

Correspondence

Are free light chain immunoglobulins related to nasal local allergic rhinitis?

To the Editor:

The study by Powe et al.¹ is the first to demonstrate the localization of free light chains (FLCs) in patients with allergic and nonallergic rhinitis in both tissue and nasal secretions by using immunohistochemical and ELISA techniques and opens up new avenues for the study of the nasal local hypersensitivity response.

Three large groups of subjects are compared: patients with persistent allergic rhinitis (n = 90) who had a positive skin prick test response to house dust mite and serum specific IgE, patients with nonallergic rhinitis with eosinophilia syndrome (NARES; n = 90), and nonatopic healthy control subjects (n = 90). A separate group of patients with idiopathic rhinitis of unknown cause (n = 10) who were retrospectively identified from tissue archives was also included.

The authors provide evidence of “significantly increased FLC-expressing cells in the mucosa of patients with persistent allergic and idiopathic rhinitis, localized in mast cells and plasma cells,” and increased FLC serum levels in subjects with NARES. They propose that “FLC may provide an additional non-IgE immune pathway to augment or replace IgE-mediated hypersensitivity in chronic mucosal inflammatory disease.” Accepting that the mechanisms involved require further study, we nevertheless believe that a few comments are warranted, given the relevance in this context of both idiopathic rhinitis and NARES.

We wonder whether some of the patients with allergic and nonallergic rhinitis included in this study could have had local allergic rhinitis (LAR)? A number of studies have shown the existence of a new form of LAR,² or entopy,³ with local production of specific IgE and a positive nasal allergen provocation test response in patients previously given diagnoses of idiopathic rhinitis and NARES.^{2,4} These patients might present with normal or low levels of total IgE in nasal secretions,⁴ but we do not believe this is sufficient to discriminate the existence of nasal IgE-mediated hypersensitivity. In the report by Powe et al.,¹ no study was made of the response on nasal allergen provocation tests or the determination of specific IgE to aeroallergens in nasal

secretions of the allergic and nonallergic patients studied. Might there have been a local production of specific IgE, as we have seen in our studies?

Another question concerns whether FLC levels are associated with nasal mucosal inflammation or a local hypersensitivity response to aeroallergens. It would be interesting to conduct further comparative studies between patients with LAR or entopy and patients with idiopathic rhinitis or NARES without a local hypersensitivity response.

The authors suggest that “mast cell activation and degranulation could be induced by a FLC-mediated mechanism.” We recently detected that patients with LARs who were sensitized to grass pollen have an important and persistent mast cell activation specifically induced by nasal exposure to the aeroallergen.⁵ Thus further studies are needed to investigate the possible function of FLCs in local hypersensitivity to aeroallergens.

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