Review

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Celiac disease in various types of diabetes mellitus

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ABSTRACT. The co-occurrence of celiac disease and diabetes mellitus, particularly in young children, is characterized by a severe clinical course, extreme metabolic lability, a tendency toward ketosis, and recurrent severe hypoglycemia, which requires modifications to the standard treatment protocol. Timely diagnosis of celiac disease in patients with diabetes mellitus is essential for correcting overt and potential metabolic disorders caused by malabsorption syndrome. This article presents a literature review that examines, from an evidence-based medicine perspective, the shared genetic factors and autoimmune mechanisms common to both celiac disease and type 1 diabetes mellitus. Particular attention is given to the epidemiological and pathogenetic aspects of the relationship between celiac disease and type 1 diabetes mellitus. The central focus is on the alteration in the clinical presentation of these diseases under conditions of comorbidity. The authors review the association of celiac disease with diabetes mellitus in autoimmune polyglandular syndrome and with latent autoimmune diabetes in adults. Special emphasis is placed on topics debated in the literature, including early diagnosis of celiac disease in various types of diabetes mellitus and the impact of malabsorption syndrome on patients' quality of life when control of either or both conditions is poor.

KEYWORDS: *diabetes mellitus, malabsorption syndrome, celiac disease, gluten-free diet, autoimmune diseases*

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Обзор

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Целиакия при различных типах сахарного диабета

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РЕЗЮМЕ. Сочетание целиакии и сахарного диабета, особенно у детей раннего возраста, характеризуется тяжестью течения и крайней лабильностью, склонностью к развитию кетоза, повторяющимися тяжелыми гипогликемиями, что вносит коррективу в лечебный протокол. Своевременная диагностика целиакии у пациентов с сахарным диабетом необходима для коррекции явных и потенциальных метаболических нарушений, вызванных синдромом мальабсорбции. В статье представлен обзор литературы, описывающий с позиций доказательной медицины общность генетических факторов и аутоиммунных процессов, характерных для целиакии и сахарного диабета 1-го типа. Особое внимание уделено эпидемиологическим и патогенетическим аспектам взаимосвязи целиакии и сахарного диабета 1-го типа. В фокусе проблемы – изменение клинической картины этих заболеваний в случае коморбидности. Авторами представлены литературные данные о сопряженности целиакии и сахарного диабета в структуре аутоиммунного полигландулярного синдрома, а также целиакии и латентного аутоиммунного сахарного диабета у взрослых. Особый акцент сделан на обсуждаемых в литературе вопросах ранней диагностики целиакии при различных типах сахарного диабета, влиянии синдрома мальабсорбции на качество жизни пациентов в случае нарушения компенсации той или иной патологии. В статье приведены рекомендации, касающиеся дифференциальной диагностики, диетотерапии и медикаментозного лечения пациентов при сочетании у них целиакии и сахарного диабета.

КЛЮЧЕВЫЕ СЛОВА: *сахарный диабет, синдром мальабсорбции, целиакия, безглютеновая диета, аутоиммунные заболевания*

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

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INTRODUCTION

Malabsorption syndrome (MS) is a major cause of impaired metabolic control in patients with diabetes mellitus, particularly in children [1–4]. At the same time, malabsorption syndrome in diabetes mellitus may be a consequence of autonomic neuropathy [1]. Along with pancreatic insufficiency, liver dysfunction, changes in the gut microbiota, and functional impairments in small intestine enzyme production, celiac disease may be a cause of MS [3–5]. Celiac disease is currently considered a systemic immune-mediated disorder manifested by gluten-dependent symptoms and specific antibodies in genetically predisposed individuals [5, 6]. Taking into account that clinical manifestations of this disease are characterized by significant polymorphism, the diagnosis of celiac disease in patients with diabetes mellitus is often difficult [7].

