



Current Diagnostic Capabilities for Gastrointestinal Food Allergies

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Abstract

Food allergies remain an urgent problem in children. Interest in it is growing every year, which is associated not only with its high prevalence, but also with difficulties in diagnosis and treatment. In early childhood, gastrointestinal forms of food allergies are more common, which often occur under the "masks" of other diseases, which further complicates the diagnostic search for causes. The review examines the causes and mechanisms of food allergy development, and presents its main forms. Modern approaches to diagnosis using clinical and anamnestic criteria and laboratory methods available in the doctor's arsenal are discussed.

Keywords: food allergy, children, gastrointestinal symptoms, diagnosis.

Interest in the problem of food allergy (PA) in pediatric practice continues to this day, due to the growing incidence in the pediatric population, the variety of clinical forms and diagnostic difficulties, especially in non-IgE-mediated mechanisms and isolated gastrointestinal manifestations. Food allergy is a pathological reaction caused by ingestion of a food product, which is based on immune mechanisms. [1]. An accurate assessment of the prevalence of PA in the pediatric population is quite problematic, since in clinical practice, toxic reactions to food, food poisoning, and digestive enzyme deficiency are often overestimated. Therefore, the number of children diagnosed with food allergies is not known for certain [2, 3]. However, according to the World Health Organization, PA is registered in 6-8% of children, young children and 2-4% of adolescents in the world [4, 5]. It is assumed that the prevalence of PA in children in the European population is 5-8%, while in the European population it is US \$ — 5% [6-8]. At the same time, according to self-assessment data, up to a quarter of the population indicates the presence of allergies to certain foods [9]. The European Academy of Allergy and Clinical Immunology (EAACI) predicts that by 2029, more than half of the European population will suffer from allergies and related diseases [10]. PA can occur by various pathogenetic mechanisms and is manifested by a large number of clinical syndromes and diseases. IgE- and non-IgE-mediated mechanisms, as well as immune complexes, may be involved in the development of the pathological process. delayed reactions and hypersensitivity. According to literature data, the pathogenetic basis of PA in most cases is a combination of different types of immunological reactions [11, 12]. Depending on the type of immune pathological reaction, there are differences in the time of occurrence, as well as the nature of clinical symptoms. Clinical manifestations of PA are polymorphic and may be localized in the skin, respiratory system, and gastrointestinal tract [2, 13, 14]. In general, up to 95% of PA cases in children are associated with skin manifestations, in 10-12% - with respiratory symptoms, gastrointestinal forms of PA are most characteristic of young children, reaching 60-70% in the structure, while in older age their share decreases to 10%, with age the share of combined forms of PA increases [15]. Features of clinical manifestations of gastrointestinal PA According to the classification proposed by the European Academy of Allergy and Clinical Immunology (EAACI), gastrointestinal forms of PA are usually divided according to pathogenetic mechanisms [16, 17] IgE-dependent: oral allergic syndrome, gastrointestinal



anaphylaxis cell-mediated: enteropathy caused by cow's milk proteins (BCM) and cereals allergic proctocolitis; mixed IgE-and non-IgE-mediated: eosinophilic esophagitis, allergic gastritis and gastroenteritis. Allergic lesions of the gastrointestinal tract do not differ in specific symptoms from non-allergic diseases of the digestive system; they are characterized by the same symptoms: nausea, vomiting, abdominal pain, diarrhea, constipation. In this regard, the diagnosis of gastrointestinal PA is very difficult; it is necessary to use various additional research methods to prove or disprove the allergic genesis of the disease. Suspect the allergic nature of the gastrointestinal tract damage: if gastrointestinal symptoms appear a few hours (2-4 hours) after consuming a possible allergenic product; if there are no symptoms during the elimination diet and resume after provocation; if there is a hereditary predisposition to allergic diseases; if there are concomitant allergic diseases (atopic dermatitis, bronchial asthma, urticaria, etc.);

