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Clinical masks of celiac disease in children: examples from practice

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Celiac disease is a chronic autoimmune disease, the basis of its pathogenesis is damage of the mucous membrane of the small intestine by gluten. The prevalence of celiac disease is steadily rising and occurs in both children and adults. Clinical manifestations of celiac disease can be typical, i.e. the development of diarrhea, steatorrhea, malabsorption, and atypical ones.

The aim: to deepen the knowledge of doctors about the polymorphism of clinical forms of celiac disease in children.

Clinical case. The article highlights three cases of atypical course of celiac disease and gluten-sensitive disease. Various clinical forms of gluten intolerance in children are shown, which were manifested by recurrent alopecia, persistent seborrhea, pronounced abdominal syndrome after taking a gluten-containing product in combination with rapid physical activity.

Conclusions. In addition to celiac disease, there are a number of that make their recognition complicated and delay their treatment. That is why timely clinical and laboratory diagnosis of gluten-sensitive diseases is important, and it allows us to recognize the disease on time, to prescribe treatment and thereby to optimize the prognosis.

The research was carried out in accordance with the principles of the Declaration of Helsinki. The informed consent of the patient was obtained for conducting the studies.

No conflict of interests was declared by the authors.

Key words: celiac disease, children, gluten intolerance, diagnostics.

Клінічні маски целіакії у дітей: приклади з практики

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Целіакія є хронічним автоімунним захворюванням, в основі патогенезу якого є пошкодження слизової оболонки тонкої кишки глютеном. Поширеність целіакії невпинно зростає та зустрічається як у дітей, так і в дорослих. Клінічні прояви целіакії можуть бути класичними – з розвитком діареї, стеатореї, мальабсорбції, так і атиповими.

Мета: поглибити знання лікарів різних спеціальностей щодо поліморфізму клінічних форм целіакії у дітей.

Клінічні випадки. Відображено три випадки атипового перебігу целіакії і глютен-чутливих захворювань без целіакії. Виявлено клінічні форми непереносимості глютену у дітей, які проявилися рецидивуючою алопецією, стійкою себореєю, абдомінальним болем при вживанні глютенвмісних продуктів в поєднанні з фізичним навантаженням.

Висновки. Окрім класичних форм целіакії існують інші нозологічні форми глютен-чутливих захворювань, що утруднює їхнє вчасне розпізнання і відтерміновує лікування. Тому важливим є їхня своєчасна клініко-лабораторна діагностика, що дасть змогу вчасно призначити лікування і тим самим оптимізувати прогноз.

Дослідження виконано згідно з принципами Гельсінської декларації. На проведення досліджень отримано інформовану згоду батьків дитини. Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: целіакія, діти, непереносимість глютену, діагностика.

Introduction

In recent years, there has been an increase in interest in the problems of gluten-dependent diseases both among the scientific medical community and among practising doctors [3,13]. More recently, the range of disorders caused by a pathological reaction to gluten has included only classical celiac disease and food allergy to wheat. At present, another form of gluten intolerance has been identified, i.e., gluten intolerance without celiac disease, which, according to the diagnostic criteria adopted in Oslo in 2012, is allocated to a specific nosology [3,8–10,12].

The most severe form of gluten intolerance is celiac disease, which is a chronic autoimmune disease that affects the digestive tract of genetically predisposed (HLA-DQ2, HLA-DQ8) people who have intolerance to the main cereal protein (gluten). Celiac disease causes chronic inflammation of the mucous membrane (MM) of the small intestine, which leads to its atrophy, malabsorption syndrome, with the development of various clinical symptoms, with the possibility of complete recovery of the intestinal mucosa after cessation of its contact with gluten [9,10,13].

Celiac disease used to be classified as a rare disease that occurred in children and was presented by typical diarrhea and malabsorption, but due to the use of special diagnostic tests in the 60s of the previous century, it has been possible to assess the prevalence of the disease in a different way. At present, the prevalence of gluten enteropathy in the adult population is 1:100 – 1:250 or 0.5–1% of the total population [8,9,11].

