

AUTOIMMUNE GASTRITIS AND GASTRITIS ASSOCIATED WITH CELIAC DISEASE

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Abstract

Celiac disease is one of the most common autoimmune diseases in the world, affecting approximately 1% of the population. The typical form is manifested by malabsorption syndrome ; in childhood, the disease leads to disturbances of physical, psychomotor and sexual development. Celiac disease has been actively studied for 120 years, but only in the last decade have significant results been achieved that have changed the view of this disease. It was found that celiac disease is a systemic autoimmune disease affecting not only the small intestine, but also the organs of the endocrine and digestive systems. At the same time, the mechanisms of development of associated autoimmune diseases in celiac disease remain insufficiently understood. Thus, the study of changes in other organs and systems of the body in celiac disease is a priority today. In recent years, interest has increased in studying the involvement of the stomach in the pathological process in celiac disease. Based on morphological data, it was found that the prevalence of chronic gastritis (CG) in individuals with celiac disease is 80%-97.8%. The most studied type of chronic gastritis in celiac disease is lymphocytic . At the same time, other forms of chronic gastritis in celiac disease have been studied less. Morphological descriptions of chronic gastritis in celiac disease are quite contradictory. Thus, in lymphocytic gastritis in children with celiac disease, the antral part of the gastric mucosa (GM) is most often described as affected . Some researchers find that in other forms of chronic gastritis against the background of celiac disease, the fundus of the stomach is affected. In isolated studies, a relationship between celiac disease and autoimmune gastritis is traced. Thus, the etiological structure, clinical, functional, morphological, and other features of chronic gastritis in celiac disease still remain unclear. The prevalence, etiology, and pathogenesis of autoimmune gastritis in celiac disease are unknown. The question of the effect of a gluten-free diet (GFD) on the gastric mucosa remains open.

In celiac disease, damage to the gastric mucosa is detected in 80%-97.8% of cases. Since 2016, lymphocytic gastritis characterized by pronounced infiltration of the superficial and pitting epithelium, as well as the lamina propria by T-lymphocytes has been associated with celiac disease . Today, lymphocytic gastritis, chronic active gastritis (CAG), and chronic inactive gastritis (CIG) have been described in celiac disease. It has been found that these three types of gastritis have different localization, different morphological picture and different response to a gluten-free diet, but the reasons for the differences are unknown. Isolated studies report the possibility of developing autoimmune atrophic gastritis in celiac disease, but there are no

data on the prevalence of autoimmune gastritis (AIG) among children with celiac disease. To date, there are no consensus documents on uniform criteria and standards for the diagnosis of AIG. It has been shown that the presence of antibodies to gastric parietal cells is a reliable predictor of fundic atrophy of the gastric mucosa associated with AIH. It is known that at the early stages of AIH, atrophy of the gastric mucosa is absent; however, the morphological, clinical and functional features of autoimmune gastritis in children have not been studied, and the criteria for its diagnosis have not been developed. Pathogenetic mechanisms of the relationship between different types of CG and celiac disease are unclear.

The aim of the study. Based on the study of etiological, pathogenetic, morphological and functional features of gastric lesions in children with celiac disease, to improve the diagnosis of chronic gastritis in celiac disease.

Research objectives

1. To identify clinical and morphofunctional features of chronic gastritis in children with celiac disease.
2. To determine the etiological features of chronic gastritis in children with celiac disease
3. To identify the pathogenetic mechanisms that cause morphological changes in the gastric mucosa in children with celiac disease.

Results of the study

The true prevalence of celiac disease has not been sufficiently studied, which is associated with the diversity of clinical manifestations of this disease and the difficulties of its diagnosis. Numerous publications in recent years are mainly devoted to the clinical manifestations of celiac disease in children, while among the adult population they require further research. The criteria for differential diagnosis of celiac disease have not been sufficiently developed. The socio-medical problems of this disease have also been poorly studied. There are no unified approaches to organizing optimal medical, rehabilitation and psychological care for these patients. Despite some progress in the study of the mechanisms involved in the development of celiac disease, the genesis of extraintestinal symptoms of the disease remains poorly understood. A point of view is being formed according to which the main cause of the clinical manifestations of this disease are immune changes followed by the development of membrane digestion and nutrient absorption disorders. There is some information on the provoking role of *Helicobacter pylori* in the development of immune responses in patients with celiac disease. The question of the possible role of *Helicobacter* is actively discussed *pylori* in the development of atrophic processes in the mucous membrane of the stomach and duodenum. It is generally recognized that one of the prerequisites for impaired intestinal barrier

permeability and the development of food allergies is a change in the microbial composition of the intestine. In this regard, the search for ways to correct intestinal microflora, as well as the study of various approaches to maintaining intestinal microecology in a state of physiological balance in patients with celiac disease, are relevant for medical science. Some researchers associate the genetic basis of predisposition to celiac disease with the histocompatibility system (HLA system). Its role in the regulation of immune homeostasis of the human body, as well as in predisposition to various diseases, has been proven. The carriage of certain alleles of the HLA system in patients with celiac disease has been noted, which with a high probability indicates a genetic determination of the disease. Questions about the associations of alleles of the HLA system and their role in the regulation of immunity are not sufficiently covered in the scientific literature. The relationship between different variants of the clinical course of celiac disease and the histomorphological picture of biopsy specimens of the duodenal mucosa has not been sufficiently studied. There are no data from studies of the secretory activity of the stomach in patients with this disease. At present, the main task is to improve immunological methods for diagnosing and treating metabolic disorders in celiac disease, as well as their use in the complex therapy of patients. The above indicates the need for further scientific research to study the pathogenetic mechanisms of recurrence and progression of the disease.

Conclusions

1. For the first time, morphological changes in the mucous membrane of the fundus, body and antrum of the stomach in children with celiac disease were identified, their comparative assessment and assessment of the dependence on the presence or absence of a gluten -free diet were carried out.
2. Based on the definition of antiparietal autoantibodies , the prevalence of autoimmune gastritis in children with celiac disease was determined for the first time to be 12.1%
3. For the first time, it has been shown that autoantibodies to the gastric mucosa are gluten-dependent.
4. It has been established that in children with celiac disease, autoimmune gastritis is an additional risk factor for anemia.

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