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RARE FORMS OF ATOPIC DERMATITIS

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Atopic dermatitis (AD) is one of the most common dermatoses of childhood. The disease is a chronic, hereditary allergic inflammation of the skin, based on immune mechanisms of formation, clinically characterized by itching, morphology of rashes, and localization [8, 15, 19]. In recent years, the growth of allergic diseases in children continues. Among these, atopic dermatitis has the largest share. It affects 1 to 5% of the child population. This constitutes a significant proportion of outpatient and inpatient children with dermatoses - from 20% to 50% or more [1, 2, 5, 6, 20].

The diagnostic criteria for AD in children include: itching of the skin, onset of the process in early childhood, hereditary predisposition, typical age-related morphology of rashes and localization, chronic recurrent course, and nonspecific skin hyperreactivity [13]. In the formation of the disease, 3 stages are distinguished: infantile (0-2 years), childhood (2-12 years), and adolescent stages (12-23 years), which can be separated by periods of remission or continuously transition from one to another [14]. The assessment of the severity of clinical manifestations of AD is carried out using scales: SCORAD (Severity Scoring of Atopic Dermatitis) [14].

In recent years, a new concept of the pathogenesis of AD has been created and proposed, including a triad of leading mechanisms: genetic predisposition to atopy, disruption of the integrity of the epidermal barrier, and a cascade of immune reactions implementing allergic inflammation in the skin.

It is known that AD is a hereditary skin disease of a multifactorial nature, based on the polygenic inheritance of additive gene actions with a threshold effect [9, 10]. In patients with AD, a polygenic mode of inheritance is present, with genetic and environmental factors occupying the main place in its formation. The manifestation of AD reflects the genetic nature of the disease, and numerous exogenous environmental factors disrupt the threshold effect and contribute to the splitting of the genetic information of the patient's phenotype. According to this concept, genetic disorders determine the onset and frequency of AD manifestations in the population. This confirms the early onset of the disease and the high degree of concordance, as well as a twofold increase in the risk of the disease in children. It is known that a family history burdened with allergic diseases is detected in 60-80% of AD patients. It is shown that the risk of developing this disease in children of healthy parents is 10-20%. If one of the parents is ill with an atopic disease, the probability of the disease increases to 45-55%, if both are ill - to 60-80%. The probability of developing AD in a child is higher if the mother suffers from an atopic disease. It is assumed that the inheritance of AD is carried out according to the polygenic type [3, 4, 7].

A new pathogenetic factor in the formation of AD is the insufficiency of the skin's barrier function, caused by a mutation of the gene encoding the synthesis of filaggrin [5, 16, 18]. Filaggrin is a key hydrophilic protein of the epidermis that aggregates keratin filaments and creates a keratin structural matrix, directly participating in the implementation of its barrier function [5, 11, 12, 17].

Subsequently, as a result of molecular and cellular disorders of the structural and functional state of the skin barrier, a cascade of subsequent phases of allergic inflammation of the skin is triggered when it interacts with allergens.

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The polymorphism of the clinical picture of AD in different age periods of life and the individual characteristics of each patient cause certain difficulties in the diagnosis of this disease. Such a complex picture of the disease is not always recognized and identified in a timely manner, which subsequently leads to diagnostic errors. All this indicates the feasibility of studying the clinic of AD, its diverse forms, taking into account the age and constitutional characteristics of the child.

Pruriginous form of atopic dermatitis.

