SKIN CARE IN ATOPIC DERMATITIS

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Summary

Atopic dermatitis (syn.: atopic eczema, AD) is the commonest childhood inflammatory skin disease. Many factors can influence the condition of the skin barrier in AD. They are linked with external environment as well as the general body condition. In earlier publications concerning AD pathogenesis, changes in barrier functioning were considered nearly solely as a consequence of inflammatory state. Now, alternative approach to this subject is postulated perceiving changes in epidermal lipids composition and disorders in epidermal cells differentiation as a group of crucial factors in pathogenesis of the disease.

The proper skin care, including omitting the factors responsible for barrier’s damage (i.e. cleansing preparations based on aggressive surfactants, not containing refatting agents) as well as regular use of emollients, provide significant improvement in life quality of patients suffering from AD. It seems that emollient preparations for people with AD will be in the future based mainly on physicochemical forms that do not contribute to forming water-impermeable occlusive layer (o/w emulsions, lipid nanoparticles, w/o emulsions containing “light” emollients).

Riassunto

La dermatite atopica (DA) è la più comune affezione a carattere infiammatorio che colpisce i bambini. Legata ad una alterazione della barriera cutanea è influenzata sia dalle condizioni ambientali che dalle condizioni generali dell’organismo.

Mentre anni fa si pensava che il processo infiammatorio fosse esclusiva conseguenza dei cambiamenti verificatisi a livello del cattivo funzionamento della barriera, ora è stato dimostrato come le
composizione dei lipidi delle lamelle e la differenziazione cellulare rappresentano fattori cruciali di questa forma patologica. Così l’uso corretto di prodotti emollienti e l’eliminazione di detergenti aggressivi sono considerati molto utili a migliorare la qualità di vita delle persone affette da DA. Soprattutto tra gli emollienti si utilizzeranno nuove forme cosmetiche nanostrutturate risultate più adatte allo scopo.
INTRODUCTION

Atopic dermatitis (syn.: atopic eczema, AD) is the commonest inflammatory skin disease of childhood, affecting 15-20% of children in industrialized communities at any one time. Adults make up about one-third of all community cases. Moderate-to-severe atopic eczema can have a profound effect on the quality of life for both sufferers and their families.

Major milestone in describing the main clinical features of AD was the Hanifin and Rajka diagnostic criteria published in 1980. According these criteria, in order to qualify as a case of atopic dermatitis, the person must have:
- an itchy skin condition plus three or more of:
  - past involvement of the skin creases, such as the bends of elbows or behind the knees
  - personal or immediate family history of asthma or hay fever
  - tendency towards a generally dry skin
  - onset under the age of 2 years
  - visible flexural dermatitis as defined by a photographic protocol.

EPIDERMAL BARRIER IN AD

Atopic dermatitis is apart from psoriasis one of the most frequently occurring dermatological disease that is accompanied by serious epidermal barrier disorders.

Many factors can influence the state of the skin barrier. They are linked with external environment (UV, decrease in humidity, etc.) as well as the general body condition (changes in barrier lipids composition at elderly, stress, pathological states). In many cases it is difficult to separate unequivocally the extent of the influence, which particular factors have on skin barrier condition and functioning. In case of atopic dermatitis the fact of occurrence the disease itself (excepting inflammatory state development, disorders in synthesis of barrier lipids, etc.) generates a number of factors that additionally deteriorate barrier condition (stress, itching and stratum corneum damage related to it).

In earlier publications concerning AD pathogenesis, changes in barrier functioning were considered nearly solely as a consequence of inflammatory state. Now, alternative approach to this subject is postulated perceiving changes in epidermal lipids composition and disorders in epidermal cells differentiation as a group of crucial factors in pathogenesis of the disease [39]. In spite of this, there is still no theory that explains unambiguously the mechanism of arising changes in epidermal barrier structure in atopic dermatitis. The basic dysfunctions in the structure of epidermal barrier and homeostasis of epidermis are presented in Table I.

The result of disorders in epidermal barrier structure and homeostasis is a number of changes typical for AD. The main symptom is obviously skin dryness (increase in TEWL, decrease in values of electrical parameters measurements) [13, 16 - 20, 37, 40] related directly to the increase in water permeability of the barrier and disorders in mechanisms restoring the correct structures of the barrier. An increase in TEWL value is positively correlated to AD exacerbation [45]. Water deficiency in stratum corneum leads to disturbances in desmosomes degradation, abnormalities in desquamation and the decrease in epidermis elasticity [19]. Dysfunctions of skin barrier functioning result also in obviously higher susceptibility to irritations [36] related to more effective penetration of xenobiotics through the stratum corneum.
Table I  

Table: The basic dysfunctions in the structure of epidermal barrier and homeostasis of epidermis in AD

<table>
<thead>
<tr>
<th>Type of change</th>
<th>Source</th>
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<tr>
<td>Increase in epidermis thickness</td>
<td>[Nassif 1994]</td>
</tr>
<tr>
<td>Decrease in stratum corneum thickness</td>
<td>[Al-Jaberi 1994]</td>
</tr>
<tr>
<td>Barrier damage may occur not only in atopic lesions but also beyond them (in the stage of acute changes)</td>
<td>[Seidenari 1995], [Melnik 1989]</td>
</tr>
<tr>
<td>Decrease in surface lipids amount</td>
<td>[Jakoza 1981], [Barth 1989], [Sator 2002]</td>
</tr>
<tr>
<td>Increase in proliferation area, disturbances in differentiation. Faster turnover, smaller cornocytes.</td>
<td>[Van Neste 1979], [Ekanayake-Mudiyanselage 1998], [Watanabe 1991]</td>
</tr>
<tr>
<td>Disturbances in epidermal lipids metabolism, excessive phospholipase A2 activity</td>
<td>[Schafer 1991]</td>
</tr>
<tr>
<td>Changes in barrier lipids composition</td>
<td>decrease in the amount of ceramides, especially ceramide 1 and ceramide 3. Decrease in the amount of omega-hydroxy ceramides bound to the cornocyte envelope. Disorders in proportions between individual classes of barrier lipids, increase in FFA and free sterols</td>
</tr>
<tr>
<td>Increase in the share of gel phase (hexagonal) with relation to crystalline phase (orthorhombic) in the intercellular cement</td>
<td>[Pilgram 2001]</td>
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COSMETICS IN AD THERAPY

