classification of gout that may be used by different studies and primary care physicians.[13] Surely, improved detection of this disease will be crucial in the reports of its frequency.

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Eosinophlic pneumonia in a patient with anticentromere antibody

Neumonía eosinofílica en pacientes con anticuerpos anticentroméricos

We read with interest the article by Jaimes-Hernández et al. (Reumatol Clin 2012 May–June issue) on eosinophilic pneumonia in patients with autoimmune phenomenon or immunoallergic disease.[1] We would like to share our experience with a patient whose condition was similar to that reported by Jaimes-Hernández et al.[1]

A 75-year-old woman was admitted to our hospital because of one-week history of left chest pain. She was never smoker. She had a seven-year history of atrial fibrillation and, thereafter, was prescribed warfarin. On admission, she had no rales in both lungs, and the musculoskeletal examination was also unremarkable. She had no Raynaud's phenomenon, screloderma, and dysphagia. The chest X-ray and computed tomography revealed bilateral nonsegmental peripheral infiltrates mainly in the left lung. Laboratory data on admission were as follows: white blood cell 4900/μL (eosinophils: 245/μL), C-reactive protein 3.77 mg/dL, anti-nuclear antibody 1:640, anticentromere antibody 1:640, rheumatoid factor 4 U/mL. RP3-ANCA, MPO-ANCA, anti-ribonucleoprotein antibody, and anti-topoisomerase I antibody were negative. All tests for acid-fast bacilli including culture, and serologic and microscopic testing for fungi was negative. A bronchoalveolar lavage obtained from left upper lobe showed total cell count 8.4×10^5/mL with 16.7% eosinophilia. Transbronchial biopsy was not performed because the patient had warfarin for atrial fibrillation. The patient was diagnosed as having eosinophilic pneumonia and was started on 30 mg prednisolone per day. After two weeks of treatment pulmonary infiltrates had normalized. She was successfully weaned off the prednisolone over a period of two months and followed up without recurrence of eosinophilic pneumonia.

Although very rare, there have been some reports with regard to marked eosinophilic pulmonary infiltration in patients, who had high titers of antiautoimmune antibodies.[2,3] Both of them were diagnosed as having Churg-Straus syndrome.[2,3] Our patient had no sign and symptoms of Churg-Straus syndrome nor any autoimmune diseases.

Our patient had a high titer of anticentromere antibody in her serum without any symptoms of CREST syndrome. There might be a possibility that eosinophilic pneumonia developed incidentally in a patient with high titer of anticentromere antibody in serum. However, the case reported by Jaimes-Hernández et al.[1] and ourselves suggested that a certain type of eosinophilic pneumonia might have some relationship with autoimmune phenomenon.

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