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MODERN CONCEPTS OF THE CAUSES AND MECHANISMS OF DYSPEPSIA SYNDROME, THE ROLE OF ALLERGY IN THE DEVELOPMENT OF DYSPEPSIA

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Abstract

The relationship between gastrointestinal tract (GIT) lesions and allergic diseases is now widely recognized. Epidemiological studies have demonstrated a high frequency of their association, and the mechanisms underlying the development of allergies in gastrointestinal pathology have been largely elucidated. For example, the role of digestive disorders in sensitization and the development of allergies has been demonstrated [1]. Furthermore, the effects of biologically active substances that promote the development of allergic inflammation of the gastrointestinal mucosa are also known [5]. Atopic dermatitis (AD) is one of the most common allergic diseases in children, often accompanied by gastrointestinal lesions [7]. The most common form of these lesions includes dyspepsia syndrome (DS)—a symptom complex of pain and discomfort in the epigastrium and periumbilical region. However, the clinical presentation and pathogenesis of this condition in the setting of atopy have several distinctive features. These include more pronounced symptoms, a sluggish course, and a significant incidence of erosive and ulcerative lesions [1]. This is due to the fact that the action of allergy mediators is not limited to their involvement in the formation of an inflammatory response. Studies have demonstrated their influence on the motor and secretory activity of the upper gastrointestinal tract (UGT) [4]. Thus, biologically active substances are a distinct pathogenetic factor in functional disorders associated with gastrointestinal pathology. Children with allergic diseases are characterized

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by hyperreactivity of the UGT, which is expressed as increased basal and stimulated acidity and impaired motility [3].

Another common etiopathogenetic mechanism that can cause syntropy of diabetes and hypertension is Helicobacter pylori (HP). The ability of H.pylori to cause chronic inflammation of the gastric mucosa, increase its acid-forming function, and affect motility is well known [4]. However, in recent years, the extragastrointestinal effects of this microorganism have been actively discussed . In addition to the direct sensitization effect, H.pylori is capable of inducing immune responses of both general and local types [1]. Histopathology of the gastric mucosa chronically infected with H.pylori is characterized by a high number of macrophages, neutrophils, eosinophils, and lymphocytes. Macrophages secrete proinflammatory cytokines that direct the T-cell response along the proinflammatory pathway. Th1 - type [2]. Under the influence of H.pylori, the synthesis of tumor necrosis factor-α (TNF-α) increases , which is considered a cytokine capable of inhibiting cell division, slowing down the processes of tissue regeneration and repair, thus probably participating in the pathogenesis of AD. Many authors note the parallelism of the course of diseases in the gastric mucosa and in the gastric mucosa, as well as the correlation between the degree of Helicobacter pylori infection and the degree of dermal manifestations [1]. The nature of the gastric mucosa infiltrate in these cases has features of a combination of chronic inflammation in the acute stage and an allergic component. It has been established that with an increase in the degree of H.pylori contamination In the gastric mucosa, a progressive increase in the content of eosinophils and a decrease in the density of mast cells are observed [3].

Thus, there is sufficient reason to believe that a comprehensive approach to treating diabetes in children with AD will improve the effectiveness of these patients. However, in the available literature, we have not found pathogenetically substantiated recommendations for a differentiated approach to the diagnosis and treatment of these diseases when they coexist in children.

The aim of the study was to develop a differentiated approach to the diagnosis and treatment of dyspepsia syndrome in children with atopic dermatitis.

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Research Objectives

1. To determine the clinical and laboratory features of dyspepsia syndrome occurring against the background of atopic dermatitis in children.
2. To study gastric acid production and motility of the upper gastrointestinal tract in children with a combined course of dyspepsia syndrome and atopic dermatitis.
3. To identify the characteristics of the course of atopic dermatitis in children with dyspepsia syndrome depending on H. pylori infection and the state of the acid-forming and motor functions of the stomach.

Research Results

Dyspepsia syndrome in children with atopic dermatitis is characterized by a discrepancy between subtle pain and dyspeptic symptoms, on the one hand, and pronounced endoscopic and morphological changes in the gastric mucosa, on the other. Children with combined dyspepsia and atopic dermatitis have more pronounced disorders of the secretory and motor functions of the upper gastrointestinal tract compared to children without a history of allergies. Persistent H. pylori and increased acid production are factors that determine a more severe course of atopic dermatitis according to the BSOYUSH scale, progression of the process, and the early appearance of lichenoid and prurigo-like forms. A differentiated approach to the diagnosis and treatment of dyspepsia syndrome in children with AD, taking into account the identified clinical and pathogenetic characteristics, can improve treatment outcomes for atopic dermatitis and reduce the incidence of relapses.

For the first time, the characteristics of gastric acid production and motility in children with AD were studied depending on the stage and type of skin disease. Increased gastric acid production was found in children with AD during exacerbations, more pronounced in the lichenoid form of the skin disease.

For the first time, the influence of H.pylon on the course of AD in children has been established, manifested in a more severe course of AD in children with diabetes associated with H.pylon. It has been shown that a combination of diabetes and intestinal disorders is characteristic of children with AD.

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The clinical manifestations of dyspepsia syndrome in atopic dermatitis do not correspond to the severity of damage to the mucous membrane of the upper gastrointestinal tract, which is manifested in less pronounced pain and dyspeptic symptoms (%2=9.5) and more pronounced endoscopic and morphological changes (X - 10.7) than in children without an aggravated allergic background.
In the combined course of dyspepsia syndrome and atopic dermatitis, there are more pronounced complex disorders of secretory and motor function according to the Be Meesler index (30.3 + 10.3; 95% CI 26.1-34.5) compared to dyspepsia syndrome without an aggravated allergic background (16.6 + 3.6; 95% CI 15.4-17.8), the difference is reliable ($p < 0.05$).
Increased acid production is a factor associated with a more severe course of atopic dermatitis according to the 8 C (FAD) scale (K correlation = 0.31), which justifies the use of antisecretory drugs in the complex treatment of exacerbations of dermatitis in such children.

Conclusions

Atopic dermatitis in children with HP-associated dyspepsia is characterized by more frequent exacerbations, progressive course, early appearance of lichenoid and prurigo-like forms (X2 = 9.6, C = 0.32, at Cmax = 0.7). A differentiated approach to the diagnosis and treatment of dyspepsia syndrome in children with AD, taking into account the identified clinical and pathogenetic features, contributes to an improvement in the clinical course of atopic dermatitis in 29.9% (3.7-55.9) (OR, 95% CI - 3.5 (1.1 - 11.8)) and a 50% decrease in the risk of probable relapses (OR, 95% CI - 0.27 (0.08 - 0.88).

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