The literature discusses associations between celiac disease and such autoimmune diseases of the endocrine system as type 1 diabetes mellitus (T1D), autoimmune thyroiditis (AIT), and gonadal involvement, as well as pathologies of the gastrointestinal tract (GIT): autoimmune gastritis, hepatitis, cholangitis, and others [8–12].

AIM

The aim of this review is to describe main mechanisms underlying the relationship between celiac disease and various types of diabetes mellitus, as well as specific features of therapeutic management in patients with celiac disease and diabetes.

MATERIALS AND METHODS

A literature search was conducted in international databases (PubMed, eLIBRARY, NCBI) using such keywords as “diabetes mellitus,” “malabsorption syndrome,” “celiac disease,” “gluten-free diet,” and “autoimmune diseases.” A total of 76 medical literature sources were selected for the literature review.

RESULTS AND DISCUSSION

Celiac disease, being a chronic immune-mediated condition, may be accompanied by extraintestinal manifestations and diseases affecting various organs. Moreover, exocrine pancreatic insufficiency (EPI) may be observed in one-third of children diagnosed with celiac disease and, as a rule, resolves after switching to a gluten-free diet (GFD). At the same time, chronic pancreatitis, including autoimmune forms, is an extremely rare comorbid condition in children with celiac disease, and very few cases in children have been described in the literature. Type 1 diabetes mellitus (T1DM) is a well-known and common comorbid condition in children with celiac disease. The main determinant of this association is a common predisposing factor related to HLA, although non-HLA-related genetic and environmental factors (viruses, gut microbiota, etc.) have also been identified and may be involved in the development of both autoimmune diseases [12].

EPIDEMIOLOGY OF CELIAC DISEASE ASSOCIATED WITH TYPE 1 DIABETES

The prevalence of T1D is 86.73 per 100,000 children and 203.29 per 100,000 adolescents [13]. The prevalence of celiac disease in T1D, according to various authors, ranges from 3–10% [14, 15]. Antibodies to tissue transglutaminase (anti-tTG) are detected in children with T1D with the same frequency [16–19]. The incidence of T1D has been rising in recent years parallel to celiac disease [20, 21]. In most patients, T1D diagnosis precedes celiac disease. However, there is evidence suggesting that early-onset celiac disease (before 20 years of age) is associated with a higher risk of developing diabetes [21]. Studies have shown that T1D develops in 0.14% of individuals with celiac disease compared to 0.06% in a control group of patients without celiac disease or autoimmune diseases, with the highest risk observed among children under 18 years of age. These results confirm that it is necessary to screen individuals at high-risk groups [18].

PATHOGENIC LINKS BETWEEN CELIAC DISEASE AND TYPE 1 DIABETES

The relationship between type 1 diabetes and celiac disease still requires further study. A common autoimmune mechanism is hypothesized, but it is associated with various genetic features determined by differences in the major histocompatibility complex (MHC), including *HLA* and *non-HLA* genes [22, 23].

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According to the European Society of Gastroenterology Guidelines and the Draft Clinical Recommendations for the Diagnosis and Treatment of Celiac Disease in Children, it is important to note that the HLA-DQ2 haplotype is detected in 90–95% of patients, and HLA-DQ8 in the remaining 5–10%. The absence of alleles typical of celiac disease in the genotype makes the development of the disease extremely unlikely [2, 3]. A strong link has been established between this disease and human HLA antigens located on chromosome 6p21. Literature analysis indicates that inflammatory bowel diseases, rheumatoid arthritis, and type 1 diabetes often involve mutations in the regulatory regions of genes located near HLA genes, which may explain the association of celiac disease with autoimmune processes [2, 4, 24].

A study conducted in Southern Iran among children with celiac disease showed that the DQ4a*4b allele also has a high frequency in samples from the main group of patients (78%, $p < 0.01$), followed by the DQ5a*5b allele (12%, $p < 0.01$). In other words, a higher prevalence of DQ4/DQ5 is observed in children with celiac disease compared to the control group (odds ratio 6.5, confidence interval 0.84–69.46, $p < 0.01$), which indicates the need for more extensive genetic studies in celiac disease, not limited to identifying HLA-DQ2/DQ8 haplotypes [25].

