

Editorial

Do local Th2-driven or local IgE-mediated diseases exist without systemic IgE-sensitization?

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In this issue of *Allergy* is a paper about vernal keratoconjunctivitis (VKC) by Montan et al. (1). In this paper the authors report that only 60% of the patients with VKC had an IgE sensitization evidenced by skin tests and/or presence in serum of allergen specific IgE antibodies. In addition, the amount of messenger RNA encoding cytokines and inflammatory cell markers in conjunctival tissue biopsies of patients with VKC did not correlate with (systemic) IgE sensitization.

In the EAACI position paper "A revised nomenclature for allergy" (2), all allergic diseases were classified into "IgE mediated" and "non-IgE mediated". This was done to create a simple classification that can be used by all medical practitioners and scientists, not being limited to the single medical discipline concerned. It was never the aim of the authors that this document would be the final one. First of all, this EAACI position paper, which reflects the opinion of European allergologists, should be presented to the allergology societies of other continents and to the World Allergy Organization (WAO), so that an internationally accepted allergy nomenclature can emerge.

With regard to VKC, the EAACI position paper ranks it, together with "atopic" keratoconjunctivitis, among the IgE-mediated allergic conjunctival diseases, although it is mentioned that it is only "in part explained by IgE-mediated reactions". This implies that other mechanisms may be involved, which is neatly demonstrated by the paper of Montan et al. This illustrates the difficulty of classification of "allergic" diseases into "IgE-mediated" and "non-IgE mediated". It seems that

in the conjunctiva of patients with VKC there is a kind of local Th2-driven reaction, without any systemic IgE-mediated process being demonstrable. Similar findings were reported by Bachert et al. (3) in nasal polyposis, where a high total (polyclonal) IgE was found in the polyps. However, this was not reflected by the skin prick test or by allergen specific IgE antibody in the serum, especially in the worst cases of polyposis. I would not be surprised if in the near future similar findings will be reported for so-called non-IgE-mediated mucosal and skin diseases. *Staphylococcus aureus* enterotoxins acting as superantigens have already been suggested as a possible trigger for local IgE production in the atopic eczema/dermatitis syndrome (AEDS) and for nasal polyposis. Ongoing and future research will hopefully clarify the finer role of these *S. aureus* enterotoxins in the physiopathology of diseases that are still not yet fully understood, including AEDS, nasal polyposis, and (why not) VKC, "idiopathic" nonallergic rhinitis, otitis media with effusion... and are these diseases really local IgE-mediated or IgE-accompanied, and to what extent? Are there other superantigens involved in these diseases, e.g. fungi and viruses in nasal polyposis? Are there any implications for the treatment of these – usually difficult to treat – diseases? Do we need another class of mechanisms to be included in our nomenclature and classification? It is certain that these questions already receive great attention in some scientific centres and will receive even more attention from other groups in the near future. We look forward to reading exciting reports about it in this and other scientific journals.

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References

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