

КЛИНИКО-ЛАБОРАТОРНЫЕ ОСОБЕННОСТИ И ЭТИОЛОГИЯ БРОНХИАЛЬНОЙ АСТМЫ С АЛЛЕРГИЧЕСКИМИ РИНОСИНУСИТАМИ У ДЕТЕЙ

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КЛИНИКО-ЛАБОРАТОРНЫЕ ОСОБЕННОСТИ И ЭТИОЛОГИЯ БРОНХИАЛЬНОЙ АСТМЫ С АЛЛЕРГИЧЕСКИМИ РИНОСИНУСИТАМИ У ДЕТЕЙ. ЖКМП.-2025.-Т.3.-№3.-С

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Аннотация: При проведении комплексного сравнительного клинического, аллергологического, иммунологического, функционального исследования 120 детей, страдающих atopической бронхиальной астмой, сочетающейся с аллергическими риносинуситами, установлены следующие научные факты: 1) аллергические риносинуситы определяют формирование и тяжесть клинического течения бронхиальной астмы; 2) в этиологии atopической бронхиальной астмы, сочетающейся с аллергическими риносинуситами, имеют значение региональные аллергены: домашняя пыль, пылевые (полынь, лебеда, айлантус и др.). инсектные (*D.pteronissinus*, *D.farinae*), эпидермальные (шерсть кошки, собаки), пищевые (коровье молоко, яичный белок и др.).

Ключевые слова: бронхиальная астма, аллергены, гиперреактивность бронхов.

BOLALARDA BRONXIAL ASTMANING ALLERGIK RINOSINUSITLAR BILAN UYG'UN SHAKLINING KLINIK-LABORATORIY VA ETIOLOGIK XUSUSIYATLARI

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Annotatsiya: 120 nafar atopik bronxial astmaning allergik rinosinusitlar bilan uyg'unlashgan shakli bilan xastalangan bemor bolalarda klinik-allergologik, immunologik, funksional tekshiruvlar o'tkazildi va quyidagi natijalar olindi: allergik rinosinusit bronxial astmaning og'ir kechishini taminlaydi 2) bronxial astmaning allergik rinosinusitlar bilan mintaqaviy etilogik allergenlar: uy changi, o'simliklar changi (yantoq, sho'ra, aylantus), insekt allergenlar (*D.pteronissinus*, *D.farinae*), epidermal allergenlar (hayvonlar yungi), ovqat allergenlari (sigir suti, tuxum sarig'i, v.b.).

Kalit so'zlar: bronxial astma, allergenlar, bronxlar giperreaktivligi.

CLINICAL AND LABORATORY FEATURES AND ETIOLOGY OF BRONCHIAL ASTHMA WITH ALLERGIC RHINOSINUSITIS IN CHILDREN

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Abstract: During a comprehensive comparative clinical, allergological, immunological, and functional study of 120 children suffering from atopical bronchial asthma combined with allergic rhinosinusitis, the following scientific facts were established: 1) allergic rhinosinusitis determines the formation and severity of the clinical course of bronchial asthma; 2) in the etiology of atopical bronchial asthma combined with allergic rhinosinusitis, regional allergens play a significant role: house dust, pollen (wormwood, goosefoot, aithus, etc.), insect (*D.pteronissinus*, *D.farinae*), epidermal (cat, dog hair), food (cow's milk, egg white, etc.).

Keywords: bronchial asthma, allergens, bronchial hyperreactivity.

Introduction: Bronchial asthma (BA) in children ranks among the top in prevalence in many countries worldwide and therefore has significant medico-social importance [1-3]. The widespread application of achievements in modern allergology, immunology, biochemistry, genetics, and other related sciences has enabled the elucidation of various aspects of the clinical features, etiology, and pathogenesis of BA in children [6,10]. Nevertheless, many questions related to the BA problem remain open and require solutions. Among them, in our opinion, the problem of regional features of the clinical presentation and etiology of BA, combined with allergic rhinosinusitis (ARS), is of great importance [4-6]. Pathology of the ENT organs significantly influences the formation and development of allergic reactions and diseases [2-4].

The occurrence of pathological processes in different parts of the respiratory tract may be one of the reasons leading to a decrease in the barrier function of the mucous membrane of the respiratory tract in eliminating exogenous allergens. The frequency of various clinical forms of ENT organ lesions (tonsillitis, sinusitis, adenoids, nasal polyps, nasal septum deviation) in children varies widely (from 4.1 to 66%) and depends on the clinical forms of BA and the pathology of the ENT organs themselves (2.20). Currently, the upper and lower respiratory tracts are considered as a single functional unit. It is believed that patients with allergic rhinitis have a sharply increased risk of developing BA. Along with this, nasal and paranasal sinus pathology is often observed in BA [1,3,5]. Therefore, studying various aspects of the problem of BA combined with ARS presents certain scientific and practical interest.

Materials and methods: 120 children aged 7-14 years were under observation, including 60 suffering from atopic BA combined with ARS, and 60 children with BA not combined with ARS. The diagnosis of the main and concomitant diseases was made on the basis of clinical and allergological, functional, laboratory, and radiological studies [1,2,9]. Forced expiratory volume was determined using a pneumotachometer, the threshold of sensitivity of bronchial receptors to histamine and acetylcholine, nasal respiratory function, the number of eosinophils in peripheral blood, and nasal mucosa secretions [7,8]. The cause of sensitization of the body was determined by setting allergic skin tests 171 (Table 1).