When taking antihistamines has a positive effect; when the level of eosinophils in the blood test increases; when eosinophils are detected in the contents of gastric juice or in feces. If the complaints are directly related to a single intake of certain food allergens or medications, then the diagnosis is greatly facilitated. It is much more difficult to establish a link between symptoms from the gastrointestinal tract and the use of everyday meals. In these cases, a carefully collected medical history, long-term follow-up, and examination of the patient using modern methods of allergological and instrumental diagnostics helps to determine the cause of the disease. In early childhood, PA may present with isolated gastrointestinal symptoms such as abdominal pain, persistent regurgitation, colic, or stool disorders. Such children are treated for functional disorders of the gastrointestinal tract for a long time and often without lasting improvement, although these symptoms may be caused solely by an allergy to BCM [1, 2, 9]. Eosinophilic gastrointestinal pathology differs depending on the localization of the process. There are eosinophilic esophagitis, eosinophilic gastroenteritis and eosinophilic colitis. Often, eosinophilic inflammation can affect all parts of the gastrointestinal tract. In cases where non-IgE-mediated mechanisms of delayed-type reactions predominate in eosinophilic gastrointestinal pathology, the association of clinical symptoms with food is not obvious, and therefore it is difficult to identify causally significant food products [11]. That is why such children are often observed for a long time for chronic gastroenterological pathology, which is especially relevant in recent times, inflammatory bowel diseases and celiac disease. It is important to note that the distinguishing features of such children are the presence of a burdened allergic history and the lack of effect from standard therapy. Allergic eosinophilic esophagitis and allergic eosinophilic gastroenteritis develop by both IgE-mediated and non-IgE-mediated mechanisms; variants with mixed mechanisms are possible [15]. Morphological diagnosis of this pathology is eosinophilic infiltration of the esophageal, gastric, and/or intestinal mucosa, basal hyperplasia, and villi enlargement may occur [16]. In non-IgE-mediated cases, isolated symptoms of gastroesophageal reflux or pyloric stenosis may occur: regurgitation, vomiting, epigastric pain, and dysphagia, which also complicate the diagnosis of PA [15]. Allergic enterocolitis is clinically manifested by insufficient weight gain or loss, diarrhea or constipation, and deficient conditions (iron deficiency anemia, hypoproteinemia) [2, 18]. Food-induced proctitis and proctocolitis are more often observed in children of the first months of life who are not only artificially fed, but also breastfed; up to 50% of cases of allergy to BMD develop when fed exclusively with breast milk [13]. Clinically, proctitis and proctocolitis induced by BCM are manifested by mucus and blood in the stool with a satisfactory condition of the child and normal nutritional status. Endoscopic examination of such patients reveals edema of the colon mucosa, morphologically eosinophilic infiltration of the epithelium and its own lamina is noted [1, 2]. At the same time, in some children, allergic proctitis may have a pattern similar to Hirschsprung's disease, manifested by persistent constipation [19]. Food-induced enteritis or allergic enteropathy develops by a cell-mediated mechanism and is more common in children of the first three months of life [10, 17]. Its triggers at this age are also BCM allergens that enter the



child's gastrointestinal tract from the mother's milk during breastfeeding or from its substitutes during early artificial feeding. The possibility of developing allergic enteropathy during exclusive breastfeeding is explained by the fact that studies have proven the possibility of an allergic reaction when using 0.3 g of allergen per 1 kg of body weight [2]. Clinically, the disease is manifested by repeated vomiting and diarrhea, malabsorption with weight loss, and peripheral blood is characterized by eosinophilia [2, 10]. Some children (up to 20%) have hypotension due to hypovolemia and increased TNF-alpha secretion [17]. Red blood cells, neutrophils, and eosinophils are detected in the coprogram, and sometimes moderate steatorrheas detected. A biopsy reveals inflammation in the mucosa of the small intestine, lymphocytes, mast and plasma cells containing IdM and IdA are found in the lamina proper, and in some cases villi atrophy develops during a long course. The severity of morphological signs, with the exception of atrophy, is reduced 72 hours after allergen elimination [18]. Allergic enteropathy is often accompanied by the development of secondary lactase deficiency. Intestinal colic is a common problem in young children, 25% of children have a prolonged course of colic, in this case it is often based on an allergy to BCM [20, 21]. When such children are prescribed mixtures based on complete protein hydrolysate or when BCM is excluded from the diet of a nursing mother, there is a significant reduction and even relief of intestinal colic. Thus, PA can manifest itself in a variety of non-specific symptoms; however, a thorough analysis of complaints, the collection of anamnesis, the use of modern diagnostic methods, and the positive effect of an elimination diet indicate an allergic etiology of gastrointestinal tract damage. Laboratory diagnostics of the gastrointestinal form of PA Laboratory diagnostics of true PA is aimed at establishing causally significant allergens, specific allergens, and other diseases. Allergic antibodies or products of specific interaction of antibodies with antigens, as well as to detect reactions to food products that occur by the mechanism of delayed hypersensitivity [23]. One of the well-established diagnostic methods is skin tests, this is due to the simplicity of application, reproducibility results, and above all, this is a less expensive option compared to laboratory tests. Testing with food allergens can take the form of prick and patchtests. The prick skin test is considered a highly specific and affordable method for the diagnosis of PA, which proceeds according to the IgE-dependent mechanism. However, it should be borne in mind that a number of patients may have false positive or false negative results, and some patients have contraindications to the use of the test. In addition, the information content of the size of the papule, to confirm the reaction to food allergens, is different. The result of a provocative test is considered positive if the size of the papule in children under two years of age with the introduction of cow's milk exceeds 5 mm, chicken egg-4 mm, peanuts-3 mm [24]. Pricktests are recognized as the most reliable method of examination, in particular in patients with oral allergic syndrome [25]. Patchtests are used to diagnose PA by a non-IgE-mediated (cell-mediated) or mixed mechanism: eosinophilic esophagitis, enterocolitis, and atopic dermatitis [26-28]. The method consists in applying an occlusive dressing with food allergens applied to the patient's skin for 24 hours, followed by an assessment of the reaction (in the form of erythema and papules) after 24-72 hours. Patch tests are currently used very rarely, although it has been shown that in children under two years of age with atopic dermatitis and normal blood IgE levels prick-the test was positive in 4.6% of cases, while the patch test was positive in 68% of cases [29]. In foreign clinical practice, the gold standard for the diagnosis of PA is a double-blind placebo-controlled food provocation test. During the test, the patient receives an allergen or placebo, then, depending on the occurrence of a reaction in response to the received allergen, a conclusion is made about the presence or absence of PA. However, this procedure has a number of limitations, among which the most serious is the risk of developing severe reactions, including anaphylaxis. According to a multicenter study, during diagnostic provocative tests, anaphylactic reactions were recorded in 2% of the examined patients [30]. A special in vivo examination vivo is performed only in an allergological office, and provocative tests are performed only in a hospital setting [31]. The essence of provocative tests is to evaluate the patient's reactions when gradually increasing