Before treatment

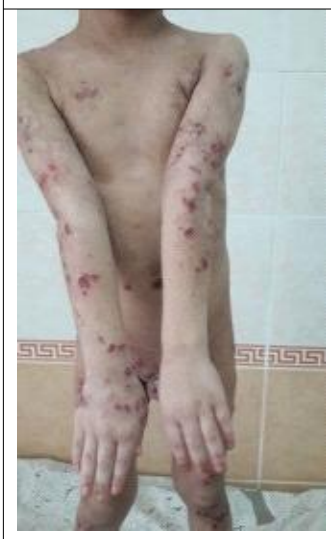

Foto. 1

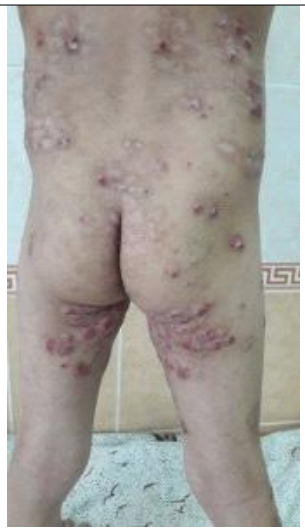

Foto. 2

Pruriginous form of atopic dermatitis.

After treatment, after 2 weeks

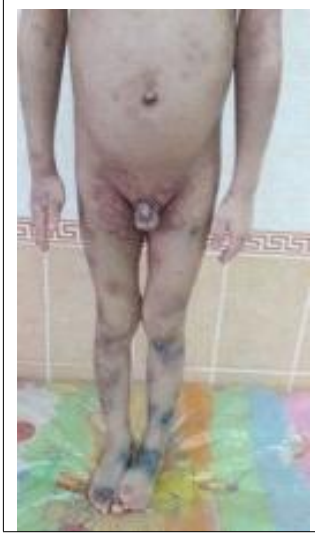

Foto. 3

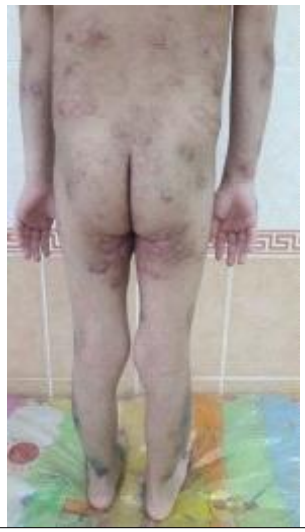

Foto. 4

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In children, the pruriginoid form of AD is diagnosed in 1-2% of cases. This form is rare, usually occurring in the age range of 7-15 years. It is characterized by multiple, isolated, rather large (pea-sized) nodules in the area of the posterior surface of the neck, extensor surfaces of the upper and lower extremities, lumbosacral and lumbar regions, often with bloody crusts and excoriations. Marked lymphadenitis is observed - multiple lymph nodes up to the size of a walnut, intense, persistent, biopsying itching.

The clinical picture of AD in children has age-related characteristics of the course, which can be considered as clinical forms (stages) of the disease, due to age-related anatomical and physiological features of the skin, the state of reactivity of the child's body.

We present our example. On January 22, 2024, a sick child, Ergashev Atkhamzhon, 8 years old (medical record No. 25/54), was admitted to the pediatric dermatology department of the TashPMI clinic. He was born from an inbred marriage and resides at 11 Bulokkuchasi Street, Mavlonova Village, Bekobad District, Tashkent Region. The mother reported his presenting complaints as rashes all over the body, anxiety, and poor sleep.

The child was born from the third pregnancy at term with a normal delivery. His birth weight was 4200 grams, and his length was 55 cm. The child was born from a consanguineous (inbred) marriage (the parents are second cousins on the mother's side). During the pregnancy, the mother experienced stress (due to the death of a second child), did not follow a diet, and consumed sweets, spicy, and salty foods. No other illnesses during pregnancy were noted. The child was breastfed for 2 months and started complementary feeding at 3 months. He experienced frequent colds, and a citrus allergy was noted. The child's father has an allergy to legume products (mung beans).

Patient History: The child first became ill at 6 months of age. The condition began with redness and swelling of the skin on the face, accompanied by nodular-vesicular elements, exudation, and weeping. By one year of age, the skin process had spread to the neck, elbow creases, and popliteal fossae. He was treated on an outpatient basis and in the hospital at his place of residence, receiving antihistamines and hyposensitizing drugs. Local therapy with corticosteroid preparations was also administered. The effect of the treatment was temporary. Exacerbations were observed during the winter and spring periods, 5-6 times per year, lasting 1-1.5 months. Remissions occurred during the summer for 2-3 months. The last exacerbation had been ongoing for 2 weeks, with a worsening of the skin condition, which prompted the child's mother to seek medical attention at the Consultative and Diagnostic Polyclinic (CDP) of the TashPMI clinic. Following examination by a dermatovenereologist, he was hospitalized in the pediatric dermatology department with a diagnosis of atopic dermatitis.

