To the editor:

IgE produced by B cells after sensitization with allergens has a central role in allergic reactions and elevated serum IgE causes bronchial hyperactivity in atopic individuals. Although elevated serum IgE is accepted as a sign of atopy, high total IgE level may accompany with non-allergic asthma and contribute to pathogenesis and severity of the disease independently from atopy [1]. Furthermore, inflammatory cytokines and high-affinity IgE receptor expression have similar features on bronchial biopsies of patients with allergic and non-allergic asthma [2]. It was suggested that most asthmatic patients may have an atopic component with increased production of IgE but these allergens cannot be identified yet.

Humanized monoclonal anti-IgE antibody omalizumab reduces levels of circulating free IgE by binding to the constant region (ce3) of the IgE molecule, thus preventing free IgE from interacting with IgE receptors (FceRI and FceRII) and downregulates expression of IgE receptors (FceRI) on mast cells and basophils. The downregulation of FceRI expression is associated with a loss of sensitivity to allergen challenge and a reduction in mediator release.

Although indication of Omalizumab restricted with moderate to severe persistent asthma who have a positive skin test or in vitro reactivity to a perennial aeroallergens, some case reports show that omalizumab can be effective in patients with high or even low IgE level non atopic severe asthma [3].

In recent years, evidence has emerged showing that increasing number of patients previously diagnosed with nonallergic or idiopathic rhinitis developed a local allergic response with nasal-specific IgE (sIgE) production and positive nasal allergen provocation test [4]. These findings suggest the presence of local allergic rhinitis (entopy) which could not be revealed by the classic skin tests. Local IgE production was also shown in bronchial mucosa of patients with atopic and non-atopic asthma. Criticism against the presence of local allergic reactions was supported by the detecting IgE and undetectable allergens; however, the data about local allergic reaction limited to nasal mucosa are increasing.

We think that local allergy (entopy) can also be seen in asthma patients with negative skin-prick test and serum specific IgE and these patients therefore may benefit from anti-IgE treatment.

Non atopic patients with severe asthma having dramatic beneficial effect from omalizumab treatment show us that concept of local allergy (entopy) is worth discussing in asthma.

References

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