amounts of suspected foods are administered, and they evaluate objective classical symptoms of immediate reactions (urticular rash, rhinitis, nausea and vomiting, asthma attack, anaphylactic shock) and subjective symptoms (itching, abdominal pain, hyperactivity or lethargy, headache, migraine, arthritis) [32]. Provocative food samples are carried out quite rarely in the world, and they are not certified at all in the Russian Federation [1]. Currently, there is a question of safer alternatives [33]. The main indications for the appointment of laboratory methods of allergodiagnosics are: early childhood, a high degree of sensitization of the patient, continuous relapsing course of the disease without periods of remission, the inability to cancel antihistamines and other drugs, polyvalent sensitization, sharply altered skin reactivity, urticular dermatographism [34]. For the specific diagnosis of PA with the detection of IgE antibodies to food allergens in vitro, various methods are used: enzyme immunoassay (ELISA); radio allergosorbent test (RAST); multiple allergosorbent test (MAST). Its diagnostic significance depends on the patient's age: young children have a low level of specific IgE (at the age of less than two years, 5kU/I has 95% diagnostic significance for BCM). An increase in the level of specific IgE indicates that the child is sensitized to the food allergen, while a high antibody titer indicates a higher probability of PA and the need for prolonged elimination of the allergen. The data obtained should be interpreted in conjunction with the clinical picture and results of dietary diagnostics, taking into account the possibility of false positive and false negative results. In particular, children with gastrointestinal symptoms often have a normal level of specific IgE, which does not exclude the presence of PA [35]. Such tests as antigen stimulation of cells (cellular allergen stimulation test, ST), basophil stimulation FAST (flow-cytometric basophil stimulation test), the immunoblot method (based on the separation of protein mixtures depending on their molecular weight, allows you to identify allergen-specific antibodies to various proteins), the inhibition reaction have not found application in practical healthcare. leukocyte migrations that reflect delayed hypersensitivity, the reaction of blast transformation of leukocytes, the basophil test (Shelley test) direct and indirect, which determine the presence of sensitization in the immediate type of hypersensitivity [36]. Methods for the diagnosis of PA that do not have a high-quality evidence base (not recommended by the EAACI, 2008; American Academy of Allergy, Asthma and Immunology (AAAAI) and the Canadian Society of Allergy and Clinical Immunology (CSACI)) are—measurement of the level of the 4th sub- class of specific immunoglobulins G (IgG), or IgG4 antibodies, to food antigens [37]; cytotoxic test with food antigens; vegetative resonance testing (VEGA); irigodiagnosics; analysis of hair composition; testing using applied kinesiology techniques [38, 39]. Speaking about the role of serological studies in detecting allergy to cow's milk proteins in children, it is worth noting that modern publications indicate the role of antibodies to free light chains of immunoglobulins (Ig-FLC) in the complex of allergodiagnosics [41]. Fecal calprotectin (a calcium - and zinc-binding protein of the S100 calgranulin family contained in the cytoplasm of neutrophils, to a lesser extent monocytes, macrophages, bone marrow cells, squamous epithelium, mucosal epithelium, fibroblasts, and some other cells) also acquires a scientific basis indicating diagnostic significance in allergic diseases. The formation and release of this protein increases dramatically during an inflammatory reaction, which makes it possible to use it as the main indicator of intestinal inflammation, including allergic. A direct correlation was found between the level of fecal calprotectin in feces and the burdened allergic state. medical history in children [42, 43]. However, this indicator is not specific for allergic inflammation and may increase in autoimmune diseases, functional disorders of the intestine, intestinal infections and invasions [44]. Currently, the attention of researchers is drawn to such an allergy marker as eosinophilic cationic protein (ECP), which is one of the main mediators of eosinophils present in the mucous membrane of the gastrointestinal tract and other organs and released from their granules in response to the interaction of allergen and IgE— immunoglobulin. ECP has immunomodulatory properties, as it affects lymphocytes and stimulates the Th2-type immune response, its significance is especially pronounced in allergic diseases. The concentration of ECP in the blood increases with the



