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Coeliac Disease and Connection with Iron Deficiency Anemia: A Literature Review

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ABSTRACT

Introduction: Celiac disease (CD) is one of many autoimmune diseases occurring in children and adults, more often among women, which presents a lot of forms, symptoms. Celiac disease can manifest on the skin, as nutrient deficiencies, or with gastrointestinal symptoms, as well as many other symptoms. One of them is an iron deficiency presented in over half patients with CD related to destroyed absorbing surface - proximal part of small intestine. Many patients with CD present anemia as only one sign. Iron is important microelement in human body essential, among other functions, for oxygen transport in the body and involvement in enzymatic processes and its absorption is regulated by liver protein hepcidin and transport by transferrin in plasma. There are many causes of iron deficiency anemia (IDA), but celiac disease should always be considered as one of them. The immune response to gluten proteins is the underlying mechanism of the disease. The implementation of a gluten-free diet as the primary treatment for celiac disease leads to the improvement and regeneration of the intestinal mucosa, which

results in a reduction of the symptoms of the underlying disease, including IDA. Therefore, it is important not to overlook the symptoms of the disease, as its complications can be mitigated.
Materials and methods: The analysis is based on a review on studies found in PubMed and ScholarShip. Keywords used were „celiac disease and IDA", „celiac disease", „iron metabolism", "symptoms of IDA", "iron deficiency anemia".
Aim of the study: The aim of this study is to review research on the relationship between celiac disease and iron deficiency anemia, as well as to analyze this relationship, evaluate the mechanisms linking these diseases, and their symptoms and treatment.
Conclusions: IDA associated with celiac disease can occur in up to half of patients and is the most common hematologic complication. Damage to the duodenal mucosa affects the absorption of iron. Additionally, bleeding may contribute to the loss of the element. Besides a gluten-free diet, patients may undergo therapy with iron supplements in various forms, which improves prognosis.
Keywords: Iron deficiency, Celiac disease, Anemia, Iron absorption, Gluten, Hepcidin, Gluten-free diet

Introduction

Celiac disease (CD) is a chronic autoimmune disorder caused by a gluten intolerance with general population prevalence about 0,5-2% (average 1%) with mostly female predominance (1)(2) Gluten is a mix of hundreds of proteins which can be found in storage cells in plants like wheat grains, rye, barley, oats. (3) CD is a reason of intestinal problems (dominating ones are diarrhoea, steatorrhea, weight loss or failure to thrive) and also extraintestinal problems like neurological manifestations - headaches, migraine, peripheral neuropathy, gluten ataxia, epilepsy, psychiatric conditions, ocular and dermatologic, oral, cardiovascular, pulmonary, renal, reproductive system manifestations, endocrine and hematologic disorders and many others.(4) The most common hematologic manifestation of CD is anemia secondary to iron deficiency (IDA). (5) Over half of clinical and subclinical patients with CD presents IDA.(6) Both children and adults may present signs of anemia and it may be the only one symptom of

CD. (7)(8)(9) Patients with refractory IDA may present CD with prevalence as high as 20%.(10)

Etiology

Celiac disease is frequent chronic auto-immune enteropathy occurring in genetically predisposed people. It is associated with the haplotypes HLA-DQ2 and HLA-DQ8. Over all patients are positive to this genes.(11) Despite of genom-wide studies no genes more strongly associated with the disease have been found.(12)(13) However during whole life many people with HLA-DQ2 or HLA-DQ8 will never develop celiac disease. Moreover, environmental triggers are also important in development of CD. Diet rich of gluten stimulates an imbalanced immune reaction in predisposed individuals. Gluten is degraded into smaller peptides, like gliadin, which are deamidated by the enzyme transglutaminase-2 (tTG2). This change boosts their ability to bind to HLA-DQ2 and HLA-DQ8 molecules, triggering T-cell activation and an inflammatory response that leads to damage in the small intestine. (14)

Symptoms of CD

Gastro-intestinal manifestations

The pathologically altered epithelium of the small intestine generates a wide range of symptoms resulting from its damage. There are differences between the symptoms in children and adults. Children under 3 years of age most commonly present with loss of appetite, diarrhea, and bloating, while older children additionally report abdominal pain and constipation. (15) In adult patients, symptoms resembling irritable bowel syndrome are predominant, including dyspepsia, constipation, and sometimes symptoms leading to cachexia. Due to malabsorption and impaired activity of certain digestive enzymes associated with the intestinal villi, patients may experience symptoms of deficiencies in various vitamins and trace elements. Low level of iron, folate, zinc and vitamin B12 and D are frequent in CD. (16)