For decades, topical corticosteroids were used in AD therapy. They brought good therapeutic effects, but sometimes they also gave a number of side effects such as: suppression of the pituitary-adrenal axis, Cushing syndrome (systemically), spread of untreated fungal infection, irreversible stretch marks and prominent fine blood vessels, contact dermatitis, perioral dermatitis and worsening of acne and mild loss of pigmentation (locally) as well as skin thinning considered as the most serious local side-effect [11]. Nowadays, as the basic medicines for external use immunosuppressive macrolide lactones isolated from the Streptomyces spp. – tacrolimus and pimecrolimus are used the most often [11]. The external use of preparations of emollient activity is commonly considered as essential supplementation for both types of therapies. The proper skin care, including omitting the factors responsible for barrier’s damage (i.e. cleansing preparations based on aggressive surfactants, not containing refatting agents) as well as regular use of emollients, provide significant improvement in life quality of patients suffering from AD. One of the factors influencing the improvement is soothing of itching and limitation of scratching (and its consequences: excoriation, etc) [43] linked with an increase in the amount of water in epidermis and stratum corneum. Moreover, improvement in life quality (stress reduction) may result in benefits in the skin barrier homeostasis.

Cleansing procedures is of great importance in AD skin care. Negative influence on skin condition in AD may exhibit hard water and water from water-supply system containing big amounts of chlorine [30]. Some authors suggest that chlorine in water from water-supply system may be one of the factors inducing exacerbation of skin changes [34]. Tendency to use (in case of
hard water) surfactants excess (to obtain “better” cleansing acting, better foaming, etc.) can lead to significant deterioration of epidermal barrier as well as exacerbation or prolongation of skin lesions [30]. For that reason, cleaning cosmetics for atopic skin should not contain typical, irritating and damaging skin surfactants (SLES), and they should contain additives in the form of refatting ingredients. Good effects were obtained if during atopic skin cleaning the addition of rice starch to a bath was used. It was noted that starch molecules exhibit high affinity to horny layer structures and remain on the skin surface after bathing, providing moisturizing and reducing TEWL. A similar effect was not observed for healthy skin [7].

PUFA and their derivatives are a group of emollients in which faith has been put for several years. The interest of this group of compounds results from good effects of PUFA in therapy of other skin barrier damages as well as good effects of oral supplementation in AD [47]. Clinical trials carried out up to now, were based mainly on application of plant oils rich in linoleic acid (LA) and gamma-linolenic acid (GLA); in most cases satisfactory results were obtained.[11, 47]. It was also observed that systematic application of typical cosmetic emulsions containing 12% evening primrose oil allows to reduce risk of skin changes exacerbation after contact with an allergen [5].

In AD therapy, emollients of no biological activity are also used (Table II). They allow to improve barrier condition through replenishment with stratum corneum lipids and linoleic acid or on the way of simple occlusive activity (mostly based on petrolatum). Most probably in this case, the normalization of water content in stratum corneum is of the greatest importance for therapeutic effect. It is confirmed by the fact of good therapeutic effects obtained also in case of formulations containing especially hydrophilic moisturizing compounds. A small number of research studies comparing long-term effects of using creams of strong emollient activity as well as the creams based on hydrophilic substances revealed the lack of significant differences in TEWL reduction in atopic lesions for both types of cosmetics [28].

For the sake of disadvantageous influence of full (continuous) occlusion on epidermal homeostasis and the state of skin in AD [15, 29], it seems that emollient preparations for people with AD will be in the future based mainly on o/w emulsions, lipid nanoparticles or w/o emulsions containing “light” emollients, that do not contribute to forming water-impermeable occlusive layer. However, the mechanism underlying the “light” emollient efficacy in the treatment of dry, atopic skin have not been well elucidated.

<table>
<thead>
<tr>
<th>Cosmetic preparations evaluated in clinical and laboratory trials</th>
<th>Source</th>
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<tbody>
<tr>
<td>Moisturizing, urea based emulsions</td>
<td>[Loden 2001], [Loden 2002]</td>
</tr>
<tr>
<td>Moisturizing, glycerine based emulsions</td>
<td>[Loden 2001], [Loden 2002]</td>
</tr>
<tr>
<td>Moisturizing emulsions</td>
<td>[Matsumoto 2005]</td>
</tr>
<tr>
<td>w/o emulsions</td>
<td>[Tabata 2000], [Loden 2003]</td>
</tr>
<tr>
<td>Emollient-type, petrolatum based emulsion</td>
<td>[Lucky 1997]</td>
</tr>
<tr>
<td>Nanolipid preparations</td>
<td>[Matsumoto 2005]</td>
</tr>
<tr>
<td>Emollient preparations containing “physiological lipids”</td>
<td>[Lerardesca 2001]</td>
</tr>
<tr>
<td>Emollient preparations containing GLA-rich oils</td>
<td>[Mao-Quiang 1995], [Chamlin 2001]</td>
</tr>
<tr>
<td>Emollient-type emulsions containing GLA-rich oils</td>
<td>[Anstey 1990], [Ferreira 1998], [Gehring 1999]</td>
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References


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