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Iron Deficiency Anemia in Inflammatory Bowel Disease

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Abstract

Inflammatory bowel disease (IBD) is a chronic inflammatory disease of the gastrointestinal tract. It includes Crohn's disease (CD) and ulcerative colitis (UC). It occurs with periods of exacerbations and remissions. One of the most common complications of IBD is iron deficiency anemia (IDA). In IBD, iron deficiency anemia (IDA) often occurs concomitantly with anemia of chronic disease, caused by chronic inflammation of the gastrointestinal tract. Disturbance of iron homeostasis in IBD results from inflammation of the intestinal mucosa, which causes increased blood loss from the gastrointestinal tract and poor iron absorption. A key role is played by hepcidin, which, by acting on ferroportin, inhibits iron absorption in the intestines, despite its deficiency. Less common causes of anemia in IBD include vitamin B12 deficiency, folate deficiency, hemolysis, drug-induced aplasia, or liver disease such as primary sclerosing cholangitis. Symptoms of iron deficiency depend on the severity and chronicity of the anemia. Anemia reduces the quality of life of patients, so diagnosis and treatment of iron deficiency anemia in IBD is essential. Regular monitoring of anemia and iron homeostasis is recommended. Tests should be performed at the time of IBD diagnosis, every 3 months in active disease and every 6-12 months in periods of remission. In all patients with IBD, it is necessary to follow an appropriate diet, but also to treat anemia with iron preparations, oral or intravenous. The choice of the method of iron administration, oral or intravenous, depends on the hemoglobin level,

IBD activity, and the patient's tolerance to oral preparations. Regular monitoring is essential because anemia in IBD often recurs.

Aim

This article aims to present the pathomechanism, symptoms and methods of treatment of iron deficiency anemia in inflammatory bowel diseases.

Materials and Methods

A comprehensive literature review was conducted using PubMed, focusing on articles and research papers published between 2014 and 2024. Search terms included "inflammatory bowel disease", “iron deficiency anemia”, "Crohn’s disease", ‘’ulcerative colitis’’, ‘’iron” and " iron supplementation".

Keywords: Inflammatory bowel disease, iron deficiency anemia, Crohn’s disease, ulcerative colitis, iron supplementation, iron.

Introduction

Inflammatory bowel disease (IBD) is a chronic disease of the digestive tract in which inflammation occurs. It includes Crohn's disease (CD) and ulcerative colitis (UC). It proceeds with periods of exacerbations when symptoms of varying intensity appear, and periods of remission, when symptoms disappear. [1,2,3]

IBD worsens the quality of life of patients and is associated with many symptoms, not only from the digestive tract but also extraintestinal, involving other systems and organs. Extraintestinal symptoms are present in about 25% of patients. The most common extraintestinal symptom is anemia, which can occur at any stage of IBD or be the first symptom of this disease. Anemia is more prevalent in Crohn's disease than in ulcerative colitis and is more frequent in active disease than in remission, which indicates that the risk of developing anemia is associated with IBD activity. [1,2,3]

The anemia in IBD is primarily caused by iron deficiency, which is triggered by inadequate intake and chronic blood loss from damaged mucosa. There is also anemia of chronic disease. Chronic inflammation impairs intestinal function and, in addition to blood loss from the inflamed gastrointestinal tract, leads to inhibition of iron absorption by hepcidin and iron sequestration in the body. These two types of anemia often occur together. [4]

Less typical causes of anemia in IBD include vitamin B12 deficiency, folate deficiency, hemolysis, drug-induced aplasia, or liver disease such as primary sclerosing cholangitis.[1]

Symptoms of iron deficiency depend on the severity and chronicity of the anemia. In addition to the standard symptoms of anemia, such as pale skin, fatigue, headaches, dizziness, memory impairment, and learning disorders, other less specific symptoms involve various body systems. These include shortness of breath, tachycardia, palpitations, hypotension, cold intolerance, depression, fainting, angular stomatitis, immune system disorders, and menstrual and libido disorders. [1, 5]

Numerous symptoms of anemia affecting various systems and organs occur because iron is essential for many physiological processes, including energy metabolism, oxygen transport, immune system function, and neurotransmitter synthesis. [6] According to patients, IDA significantly reduces the quality of life and contributes to reduced physical activity, but despite this, it remains undiagnosed in many patients, which poses a significant problem. [2]

Pathophysiology of anemia in IBD

The pathophysiology of anemia in IBD is multifactorial. There are two major types of anemia in IBD, iron deficiency anemia and anemia of chronic disease. Both anemias often occur concurrently. The disturbance of iron homeostasis in IBD results from inflammation of the intestinal mucosa, which causes increased blood loss from the gastrointestinal tract and poor absorption of this element. In addition, the patient's diet is important, but it is often unbalanced and provides too little iron. [7,8]