Extraintestinal manifestations

In addition to a variety of gastrointestinal symptoms, celiac disease also presents with osteopenia or osteoporosis, conditions that affect up to 70% of patients. Impaired absorption of vitamin D3 and calcium results in reduced bone mineralization. (17) In children, a deficiency of vitamin D3, along with general malnutrition, can lead to growth restriction or growth failure. Joint's pain is also frequently a common symptom occurring in 20-30% of patients with celiac disease. (18) Musculoskeletal symptoms, such as muscle pain, are more frequently observed in

patients with celiac disease and are associated with nutrient deficiencies. In half of adult patients, the pain decreases within 24 months after the introduction of a gluten-free diet. (19) Neurological symptoms occur relatively frequently in patients, with an incidence of 6-10% in treated individuals and up to 48% in untreated patients. The manifestation of these symptoms involves several mechanisms, such as deficiencies in nutrients and vitamins, as well as cross-reactive immune responses. Headaches, including migraines, may occur in approximately 26% of patients with celiac disease (CD), representing one of the complications of this condition. Additionally, patients with CD may experience peripheral nerve polyneuropathy, epilepsy, and gluten-dependent ataxia. This ataxia typically develops in middle-aged individuals and is believed to result from a cross-reaction between antibodies directed against Purkinje cells in the cerebellum and gluten antigens. This phenomenon is part of a broader spectrum of neurological symptoms associated with celiac disease. (16) Chronic fatigue is an indication to expand the diagnostic workup to include celiac disease, as it is a common yet subjective and difficult-to-measure symptom.(20) An important dermatological manifestation of celiac disease is dermatitis herpetiformis, which, according to studies, occurs equally often in both women and men with celiac disease. Characteristic of dermatitis herpetiformis are polymorphic, pruritic skin lesions with blisters, typically located on the extensor surfaces of the elbows, knees, and buttocks. They may also appear on other, less typical areas of the skin, including the oral cavity.(21) Celiac disease is significantly more likely to coexist with other autoimmune disorders, such as alopecia areata, autoimmune endocrinopathies, and psoriasis.

Detection of coeliac disease

Celiac disease is an inflammatory bowel disease, the diagnosis of which is based on the histopathological appearance of a biopsy taken from the patient's mucosae duodenum, serological results and symptoms. The biopsies taken during gastroscopy are analyzed and assessed according to the Marsh classification modified by Oberhüber by a pathologist. (22) In order to properly assess the condit ion of the duodenal mucosa, six biopsies should be taken: four from the postbulbar part and two from the bulb.(23) This allows for the comparative assessment of inflammatory changes over time during subsequent examinations, as well as the analysis of disease progression and treatment effectiveness. The second element of the diagnostics is serology. The most specific antibodies for celiac disease (CD) are IgA antibodies; however, 2-3% of individuals with celiac disease exhibit a deficiency in this antibody class. Therefore, during diagnostic evaluation, the levels of these antibodies should always be measured. In the case of a deficiency, further diagnostics should include IgG class

antibodies.(24) Serum concentrations of immunoglobulin (Ig)A anti-tissue transglutaminase (TG2) are the preferred method for screening celiac disease, showing the highest sensitivity (up to 98%) and specificity (around 96%).(25)The most specific test is the measurement of IgA-EMA(endomysium) antibodies, which is therefore used as a confirmatory test for celiac disease diagnosis in cases of low IgA-TG2 titres. Other antibodies, such as IgA-anti-gliadin antibodies (AGA) are currently used less frequently in diagnostics due to their lower sensitivity and specificity. (24)

Biological roles of iron

Iron is one of the most important microelement in human body. Many processes are addictive to appropriate level of iron. Participation in many enzymatic processes, transport oxygen binded with heme in the erythrocytes, deoxyribonucleic acid synthesis, metabolic energy, and cellular respiration are essential for the survival of each organism. Iron is a component of hemoglobin, myoglobin, cytochrome proteins, myeloperoxidase, nitric oxide synthetases, respiratory complexes I-III, coenzyme Q10, mitochondrial aconitase, DNA primase. (26) Too low level of iron carries serious consequences, but its excess is also harmful like production of reactive oxygen species (ROSC). This stressful elements lead to destroy cells. (27)