Some interesting data on the predicted prevalence of celiac disease in the USA were published by the National Institutes of Health in 2006. According to them in the USA 500,000 Americans suffered from non-specific ulcerative colitis, 500,000 ones suffered from Crohn's disease, 333,000 ones suffered from multiple sclerosis, 30,000 ones suffered from cystic fibrosis, and at least 3 million people were estimated to be affected by celiac disease, among them only 3% of patients had been diagnosed and were receiving adequate treatment of [6,10,12].

Celiac disease (gluten enteropathy) can occur both in childhood and in adulthood. The ratio of sick women to men is 2:1. The risk of celiac disease is especially high in relatives of the first line of kinship, i.e. 1:10, in the second line it is 1:39; in persons with so-called «associated» pathology it is 1:56 (for example, autoimmune diseases, type 1 diabetes, Down's disease, etc.) [1,8,10,12].

Celiac disease is considered an ideal model of HLA, which is an associated disease. Almost all patients (95%) with celiac disease are carriers of HLA-DQ2 and HLA-DQ8 alleles (5–10%), which are heterodimers of the corresponding DR-DQ haplotypes. These alleles are able to present deaminated gliadin peptides to T-lymphocytes, and therefore, they play a central role in the pathogenesis of the disease. At present, genetic predisposition is considered an important factor in the development of the disease, though it is not the only one. For example, Chinese and Japanese populations do not have DQ2 genes and do not suffer from celiac disease, except for DQ8 carriers [1,10,14].

The basis of the pathogenesis of celiac disease is damage to the mucous membrane of the small intestine by gluten. The most toxic fragment of the gluten molecule is its alcohol-soluble fraction, i.e., gliadin. The interaction of a genetically predisposed organism with gluten leads to the activation of the T- and B-cell immune response, the initiation of a cascade of pathological autoimmune reactions that cause and support inflammation of the mucous membrane of the small intestine, as well as damage to the other organs and systems of the body [1,9,10].

In response to the introduction of gluten/gliadin into the alimentary canal of a genetically predisposed person, specific antibodies, anti-gliadin (AGA), are produced. Thanks to the laboratory test, it became possible to diagnose diseases earlier and treat them in time, which has contributed to the reduction of mortality from celiac disease and its complications. Unfortunately, this test has low sensitivity and specificity, i.e., 50–60%. Moreover, there were discovered antibodies that are produced in the connective tissue elements of the mucous membrane of the small intestine in response to its damage, such as reticulin, endomysium, and tissue transglutaminase, associated with it. Endomysial antibodies (EMA) have high sensitivity and specificity (87–95%). Tissue transglutaminase (TSH) is directly related to EMA; it is an autoantigen that is recognized by EMA. The examination of TSH is recognized as a key laboratory test for the diagnosis of celiac disease, as it has high specificity and sensitivity [3,10].

However, new laboratory markers of celiac disease have recently been discovered that have demonstrated the highest sensitivity. These are antibodies to deaminated peptides of gliadin (DPG) and tissue transglutaminase. The advantage of modern markers of celiac disease is their accuracy, the possibility of use in the most complex and controversial clinical and laboratory cases (for example, with IgA-immunodeficiency, in patients with Dühring herpetiform dermatitis). The sensitivity of total antibodies to DPG (IgA + IgG) is up to 98.3–100%. So, perhaps, in the near future, serology might become not only a supplement, but also an alternative to traditional biopsy [3].

The diagnostic algorithm for celiac disease includes the selection of patients from the high-risk groups suffering from celiac disease, proper anamnesis collection, analysis of complaints, both from the gastrointestinal tract and extraintestinal ones, detection of associated pathology, as well as relatives with celiac disease. The laboratory diagnostic algorithm is based on enzyme-linked immunosorbent assay (ELISA) for determining specific serological biomarkers of celiac disease, i.e., antibodies of the IgA class to TSH and EMA, as well as «modern» antibodies to deaminated gliadin peptides (IgA) for simultaneously determining the total level of antibodies of the class IgA. When the titers of IgA class immunoglobulins decrease, specific IgG class antibodies should be examined [3,12].

