THE TREATMENT OF ATOPIC DERMATITIS

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ABSTRACT

Data on the mechanisms of formation and methods of effective treatment of atopic dermatitis (AD) are presented. The main directions of pathogenetically based treatment of blood pressure, 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.

Keywords: Atopic dermatitis, treatment, tacrolimus.

INTRODUCTION

Atopic dermatitis (syn.: atopic eczema, constitutional eczema, diffuse neurodermatitis) is a chronic recurrent skin disease manifested 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 the hyperactive state of the Th2 helper system and a phyllagrin-dependent defect of the barrier function of the skin. According to numerous epidemiological studies, about 5% of the world's population suffer from AD, and in the age group of early childhood, the severity of atopic skin lesions is diagnosed in almost 30-50% of observations. 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 primarily concerns the functioning of the immune system, which is characterized by hyperactivity of T-helpers, which tend to differentiate with antigenic irritation more towards type II T-helpers (Th2). In addition, the genotypic mechanism is also explained by the impaired barrier function of the skin in patients with AD, which is clinically manifested in varying degrees of severity of dry skin (xerosis). The development of exacerbation of blood pressure is closely related to the production of Th2 cytokines, primarily IL-4 and IL-13, the content of which is higher in patients than in healthy people. These interleukins lead to hyperproduction of IgE antibodies and increase the expression of adhesion molecules on endothelial cells. They are given significance in the development of the initial phase of tissue inflammation, while IL-5, which causes the maturation of eosinophils and determines their survival, prevails in the chronic phase of blood pressure, 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 expressed mainly in chronic forms of the disease [1]. Approximately 80% of adult patients with blood pressure have an increase in the content of serum IgE antibodies (IgE-dependent blood pressure, or exogenous), sensitization to air and food allergens and/or comorbid allergic rhinitis and asthma. However, in 20% of adult patients with blood pressure, 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 patients with AD, 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 the 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 in patients with AD, a

decrease in the amount of ceramides is detected. These changes, clinically manifested by noticeable dryness of the skin, facilitate the invasion of antigens into the skin (microbes, viruses, fungi, etc.) and, thus, a vicious circle is created, leading to the subsequent activation of the immune system and the maintenance of chronic inflammation, which further aggravates the barrier defect. Recently, a defect in the barrier function of the skin 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 effects.

AIM

To study the effectiveness of the combined treatment regimen for atopic dermatitis and to study the safety of the use of pelemetazone ointment (mometazone and furoate) and beponten ointment (dexpanthenol, protegin).

MATERIALS AND METHODS OF THE STUDY

Evaluation of the effectiveness of treatment was carried out in 7 patients aged 7 to 16 years with a diagnosis of atopic dermatitis, children's period of moderate severity. Those who applied for outpatient treatment at the regional dermatovenereological dispensary. At the time of examination, hyperemia and pronounced dryness of the skin with a large number of bran-like scales were observed in children; increased skin pattern, hyperkeratosis, abundant peeling, painful cracks, persistent itching with an increase at night. Skin changes are located mainly on the flexor surfaces of the extremities (elbow bends, popliteal pits), palmar-plantar surface, inguinal and gluteal folds, back of the neck. At the beginning of treatment, pelemetazone ointments were used 1 time a day, the duration of use was from 5 to 10 days. In the last two days of applying pelemetazone ointment, bepanten ointment was additionally applied 1 time a day, and then after the cancellation of pelemetazone ointment, beponten was applied 2 times a day for an average of 10 days, then the use decreased to 1 time a day until the symptoms disappeared completely (on average 12 days).

RESULTS

Withdrawal of symptoms occurred in 100% of patients on the 12th day of treatment. Minor symptoms of skin irritation to the use of pelemetazone ointment were observed in 5 out of 7 patients at the beginning of use, but this did not require the drug to be discontinued. After the treatment, the patients were monitored for 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 pelemetazone ointment, skin atrophy was not observed.

CONCLUSIONS

Consistent use of pelemetazone ointment and beponten ointment in the treatment of manifestations of atopic dermatitis showed high effectiveness in relieving symptoms in most patients, long-term remission was achieved, good tolerability was noted, side effects and there were no complications.

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