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The Role of Antihistamine Treatment Allergies in Children and Adolescents

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Abstract: The trend of increasing prevalence of food allergies observed over the past decades has prompted the European Academy of Allergy and Clinical Immunology (Allergy and Clinical Immunology EAACI) to develop a Guide on food allergy and anaphylaxis – EAACI Food Allergy and Anaphylaxis Guidelines. The document was published this year [1,3,4]. Foodallergy refers to adverse reactions to a food product caused by immunological mechanisms (IgE, non-IgE, or both) that develop in sensitized individuals. Food allergies, known since the time of Hippocrates, and possibly even earlier (the Babylonian Talmud mentions egg white intolerance and suggests treatment options), are the most obvious type of allergy for patients, which is confirmed by clinical and diagnostic procedures and medical reports much less often than patients claim. The results of epidemiological studies indicate a high prevalence of food allergies according to self-assessment using questionnaires and several times lower prevalence according to the results of a specific allergological examination.

Keywords: allergy, food, treatment, prophylaxis.

Data from the latest European Systematic Review of Epidemiological Studies of Food Allergies [2] showed that, on average, in Europe, the prevalence of food allergies according to self-assessment data at the time of the study reaches 6%, and the prevalence of food allergies observed at any time in life is 17%. At the same time, the frequency of food allergies confirmed by a provocative test is only 1%. Its frequency is higher among the children's population, as well as in the countries of North-Western Europe. In Southern Europe, the incidence of food allergies is significantly lower, although the review points to a lack of studies in Eastern and Southern Europe using a standardized double-blind placebo-controlled provocation test to confirm the diagnosis of food allergies [2]. Note that the prevalence of food allergies in some selected populations is significantly higher than in the general population. People with food allergies have:

- from 30 to 50% of children with atopic dermatitis [3, 4];
- from 2 to 8% of people with bronchial asthma [5];
- 1-5% of patients with chronic urticaria [6];
- More than 10% of people who have suffered anaphylactic shock [7].

Food allergy refers to adverse reactions to food, namely food hypersensitivity reactions, which can be allergic if caused by immunological mechanisms, and non-allergic [8].Non-allergic food hypersensitivity often results from metabolic disorders. In some cases, the mechanism of hypersensitivity is known. These are intolerance to milk and dairy products due to a deficiency of the enzyme lactase, hyperreactivity to vasoactive amines that are part of a number of food products (caffeine – in coffee and tea, histamine - in fermented foods and beverages, canned fish, tomatoes, spinach, tyramine-in fermented cheeses, brewer's yeast, pickled herring, serotonin - in pineapples, bananas, peanuts, nettles, pheny lethylamine-in

chocolate, bananas, cheeses, red wines, beans). However, in many cases, the mechanism of hypersensitivity remains unclear. One thing is clear: immune mechanisms are not involved in the formation of non-allergic food hyper sensitivity. Thus, food hypersensitivity caused by immunological mechanisms is called a food allergy. This reaction is not associated with a number of physiological or toxic effects of food and can occur after consuming a small amount of the product. Food allergies can be mediated by IgE antibodies. It is allergic reactions of the first type that are induced by the majority of identified food allergens (more than 170 food products). Recently, special attention has been paid to non-IgE-related (cell-mediated) and combined IgE- and cell-mediated food allergies with such clinical manifestations as atopic dermatitis, eosinophilic esophagitis, and eosinophilic gastroenteritis.

