

M. SCHIAPPOLI¹, C. LOMBARDO¹, O. BORTOLAMI³, B. CARUSO², G. SENNA¹

IgE to staphylococcal enterotoxins are undetectable in sera from patients with nasal polyposis

¹Allergy Unit, Verona University Hospital (Italy) - E-mail: michele.schiappoli@ospedaleuniverona.it

²Laboratory of Clinical Chemistry and Haematology, Verona University Hospital (Italy)

³Research Support Unit and Biostatistics, Verona University Hospital (Italy)

KEY WORDS

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Corresponding author

Michele Schiappoli
Allergy Unit
University Hospital of Verona
Piazzale A. Stefani 1
35126 Verona (Italy)
E-mail:
michele.schiappoli@ospedaleuniverona.it

SUMMARY

Nasal polyposis is a frequent disease, sometime associated with asthma and non steroidal anti-inflammatory drugs intolerance. Staphylococcus aureus colonization can play a pathogenetic role in some cases by a severe eosinophilic inflammation, which can suggest new therapeutic approaches. Staphylococcus aureus colonization has been demonstrated by local specific enterotoxins IgE dosage in polyps homogenates. This study demonstrate lack of detection of serum enterotoxins specific IgE to staphylococcal in patients with nasal polyposis, compared with healthy subjects.

Nasal polyposis (chronic rhinosinusitis with nasal polyps) is a fairly common disease, frequently associated with asthma and hypersensitivity to non steroidal anti-inflammatory drugs. It is characterized by eosinophil infiltration and a T-helper 2 cell dominated cytokine pattern that includes interleukin 5 and IgE production (1). However, the role of atopy and concomitant sensitization to common aeroallergens is still debated, as its prevalence is generally not increased in subjects with nasal polyposis (2). *Staphylococcal enterotoxins* have been demonstrated to activate T cells, which can orchestrate severe eosinophilic inflammation as well as polyclonal B cell activation (3). An increased colonization rate by *Staphylococcus aureus* (*S. aureus*) has also been demonstrated in nasal polyposis, as well as the presence in nasal tissue homogenates of IgE specific to classical staphylococcal enterotoxins (*S. aureus* derived endotoxins: SAEs) (4). Of note, the involvement of *S. aureus* and the related IgE response to SAEs in the pathogenesis of some cases of polyposis has practical im-

plications. In fact it could suggest new therapeutic approaches in the long-term management of the disease (1). However the determination of specific IgE to SAEs in nasal mucosa or in polyp homogenates is time consuming, needs expertise and therefore it is not affordable in daily routine practice. Serum determination is an easier method to measure these antibodies and previous studies have reported an increase of specific IgE to SAEs in perennial rhinitis (5) as well in aspirin intolerant asthma (6).

In the present study we measured the presence of serum specific IgE antibodies to SAEs in a population of 34 patients with nasal polyposis, [14 subjects with aspirin intolerance and asthma (AIT) and 20 subjects tolerant to aspirin and NSAIDs (AT)] and in 34 age- and sex-matched healthy controls (Tab. 1).

Specific IgE for SEA (*S. aureus* endotoxin A), SEB (*S. aureus* endotoxin B) and SEC (*S. aureus* endotoxin C) were measured by ImmunoCAP (Phadia, Uppsala, Sweden).

Table 1 - Demographic and clinical characteristics of patients

Characteristic	Patients with nasal polyposis	Control population
Gender (f/m)	14 patients / 20 patients	18 patients / 16 patients
Age-yr (mean \pm sd)	50 \pm 18	52 \pm 11
AIT patients/AT patients	14 patients/20 patients	

Nasal polyposis was endoscopically diagnosed, whereas aspirin intolerance was diagnosed in the presence of a clear-cut history of more than two episodes of asthma exacerbations after aspirin or non COX2 selective NSAIDs. Thus, for ethical reasons, aspirin challenges were not carried out.

As variables presented a skewed distribution according to skewness–Kurtosis test, differences between specific SEA, SEB and SEC IgE levels were evaluated by Mann–Witney Wilcoxon Rank-sum test. No significant differences were observed in the level of specific IgE to SAEs between subjects with nasal polyposis (median: 0.03; IQR: 0–0.07) and controls (median 0; IQR: 0–0.07) ($p=0.19$), as well as between AIT (median: 0.035; IQR: 0.01–0.07) and AT (median: 0.02; IQR: 0–0.07) groups ($p=0.43$). Further, no significant difference in SEB ($p=0.53$) and SEC ($p=0.7$) specific IgE was observed between study population and controls.

There is increasing evidence of the involvement of *S. aureus* and related IgE in several allergic diseases, such as atopic dermatitis, allergic rhinitis and asthma (7), suggesting a possible phenotype-driven treatment. However, so far the identification of SAEs-specific IgE involvement has been carried out by methods requiring expertise and technical resources that not widely available.

The aim of our study was to establish the involvement of *S. aureus* and its superantigens in nasal polyposis, using a

routinely available method. According to our data, serum SAEs-specific IgE are unable to confirm the involvement of *S. aureus*, possibly because the low levels of antibodies produced can be detected only locally.

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