pathway, thereby increasing airway remodeling. By blocking this action, muscle proliferation and the deposition of pro-inflammatory substances can be reduced.11

Our patient’s progress not only demonstrates the favorable effect of omalizumab, but also suggests that it is more than just an IgE immunomodulator, and that its effects on eosinophilic inflammation are also important. These findings are supported by the results of basic research, and have been confirmed by reports of favorable responses such as in our patient, in other conditions such as Churg-Strauss disease or chronic rhinosinusitis, in which eosinophilic inflammation is a key component.12

References


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A 60-Year-Old Male Smoker With Chronic Obstructive Pulmonary Disease and Hypereosinophilia

Varón de 60 años de edad con enfermedad pulmonar obstructiva crónica y eosinofilia

To the Editor,

In clinical practice, a diagnosis of pulmonary eosinophilia is suspected in patients with respiratory symptoms (dyspnea, cough or wheezing), migratory pulmonary infiltrates on chest X-ray, and eosinophilia in peripheral blood, or preferably, in the lung.1,2 Hypereosinophilic syndrome (HES) is a rare entity with different forms, one of the most exceptional of which is myeloproliferative HES. We report a case of FIP1L1/PDGFRα-positive myeloproliferative HES diagnosed in a patient with pulmonary eosinophilia.

A 60-year-old man, smoker of 50 pack-years, attended the respiratory medicine clinic with a 2-month history of worsening of his habitual cough and expectoration, and no other symptoms. He had been diagnosed 2 years previously with grade II COPD (forced expiratory volume in 1 s [FEV1] 79%), well managed after giving up smoking and receiving treatment with fluticasone/salmeterol 25/250 mcg.

No lymphadenopathies were observed on physical examination, and auscultation revealed some rhonchi, and a respiratory rate of 16 breaths/min and heart rate of 90 beats/min. No other significant findings were noted.

Blood tests showed leukocytosis 12 590 cells/mm with eosinophils 8560 (61%). Other blood counts, coagulation, and biochemistry results were normal. Lung function tests: FEV1 2610 l (87%), forced vital capacity (FVC) 4090 l (109%), FEV1/FVC 63.78%. Bronchodilator test and methacholine challenge were negative. Chest X-ray showed radiological signs of COPD. Chest and abdomen CT showed centriacinar and paraseptal emphysema, micronodules in the upper lung lobes, middle lobe and lingula, and thickening of the bladder, with suspected malignancy (Fig. 1). Echocardiogram was normal. Given these findings, the patient was referred to the urology department, where he was diagnosed with transitional cell carcinoma, and treated accordingly. The high number of eosinophils in blood prompted us to undertake a detailed differential diagnosis. Tests for parasites, helminths (Ascaris lumbricoides, Taenia solium, hydatidosis, Toxocara canis, Leishmania) and fungi, and stool culture were all negative. Hepatitis A, B and C and human immunodeficiency virus serologies, and Mycobacterium and Bordetella testing were also negative. In view of these findings, fibreoptic bronchoscopy was performed, and bronchial aspirate, bronchoalveolar lavage (BAL), and transbronchial biopsy (TBB) were obtained. Bronchial aspirate was negative for malignant cells. The BAL cell count was: lymphocytes 6%, polymorphonuclear cells 69%, and eosinophils 20%. Pathology study of TBB reported bronchial mucosa and pulmonary parenchyma with eosinophil infiltration, confirming pulmonary eosinophilia. The immunological study, including antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, and rheumatoid factor, was also negative.

The urology department was consulted, in view of the patient’s persistently high blood and pulmonary eosinophil levels (11.330/ul and 20%, respectively). A bone marrow biopsy was performed which showed marked eosinophilia with predominantly mature cells. Flow cytometry revealed a predominance of CD3.
There are 2 variants of HES: lymphoproliferative, occurring in 90% of cases, and myeloproliferative, in 10%. This variant is due in most cases to rearrangement of the FIP1L1/PDGFRα gene, although other genetic disorders have also been observed. It is detected using the FISH technique, which can identify chromosomal abnormalities by capturing the fluorescent point on the chromosome to which each FISH probe binds. This mutation causes continuous activation of a tyrosine kinase that leads to the clonal proliferation of eosinophils.8–10

Although HES is a very rare entity that is still relatively unknown, we do not believe that it was associated with the transitional cell carcinoma detected in our patient, although we cannot confidently rule this out.

To conclude, in the evaluation of pulmonary eosinophilia when eosinophil concentrations are very high, the possibility of rare hematological diseases, such as FIP1L1/PDGFRα-positive myeloproliferative HES, must be considered.

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