A 49-year-old woman had been treated with carbamazepine for 2 years because of epilepsy. She was referred to us for progressive exertional dyspnea and prolonged productive cough. Chest computed tomography (CT) scan showed bilateral infiltrates including ground glass opacities and consolidations predominantly in the lower lung fields. Her laboratory findings showed severe hypogammaglobulinemia, that is, immunoglobulin (Ig) G 418 mg/dl (normal, 748–1694 mg/dl), Ig A 20 mg/dl (91–391 mg/dl) and Ig M 51 mg/dl (33–254 mg/dl). Carbamazepine and other suspected antibiotics were all negative for drug-induced lymphocyte stimulation tests. Histological examination by trans-bronchial lung biopsy showed intraluminal fibrosis of distal airspaces with foamy alveolar macrophages, suggesting bronchiolitis obliterans organising pneumonia (BOOP). After the cessation of carbamazepine, all abnormalities in gammaglobulins and roentgenogram findings gradually improved without any medication. This good clinical course also considerably supports the diagnosis of BOOP.

BOOP may result from diverse causes such as drugs, acute respiratory infections and radiation treatment, or appear idiopathically. Here, we show a case of secondary BOOP, which was associated with repeated respiratory infections caused by carbamazepine-induced hypogammaglobulinemia. Although the exact mechanisms of carbamazepine-induced hypogammaglobulinemia are unknown, they can be classified into three groups, that is, an absence of B cells, an extensive impairment of the synthesis of Igs in B cells, and a disorder of the class-switch of Igs in B cells. Our case described above would belong to the second group. Generally, drug-induced BOOP often develops within several weeks or less. However, our report indicates that even in the case of several years after use, anticonvulsants such as carbamazepine may have some adverse effects on the immune system and cause frequent airway infections, resulting in the development of secondary BOOP.

**ACKNOWLEDGEMENTS**

The authors acknowledge Mr Brent K. Bell for reading the manuscript.

T Tamada, M Nara, M Tomaki, Y Ashino, T Hattori, Division of Infectious and Respiratory Diseases, Department of Internal Medicine, Tohoku University Hospital, Sendai, Japan

Correspondence to: Tsutomu Tamada, Division of Infectious and Respiratory Diseases, Department of Internal Medicine, Tohoku University Hospital, Seiryo-machi, Aoba-ku, Sendai 980-8574, Japan; tamada@rid.med.tohoku.ac.jp

doi: 10.1136/thx.2006.063842

Competing interests: None declared.

**REFERENCES**