Allergen specific immunotherapy in atopic dermatitis

A. Kapitány[1], S. Baráth[2], G. Béke[1], Zs. Dajnoki[1], A. Khasawneh[1], Z. Káplár[1], A. Szegedi[1], K. Gáspár[1]

[1]Division of Dermatological Allergology, Department of Dermatology, Faculty of Medicine, University of Debrecen

[2]Department of Laboratory Medicine, Faculty of Medicine, University of Debrecen

Atopic dermatitis (AD) is a chronic inflammatory skin disease prone to relapse, having both genetic and environmental factors (e.g. allergens) as underlying causes for its development. The use of allergen specific immunotherapy (ASIT) has been restricted to insect venom allergy, allergic rhinitis and mild extrinsic asthma; however a few contradicting results are available on the use of ASIT in AD. Our aim was to analyze the effect of ASIT on clinical and subclinical variables in patients sensitized by house dust mite and suffering from both allergic rhinitis and AD. We examined the patients clinical (physical status, disease specific questionnaire), immunological laboratory (defining regulatory and effector T cells and also blood dendritic cells – flow cytometry; determining serum allergen specific IgE levels – ELISA; atopy patch test, prick test) as well as skin barrier (specifying Filaggrin mutation – molecular genetics; determining serum TSLP levels – ELISA; measuring TEWL – Tewameter) parameters prior to and during the ASIT treatment in comparison with diseased control groups. As a result of ASIT the measured clinical and skin barrier variables displayed improvement compared to the initial values as well as to the control group, although the differences were not significant. However when only patients without filaggrin mutation where compared, the ASIT treated patients showed significant improvement in barrier (TEWL) function (38,96+17,42 g/m^{2}h vs. 19,97+2,077 g/m^{2}h; p=0,0357). This result predispose that the modified immune status may improve the skin barrier in the mild-to-moderate patients without filaggrin mutation. Perhaps this AD population may benefit of ASIT. There is an intricate pathogenesis underlying AD, which requires a complex approach in therapy. Additionally to the previously described immunological changes attributed to ASIT, analysis of other physicochemical barrier parameters is crucial.