Foods that most often cause allergic reactions

Theoretically, any food product can cause an allergic reaction. However, in practice, the symptoms of clinically significant allergies are more often caused by several foods: cow's milk, chicken eggs, wheat, soy, peanuts, hazelnuts, fish and crustaceans. This was once again demonstrated in a recent European Systematic Review [9]. As a rule, allergy to cow's milk and chicken eggs was observed in young children, and allergy to peanuts, hazelnuts, fish and crustaceans-in the population of older children, adolescents and adults [9].Food traditions that exist in different countries and regions of the world can influence the frequency of allergic reactions to different foods. For example, in Italy, as in other European Mediterranean countries, allergic reactions to fresh fruits and vegetables often occur, in the Nordic countries – allergic reactions to fish (cod), in the United States-to peanuts. There is an opinion that the frequency of food allergies increases as a result of the use of genetically modified foods. However, there are no reliable data confirming this fact, and the results of studies show that people who are allergic to a native product react in a similar way to a genetically modified product [10]. At the same time, the removal of the main allergenic protein, for example, from peanuts, can lead to the creation of a safe product [10].Another common myth is the association of food allergies with food additives: preservatives, colorants, flavorings, etc. Allergic reactions to food additives are rare: from 0.01 to 0.23% in the general population, since most food additives have a simple chemical structure and low molecular weight [11]. True IgE-related allergies are caused only by enzymes and certain proteins derived from animals and plants and used in the cooking process. Such reactions can be induced by papain, gelatin, alpha-amylase, carmine, and annatto [11, 12,22].The molecular weight of most food allergens is between 10 and 70 kD. Allergens with a higher molecular weight should be fragmented. These are the so-called hidden allergens. Depending on the temperature effect, food allergens are divided into two groups: thermostable (resistant to heat) andthermolabile (not resistant to heat) The thermostable group of allergens characterized by a linear protein structure includes allergens of peanuts, fish, milk lacto globulin, egg albumin. All of them are resistant to high temperatures and can cause allergic reactions even after cooking the product.Food allergens of the thermos labile group have a spatial protein structure. Under the influence of high temperature, the three-dimensional structure of the protein changes and allergens lose their activity. This group includes allergens of fruits, vegetables, and meat, which often lose their allergenicity during heat treatment, but are active in raw fruits and vegetables.

Immunological mechanisms and clinical manifestations of food allergy

Table 1 shows the immunological mechanisms and clinical manifestations of food allergies that develop on their basis [22,24]. you can see, the clinical manifestations of food allergies are diverse and non-specific. Symptoms of a food allergy may appear a few minutes or hours after eating the "guilty" food product and vary in severity – from mild to life-threatening.

Clinical manifestations of the most common IgE-related food allergy occur, as a rule, within a few minutes or one to two hours after eating a food allergen and can be:local (oral allergic syndrome);

- ✓ systemic (anaphylaxis);
- ✓ gastrointestinal (nausea, vomiting, abdominal pain of a stabbing or spastic nature, diarrhea);
- ✓ respiratory diseases (rhinitis, bronchial asthma);
- ✓ skin diseases (pruritus, erythema (redness of the skin), urticaria and / or angioedema, atopic dermatitis (T-related reactions are involved), a crust-like rash).

The severity of the clinical manifestations of food allergy depends on the amount and type (raw or cooked) of the product eaten, the accompanying circumstances (physical activity, medication, alcohol consumption). The patient's age and underlying medical conditions (such as asthma, atopic dermatitis, etc.) also affect the severity of symptoms. For example, the development of the most severe clinical manifestation of food allergy – anaphylaxis-is often determined by the presence of such risk factors as the age of patients (most often in children, adolescents, and young adults), the presence of atopic diseases (primarily bronchial asthma), and / or adjuvant factors (physical activity, stress, hormonal changes during the menstrual cycle,etc.). medicinal products: acetylsalicylic acid, nonsteroidal anti-inflammatory drugs, codeine, angiotensin-converting enzyme inhibitors, beta-blockers, etc.).One of the most common clinical manifestations of food allergy in adults is urticaria. It can be accompanied by angioedema, which develops as a result of edema of the deep layers of the dermis, subcutaneous tissue or submucosal tissues. Common spontaneous urticaria can be acute if it lasts less than six weeks, and chronic if the rash persists for more than six weeks [13,16].In the pathophysiology of urticaria, a key role is played by mast cells of the skin, the activation of which by immuno- or non-immuno-conditioned triggers leads to the release of preformed and newly formed mediators from them. In food allergies, such a trigger is IgE antibodies to the food allergen. Histamine, bradykinin, leukotriene C4, prostaglandin D2, and platelet activation factor (FAT) are the main biologically active substances secreted by the mast cell in response to the interaction of a food allergen with specific IgE fixed on its membrane. These mediators cause extravasation of plasma into the dermis, which leads to the formation of a blister. The intense pruritus that accompanies this process is the result of histamine activation of unmyelinated skin cells. C-fibers of nerve endings in the skin. Histamine is a natural ligand for four types of membrane-bound receptors-$_{H1}$, H_2, H2, $H3_{,and\ H4}$. The most studied and significant$_{H1}$- and$_{H2}$-receptors. They are present on many cell types. $_{H1}$-receptors (postsynaptic) are present in the cells of the smooth muscles of the bronchi, digestive system, bladder, heart and blood vessels, and brain. $_{H2}$-receptors (postsynaptic) are found on the cells of the gastric mucosa, uterus, and brain. Activation $_{H1}$-histamine receptors on endothelial and smooth muscle cells lead to an increase in capillary permeability. Activation $_{H2}$-histamine receptors lead to vasodilation of arterioles and venules.How It is known that histamine is the most important mediator of immediate allergic reactions, which has a wide range of biological activity carried out by activating cellular surface specific receptors. The main pharmacological effects of histamine are manifested by increased vascular permeability, edema, hypersecretion and smooth muscle spasm. It is these effects of histamine that antihistamines block. However, other mediators are also secreted from the mast cell, which are also pharmacologically active substances and contribute to the formation of symptoms of an allergic reaction. In particular, such a pharmacologically active mast cell mediator is FAT.