Further diagnosis is based on the analysis of the condition of the mucous membrane of the small intestine in patients with positive serological test results. They undergo an upper endoscopy with a biopsy of the mucosa of the duodenum in its extrabulbar section. A negative result of the morphological examination does not exclude celiac disease. Repeated laboratory diagnostics, in particular, EMA determination, are indicated for patients with a high risk of celiac disease and high titers of organ-specific antibodies (in particular tTG), in the case of questionable or negative biopsy data. It is also necessary to remember the importance

of the correct collection of biopsy material, i.e., at least 4–6 fragments of the intestinal mucosa should be taken, while 3–4 fragments should be taken from the large papilla of the duodenum, and at least one should be taken from the bulb [1,3].

Clinical manifestations of celiac disease are quite polymorphic, and the disease itself occurs in different forms, i.e., typical, atypical, and latent. The typical (classical) form develops at any age, is presented as severe diarrhea with polyfecality, steatorrhea, anemia, with the subsequent development of malabsorption syndrome. At present, the classical form is rare; it occurs in 10–30% of all cases of celiac disease [1,9].

The atypical form occurs in most cases. It is characterized by the predominance of extraintestinal signs of the disease, i.e., a headache resembling a migraine; chronic fatigue; various skin changes, unmotivated weight loss, depressive disorders; stable course of iron deficiency anemia; fibromyalgia and artralgia, numbness of limbs; presence of other autoimmune diseases [1,2,9].

Herpetiform dermatitis of Dühring, chronic anemia, autoimmune thyroiditis, type 1 diabetes, autoimmune hepatitis, early osteoporosis, arthropathies, cerebral ataxia, some forms of epilepsy, autism, disorders of reproductive function (female and male infertility, spontaneous abortions) are considered to be «associated» with celiac disease, and their detection is an indication for examining a patient with celiac disease [1–3,11,10].

A nosological form is identified separately as gluten allergy, which is a classical allergic reaction caused by eating cereal products, in particular, wheat. The detection of high titers of specific IgE and the formation of hypersensitivity reactions with clinical manifestations occurring in response to cereal consumption are characteristic of the wheat allergy [2,3,5,6,8,11,14].

An IgE-mediated reaction to wheat can develop from eating wheat (food allergy) or inhaling its pollen or flour (respiratory allergy). Wheat allergy is presented by many allergic symptoms, i.e., urticaria, angioedema, and less often by asthma, allergic rhinitis, pain in the abdominal cavity, vomiting, aggravation of atopic dermatitis, as well as anaphylaxis associated with physical activity (wheat-dependent exercise-induced anaphylaxis, WDEIA) [4–6,8,13].

In a population-based cohort study (Stockholm), the prevalence of sensitization to wheat in 4-year-old children (n=2336) was 4%. In the Multicenter Study in Germany (MAS), they analyzed data from long-term serum studies of 273 children aged 2 to 10 years. The prevalence of sensitization to wheat in the children increased with age from 2% to 9% [3–5,8,14].

At present, the theory of violation of oral tolerance is considered, which leads to a shift of the immune response towards Th2 and to the synthesis of specific IgE. Many factors determine the severity of the wheat allergy signs, they range from genetic predisposition, environmental conditions, and the properties of the allergen itself. Physical activity increases the process of absorption in the gastrointestinal tract [3,4,14].

The gold standard in the diagnosis of this condition is a provocation test followed by physical exertion. However, this type of diagnosis carries a high risk, because the amount of food and the intensity of exercise required to develop a reaction cannot be controlled, and serious anaphylactic reactions have been reported in some studies [4,8]. Thus, the diagnosis of IgE-mediated wheat allergy is based on *a careful medical history collection* to identify specific symptoms for this type of sensitization, *carrying out skin tests* with wheat extract *and determining specific IgE (sIgE)* to wheat extract (high sensitivity). It is recommended to perform the ISAC test in case sIgE results are negative, and its clinical manifestations occur after physical exertion [4,8,14].

Clinical signs of an allergic reaction of the immediate type, which develop due to wheat consumption, are caused by the release of mediators (histamine, platelet-activating factor, and leukotrienes) from mast cells and basophils [4,5,8,14].