The inflammatory response significantly affects iron metabolism in the human body. In chronic inflammation in IBD, cytokines and acute-phase proteins are produced, which affect homeostasis. A key role here is played by hepcidin from the liver, which is made in the case of inflammation. Its production is induced, among others, by interleukin 1 or interleukin 6. Hepcidin inhibits ferroportin, the main cellular iron exporter found in the intestinal epithelium,

macrophages, and hepatocytes, which allows the transport of absorbed iron through the intestines into the bloodstream. Hepcidin binds to ferroportin in the presence of iron and, by inducing its internalization and degradation, inhibits the absorption and transport of iron from enterocytes to the bloodstream and the retention of iron in monocytes and macrophages. This leads to a decrease in serum iron concentration. Additionally, hepcidin reduces iron absorption from the duodenum by inhibiting DMT1 (divalent metal transporter). [9,10]

Hepcidin is usually produced at high iron concentrations and its production is reduced in iron deficiency or hypoxia. However, patients with IBD often have elevated hepcidin levels, which inhibits iron absorption even in iron-deficient states. For this reason, hepcidin is considered the most important factor in iron homeostasis regulation. [9,10,11]

In addition, it should be noted that vitamin deficiencies, such as vitamin B12 and folic acid, bowel resection, and drugs used to treat IBD such as methotrexate, sulfasalazine, and thiopurines also contribute to anemia in IBD. [1]

Diagnosis of anemia

Due to the high prevalence of anemia in patients with IBD, regular monitoring of anemia and iron homeostasis is recommended. Testing should be performed at the time of IBD diagnosis, every 3 months in active disease, and every 6-12 months in periods of remission. [12]

According to ECCO recommendations, if a decreased hemoglobin concentration is detected (below 12 g/dl in women, below 13 g/dl in men), the erythrocyte count, ferritin, C-reactive protein (CRP) concentration, transferrin saturation, reticulocyte count, erythrocyte width distribution and mean corpuscular volume should also be monitored. [13]

Because ferritin, in addition to its function as a protein storage protein in the body, also plays the role of an acute phase protein, and its level increases in inflammatory conditions, the current condition of the patient should be taken into account when diagnosing anemia in IBD. IBD activity should be assessed using the Crohn's Disease Activity and Mayo score and the concentration of CRP in serum and calprotectin in feces. [1]

In the case of IBD remission, normal CRP level, iron deficiency anemia should be diagnosed if the ferritin level is <30 ug/l. In the case of active IBD, if clinically or endoscopically we find

the presence of inflammation and CRP is elevated, the ferritin level indicating iron deficiency is <100 ug/l. [13]

In the case of anemia in IBD caused by chronic inflammation, if the ferritin level is greater than 100 ug/dl, it may also be helpful to test transferrin saturation. In such cases, anemia is indicated by a transferrin saturation of less than 20%. [14]

However, if the ferritin concentration is between 30 and 100 µg/l, it can be assumed that the patient has both types of anemia, iron deficiency, and inflammatory anemia. [15,16]

Other markers can help diagnose anemia in IBD, these are hepcidin and soluble transferrin receptors (sTfR). Hepcidin is a protein that regulates iron absorption and its release from stores in the reticuloendothelial system. At high hepcidin levels, iron absorption is reduced. In turn, soluble transferrin receptors are elevated in iron deficiency and are much less affected by inflammation in the body than, for example, ferritin. Therefore, both hepcidin and soluble transferrin receptors (sTfR) can be used in combination with other parameters to diagnose iron deficiency anemia, because, as noted, other parameters routinely used in the diagnosis of IDA are affected by inflammation, which makes it difficult to assess the patient's iron stores and metabolism. [17,18,19]

Treatment of IDA in IBD

If iron deficiency anemia is detected in all patients with IBD, iron supplementation should be initiated. However, in the case of iron deficiency without anemia, as indicated by the hemoglobin level (below 12 g/dl in women, below 13 g/dl in men), iron supplementation should not be routinely initiated. The decision to initiate iron supplementation in this situation should be considered after analyzing the individual clinical condition of the patient. [20]

Despite existing recommendations, it is observed that anemia in IBD still often remains an underdiagnosed and undertreated disease, about 30 to 50% of patients do not receive appropriate therapy. Undertreatment of IDA can worsen the clinical condition of the patient with IBD. Patients with chronic or recurrent anemia have higher rates of disease activity, complain of a lower quality of life, and need significantly more health care resources. [21]

It should also be noted that anemia in IBD is multifactorial and correcting iron levels alone may not be sufficient to correct the anemia, therefore all possible causes of anemia in a given patient should be considered. [22]

The purpose of IDA therapy is to normalize hemoglobin levels, resolve anemia symptoms to improve the quality of life of patients, replenish iron stores, control disease activity, and prevent the recurrence of anemia. [22,23]

An adequate response to treatment is considered to be an increase in hemoglobin concentration by 2 g/dl or transferrin saturation of >30% within 4 weeks. [20]
Iron supplementation can be administered orally or intravenously. The method of iron administration depends on the degree of iron deficiency, the patient's tolerance of oral preparations, the response to treatment, and access to the drug. Studies conducted so far comparing oral and intravenous iron administration have confirmed that gastrointestinal side effects are much less common with intravenous iron. Some authors believe that intravenous administration should be the preferred way of treating anemia in IBD. The others claim that both routes are effective because newer forms of oral iron have fewer side effects. [24,25]