development of allergic inflammation, so it is considered as a marker of exacerbation of allergic diseases and can be used both for assessing activity and for monitoring treatment [45]. A non-invasive test for the diagnosis of allergic inflammation is the detection of eosinophilic neurotoxin (EDN) in the stool. EDN is released from eosinophil granules in places of their accumulation, that is, allergic inflammation. The method allows you to diagnose PA even in cases where the level of IgE in the blood does not increase. In addition to diagnosing food allergies, EDN is used to differentiate between food allergies and non-allergic food intolerances, as well as to evaluate the effectiveness of an elimination diet and anti-allergic therapy. Since the test characterizes only eosinophilic inflammation, it is more specific for the diagnosis of PA, although an increase in EDN is also observed in inflammatory bowel diseases of a different genesis [1]. When the inflammation subsides, EDN also gradually normalizes, so it is used to monitor the treatment of allergic diseases. Kalach N. et al. In 2012, a study evaluated the diagnostic parameters of intestinal permeability (specific protein IgE and IgG, priktest, patchtest, provocative test for BCM) and fecal markers (eosinophilic protein), compared with standard allergic diagnostics in children. In newborns with manifestations of allergic enteropathy, the severity of clinical manifestations was associated with an increase in the level of eosinophilic neurotoxin [43]. Endoscopic examination of the gastrointestinal tract with morphological examination of biopsies is indicated for patients with persistent gastrointestinal complaints, delayed physical development, iron deficiency anemia that cannot be explained by other causes, diarrhea that persists after the child is transferred to parenteral nutrition and worsens after eating [1]. Macroscopically detect erythema, erosions, and nodularity or полипоидные polypoid growths in the intestine. With eosinophilic lesions of the gastrointestinal tract, a histological examination allows you to establish a diagnosis. In one biopsy of the gastrointestinal mucosa, the diagnostic criterion for eosinophilic pathology is the presence of at least 15-20 eosinophils in the field of view of a high-resolution microscope [400] [47, 48]. Due to the fact that the pathological changes are of a focal nature, it is necessary to take at least 5 biopsies from each part of the digestive tube, and a biopsy should be performed from both altered and apparently normal areas [49]. The multiplicity of clinical manifestations, complexity in the diagnosis of food allergies, as well as a lack of knowledge about the pathogenesis of the disease leads to inappropriate treatment of food allergies, deterioration of the quality of life of the whole family (economic costs of treatment, specialized food products) [50]. This makes the search for new ways to solve the problem of food allergies, the introduction of minimally invasive diagnostic methods, and modern approaches to prevention and treatment relevant. Conclusions Gastrointestinal forms of PA are quite diverse in their clinical manifestations and pathogenetic mechanisms, so they can often occur under the "masks" of various diseases. Timely diagnosis of PA is extremely important for the correct diagnosis, management tactics, and the ability to avoid fruitless and often negatively affecting the quality of life of the child diet therapy and drug treatment. Despite the large arsenal of laboratory and instrumental research methods, diagnostic examination of a patient with a gastrointestinal form of PA is a complex problem. Due to the etiopathogenetic features of the development of this pathology in childhood, most methods of allergological examination are uninformative (the probability of obtaining both false-negative and false-positive results) and have age restrictions. Endoscopic examination with morphological examination of a biopsy sample in gastrointestinal forms of PA is currently an accurate diagnostic method, but it is considered quite invasive, which also limits its use in children. One of the promising areas in the diagnosis of gastrointestinal PA in young children is the determination of eosinophilic neurotoxin. When examining a patient with a gastrointestinal form of PA, the scope and methods of examination should be determined by the clinical manifestations of the disease. The significance of a particular food allergen in the development of the disease can be considered proven if the results of an allergological examination coincide with the data of an anamnesis and the results of diagnostic elimination diets. The use of modern capabilities in the diagnosis of gastrointestinal PA makes it possible to establish clinically significant sensitization in a timely



manner, avoid time-consuming and potentially dangerous oral provocative testing, prescribe a rational diet, and prevent the development of severe complications.

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