About 13% of people suffer from gluten intolerance without celiac disease. This pathology was also allocated to a single nosological form in 2013 at the annual meeting of the United European Gastroweek in Berlin, which was recognized as one of the most significant discoveries in the field of gastroenterology. It differs from celiac disease and wheat protein allergy, and it is usually not accompanied by classic gastrointestinal disorders, but it is more often presented by extraintestinal symptoms [1,8].

At present, the diagnosis of gluten intolerance without celiac disease is a diagnosis of exclusion. Gluten intolerance without celiac disease is characterized by the absence of antibodies to tTG and EMA, which excludes celiac disease, but high titers of IgA and IgG antibodies to gliadin are detected [3,4].

The aim of CD treatment is to restore the structure of the mucous membrane of the small intestine, to increase the duration and quality of life, and to prevent life-threatening complications, such as intestinal bleeding and malignant formations. The only method of treatment for patients with CD is the administration of a gluten-free diet, based on the complete and lifelong

avoidance of all products that contain wheat, rye, barley, and oats. Patients with CD should pay attention to products containing «hidden gluten». Complete lists of products approved for use by celiacs are usually published on special official websites of celiac disease societies. Treatment of CD is considered effective if, within 1–2 years of a gluten-free diet, the patient doesn't present any clinical signs of the disease, their laboratory indicators return to normal, and the structure of the mucous membrane of the small intestine is restored. Along with this, new directions of scientific research have arisen, such as creating gluten-free varieties of cereals, producing vaccines for celiac disease, and searching for new medicinal substances, i.e., blockers of tissue transglutaminase, blockers of gliadin deamination [2,12,13].

The aim: to deepen the knowledge of doctors about the polymorphism of clinical forms of celiac disease in children.

Gluten-sensitive diseases often develop without any gastroenterological symptoms, which makes their diagnosis quite difficult. In recent years, the specialists from KE «KhMDH» of the Khmelnytskyi City Council have diagnosed 5 patients with celiac disease, and 14 children have been diagnosed with gluten intolerance without celiac disease. All the patients have been prescribed a gluten-free diet and are under medical supervision.

The patients with gluten-sensitive diseases, atypical forms with rare clinical manifestations, have been shown in three cases. It was the reason to share our experience with other scientists and practising doctors. We hope that the results we have gained will help other specialists in their clinical practice.

The research was carried out in accordance with the principles of the Declaration of Helsinki. The informed consent of the patient was obtained for conducting the studies.

Clinical case 1

A *boy N.*, 6 years old. The patient's mother informed that the alopecia areata in the child had appeared when he was 4. The boy had been examined by a pediatrician, a gastroenterologist, and a dermatologist. Ascariasis had been detected. The child had been prescribed anti-parasitic treatment, long-term local treatment, but the foci of alopecia had not disappeared. Two years later (when the boy was 6), their number increased with a tendency to merge (Fig. 1).

The boy was brought by his mother to a gastroenterologist at Khmelnytsky City Children's Hospital. The child was re-examined. The results of the examination

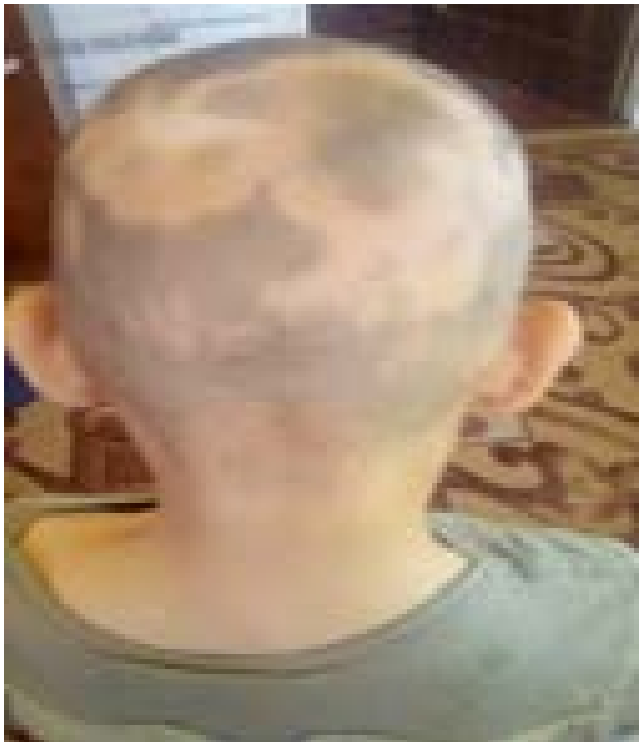
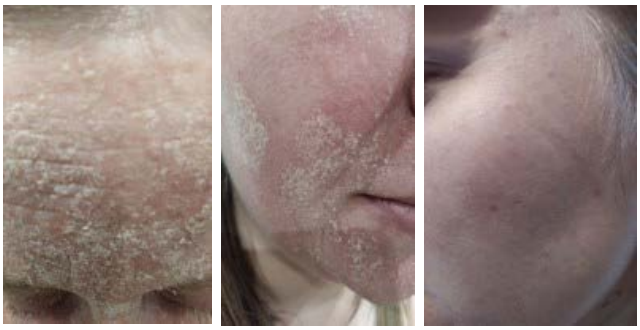