With all the evidence and international guidelines, it can be concluded that oral iron is sufficient in some patients, and intravenous iron should be used in other situations, depending on the level of anemia, side effects of oral iron, and IBS activity. [24,25]

Oral supplementation should be used in patients with mild IDA (hemoglobin >10 g/dl) who have normal CRP values and are in clinical and endoscopic remission. Intravenous preparations are recommended in patients with severe anemia (Hb<10 g/dl), intolerance to oral iron, or inadequate response to oral iron, and in patients with active IBD because intravenous iron does not worsen disease activity. Intramuscular iron is not recommended due to pain and tissue damage at the injection site. [23]

Oral iron, despite its advantages, such as easy availability, ease of use, and low cost, has side effects on the gastrointestinal tract: nausea, abdominal pain, diarrhea, and constipation. Its absorption is affected by some food products: coffee, tea, dairy products, and some medications, such as proton pump inhibitors, H2 blockers, and antacids. In addition, only part of the oral iron is absorbed from the gastrointestinal tract. It is commonly believed that the remaining unabsorbed iron causes damage to the mucous membranes and can increase intestinal

inflammation and affect the intestinal microbiota. To minimize the side effects of oral iron it is recommended to use small doses of iron, not exceeding 100 mg, and to use ferric maltol instead of divalent iron preparations in a complex with sulfate, gluconate, or fumarate. [1,2,4,26] In a prospective, randomized study conducted over three years at 56 sites in the United States, Germany, Spain, France, Belgium, and Hungary, ferric maltol provided significant improvements in hemoglobin levels compared with placebo over 12 weeks in patients with inactive IBD or mild-to-moderate active IBD and mild-to-moderate IDA. In addition, the incidence of adverse gastrointestinal events was low and comparable to the placebo. [26]

Intravenous iron is considered more effective than oral preparations. It does not affect disease activity or mucosal health. In addition, higher ferritin levels can be achieved after intravenous treatment, which results in a lower risk of recurrence of anemia. In addition, patients who receive intravenous iron are less likely to discontinue treatment due to adverse effects compared to patients who use oral iron. [27] There are several parenteral iron preparations available, which can be divided into three generations. The first generation includes high-molecular-weight iron dextran. The second generation is low-molecular-weight iron dextran (ferrous gluconate and iron sucrose), and the third generation is ferumoxytol, iron carboxymaltose, and iron isomaltose. The use of first-generation parenteral iron is associated with a higher risk of anaphylactic reactions, so this form of intravenous iron should currently be avoided. The use of higher-generation preparations is recommended. [27,28]

Iron sucrose is a widely studied preparation in patients with IBD and is well tolerated. Iron sucrose has a significantly lower rate of adverse events, including allergic reactions, compared to iron dextran and gluconate. In addition, sucrose can be administered to patients with intolerance to iron dextran or iron gluconate. [28]

Third-generation preparations are preferred, because they have few side effects and better efficacy. Studies comparing the third-generation preparation, iron carboxymaltose, and iron sucrose found that patients treated with iron carboxymaltose had a better increase of ferritin and transferrin saturation with iron. The increases in hemoglobin levels were similar in both groups. [28, 29]

In addition, third-generation preparations such as iron carboxymaltose or iron isomaltose can be administered in higher doses of iron and at a faster rate than other intravenous iron preparations. However, it should be noted that a side effect of iron carboxymaltose is

hypophosphatemia, which can cause bone weakness. Therefore, phosphate levels must be monitored and supplemented if hypophosphatemia occurs during iron carboxymaltose therapy. [30,31]

In the treatment of iron deficiency anemia, which is so common in IBD, the total cost of treatment must also be taken into account. A retrospective study from Germany analyzed the healthcare costs of patients with IBD. The analysis showed that intravenous iron therapy was associated with fewer hospitalizations for IDA and lower overall costs compared to oral iron therapy, despite the higher pharmaceutical costs of intravenous iron. [29,31,32]

Relapses of anemia in IBD occur frequently, so it is recommended to monitor iron levels after treatment every 3 months for the first year, and every 6-12 months thereafter. [25]

Conclusion

In summary, iron deficiency anemia is one of the most common complications of inflammatory bowel disease. It should undoubtedly be an important aspect of patient care because it causes a decrease in the quality of life of patients. Unfortunately, IDA in patients with IBD still often remains undiagnosed and undertreated. Regular monitoring of anemia and iron homeostasis in patients with IBD is necessary. If iron deficiency anemia is diagnosed, iron therapy should be started in every patient with IBD. The choice of the method of iron administration, oral or intravenous, depends on the hemoglobin level, IBD activity, and the patient's tolerance to oral preparations. Further monitoring of anemia is necessary after treatment because anemia in IBD often recurs.

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