At the same time, T1D is a complex polygenic disease for which more than two dozen genetic regions have been identified, each designated IDDM (insulin-dependent diabetes mellitus) and assigned a serial number. The IDDM1 (MHC) and IDDM2 (insulin gene) loci have the most significant influence on T1D [23]. Various genetic markers also confirm the role of genetic predisposition in type 2 diabetes [26]. Genetic testing for gene polymorphisms that contribute to impaired carbohydrate and/or fat metabolism in patients with overweight and obesity is currently relevant as well. It is possible to create personalized recommendations for dietary therapy and physical activity to prevent diabetes in patients with a family history of the disease [27].

HLA molecules play a role in celiac disease pathogenesis due to their involvement in antigen presentation to antigen-presenting cells (T-lymphocytes, macrophages, dendritic cells) in the small intestinal mucosa (SIM), which begin to produce numerous biologically active substances (e.g., interleukin-15). These substances have a damaging effect on the intestinal epithelium and ultimately enter the systemic circulation. Genetic typing of *HLA* genes is diagnostically significant in identifying the disease among individuals belonging to risk groups (first-degree relatives) or exhibiting signs of an atypical form of celiac disease [3–5].

Type 1 diabetes is characterized by destruction of β-cells, which typically leads to absolute insulin deficiency. Autoimmune processes play a crucial role in the pathogenesis of T1D. Type 2 diabetes mellitus is a group of heterogeneous disorders of carbohydrate metabolism characterized by a predominance of either insulin resistance or impaired insulin secretion. In children, T2D is usually detected incidentally during routine screening for obesity or a family history of diabetes [13]. In individuals over 35 years of age, T2D is often accompanied by T1D, which is characterized by very slow progression. This distinct subtype has been termed LADA (latent autoimmune diabetes mellitus in adults). In the World Health Organization classification, this subtype of diabetes is considered a variant of T1D [23]. The autoimmune process in LADA has several distinctive features. The main types of autoantibodies in patients with LADA are antibodies to glutamic acid decarboxylase (GAD) and islet cell cytoplasmic autoantibodies (ICA), with the former being detected much more frequently. Other autoimmune diseases are frequently detected in LADA. For example, AIT occurs in 4% of patients with T1D and in 25% of patients with LADA. Celiac disease is detected in 10% of patients with T1D and in 19% with LADA [13, 14].

It should be noted that T1D may occur alongside other endocrine and non-endocrine diseases of autoimmune origin. This cluster of diseases is known as “polyglandular autoimmune syndrome (PAS). Decompensation of autoimmune thyroid disease, the development of adrenal insufficiency, and worsening of celiac disease, autoimmune gastritis, and pernicious anemia in patients with T1D can lead to impaired metabolic control. The increased risk of these diseases is associated with the presence of genetic markers such as HLA class II complex haplotypes, and the *CTLA-4, PTPN22, FOXP3* genes, etc. [14]. According to current data, PAS-1 is a rare hereditary autoimmune disease resulting from mutations in the Autoimmune REgulator (AIRE) gene and characterized by multiple organ dysfunction. Diabetes is a compo-

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nent of this disease. The prevalence of diabetes in patients with PAS-1 in Russia is high (14.1%) [15]. According to a Finnish study, which includes the largest number of patients with Type PAS-1 and the longest follow-up periods, 18% of patients with PAS-1 have diabetes. A low prevalence of diabetes is observed in countries such as China, Thailand, Korea, and Japan [14].

Almost all forms of diabetes are characterized by potential complications resulting from impaired glucose and lipid metabolism. Elevated levels of total lipids, triglycerides, and phospholipids are frequently detected in patients with diabetes and enteropathy. Steatorrhea is observed in the stool lipid profile of 50% of patients. This is associated both with increased triglyceride excretion due to impaired pancreatic exocrine function and insufficient lipase secretion, as well as with impaired absorption of phospholipids and monoglycerides in the small intestine resulting from disruption of the gut microbiota [1].

