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In this issue...

Old treatment comes true

In our department we have an "old" treatment, where we boil 1–2 kg of non-peeled wheat (containing a lot of starch) for 1 hour and put both the sack of wheat and the boiled water with starch into the bathtub. We use this bath for patients having extremely dry and itchy skin. And – the patients love it. However, evidence-based proof for its physiological efficacy is lacking!

In this issue Dr. Paepe and colleagues (pp. 184–186) demonstrate that soap-damaged skin in healthy subjects will have a quicker barrier recovery as measured using transepidermal water loss (TEWL) if starch is added to the bath water. Similar observations were seen in atopic dermatitis patients, where addition of starch to bath water used as 2×15 min per day will improve barrier recovery with approx. 20%. Thus, old remedies which have been used for decades can be demonstrated to have a beneficial function on skin barrier recovery. The observations presented are arguments for a clinical trial especially among children with mild to moderate atopic dermatitis.

TIM's and herpes

In this issue (pp. 224–225) one of the Letters to the Editor describe a female patient suffering from severe head-and-neck dermatitis who succesfully used tacrolimus ointment. However, this led to Kaposi's varicelliform eruption – also called eczema herpeticum. The author therefore warns against unrestricted use of tacrolimus.

Tacrolimus and pimecrolimus are new TIM's (= topical immunomodulators). They are unique new compounds specifically inhibiting activated T lymphocytes. They are likely going to be widely used by the dermatological community as they have proven very efficacious for atopic dermatitis. Already now there is hope that the TIM's may also prove helpful in the control of e.g. hand eczema, psoriasis, severe lichen planus etc..

Is tacrolimus dangerous to use? A 1-year study on 316 adult patients with atopic dermatitis has demonstrated a remarkbable success rate (1). Among side effects, 5.7% of the patients developed herpes simplex in the treated skin areas over the 1-year period.

The patient data published here is in many ways unique: three episodes of Kaposi's varicelliform eruption before starting on tacrolimus therapy. The patient had a very low number of antibodies towards herpes virus in her blood before and after treatment with tacrolimus, meaning that even though she had suffered from herpes simplex infection several times, she didn't develop high titers of antibodies. However, she apparently didn't suffer from a major immune deficiency.

This particular patient must – somehow – have a "specific immune deficiency" towards herpes virus. She was repeatedly – and successfully – treated with anti-viral compounds, which are efficaceous and easy to use. Tacrolimus is a new compound that deserves the interest of dermatologists and their patients. However, it is recommendable to ask your patient if herpes infections have been prominent previously and then to inform your patient about the available anti-viral therapy, before starting tacrolimus therapy.

REFERENCE

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Kristian Thestrup Pedersen
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Preventing corticosteroid-induced skin atrophy

Skin atrophy caused by topical corticosteroids is a major problem when potent steroids are applied to sensitive skin areas for prolonged periods, especially when used together with occlusion. Despite various attempts to reduce this unwanted effect by modifying the steroid molecule (i.e. by rendring it less inhibitory to fibroblasts) or to use additional agents (e.g. topical retinoic acid) which by themselves may cause epidermal hyperplasia and stimulate collagen synthesis, there is currently no effective remedy against skin atrophy due to steroids. It is interesting therefore that Faergemann and co-workers (pp. 179–183) have set out to investigate a novel strategy which might prove useful also in humans with steroid atrophy provided it stands the test of clinical trials. The authors have studied the effects of various thyroid hormone analogues on betametasone-induced skin atrophy in haired mice. A dose-dependent and significant inhibition of skin atrophy was noted when tri-iodoacetic acid (triac) and two other thyroid analogues were combined with the topical betametasone preparation. Although the study can be criticized for the choice of animal model (haired mouse skin is very different from human non-hairy skin) and for using a relatively short observation period (7 days instead of several weeks), there is reason to believe that thyroid analogues might also be effective in humans to prevent steroid atrophy of the skin. Thyroid analogues probably exert their effects on the integument by interacting with the nuclear thyroid receptor which, in turn, acts in concert with a superfamily of receptors for retinoids, vitamin D and oestrogen to control cellular proliferation and differentiation. But exactly how the anti-atrophogenic effect of thyroid hormones is accomplished is not fully understood. Since Triac has been around for decades and appears to be safe at least when used topically, clinical

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trials which are underway should soon tell us whether this treatment rational is useful also in man.

Many family members with a rare genodermatosis

On pages 187–191 in this issue, a research group lead by Prof. Xuejun Zhu at the Peking University First Hospital describes a novel collagen type VII mutation causing a peculiar subtype of dominant dystrophic epidermolysis bullosa (EB) with intense pruritus especially on the extremities. The authors report clinical, skin biopsy and molecular data of a five-generation family with EB pruriginosa, a condition first described by McGrath et al. (1). The disease-causing missense mutation COL7A1 (6899A > G) changes a glutamine residue to argenine. This type of mutation is not a typical glycine substitution as reported in many other cases of DEB. The nucleotide change occurs in the penultimate nucleotide on exon 87 and therefore might also have an influence on splicing. What is so unusual with this report, apart from the new mutation, is that clinical data from five generations have been collected allowing the phenotype variations in 19 individuals carrying the same dominant mutation to be illustrated. Clearly, the degree of pruritus and blistering varied among the family members suggesting that other factors than the mutation participate in the pathogenesis. Indeed, atopy is usually a prominent feature which may contribute to the itching and cause a vicious circle with increased blistering. Because of this, EB pruriginosa was not accepted as a subgroup in its own right in the most recent classification of EB (2) but was included among other variants of dominant dystrophic EB, all caused by defects in the anchoring fibrils below the lamina densa.

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2. Fine J-D, Eady RAJ, Bauer EA, Briggman RA, Bruckner-Tuderman L, Christiano A, et al. Revised classification system for inherited epidermolysis bullosa: report of the Second International Consensus Meeting on diagnosis and classification of epidermolysis bullosa. J Am Acad Dermatol 2000;42: 1051–1066.

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