Iron deficiency anemia

Diagnosis

Across the world, low level of iron is the most common cause of anemia and micronutrient deficiency. (28) To tell about anemia hemoglobin level must decrease under <2 SD of correct range related to sex, age or pregnancy. (29) World Health Organization (WHO) recommends diagnosing anemia in non-pregnant women with a hemoglobin level below 12 mg/dL, in pregnant women below 11 mg/dL, and in men and boys over 15 years old below 13 mg/dL. Anemia is diagnosed in children based on age-specific criteria. In children aged 0.5 to 4 years, anemia is diagnosed with a hemoglobin level below 11 g/dL, in the 5 to 11-year age group with a hemoglobin level below 11.5 g/dL, and in children aged 12 to 14 years with a hemoglobin level below 12 g/dL. (30) Ferritin is an iron storage in human body and may facilitate the diagnosis of IDA. An appropriate level of ferritin in blood is >30 μg L^{-1} and a result below indicates iron deficiency. During an active inflammatory response ferritin level increases because it is also an acute-phase protein. Chronic inflammation of bowel in CD makes IDA diagnosis more difficult. Other parameters that facilitate diagnosis include MCV, transferrin

levels, and TSAT (transferrin saturation). Decreased MCV, high transferrin level indicate IDA. (31)

Symptoms

IDA similar to CD is more often diagnosed in women than men. (32) Some patients with iron deficiency do not exhibit symptoms, especially when the deficiency is mild, because the body can compensate for the initial lack of this element. A significant proportion of individuals with iron deficiency develop anemia. Symptoms that may suggest the presence of anemia include weakness, fatigue, reduced exercise tolerance, irritability, depressed mood, impaired thermoregulation, and a sensation of coldness. These manifestations result from a decreased capacity for oxygen transport due to insufficient hemoglobin production, which leads to inadequate tissue oxygenation and triggers compensatory mechanisms in the body. In children with iron deficiency, neurological developmental delays, impaired concentration, decreased academic performance, and restless legs syndrome may occur. In cases of severe deficiency, pica, which is the compulsive consumption of non-food substances, is characteristic. (33)

Iron in human body

Absorption

Iron is absorbed in a proximal part of small intestine and depends on a few factors. Only ferrous form (Fe^{2+}) or bounding with protein such a heme is enabled to be incorporated to the interior of the enterocyte. Duodenal cytochrome B (Dcytb), a ferric reductase enzyme allows conversion ferric ions (Fe^{3+}) to absorbable ferrous ions. (34) Divalent metal transporter (DMT1) is a transport protein located in apical or entire brush border membrane of epithelial cells of the duodenum. Iron is transported in ferrous form through DM1 to cells interior. Also some others divalent metals like Mn^{2+}, Ni^{2+}, Cu^{2+} may be transferred as well as iron.(35)(36)

Regulation of iron absorption

Hepcidin is a hormone necessary to regulate level of iron in the body. (37) Liver is responsible for production and secretion this protein into the plasma, where is located in free form or with not essential bindings to albumin and α2-macroglobulin.(38)(39) Ferroportin is protein involved in iron absorption, located in enterocyte membrane and controlled by hepcidin.(40) This mechanism is based on negative regulation: hepcidin binding to ferroportin caused endocytosis of membrane receptor for iron. As a result absorption in proximal intestine is impaired. When level of iron is low liver products less hepcidin what stimulates absorption iron

by ferroportin. Moreover conditions that increase erythropoietin levels, such as hypoxia or pregnancy inhibit hepcidin release. (41)

Circulation

In the blood the largest percentage of iron is bound to the protein transferrin, which has the ability to bind two atoms of the element. In homeostatic conditions, transferrin is typically 20-40% saturated with iron and facilitates its delivery to tissues via transferrin receptor 1 (TFR1) or transferrin receptor 2 (TFR2) in the liver and erythrocytes. When the amount of iron in plasma exceeds the binding capacity of transferrin, it exists in a free form. Excess iron is toxic to the organism and accumulates in organs such as the heart, liver, and nervous system, resulting in cellular damage and organ dysfunction. (26)

Celiac disease and iron deficiency

Iron deficiency anemia (IDA) may be the sole manifestation of celiac disease. Impaired iron absorption from the duodenum, which is inflamed due to the disease, is the primary mechanism responsible for the development of anemia. Additionally, occult gastrointestinal bleeding from the mucosa may occur in celiac disease and contribute to IDA. However, peptic ulcer disease is a more frequent cause of such bleeding, although celiac disease should also be considered in the differential diagnosis. Celiac disease can be associated with gastrointestinal lymphomas or other autoimmune enteritis leading to ulcerations, which possess the potential for subclinical bleeding, thus providing a clue to the underlying diagnosis. (40) A considerable number of patients develop microcytic hypochromic anemia as a result of iron deficiency. (42) In many analyzed studies were shown connection between CD and IDA. Prevalence of IDA in CD patients was different from 12% to 84%. The bigger group was 727 adult patients from USA in 2013 where 21% of them was diagnosed IDA. (43) Saukkonen et al. found that 23% of patients with CD had anemia. (44) Studies show that patients with IDA experience symptoms for a longer duration, respond less favorably to treatment with GFD, and exhibit more pronounced histological changes and serological abnormalities compared to patients without IDA. (45) Annibale et al. described an inverse correlation ($p=0.0003$) between the increase in hemoglobin levels and the reduction of histopathological changes in the duodenal mucosa without iron suplemetation, suggesting confirmation of the relationship between IDA and impaired iron absorption. (46)