CHARACTERISTICS OF THE CLINICAL PRESENTATION

Clinical symptoms of celiac disease in patients with T1D may be absent or minimal. In such cases, recurrences of hypoglycemia or uncontrolled hyperglycemia may be sole manifestations of the disease, which may pass unnoticed due to dominant symptoms of T1D, particularly when the patient is on insulin therapy. Most researchers emphasize the need for timely diagnosis of celiac disease in T1D to ensure effective management of these conditions [26]. It is noted that the risk of celiac disease is higher in cases of early-onset T1D (before age 4) and late-onset T1D (around age 45) [27]. Screening is recommended in these groups [28].

Studies have shown that patients with celiac disease and T1D have an increased risk of developing diabetic retinopathy and nephropathy. At the same time, patients with T1D and undiagnosed celiac disease had a higher prevalence of retinopathy (58% vs. 25%) and nephropathy (42% vs. 4%) [22].

According to the All-Russian Consensus on the Diagnosis and Treatment of Celiac Disease in Children and Adults, patients with T1D should be screened for celiac disease, especially if they have any gastrointestinal complaints or laboratory abnormalities that suggest celiac disease [3, 5]. It is advisable to conduct periodic examinations of patients with T1D to identify signs of PAS and to promptly address potential metabolic and vascular complications [14]. Patients with T1D, autoimmune, and endocrine diseases may be classified as high risk for celiac disease and require serological screening tests [3, 28]. Point-of-care rapid tests for diagnosing celiac disease to detect a spectrum of anti-tTG antibodies, as well as antibodies to deamidated gliadin peptides (anti-DGP) and antibodies to endomysium (anti-EMA), are recommended for use in clinical practice [29]. The clinical presentation of celiac disease may be dominated by gastrointestinal manifestations, such as diarrhea (voluminous, foul-smelling stools), steatorrhea, abdominal pain, flatulence, abdominal distension, vomiting, and loss of appetite [3–5]. Nonspecific symptoms include delayed physical development, weight loss, muscle hypotonia, and apathy. Clinical symptoms of celiac disease appear gradually in most cases. Appetite disturbances, unexplained vomiting, and weight loss are noted [27].

Previously, there were four forms of the disease: typical (classic) celiac disease, characterized by clinical symptoms of malabsorption (chronic diarrhea, wasting, and “deficiency” symptoms resulting from impaired absorption of minerals and vitamins), and atypical celiac disease, in which gastrointestinal symptoms are absent or mild, while extraintestinal manifestations such as osteoporosis, anemia, infertility, neurological symptoms, and others take center stage in the clinical picture [2, 4, 12]. In modern medicine, the division of celiac disease into classic and atypical forms appears unfounded, since atypical variants occur much more frequently than classic ones. It is recommended to distinguish between cases with pronounced symptoms (both those related to the gastrointestinal tract or extraintestinal symptoms) and asymptomatic conditions. People with the asymptomatic (latent) form of the disease show no signs of the disease, and it is detected during preventive screenings or when family members are examined [3].

DIAGNOSIS

Non-invasive serological tests are currently used to diagnose celiac disease.

It is important to perform such testing before starting a gluten-free diet, when patients are con-

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suming their usual number of gluten-containing foods. Restricting or eliminating gluten from the diet can cause a rapid decrease in specific antibody levels, which may complicate or prevent further testing [2–4, 12]. For example, anti-DPG, anti-tTG, and anti-EMA antibodies (IgA and IgG) are detected in blood samples from patients consuming gluten. IgA antibodies produced by B-lymphocytes of the small intestine's own tissue are the most valuable for diagnosis. However, if the total IgA level in serum is reduced, IgG antibodies become more informative [3, 4].