Physical Examination: The cutaneous pathological process is widespread, located on the scalp in the parietal region, on the posterior surface of the neck, on the back, on the extensor surfaces of the arms and legs, and on the skin of the genitalia and buttocks (Figs. 1, 2). Pruriginous papules are observed in the lesions, ranging in size from a pinhead to lentils and beans; in some areas, they are confluent. The surface of the elements shows bloody crusts, surrounding dryness, slight scaling, and excoriations. The itching is of strong intensity, biopsying, and constant. Marked lymphadenitis is noted in the cervical, axillary, and inguinal lymph nodes. The dermographism is white, persistent, and appears after 20 seconds.

Laboratory Analyses:

- Complete Blood Count (CBC) - 01/17/2024: Hemoglobin: 100.0, RBC: 3.5, Color Index: 0.9, ESR: 6 mm/hr

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• Urinalysis - 01/23/2024: Total Volume: 56.0, Color: light yellow, Specific Gravity: 1020, Protein: absent, Squamous Epithelial Cells: 0-1-2, Leukocytes: 2-3-4, Erythrocytes: occasional, Bacteria: trace

• Stool Analysis - 01/23/2024: Quantity: 54, Form: formed, Color: light brown, Leukocytes: absent, Erythrocytes: absent, Ova and Parasites: not detected

Consultations:

• Neurologist Consultation - 01/27/2024: Conclusion - no pathology detected

• Ophthalmologist Consultation - 01/27/2024: Conclusion - allergic conjunctivitis, molluscum contagiosum around the skin of the eyelids

• Pediatrician Consultation - 01/29/2024: Conclusion - no pathology detected

Based on the history, the characteristic clinical picture of the cutaneous pathological process, and the results of laboratory studies, the patient was given a final diagnosis of: Atopic Dermatitis, pruriginoid form, Grade III activity (severe), severe course. Comorbid condition: Molluscum Contagiosum.

The patient was prescribed pathogenetic therapy. The treatment was conducted in the dermatology department in conjunction with a pediatrician and consisted of the following:

• 30% Sodium Thiosulfate solution 60.0 + 0.9% Sodium Chloride solution 0.6 mL IV drip, #5
• 10% Calcium Gluconate solution 6.0 + 0.9% Sodium Chloride solution 0.6 mL IV drip, #5
• 2% Suprastin (Chloropyramine) solution 0.5 mL IM once daily for 10 days
• Ketotifen ½ tablet once daily in the morning for 10 days
• Neuralon 1 tablet twice daily (morning and evening) for 14 days
• Pancreatin ½ tablet four times daily with meals for 14 days

Local therapy consisted of:

• Methylene blue solution twice daily

• Dermovate (Clobetasol propionate) cream + Zinc oxide ointment (1:1 ratio) twice daily for 5 days

• Allergoderm solution twice daily for 14 days

As a result of the treatment, the cutaneous pathological process showed positive dynamics, including a reduction in the intensity of itching, normalization of sleep and behavior, cleansing of elements from bloody crusts and scales, epithelialization of excoriations and fissures, and resolution of pruriginous and papular elements and lymph nodes to normal values (Figs. 3, 4).

The patient was discharged with clinical recovery and will continue to be monitored at his place of residence.

This clinical case is interesting due to the rare occurrence of the pruriginous form of atopic dermatitis in children during childhood, and the diagnostic challenges in patients of this age group. The presented case underscores the need for a thorough differential diagnosis in this patient population to guide the selection of appropriate management and treatment strategies. Timely diagnosis of the

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pruriginous form of atopic dermatitis in children optimizes patient management, minimizes the potential for complications, and reduces the length of hospital stay.

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