Fig. 1. Alopecia areata circularis

were the following: total IgE was 38 units/ml (the age norm is <50 units/ml), total IgA was 1.2 g/l (the age norm is 0.27–1.95 g/l), IgG was 7.04 g/l (the age norm is 5.04–14.64 g/l), IgM was 2.0 g/l (the age norm is 0.24–2.1 g/l); antinuclear antibodies (ANA) was 1:100. Biochemical blood analysis showed alanine-aminotransferase (ALT) was 28 mmol/l, aspartate aminotransferase (AST) was 16 mmol/l; alkaline phosphatase was 224 units/l, creatinine was 58 μmol/l, urea was 4.6 mmol/l.

The child was examined for lactase deficiency (Benedict's test was normal) and for celiac disease. IgA class antibodies to tissue transglutaminase were 16.3 (norm 0–20), IgG antibodies to tissue transglutaminase were 71.7 (norm 0–20), which made it possible to suggest celiac disease. The child did not have any dyspeptic disorders, but he was tested positive when examined for fecal calprotectin. The boy's mother didn't give her consent for the patient to undergo a biopsy of the intestinal mucosa. The child was administered a complete gluten-free diet. One month later, fluffy hair grew. 3 months later, the alopecia disappeared almost completely, except for the primary focus that had appeared on the left ear when the child was 4.

This case indicates an atypical form of celiac disease with the development of alopecia without any dyspeptic signs. The child continues to be on a gluten-free diet.



A B C

Fig. 2. Seborrhea of the scalp and face (A, B – before gluten-free diet; C – after a month's gluten-free diet)

Clinical case 2

A female *patient M.*, 17 years old. She came to a dermatologist with complaints of hyperemia of her skin on the face and on the hair area of the head, accompanied by skin peeling and itching, that had lasted for three years. The patient thought the signs appeared after she had used a new cosmetic skin care product. Seborrhea was diagnosed, and the appropriate treatment was prescribed. The condition improved, but after the end of the therapy, clinical manifestations reappeared. She was examined by various specialists, received local and general treatment, with a short-term, incomplete effect. Neither a hypoallergenic diet nor antihistamines nor local treatment was effective (Fig. 2).

The patient consulted a pediatric gastroenterologist. The tests of transaminases, thyroid hormones, bacteriological examination of the skin, a food allergy panel, and Benedict's test were administered. No deviations were found. Total IgE was 10.9 units/ml, total IgA was 3.53 g/l (the normal range is 0.61–3.48 g/l), total IgM was 1.71 g/l (the normal range is 0.23–2.59 g/l) and total IgG was 13.72 (the normal range is 7.0–16.00).

The patient was examined for celiac disease. In order to prevent a false positive result, IgA and IgG antibodies tests to tissue transglutaminase 5 units/ml and 4 units/ml, respectively) simultaneously were examined, as well as IgA and IgG antibodies to endomysium (less than 7 units/ml). Total levels of immunoglobulins A and G were tested as normal; fecal calprotectin was not detected (less than 50 μg/g). Examination of the antibodies of IgG and IgA classes to gliadin was administered; they were 23 units/ml and 75 units/ml, respectively (the age norm is <10 and <25, respectively). The increased titers of antibodies and a properly collected medical history (the patient indicated that when she ate only buckwheat porridge, the signs of seborrhea were reduced) helped to diagnose gluten intolerance without celiac disease. She was recommended to be on a gluten-free diet for three months. The positive effect was already seen three weeks later (Fig. 2 C).