The high probability of coexisting type 1 diabetes, autoimmune diseases, and celiac disease is demonstrated by the studies of E.O. Hennessy et al. (2012), M.S. Popoviciu et al. (2023) [11, 30]. For example, seropositive individuals with an anti-tTG titer >100 were significantly more frequently identified among patients with T1D and concomitant autoimmune diseases. Individuals homozygous for the DR3-DQ2 haplotype have a 33% risk of testing positive for anti-tTG, whereas those with the same haplotype but without T1D have only a 2% probability.

However, when testing for celiac disease, it should be noted that false-negative results may occur in cases of minor inflammatory processes in the small intestinal mucosa in latent forms of celiac disease, when antibody production is reduced due to low activity of type 2 tissue transglutaminase [2, 4]. Anti-tTG antibodies may not be produced even in the presence of clinical and morphological changes characteristic of celiac disease if the patient has hypogammaglobulinemia. So-called seronegative celiac disease can be diagnosed based on the results of a thorough examination, and according to the literature, it occurs in 6–22% of cases [2–5]. The rapid celiac disease test is an immunochromatographic assay designed to detect anti-tTG IgA and IgG in human whole blood. The use of a rapid celiac disease test may be useful for screening high-risk patients. Testing in a non-laboratory setting took 10 minutes and did not cause adverse effects in children. The prevalence of seropositive patients was 3.3% [30]. Earlier studies have demonstrated the high diagnostic value of such assays in screening for atypical forms of celiac disease in patients with type 1 diabetes [31, 32].

As for endoscopic markers of celiac disease, they are highly nonspecific. During endoscopy, celiac disease may be suspected upon detection of characteristic changes: flattening or absence of annular folds in the duodenum, the appearance of transverse grooves, a honeycomb pattern, or micronodularity of the mucosa. The characteristic complex of changes associated with this disease includes an increase in the number of lymphocytes between epithelial cells, varying degrees of villus shortening, and crypt hyperplasia. Nevertheless, the macroscopic appearance of the mucosa may be normal, which limits the utility of endoscopy as the primary diagnostic method [3, 33]. According to official guidelines for diagnosing celiac disease, if patients with symptoms have Anti-tTG IgA levels exceeding the upper limit of normal by more than 10-fold, along with elevated Anti-EMA levels and positive HLA DQ2/DQ8 markers, a diagnosis of celiac disease can be established without a biopsy [3]. In order to reliably assess the condition, morphological studies must be conducted while the patient continues their usual consumption of gluten-containing foods. Morphological diagnosis becomes more accurate when five or more tissue samples are taken, including the duodenal bulb region. Eliminating gluten from the diet can rapidly restore the normal structure of the mucosa, which will significantly complicate or even preclude the possibility of confirming the diagnosis through morphological analysis [3, 34].

An increase in the number of interepithelial lymphocytes may be observed in various pathological conditions (such as food allergies, viral intestinal infections, giardiasis, autoimmune diseases, and inflammatory bowel diseases) associated with changes in the histological structure of the small intestinal villi corresponding to Marsh–Oberhuber type 1. Such changes in the SIM cannot serve as a basis for diagnosing celiac disease without corresponding immunohistochemical analysisю They must be evaluated only in combination with the clinical picture and data from serological and genetic testing [3, 35]. It should be noted that a distinctive feature of interepithelial lymphocytes in celiac disease is the presence of a specific T-cell receptor on the surface of most enterocytes, which is utilized in immunohistochemical studies to determine the predominant type of lymphocytes in the SIM [2, 4]. The identification of type 2 and 3 lesions during microscopic examination (type 2—hyperplastic, type 3—destructive, in accordance with the Marsh–Oberhuber pathomorphological classification of enteropathy severity) is sufficient grounds for diagnosing celiac disease in seropositive patients,

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even in the absence of clinical manifestations of the disease [3].

In cases serological test are negative, some researchers believe that a gluten-free diet (GFD) should be initiated. If a patient responds to the diet, a diagnosis of seronegative celiac disease is rendered [36].