Patients with Bronchial Asthma with ARD were characterized by meteorological lability, low forced ex-

piration indicators (1.56 ± 0.45 l/s), hyperreactivity of bronchial receptors to histamine and acetylcholine.

Table 1. Severity of Clinical Course of Atopic Bronchial Asthma.

Clinical Course	Atopic Bronchial Asthma	
	Combined with ARD	Not Combined with ARD
Mild	21 (35,0±10,4)	40 (66,7±6,0)
Moderate	27 (45,0±10,9)	20 (33,3±6,0)
Severe	12 (20,0±11,9)	

Presence of allergic inflammation of the nasal mucosa (rhinitis) and its paranasal sinuses (sinusitis), difficulty in nasal breathing, high rhinometry values (16.8 ± 3.1 - 18.4 ± 3.2 mm water column), severity of expiratory dyspnea, eosinophilia (10.0-15.0%) of peripheral blood, the presence of a large number of eosinophils (16.2 ± 4.2) in the nasal mucosa, low phagocytic activity of neutrophils: phagocytic number - $44.6 \pm 5.1\%$, phagocytic index 3.5, phagocytosis strength 156.1 ± 10.3 , high level of IgE (850.5 IU/ml) in blood serum, hereditary allergic burden ($75.0 \pm 5.5\%$), the presence of allergic diathesis in the anamnesis ($55.0 \pm 6.4\%$). Even after the subsidence of the clinical symptoms of the disease, inflammatory phenomena in the nasal mucosa, bronchi, and lungs persist, which is consistent with the literature data (2,5,6). Among the contributing factors, toxicosis of pregnancy ($68.3 \pm 7.2\%$) and pathology were important.

According to our data, in most children (38, or 63.3%), the development of asthma began with symptoms of allergic rhinitis (AR), in 18 (30%) - with the simultaneous appearance of asthma and AR symptoms, and only in 4 (6.7%) the symptoms of the disease began with symptoms of bronchial asthma, and after some time, symptoms of allergic inflammation of the nose and paranasal sinuses appeared.

Non-infectious allergens were of great importance in the etiology of atopic asthma combined with AR. The frequency of sensitization of the body, according to the positive results of skin tests, was in the range of 8.3-30% and depended on the type of allergen and the individual reactivity of the body of sick children (Table 2).

In bronchial asthma of dust etiology (house dust), exacerbation of the main disease symptoms was more often noted in a home environment. With sensitization to house dust mites (*D.pteronissinus*, *D.farinae*), suffocation attacks predominated at night. Pollen asthma was characterized by a clear seasonality.

Exacerbation of disease symptoms was observed in the spring-summer season and coincided with the flowering time of plants to which patients' children were sensitized. The cause of food-related asthma was cow's milk, egg white, fish, etc. It should be emphasized that polysensitization was most often observed.

Bronchial asthma (BA) is related to typical allergic diseases that develop based on the pathogenesis of allergic reactions of the anaphylactic type (Type I). That is, immune reactions involving allergens and specific IgE antibodies reacting on the surface of the target cell cytoplasm. It has been established that histamine plays a decisive role in the pathogenesis of atopic BA, while serotonin is involved in the mechanism of development of infectious-allergic forms. Some forms of BA in children may develop through the pathogenesis of cytotoxic allergic reactions (Type II), mediated by immune complexes (Type III), or cellular mechanisms (Type IV). Children often exhibit mixed types of allergic reactions (1).

In the pathogenesis of allergic bronchial asthma (ABA) with allergic rhinitis syndrome (ARS), the pathology of ENT organs plays a significant role. The reduction of the barrier function of the mucous membrane of the upper respiratory tract and the function of the ciliated epithelium (slowing down or complete cessation of their movement) contributes to prolonging the contact time of the organism with an exogenous allergen, which causes sensitization and disease development.

Table 2. Causes of Sensitization in Children with Atopic Asthma Associated with Allergic Rhinitis.

Allergens	Atopic Asthma	
	Combined with Allergic Rhinitis	Not Combined with Allergic Rhinitis
Household (house dust)	14 (23,3±5,4)	12 (20,0±5,1)
Pollen	12 (20,0±5,1)	10 (16,7±4,8)
Insect: Dermatophagoides	18 (30,0±5,9)	14 (23,3±5,4)
Epidermal	16 (26,7±5,7)	13 (21,7±5,3)
Food	7 (11,7±4,1)	5 (8,3±3,5)

The following mechanisms of ABA development in the context of nasal and paranasal sinus pathology are given considerable importance: 1) development of inflammatory processes in the lungs upon entry of various allergenic substances from pathologically altered nasal paranasal sinuses; 2) enhancement of B-adrenergic receptor blockade, leading to increased irritability of the bronchial tree; 3) reflex bronchospasm due to increased excitation of the parasympa-

thetic nervous system (1,2,3,5). In the pathogenesis of ABA, a significant role is played by genetic factors.

Conclusions:

1. Allergic rhinosinusitis determines the formation and severity of the clinical course of atopic asthma in children.
2. Regional allergens such as household (house dust), pollen (wormwood, goosefoot, ailanthus, etc.), insect (*D. pteronissinus*, *D. farinae*), epidermal (cat hair, dog hair, etc.), and food (cow's milk, egg white, etc.) are important in the etiology of atopic asthma combined with ARS.

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