

Letter to the Editor

Role of omalizumab and steroids in Churg-Strauss syndrome

To the Editor:

The first case report on a patient who developed Churg-Strauss syndrome (CSS) after therapy with omalizumab, the anti-IgE recombinant humanized mAb, was described by Winchester et al¹ in 2006. Later, several similar cases were reported in the medical literature; the authors suggested a possible role for anti-IgE treatment in the emergence of CSS and recommended careful monitoring of emerging symptoms of this vasculitis in patients treated with omalizumab.²⁻⁴

In contrast, Giavina Bianchi et al⁵ presented the case of a patient with CSS with uncontrolled asthma that improved with omalizumab treatment without increase of CSS clinical severity. The authors hypothesized that steroid tapering during omalizumab therapy could have caused CSS clinical appearance.

Here we describe the case of a 42-year-old Caucasian man with a 5 year history of asthma. One year after asthma onset, blood hypereosinophilia became evident (1600/ μ L), and the patient developed eosinophilic pneumonia treated with prednisone 1 mg/kg/d. Nasal polyposis was detected, and skin prick tests were positive for many allergens, such as Gramineae, birch, *Alternaria*, *Aspergillus*, acarids, and dog and cat dander. Further blood tests documented the absence of autoantibodies such as antinuclear antibodies and antineutrophil cytoplasmic antibodies; complement factors (C3 and C4) were normal. In January 2007, the patient started omalizumab therapy (375 mg every 15 days) for uncontrolled asthma and elevated blood IgE level (580 IU/mL). The patient had a partial response to oral steroids; however, the daily prednisone dose required for clinical improvement was very high (0.25 mg/kg/d). After 16 months, without oral steroid reduction, the patient developed symmetric polyneuropathy on 4 arms. Diagnosis of CSS was then considered according to American College of Rheumatology diagnostic criteria.^{6,7}

However, given the absence of evidence supporting a causal relationship between CSS and omalizumab, it is our opinion that we observed the natural course of the disease.

The vasculitic phase of CSS manifests after a variable period in which the patient displays clinical symptoms of allergic rhinitis, asthma, and blood eosinophilia. Typically, the CSS diagnosis is made several years after onset of initial symptoms.

In the absence of an evident cause-effect link between omalizumab and CSS, we believe that omalizumab should still be considered as a potentially useful and safe treatment for asthma, even in patients at risk for developing CSS. More data are required to elucidate better any possible role of omalizumab in CSS.

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