Other authors argue that patients with seronegative celiac disease should not be prescribed a GFD because the diet does not improve quality of life, and they describe acute nutrient deficiencies in patients with seronegative celiac disease [5].

Diagnosis of seronegative celiac disease, especially when combined with type 1 diabetes, anemia, chronic gastritis of mixed etiology (autoimmune and Helicobacter pylori-associated), and a family history of autoimmune diseases such as type 1 diabetes, AIT, presents certain challenges. Differential diagnosis with other pathological conditions is necessary. For example, it is necessary to rule out gluten sensitivity not associated with celiac disease and common variable immunodeficiency [37].

When a patient has both celiac disease and T1D, adherence to a diet is indicated, as a gluten-free diet forms the basis for treating celiac disease, while controlling carbohydrate intake is crucial for T1D. The dilemma of combining diets for these conditions stems from the fact that recommended gluten-free foods often have a high glycemic index and contain elevated amounts of added sugars and fats, which can negatively impact blood glucose control [26].

However, it is believed that GFD in children with T1D and celiac disease improves control of the disease and prevents growth retardation, even though in some cases there is a slight increase in insulin dosage in response to corrected malabsorption and the higher glycemic index of gluten-free foods.

A gluten-free diet has a preventive effect on vascular complications of type 1 diabetes [38]. Low adherence to dietary therapy in patients in this group is associated with osteopenia and various disorders of osteogenesis. A gluten-free diet and improved glycemic control play an important role in preventing osteopenia complications, diagnosed by a decrease in bone mineral density [2, 4, 39, 40].

TREATMENT

Treatment of celiac disease in patients with T1D involves dietary therapy that includes foods that are gluten-free and do not worsen glycemic control. Processed gluten-free foods are low in certain vitamins and minerals, such as B vitamins, vitamin D, calcium, iron, zinc, and magnesium, which will require dietary optimization and supplemental intake of vitamins and minerals.

It is essential to avoid not only foods that contain “obvious” gluten (bread, baked goods and pastries, pasta, wheat/semolina, barley/pearl barley, bulgur, couscous, spelt, triticale, kamut), but also those containing “hidden” gluten. This refers to gluten used as a food additive during production. Safe grains for people with celiac disease include rice, buckwheat, corn, millet, amaranth, quinoa, montina, chumiza, sago, sorghum, and teff. Flours and starches made from root vegetables such as potatoes, cassava, tapioca, and sweet potatoes as well as legumes (beans, peas, soybeans) and various nuts are safe as well. Specialized gluten-free products such as substitutes for bread, pasta, and baked goods are recommended for patients with celiac disease. In Russia, the consumption of oats is not recommended for celiac disease because oat groats are often contaminated with impurities from other grains [3].

Patients with type 1 diabetes and celiac disease are advised to follow a long-term low-carbohydrate diet, while taking into account the glycemic index of foods. Insulin therapy, in conjunction with dietary therapy, must be administered under strict monitoring of blood glucose levels. Frequent, small meals (5–6 times a day) are recommended, taking into account the principles of mechanical, thermal, and chemical sparing [26]. Given the need for frequent meals and strict monitoring of carbohydrate intake, the use of an insulin pump to administer ultra-short-acting insulin is the most optimal approach for patients with type 1 diabetes, especially for children and adolescents. This method of insulin administration expands opportunities for dose adjustment in cases of gastrointestinal disorders [39, 41].

However, it is important to remember that adherence to a restrictive diet by children with celiac disease and T1D affects their quality of life. For example, children face emotional and behavioral challenges in schools as they adapt to their condition and dietary needs. Therefore, parents and teachers must work together to implement necessary changes in school practices to ensure comprehensive care for these children [41–44].

