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EDITORIAL

In this issue...

Alopecia is a troublesome disease affecting patients very much in their daily life and giving the doctor therapeutical challenges. The mechanisms behind its development are to a large extent obscure, but some degree of auto-immunity is involved according to previous clinical and investigational experience. In a rather large group of 58 patients with either severe alopecia or alopecia responding to treatment, Dr. S. Yano and colleagues (p. 82) have looked at the expression of cutaneous lymphocyte antigen (CLA) on lymphocytes in the blood. They make the interesting observation that CLA expression is significantly increased especially on the CD4+ T cells in patients with recalcitrant and severe AA – not so much in patients with AA showing clinically improvement. Another quite interesting observation is that CLA is not so much increased on the CD8+ T cells in the blood. Is that because the CD8+ CLA+ cells have already left the blood and gone in the skin of the scalp, or is it because the CD4+ T cells are a primary "player" in AA? Although the authors have tried to look at skin sections using immunohistological staining, such techniques are very difficult to quantitate in contrast to FACS analysis of blood lymphocytes. The important lesson here is that AA is a "systemic disease", i.e. it involves the immune system, even circulating T lymphocytes. This confirms that the immune system must be involved. The hunt for "antigens" towards which these activated CLA+ cells are reacting is the issue for further studies.

Dr. Yoshizawa and co-workers (p. 136) have found that application of DNCB on arms or legs in patients with alopecia areata (AA) will improve hair growth in 13 of 20 treated patients. This is quite an impressive response rate among patients often difficult to treat. The authors recognize that the results should be viewed with caution as the study is an open study and the treatment was given as additional treatment to local treatment with cryotherapy with or without topical steroids to which the patients had not shown any response. The dosage of DNCB was 5%, which is remarkably high and would in healthy persons induce a very strong eczematous reaction with swelling of the regional lymph nodes, but this was not observed in the patients. Also, previous observations document a need for sensitization of the diseased skin area, which did not seem necessary in the present study. If confirmed, this study opens new treatment strategies using contact sensitizers for the treatment of AA. However, the authors will hopefully repeat their observations in a single-blind study of patients in whom DNCB – or preferably DPCP – is used as single treatment. Readers who want an update on the use of contact sensitizers in benign dermatoses may read: Buckley DA, Du Vivier AWP: The therapeutic use of topical contact sensitizers in benign dermatoses in *Br J Dermatol* 2001; 145: 385–405.

Maria Böhme and colleagues (p. 98) present their results on atopic dermatitis and other atopic diseases and respiratory infections among 4,000 children who – prospectively(!) – were followed using questionnaires during their first two years of life. They observe that significantly more boys than girls develop AD during the first year of life, but after two years this sex difference disappeared. They confirm retrospective observations that respiratory atopic symptoms and infections are significantly increased. Interestingly, the strongest association was to "food allergy" defined as parents suspicion hereof. Questionnaire studies have drawbacks. Thus, the authors observe "atopic eczema" among 25.1% of the children even though eczema is likely to develop among more children during the 3rd year of life so that the cumulative incidence of atopic eczema may reach 30–35%, which seems very high. What about seborrhoeic dermatitis? – a diagnosis rarely used in children these days. It is strongly recommended that the authors continue to descibe their observations of this cohort of children since that will most certainly give us a deeper understanding of the course of atopic diseases including when they stop.

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