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Topical Treatment of Children with Atopic Dermatitis

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Annotation: The main directions of pathogenetically substantiated treatment of AD, differentiated correction of concomitant pathology are described in detail. The external therapy of atopic skin lesions in children with the use of modern dermatological cosmetics is presented in particular detail.

Key words: Atopic dermatitis, treatment, childrens.

Introduction: Atopic dermatitis (syn.: atopic eczema, constitutional eczema, diffuse neurodermatitis) is a chronic relapsing skin disease that manifests itself in adult patients with erythematous-papular rashes with lichenization phenomena, accompanied by constant, often painful, itching and developing in the presence of a genetic predisposition to hyperactive the state of the Th2-helper system and the filagrin-dependent defect in the barrier function of the skin. According to numerous epidemiological studies, AD affects about 5% of the world's population, and in the age group of early childhood, the severity of atopic skin lesions is diagnosed in almost 30–50% of cases. The mechanism of the development of AD symptoms today is presented as a complex interaction of a genetically determined defect in the barrier function of the skin, features of innate and adaptive immunity, on the one hand, and the environment, infectious agents and concomitant diseases, on the other. Hereditary predisposition concerns primarily the functioning of the immune system, which is characterized by hyperactivity of T-helpers, which tend to differentiate more towards T-helpers of type II (Th2) during antigenic irritation. In addition, the impaired barrier function of the skin in patients with AD is explained by the genotypic mechanism, which is clinically manifested in varying degrees of dryness of the skin (xerosis). The development of AD exacerbation is closely related to the production of Th2 cytokines, primarily IL-4 and IL-13, the content of which in patients is higher than in healthy people. These interleukins lead to overproduction of IgE antibodies and increase the expression of adhesion molecules on endothelial cells. They are given importance in the development of the initial phase of tissue inflammation, while IL-5, which causes the maturation of eosinophils and determines their survival, predominates in the chronic phase of AD, which is also accompanied by the production of Th1-cytokines IL-12 and IL-18 and other cytokines. , such as IL-11 and TGF1b, which are predominantly expressed in chronic forms of the disease [1]. Approximately 80% of adult AD patients have elevated serum IgE antibodies (IgE-dependent or exogenous BP), sensitization to air and food allergens, and/or comorbid allergic rhinitis and asthma. However, in 20% of adult patients with AD, the serum IgE content remains normal (IgE-independent blood pressure, or endogenous). This allows us to distinguish two main phenotypes of the disease - exogenous and endogenous. The main function of the skin is to protect the body from the external aggressive environment. In AD patients, regardless of the skin phenotype, due to the failure of the functioning of the epidermal barrier, an increased loss of moisture through the epidermis is observed. In turn, the stratum corneum does not retain water due to a quantitative and qualitative deficiency of lipids - ceramides, cholesterol, fatty acids produced by keratinocytes, while the content of ceramides 1 and 3 is especially reduced. Ceramides are the main molecules that retain fluid in the extracellular space, and the barrier function of these complex structures is provided by the protein matrix associated with them. In the affected and unaffected skin of patients with AD, a decrease in the amount of ceramides is found. These changes, clinically manifested by marked dryness of the skin, facilitate the invasion of antigens into the skin (microbes, viruses, fungi, etc.) and thus create a vicious circle leading to the subsequent activation of the immune system and the maintenance of chronic inflammation, which further exacerbates the barrier defect. . Recently, a defect in the skin barrier function has been considered as one of the important phenotypic signs of AD, which leads to xerosis and a decrease in the content of antimicrobial peptides in the skin, which makes it an important target for therapeutic interventions.

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Aim: to study the effectiveness of a combined regimen for the treatment of atypical dermatitis, as well as to study the safety of using pelemethasone ointment (mometasone and furoate) and Beponten ointment (dexpanthenol, protegin).

Materials and methods: evaluation of the effectiveness of treatment was carried out in 7 patients, aged 7 to 16 years, diagnosed with atopic dermatitis, children's period of moderate severity. Those who applied for outpatient treatment at the regional dermatovenerologic dispensary. At the time of the examination, the patients were observed. In children, there is hyperemia and severe dry skin with a large number of pityriasis scales; increased skin pattern, hyperkeratosis, abundant peeling, painful cracks, persistent itching with increased at night. Skin changes are located mainly on the flexion surfaces of the limbs (elbows, popliteal fossae), palmar-plantar surface, inguinal and gluteal folds, dorsal surface of the neck. At the beginning of treatment, pelemethasone ointment was used once a day, the duration of use was from 5 to 10 days. In the last two days of applying pelemethasone ointment, the use of Bepanten ointment was additionally started 1 time per day, and then after the cancellation of pelemethasone ointment, Beponten was applied 2 times a day for an average of 10 days, then the use was reduced to 1 time per day until the symptoms completely disappeared (on average 12 days).

Results: symptom relief occurred in 100% of patients on the 12th day of treatment. Minor symptoms of skin irritation on the use of pelemethasone ointment were observed in 5 patients out of 7 at the beginning of the application, but this did not require discontinuation of the drug. After the treatment, the patients were monitored for the detection of relapses for 6 months. There was no relapse in the group of patients receiving maintenance therapy. As a result of the therapy at the site of application of pelemetasone ointment, no skin atrophy was observed.

Conclusions: the consistent use of pelemethasone ointment and beponten ointment in the treatment of manifestations of atopic dermatitis showed high efficiency in relieving symptoms; most patients achieved long-term remission, had good tolerability, side effects and no complications.

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