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Drug therapy for celiac disease is secondary, but in some cases it can be vital. Long-term, repeated courses of highly active microencapsulated pancreatic enzymes are often indicated to correct digestive processes and are mandatory during periods of acute diarrhea. During exacerbations of diarrhea, adsorbents that neutralize organic acids, astringents, and coating agents are widely used; for example, Smecta, which is administered as a suspension before meals. The use of loperamide is contraindicated [3]. The most common differences in the microbiota in children with T1D compared to healthy children include changes in the abundance of *Bacteroides, Streptococcus, Clostridium, Bifidobacterium, Prevotella, Staphylococcus, Blautia, Faecalibacterium, Roseburia, and Lactobacillus* spp. [44]. Disruptions in gut microbiota in patients with T2D reach 60% [45]. To restore the microbial composition in cases of recurrent diarrhea, courses of prebiotics, probiotics, and symbiotics are indicated. According to specialists' observations, in two-thirds of patients, disturbances in the aerobic flora resolve, and in 50% of cases, Bifidobacteria reappear or their titer increases. In this regard, it is advisable to conduct repeated courses of treatment with probiotics and prebiotics during recurrent episodes of diarrhea [3]. According to leading endocrinologists, it is advisable to assess the influence of the gut microbiota not at the level of types, but of specific species and the way they cause certain changes in patients with diabetes [46].

Nutritional status disorders in children and adolescents on long-term GFD are manifested primarily by a decrease in body mass index, which was observed in 34.4%. Grade I protein-energy malnutrition and obesity are rare (8.6% and 2.8%, respectively). With long-term adherence to a low-carbohydrate diet, vitamin B6 deficiency was detected in 14.3%, decreased ionized calcium in 40.0%, decreased serum iron in 11.4%, and mild anemia in 5.7% of children [47]. To correct serious protein metabolism disorders, such as severe protein deficiency, plasma and albumin are administered intravenously. In some cases, anabolic hormones are used to improve protein absorption [45]. The use of glucocorticoid medications in celiac disease is indicated in cases of severe disease with pronounced protein-energy malnutrition and as replacement therapy to correct adrenal insufficiency [3]. The above-mentioned features of celiac disease treatment must be taken into account in patients with type 1 diabetes, as both anabolic hormones and glucocorticoids are counterinsular hormones [13].

Patients with celiac disease who have calcium metabolism disorders (rickets-like syndrome, hypocalcemic seizures, osteopenia, and osteoporosis) are prescribed calcium and vitamin D_3 supplements [48]. Studies evaluating densitometry results in children have revealed a decrease in bone mineral density not only at the time of celiac disease diagnosis but also during subsequent follow-up of these patients. It is important to note that patients with type 1 diabetes, even without celiac disease, are at risk of decreased levels of total and ionized serum calcium. In cases of long-standing decompensated diabetes, bone metabolism is impaired [48, 49].

In cases of pellagra syndrome associated with celiac disease, nicotinic acid preparations are used. Preventive treatment with nicotinic acid preparations is also relevant for patients with diabetes [50]. For the primary and secondary prevention of cardiovascular complications in diabetes among adult patients with and without dyslipidemia, statins are recommended as first-line medications. To treat and prevent vitamin deficiencies in both patients with diabetes and those with celiac disease, courses of B-complex vitamins, vitamin C, and fat-soluble vitamins (A, D, K, and E) are recommended [3, 9, 51].

CONCLUSION

The management of patients with a combination of diabetes and enteropathy requires a multidisciplinary approach involving a gastroenterologist, an endocrinologist, and a dietitian.

Even such an advanced and expensive method of treating T1D as insulin pump therapy may not be effective without proper dietary correction and medication, in case of uncontrolled celiac disease. It is impossible to predict the correlation between insulinemia and glycemia in cases of impaired intestinal absorption. Both medical and economic factors listed above and maintaining a good quality of life for require accurate and timely diagnosis of celiac disease in patients with diabetes. This is the only way to ensure patient compliance, which will improve the quality of their treatment and ensure the effective use of medical and economic resources.

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ADDITIONAL INFORMATION

Authors' contribution. All authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

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