74. **Kasuga I,** Pare PD, Ruan J, *et al*. Lack of association of group specific component haplotypes with lung function in smokers. *Thorax* 2003;**58**:790–3.
75. **Parmar JS,** Mahadeva R, Reed BJ, *et al*. Polymers of alpha(1)-antitrypsin are chemotactic for human neutrophils: a new paradigm for the pathogenesis of emphysema. *Am J Respir Cell Mol Biol* 2002;**26**:723–30.
76. **Mulgrew AT,** Taggart CC, Lawless MW, *et al*. Z alpha1-antitrypsin polymerizes in the lung and acts as a neutrophil chemoattractant. *Chest* 2004;**125**:1952–7.
77. **Chappell S,** Daly L, Morgan K, *et al*. Cryptic haplotypes of SERPINA1 confer susceptibility to chronic obstructive pulmonary disease. *Hum Mutat* 2006;**27**:103–9.
78. **Keatings VM,** Cave SJ, Henry MJ, *et al*. A polymorphism in the tumor necrosis factor-alpha gene promoter region may predispose to a poor prognosis in COPD. *Chest* 2000;**118**:971–5.
79. **Sakao S,** Tatsumi K, Igari H, *et al*. Association of tumor necrosis factor alpha gene promoter polymorphism with the presence of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2001;**163**:420–2.
80. **Kucukaycan M,** Van Krugten M, Pennings HJ, *et al*. Tumor necrosis factor-alpha +489G/A gene polymorphism is associated with chronic obstructive pulmonary disease. *Respir Res* 2002;**3**:29.

Pulmonary puzzle

ANSWER

From the question on page 290

The total white cell count of the BAL fluid was 184/mm^3 with 54% eosinophils. Figure 1 shows clusters of granulated and degranulated eosinophils within a background of septal oedema. Comprehensive examination of the BAL fluid and biopsy material yielded no clear infectious cause. These findings are consistent with a diagnosis of acute eosinophilic pneumonia (AEP), which is characterised by progressive respiratory symptoms, diffuse bilateral infiltrates and increased numbers of lung eosinophils in the absence of detectable infection.[1] Biopsy, although not necessary for diagnosing AEP, may be useful for ruling out other causes of pulmonary eosinophilia. Other entities to consider in patients with acute progressive respiratory symptoms and increased numbers of eosinophils in the BAL fluid include drug reactions and fungal and parasitic infections.

The clinical course typically begins with fever averaging 38°C, cough, tachypnoea, hypoxaemia and dyspnoea and often progresses rapidly. Plain radiographs may show an interstitial, alveolar or mixed pattern and pleural effusions are common.[1 2] Environmental, occupational and recreational exposures are thought to play a significant role in the pathogenesis of AEP. A number of authors have described the development of AEP shortly after the onset of smoking.[1 3 4] AEP has also been reported in a patient who became symptomatic shortly after participating in a bicycle race "in dusty conditions".[5] Our patient had both exposures, as he later admitted to trying cigarette smoking for the first time the evening before his symptoms developed.

At presentation the patient had increased peripheral blood neutrophils, a phenomenon previously reported in cigarette smoke-induced AEP.[4] While peripheral blood eosinophilia characteristically does not occur during the acute phase of AEP, levels may become quite high during convalescence before normalising.[2] The patient's peripheral blood absolute eosinophil count was 1380/mm^3 1 week after discharge. On rechecking 5 months later, it had returned to the normal range.

While individuals with AEP classically show clinical improvement after the initiation of steroids, most who are treated with only supportive measures also recover completely.[2] Although case reports have described the return of symptoms upon re-exposure to an inciting factor,[3] most patients develop tolerance and do not have recurrence.[1 2] We advised our patient to avoid cigarette smoke altogether. His symptoms resolved completely with no lasting pulmonary function abnormalities after a course of steroids.

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REFERENCES

1. **Allen J.** Acute eosinophilic pneumonia. *Semin Respir Crit Care Med* 2006;**27**:142–7.
2. **Philit F,** Etienne-Mastroianni B, Parrot A, *et al*. Idiopathic acute eosinophilic pneumonia: a study of 22 patients. *Am J Respir Crit Care Med* 2002;**166**:1235–9.
3. **Watanabe K,** Fujimura M, Kasahara K, *et al*. Acute eosinophilic pneumonia following cigarette smoking: a case report including cigarette-smoking challenge test. *Intern Med* 2002;**41**:1016–20.
4. **Miki K,** Miki M, Nakamura Y, *et al*. Early-phase neutrophilia in cigarette smoke-induced acute eosinophilic pneumonia. *Intern Med* 2003;**42**:839–45.
5. **Badesch DB,** King TE Jr, Schwarz MI. Acute eosinophilic pneumonia: a hypersensitivity phenomenon? *Am Rev Respir Dis* 1989;**139**:249–52.