Role of FAT in the pathogenesis of food allergies and anaphylaxis

FAT is a pro-inflammatory phospholipid synthesized by inflammatory cells (basophils, mast cells, macrophages, neutrophils, endothelial cells) under the influence of bacteria and viruses, immune complexes, complement components (C3A, C5A), as well as as a result of IgE-mediated stimulation of mast cells and basophils, including under the influence of food allergens. FAT has the following properties::

- causes platelet aggregation.
- activates the mast cells of the human lungs;
- causes degranulation of mouse and human eosinophils.
- attracts and activates eosinophils and neutrophils.
- with the inhaled route of administration, it can cause a short-term increase in vascular permeability, and this effect is 1000 times stronger than that of histamine;
- It stimulates the biosynthesis of cyclooxygenase (thromboxane A2) and lipoxygenase (cis-leukotrienes) products [26].

The pharmacological effects of FAT, as well as the effects of histamine, are realized through special receptors on cells ($_{G1}$and$_{G2}$), which are expressed on platelets, monocytes, neutrophils, B-lymphocytes, keratinocytes, cells of the lung, brain, kidneys, liver, and spleen. It is logical to assume that blockade of FAT receptors on cells will lead to a decrease or complete elimination of the pharmacological effects of this mediator. In addition, the circulating level of FAT is controlled by the activity of special FAT enzymes-acetyl hydrolases, which lead to FAT degradation

Diagnosis of food allergies

Given the non-specific nature of the clinical manifestations of food allergies, the main attention should be paid to the relationship of symptoms with food. Moreover, it should be remembered that an allergic reaction can be triggered not only by eating / swallowing a food allergen, but also when it enters the body by inhalation or in contact with the skin. Carefully collected medical history and analysis of the food intake often allow you to determine the food allergen. Figure 4 shows an algorithm for diagnosing food allergies proposed by international experts [1]. The results of studies indicate that the prognostic value of anamnesis and clinical symptoms for the diagnosis of immediate food allergies (in isolation or in combination with skin tests or determination of specific IgE in blood serum) is from 50 to 100% [26]. In the presence of clinical symptoms, their reduction as a result of the appointment of an elimination diet with the exclusion of the suspected food allergen for two to four weeks serves as additional confirmation of the diagnosis of food allergy. Skin tests and determination of specific IgE antibodies to food allergens are considered paramount in the diagnosis of immediate IgE-related allergies. However, like a medical history, these tests are not always able to accurately diagnose food allergies. That is why a diagnostic elimination diet and an oral challenge test with a suspected food allergen are necessary diagnostic techniques to confirm the diagnosis of both IgE-related and non-IgE-related food allergies. Together however, for most food allergens, a positive result of a skin pricktest with good-quality allergenic extracts is highly correlated with clinical symptoms. In the absence of such diagnostic allergens and/or in case of suspected involvement of minor or unstable allergens (fruits and vegetables), skin tests with native products can be performed [1]. Энтеропатий Endoscopic examinations and biopsies are required to confirm the diagnosis of food-induced enteropathies. Modern methods of molecular or component diagnostics allow us to identify specific IgE (SiGe) against the main (major) antigens of food products, which