After three months' diet, an attempt was made to introduce gluten. As a result, the itching of the scalp and dry skin of the face reappeared, which confirmed the diagnosis. The girl continues to be on a gluten-free diet, feels well, and has no complaints.

Clinical case 3

The parents of an 11-year-old *girl S.,* consulted a pediatric gastroenterologist with complaints of frequent abdominal pains in the child, which were accompanied by flatulence and occasional nausea. From the anamnesis, it was known that due to frequent and intense attacks of abdominal pain, the child had been often examined for helminthic infestation; multiple fecal tests for Benedict's test and fecal calprotectin had been performed. No deviations had been detected. During the ultrasound examination of the organs of the abdominal cavity, no pathological abnormalities were detected.

Fibrogastroduodenoscopy did not reveal any pathological abnormalities either. The differential diagnosis of somatoform disorders of the gastrointestinal tract in the child was carried out according to the recommendations of the Rome IV criteria [10].

She was examined for celiac disease, and no markers of celiac disease were found; in the examination of Ig A, M, G and E, elevated levels of IgE were found (186 g/l, with the age norm of 6.98–15.6 g/l). To eliminate the allergic inflammation of the mucous membrane of the gastrointestinal tract, antihistamines were prescribed, but there was no effect. Since it became known from the anamnesis that especially often the child complained of severe abdominal pain after physical exertion (dancing, swimming, etc.), it was recommended to keep a food diary, a diary for physical exertion and crises of abdominal pain.

It was found out that the child developed abdominal pain after physical exertion if she had previously taken food containing wheat. It was recommended to exercise 2–3 hours after eating food containing gluten, which led to the disappearance of abdominal pain. After 2 months of clinical well-being, while staying at the sea coast, the girl went swimming in the sea, having previously eaten a wheat flour bun. 5–7 minutes later, she came ashore with complaints of intense abdominal pain with an acute abdomen clinic, which confirmed the diagnosis «wheat allergy during physical exertion»; and, what was important, it convinced the parents of the correct tactics of their child's way of treatment.

Celiac disease is a common, clinically polymorphic, rather difficult disease to diagnose and treat. Despite

the obtained success, the search for universal diagnostic criteria and etiotropic treatment is still ongoing. The emergence of new methods and algorithms will ensure a high quality of life for patients and prevent disability due to CD.

After excluding celiac disease and an allergic reaction to gluten, in the presence of clinical symptoms in a person who notices a worsening of their condition with the use of gluten-containing products, it is possible to assume gluten intolerance without celiac disease. In such cases, agliadin antibodies should be examined. A completely new form of the disease is gluten intolerance during physical exertion.

Gluten-dependent diseases can be asymptomatic for a long time or manifest outside the intestinal manifestations, which significantly complicates their timely diagnosis and therapy. The literature mainly describes classical forms of celiac disease with damage to the gastrointestinal tract [1]. Celiac disease can manifest itself as growth and development delay, iron deficiency anemia, and in severe forms – a delay, in the future, sexual development, with the development of chronic diarrhea – protein deficiency with edematous syndrome, chronic headache, and others [1,3,9,14]. This paper presents atypical forms of gluten-dependent conditions, namely, alopecia that was not treatable by traditional methods, severe gluten-induced seborrhea, and gluten intolerance during exercise, to once again remind us of the diversity of clinical forms of gluten intolerance.

Conclusions

In order to expand the knowledge for the timely diagnosis of this pathology, the peculiarities of the course and diagnostics of each atypical case of CD should be shared in publications; demonstrations of the rare disease cases and syndromes, both in adults and children, should be carried out at various scientific forums.

Authors declare the absence of any conflicts of interests and own financial interest that might be construed to influence the results or interpretation of the manuscript.

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