Prevention

Patients with celiac disease (CD) are significantly more prone to iron deficiency and iron deficiency anemia (IDA) compared to healthy individuals. Both at the time of diagnosis and during adherence to a gluten-free diet, iron levels should be regularly monitored. The study demonstrated that as many as 31.58% of patients on a gluten-free diet had anemia, although it was conducted on a group of only 59 patients. (47) Additionally, a genotype of *TMPRSS6* was identified, which influences iron metabolism through its effect on hepcidin. The evaluation of this genotype may have clinical significance in managing iron supplementation therapy, as mutations in *TMPRSS6* can result in a poor response to iron treatment and predict persistent IDA despite iron supplementation and adherence to a gluten-free diet. (48) A significant association has been established between the occurrence of iron deficiency anemia (IDA) and the use of proton pump inhibitors (PPIs) in patients with celiac disease. Consequently, it is recommended to exercise caution when prescribing these medications, and, when feasible, to substitute them with alternative agents that reduce gastric acid secretion. This approach aims to mitigate the potential adverse impact of PPIs on iron absorption, given their role in altering gastric pH and consequently impairing the solubility and bioavailability of iron. (49)

Treatment

The primary treatment for celiac disease (CD) is the adoption of a gluten-free diet (GFD), which can improve the absorption of iron by promoting the healing of the inflamed intestinal mucosa. In cases of mild iron deficiency anemia (IDA), the GFD alone may be sufficient to restore iron levels and improve hematologic parameters. (50)

If iron deficiency anemia (IDA) persists despite the implementation of a gluten-free diet (GFD), other potential causes of anemia should be considered. After identifying and resolving these factors, iron supplementation should be recommended. The most commonly used form of supplementation is oral iron, typically in the form of ferrous sulfate. However, some patients may experience discomfort when taking the medication. Gastrointestinal symptoms such as abdominal pain, nausea, diarrhea, vomiting, and constipation may occur in approximately 50% of patients. Daily dose of elementary iron is 100-200 mg in adults and 2-6mg/kg in children. In IDA without CD treatment should last about 2 to 4 months after normalizing hemoglobin, but patients with CD (51) In patients with iron deficiency anemia (IDA) and celiac disease, a statistically significant increase in blood iron levels was observed after performing an oral iron absorption test with Feraligine ® (ferrous glycinate). These results suggest that ferrous glycinate is effectively absorbed in patients with celiac disease, indicating its potential as an

efficient source of iron in this patient group, despite the previous issues with iron absorption associated with intestinal mucosal damage. (52) In case intolerance oral iron because of malabsorption it is necessary to use an intravenous form.

Conclusions

The topic of celiac disease is a very important and current issue. As studies indicate, it affects many individuals and can manifest in various forms, which is why patients may seek help from specialists with different profiles. The most common hematological condition is IDA, which is frequently diagnosed in both pediatric and adult patients. It is important to consider the possibility of celiac disease during the diagnostic process and to measure antibodies against transglutaminase in the IgA class and total IgA. After appropriate treatment with a gluten-free diet, additionally supported by iron supplements it is possible to improve patient prognosis and enhance their quality of life. A large number of patients, especially those with celiac disease, poorly tolerate the treatment with oral iron supplements. There are preparations available on the market that generate fewer gastrointestinal side effects, but they may be less absorbed, making them less effective.

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Author's contribution

Conceptualization – Aleksandra Zielińska and Jakub Skiba; methodology – Szymon Szypulski; software ,- Natalia Tylczyńska and Zuzanna Skiba; check – Kinga Kowalik, Maria Michalska and Ignacy Maciejewski; formal analysis Sebastian Iwaniuk and Jakub Skiba; investigation – Kinga Tylczyńska; resources – Natalia Tylczyńska; data curation – Sebastian Iwaniuk and Szymon Szypulski; writing - rough preparation – Maria Michalska and Zuzanna Skiba; writing - review and editing, Ignacy Maciejewski and Kinga Tylczyńska; visualization, Natalia Tylczyńska; supervision – Kinga Kowalik; project administration – Aleksandra Zielińska; receiving funding not applicable, All authors have read and agreed with the published version of the manuscript.

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