Neutrophilic Endotheliitis in Patients With Idiopathic Pulmonary Fibrosis

To the Editor:

Idiopathic pulmonary fibrosis (IPF) is a disease of unknown cause that is characterized by the accumulation of neutrophils and mononuclear cells, followed by the progressive deposition of collagen within the interstitium and subsequent destruction of lung airspace. Patients with IPF have an expected median survival of less than 5 years. Increases in polymorphonuclear neutrophils (PMN) in bronchoalveolar lavage fluid (BALF) and in lung tissue have been shown in patients with IPF. Excessive neutrophils in BALF have been associated with a higher likelihood of disease progression and a failure to respond to immunosuppressive therapy. Therefore, IPF has been considered to be a neutrophilic alveolitis since neutrophils may play an important role for the pathogenesis of IPF. However, it was also suggested that the number or proportion of PMN in BALF does not correlate with activity of alveolitis and has limited prognostic value.

To evaluate the site of neutrophil accumulation in patients with IPF, specimens were obtained by open lung biopsy from five patients with IPF and were immunohistochemically stained using antihuman neutrophil elastase (DAKO, M752). Neutrophil elastase stains were detected around the honey-combing, as well as the alveolar septa. Interestingly, strong staining of neutrophils was observed along to the endothelium in pulmonary microvessels (Fig 1). This evidence was not observed in patients with pneumonia. These observations suggest that activation of neutrophils at the site of the endothelium of pulmonary microvessels takes place in patients with IPF. In addition, this evidence may explain the conflict of the prognostic significance of BALF neutrophils. In conclusion, we think that “neutrophilic endotheliitis” as part of “neutrophilic alveolitis” would be important pathogenic character of IPF.

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We Need More Studies on Colchicine and Airway Reactivity

To the Editor:

I read with interest the article in CHEST April 1995, Effects of Colchicine on IgE-Mediated Early and Late Airway Reactions by Dr. Kelly and colleagues (CHEST 1995; 107:985-91).

In my practice, I have seen four previously steroid-dependent patients who I have been able to discontinue taking steroids by using colchicine 0.6 mg bid as an antiinflammatory agent. Of course, when the patients have had exacerbations of acute bronchitis or pneumonia or both, I have had to reintroduce pulse therapy with prednisone after a dose of intravenous methylprednisolone sodium succinate, but these patients have at this time remained on colchicine but have not continued taking oral prednisone.

Dr. Kelly’s patients were nine “moderately allergic asthmatic subjects.” My patients are certainly not moderately allergic, but instead are the sicker patients we pulmonologists typically see in our offices referred by other physicians who just “can’t get these patients off steroids.”

Consequently, I look forward to controlled studies on this topic. In the meantime, I will continue to use this mode of therapy for my patients.

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Regular β-Adrenergic Agonists

To the Editor:

Some 30 years ago we found that mortality from asthma was rising. There was no obvious reason for this and therefore a large number of investigations were carried out all over the world. No clear cut cause emerged and thus iatrogenesis was considered as one of the possible causes. β-Agonist sympathomimetics in aerosol form and adrenal corticosteroids were the two new agents introduced at about the same time. Suspicion should logically have fallen on these two but only β-agonists were criticized. Apart from stray voices1,2 most looked on steroids as life saving agents and thus beyond re-approach. After much debate and therapeutic corrective, the