increases the specificity of the diagnostic method and can be useful in the case of inconclusive data from skin tests and SiGe determination results. For example, the determination of SiGe to the main peanut allergen Arah2 shows 100% sensitivity and 70-80% specificity [20], and the determination of omega-5-gliadin has a high diagnostic significance for exercise-induced food allergy to wheat [24]. Methods of molecular allergic diagnostics are currently being widely studied and actively implemented in clinical practice.The current European Guidelines on Food Allergy once again draw attention to the fact that such tests as bioresonance diagnostics, iridodiagnostics, kinesiology and hair diagnostics, as well as cytotoxic tests and determination of IgG and $IgG_4$are not validated methods and cannot be recommended for the diagnosis of food allergies [1]. Thus, the presence of IgG 4 specific to food $allergens_4$ only indicates that the patient, especially with an atopic disease, has repeatedly and for a long time come into contact with this food product, which is perceived by the immune system as a foreign protein. It reacts to it by synthesizing antibodies (IgG_4) that do not have sensitizing and functional activity in relation to the release of mediators of an allergic reaction.As already noted, the provocative oral test is in most cases a mandatory diagnostic technique for confirming the diagnosis of food allergies, and the double-blind placebo-controlled provocative test has long been recognized as the gold standard for diagnosing food allergies. A provocative test with food allergens can be open and blind. Currently, special guidelines for conducting an oral provocation test with food allergens have been published, containing all the details of the procedure, doses, and types of allergens .

Treatment of food allergies

Treatment of food allergies consists of symptomatic therapy during clinical manifestations and a preventive strategy to minimize the risk of future allergic food hypersensitivity reactions, primarily involving an elimination diet with the exception of an allergenic food product, patient education, and the development of a pharmacological and non-pharmacological strategy for possible future reactions. The possibilities of allergen-specific immunotherapy for food allergies are being actively investigated, which shows promising results, but is not yet recommended for clinical practice [28,29].Symptomatic treatment of food allergies in the period of clinical manifestations is determined by the type and severity of symptoms. With the development of anaphylaxis, epinephrine is considered the primary drug. Antihistamines and glucocorticosteroids (corticosteroids) are used for skin and gastrointestinal symptoms. Respiratory symptoms are effectively eliminated by antihistamines, bronchodilators, or corticosteroids.

The role of antihistamines in the treatment of food allergy symptoms

A systematic review carried out within the framework of the European Guidelines on Food Allergies and devoted to emergency and long-term treatment of food allergies showed that there is little evidence for emergency treatment of non-life-threatening manifestations of food allergies. However, the use of$_{H1}$-antihistamines can be effective. The results of three randomized and two non-randomized clinical trials demonstrated the effect of these drugs on the symptoms of cross-reactivity in patients with pollinosis [14]. Please note: the effectiveness of antihistamines in the treatment of more severe food allergy reactions has not been noted, and preventive administration of antihistamines may mask the early symptoms of anaphylaxis and lead to delayed administration of epinephrine. Therefore, antihistamines are prescribed for already developed, non-life-threatening symptoms of food allergies, such as oral allergic syndrome, urticaria, atopic dermatitis, and allergic rhinitis. There are two groups of antihistamines: the first (sedative) and second (non-sedative) generation. The use of first-generation antihistamines is limited by their side effects: pronounced sedation, cholinergic, adrenergic effects, short duration of action, rapid development of tachyphylaxis. Adverse,

potentiating interactions of this group of drugs with alcohol, psychotropic and hypnotic drugs are described. Second-generation antihistamines are highly selective blockers of$_{H1}$-histamine receptors. The antihistamine effect of these drugs manifests itself quickly (within 1-2 hours) and lasts up to 12-24 hours. Accordingly, they are applied once, or at most twice day. Antihistamines second-generation drugs are practically devoid of or have a slight sedative effect, which usually does not differ from the placebo effect, do not affect cholinergic and adrenergic receptors, or this effect is insignificant. These drugs are effective in relieving the symptoms of an immediate allergic reaction and urticaria. Moreover, this effect is dose-dependent

Conclusion

Clinical manifestations of food allergies are non-specific and diverse, but most of them are based on an immediate allergic reaction involving IgEantibodies. A carefully collected medical history and dietary analysis often suggest that skin tests and determination of specific IgE can establish a food allergen. However, only a provocative test can definitively verify the diagnosis of food allergies. Treatment of food allergies consists of symptomatic therapy during clinical manifestations and a preventive strategy to minimize the risk of future reactions, including an elimination diet with the exclusion of an allergenic food product, patient education, and the development of pharmacological and non-pharmacological strategies for possible reactions. Modern second-generation antihistamines are effective tools for relieving non-life-threatening symptoms of food allergies, such as oral allergic syndrome, urticaria, and allergic rhinitis. One of the representatives of this class of drugs is rupatadine, which has a dual mechanism of action: pronounced selective blockade $_{H1}$-receptors and significant inhibitory activity in relation to FAT-receptors, which is realized by the high clinical effectiveness of the drug against skin manifestations of allergies and symptoms of allergic rhinitis. This allows us to recommend rupatadine for the treatment of non-life-threatening food allergy symptoms in adults and adolescents from 12 years of age.

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