

Correspondence

Three months' administration of anti-IgE to a patients with Churg-Strauss syndrome

To the Editor:

Noga et al¹ demonstrated that omalizumab induces eosinophil's apoptosis in patients with asthma. By blocking IgE, omalizumab inhibits T_H2 immune response, thus reducing recruitment and activation of eosinophils and other inflammatory cells. Studies have demonstrated the anti-allergic and anti-inflammatory actions of anti-IgE, including reduction of circulating and tissue eosinophils.^{2,3}

Treatment of Churg-Strauss syndrome (CSS) remains a challenge.⁴ For 17 years, we have been following a male patient with CSS, now 46 years old. At the time of diagnosis, the patient presented with severe asthma, mono-neuropathy, epididymo-orchitis, and palpable purpura. Laboratory findings included: 18,800 leukocytes/mm³; 5,640 eosinophils/mm³ (30%); IgE of 398 IU/mL; negative parasitological stool exam; negative skin prick test; and negative *in vitro* IgE-specific test (RAST) to aeroallergens. Sural nerve biopsy revealed eosinophilic inflammatory infiltrate and necrotizing vasculitis, which confirmed the diagnosis of CSS.

Upon treatment, CSS showed remission but asthma did not improve, despite the use of high-dose inhaled corticosteroids and long-acting β_2 -agonists and several courses of steroid pulse therapy. At that point, we started the patient on omalizumab 300 mg subcutaneously every 2 weeks for 3 months. Following anti-IgE administration, the patient showed significant improvement of asthma (Table I). The effect of omalizumab on difficult-to-treat allergic asthma is well documented,⁵ but, in addition to that, we observed a marked improvement in eosinophilia. We should emphasize that the patient had not received systemic steroids during the 3 months before omalizumab treatment, implicating anti-IgE therapy as putative agent for eosinophilia control.

Although eosinophil count usually rises prior to vasculitis relapse, working as an early marker of activity, there is no sound basis to state that omalizumab may prevent CSS relapse. On the other hand, a recent report described a temporal association between anti-IgE use and the development of CSS.⁶

Our patient presented with high levels of serum IgE but also with negative results on specific IgE tests, late onset of symptoms, and no family history of atopy, which were suggestive of nonallergic asthma. This increase in IgE levels could be nonspecific, resulting from polyclonal immunoglobulin production during a T_H2 type immune response or mono-oligoclonal immunoglobulin production in response to unknown triggering antigen(s). Regardless of this feature, our results suggest that IgE may be involved in the mechanisms leading to peripheral eosinophilia in patients with CSS.

TABLE I. Clinical and laboratory findings before and after anti-IgE administration

	Before treatment	15 d after treatment initiation	3 mo after treatment initiation
Auscultation	Bronchospasm	Normal	Normal
Peak flow	420	500	500
FEV ₁ (L)	1.87 (61%*)	2.11 (68%*)	2.12 (70%*)
Leukocytes (cells/mm ³)	9.300	7.800	9.900
Eosinophil (cells/mm ³)	660 (7.1%†)	234 (3%†)	99 (1%†)

*Percent predicted.

†Percent of total blood leukocytes.

Anti-IgE therapy offers the potential to decrease asthma activity, but whether it can also impact the activity of other manifestations of CSS is an open question. In this patient, anti-IgE improved asthma control, decreased eosinophil blood count, and did not increase CSS clinical activity. However, large and long-term studies are necessary before widespread indication of omalizumab for CSS can be proposed. As far as we know, there is no report in the literature about prescription of anti-IgE to patients with CSS and nonallergic asthma.

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