar lavage fluid. Stool ova and parasite test was negative. Sputum culture showed a heavy mix of normal flora and *Candida albicans*. CT showed diffuse pulmonary centrilobular nodules bilaterally (Fig. 1A) and sinusitis. Severe asthma with airway remodeling, allergic bronchopulmonary mycosis, sinobronchial syndrome, and diffuse pan-bronchiolitis were suspected but sequentially ruled out because her lung functions deteriorated despite specific treatments for these diseases.

Finally, lung biopsy revealed chronic diffuse bronchiolitis with marked peribronchiolar eosinophilic infiltration (Fig. 1B and C), which was compatible with eosinophilic bronchiolitis. However, there was no improvement of her lung condition even with oral prednisone (60 mg/day) treatment for 2 months, which had been useful in previously reported cases.³ Methylprednisolone pulse therapy at a dose of 1 g/day for 3 days was applied with improvement

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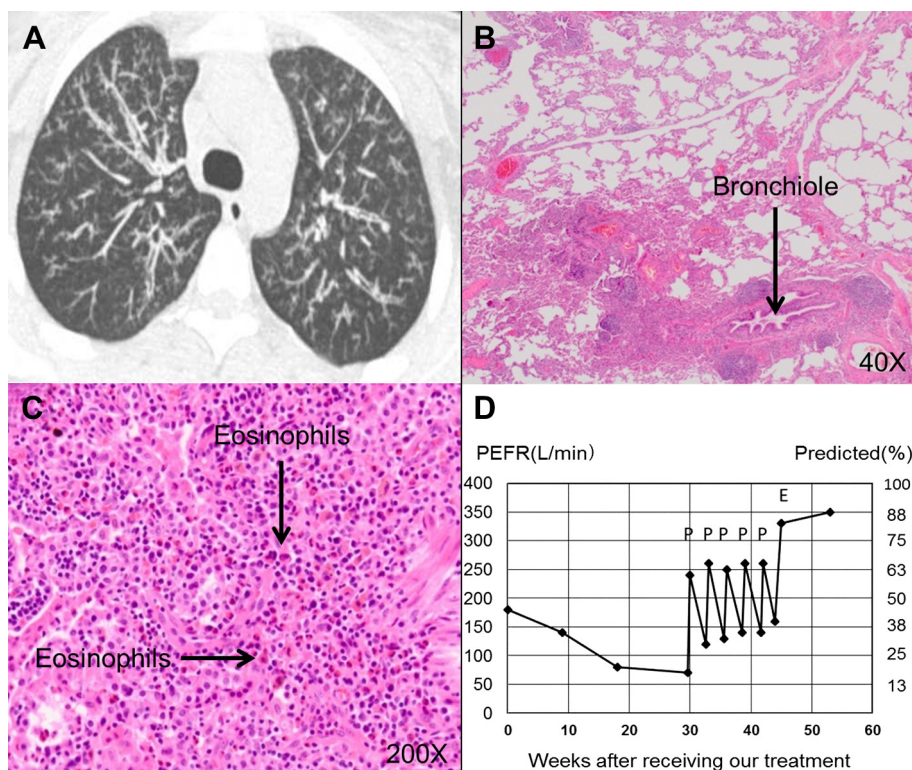


Figure 1 (A) Chest CT shows diffuse centrilobular nodules; (B,C) histology shows peri-bronchiolar infiltration with many eosinophils (hematoxylin and eosin stain), B: $\times 40$; C: $\times 200$); (D) PEFR of the patient after receiving our treatment. The predicted PEFR is 400 L/minute. P = pulse methylprednisolone therapy (1 g/day for 3 days); E = anti-IgE therapy (omalizumab 450 mg every 2 weeks); CT = computed tomography; Ig = immunoglobulin; PEFR = peak expiratory flow rate.

of lung functions and decreased serum eosinophils and IgE. However, this effect only lasted 2–3 weeks. The patient needed additional pulse methylprednisolone therapy every 3 weeks to maintain her pulmonary function. After five courses of pulse methylprednisolone therapy, her total IgE decreased to 922 IU/ml. An anti-IgE monoclonal antibody (omalizumab) was used at a dose of 450 mg every 2 weeks, which further led to significant improvement of her lung functions even without further pulse steroid therapy (Fig. 1D).

To date, this is the first case report of eosinophilic bronchiolitis that has been successfully treated by pulse steroid and anti-IgE therapy. A progressive decrease in eosinophils and IgE after pulse steroid therapy was found. Therefore, anti-IgE monoclonal antibody therapy can be applied because omalizumab is only approved in IgE between 30–1500 IU/ml.⁴ It is surprising to find significant improvement of lung function with anti-IgE therapy inasmuch that further pulse steroid therapy is not needed.

In conclusion, pathologic examination is important in the diagnosis of eosinophilic bronchiolitis. Anti-IgE may be a useful regimen for eosinophilic bronchiolitis.

References

- Jeong YJ, Kim KI, Seo IJ, Lee CH, Lee KN, Kim KN, et al. Eosinophilic lung diseases: a clinical, radiologic, and pathologic overview. *Radiographics* 2007;**27**:617–37.
- Takayanagi N, Kanazawa M, Kawabata Y, Colby TV. Chronic bronchiolitis with associated eosinophilic lung disease (eosinophilic bronchiolitis). *Respiration* 2001;**68**:319–22.
- Fukushima Y, Kamiya K, Tatewaki M, Fukushima F, Hirata H, Ishii Y, et al. A patient with bronchial asthma in whom eosinophilic bronchitis and bronchiolitis developed during treatment. *Allergol Int* 2010;**59**:87–91.
- Burton AJ, Hargadon B, Murphy AC, Brightling CE, Pavord ID, Wardlaw AJ, et al. The eligibility of patients with difficult asthma for omalizumab since the change to the treatment criteria. *Thorax* 2010;**65**:A84–5.