Staphylococcal enterotoxin specific IgE in serum is linked to severe asthma and nasal polyposis

P. Tomassen[1], L-O. Cardell[2], N. De Ruyck[1], U. Foerster[3], W. Fokkens[4], P. Gevaert[1], P. Hellings[5], V. Hox[5], E. Katainen[6], M. Kowalski[7], J. Mullol[8], H. Olze[3], A. Olzewska[7], C. Segboer[4], A. Reutherborg[2], E. Toskala[9], N. Van Bruaene[1], C. Van Drunen[4], T. Van Zele[1], C. Bachert[1]

[1]Ghent University, Upper Airways Research Laboratory, Gent, Belgium;
[2]Karolinska Institutet, Division of ENT diseases, CLINTEC, Stockholm, Sweden;
[3]Charité-Universitätsmedizin Berlin, Department of Otorhinolaryngology, Berlin, Germany;
[4]Academic Medical Centre, Department of Otorhinolaryngology, Amsterdam, Netherlands;
[5]University Hospitals Leuven, Department of Otorhinolaryngology, Head and Neck Surgery, Leuven, Belgium;
[6]Helsinki University Hospital, Helsinki, Finland;
[7]Medical University of Lodz, Department of Immunology, Rheumatology and Allergy, Lodz, Poland;
[8]IDIBAPS, Clinical and Experimental Respiratory Immunoallergy, Barcelona, Spain;
[9]Finnish Institute of Occupational Health and Helsinki University Hospital, Helsinki, Finland

Introduction

Staphylococcus aureus produces enterotoxins which have potent polyclonal immunostimulatory effects. IgE directed to these enterotoxins has been detected in serum of asthma, nasal polyp, allergic rhinitis and atopic dermatitis patients, but can also be present in serum of healthy subjects. Aim of the study was to assess the association of sensitization to enterotoxins with chronic rhinosinusitis, asthma and atopy presence and severity.

Methods: The study was designed as a multicenter, non-matched case-control study, and recruited CRS subjects with nasal polyps (CRSwNP), without nasal polyps (CRSsNP), and controls undergoing septoplasty or conchotomy. Serum was analyzed for total IgE and for SAE-IgE (SEA, SEC and TSST-1). Sensitization was defined as SAE-IgE > 0,35 kU/L. Asthma severity was classified according to medication use based on NHLBI guidelines.

Results: 794 serum samples were collected. Of these, 189 were controls, 378 has CRSsNP and 189 had CRSwNP. Asthma was present in 11.1% of controls, 19.2% of CRSsNP subjects and in 39.2% of CRSwNP subjects. Moderate to severe asthma was present in 7.4% of controls, 10.2% of CRSsNP and in 28.3 of CRSwNP subjects. SAE-IgE levels were positive in 15,7 % of controls, in 19,6% of CRSsNP and in 29,6% of CRSwNP. Non-asthmatics had in 19,4% SAE sensitization, compared to 19,1% of intermittent to mild asthmatics and 40,2 % of moderate to severe asthmatics. Binary logistic regression revealed significant and independent associations of SAE-IgE presence with moderate-severe asthma and nasal polyposis. When taking in account the titer of SAE-IgE using multinomial logistic regression, moderate levels of IgE (0.35-1 kU/L) were associated with nasal polyposis and moderate-severe asthma, whereas high levels (>1 kU/L) were associated with moderate-severe asthma. After controlling for asthma and sinus disease, atopy was not associated with SAE-IgE.

Conclusions: Serum presence of IgE to staphylococcal enterotoxins was strongly associated with moderate to severe asthma and nasal polyposis, independently of the presence of atopy. This underlines a potential role for staphylococcal superantigens in the pathogenesis of severe asthma, nasal polyposis and in their systemic interaction.