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IMPROVEMENTS IN COMPLEX TREATMENT OF PATHOLOGICAL CHANGES IN THE ORAL CAVITY IN PATIENTS WITH IRON DEFICIENCY ANEMIA

Ortikova Nargiza Khairullayevna
PhD, Associate Professor Department of Orthopedic Dentistry

Salohiddinov Mirzobek Bahodirovich
1st Year Clinical Resident

Abstract
The oral cavity has a close anatomical and physiological interaction with various body systems. Pathogenetically limited damage to the oral mucosa occurs when homeostasis is disturbed and in various somatic diseases. The stability of the chemical composition is one of the most important and inevitable conditions for the normal functioning of the body, and the oral mucosa can be an indicator or mirror of pathological processes occurring in general. Diseases caused by deviations in the content of chemical elements in the human body, caused by environmental, professional, climatic and geographical factors, lead to a wide range of health disorders, including the condition of the oral mucosa.

Keywords: Oral mucosa, Health disorders, Various somatic diseases, Iron.

Introduction

The body's need for iron increases during growth, pregnancy, blood loss (including during menstruation). Deficiency can also occur when it is not taken with food (eating disorders, etc.), with achlorhydria, chronic diarrhea, gastrointestinal bleeding after gastrectomy, including gastric and duodenal ulcers, and in other cases. Iron deficiency is defined as a deficiency of its total amount, which leads to a negative balance, an imbalance between an increase in the body's need for iron and its intake or loss.

Iron is an indispensable element, is a part of hemoglobin, myoglobin, cytochromes, participates in a number of redox reactions, plays an important role in the processes of hematopoiesis. On average, the human body contains about 3-4 g of iron (about 40 mg Fe/kg body weight in women and about 50 mg Fe/kg body weight in men). Most of this trace element (60%, or more than 2 g) is found in hemoglobin (Hb), about 9% - in myoglobin, about 1% - in heme and nohem enzymes. 25–30% of iron in the ferritin protein is also stored in combination with hemosiderin.

Anemia is a common extraintestinal manifestation of inflammatory bowel disease (IBD) and is often overlooked as a complication. Patients with IBD usually present with iron deficiency anemia (IDA) secondary to chronic blood loss and iron malabsorption due to tissue inflammation. Patients

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with iron deficiency may not always show signs and symptoms; Thus, the level of hemoglobin in patients with IBD should be regularly monitored for earlier detection of anemia. IDA in IBD is associated with poor quality of life, which requires rapid diagnosis and appropriate treatment. IDA is often associated with inflammation in patients with IBD. Thus, commonly used laboratory parameters are insufficient for diagnosing IDA, and new iron measures such as reticulocyte hemoglobin or percentage of hypochromic RBCs or zinc protoporphyrin are required to differentiate IDA from anemia of chronic disease. Signs and symptoms of iron deficiency depend on the severity and chronicity of anemia, along with the usual symptoms of anemia, including fatigue, paleness, and decreased physical performance. Cheilosis and koilonychia are symptoms of iron deficiency that develops in tissues that are rare in the modern world due to early diagnosis and timely correction. ***(Fig. 1)***

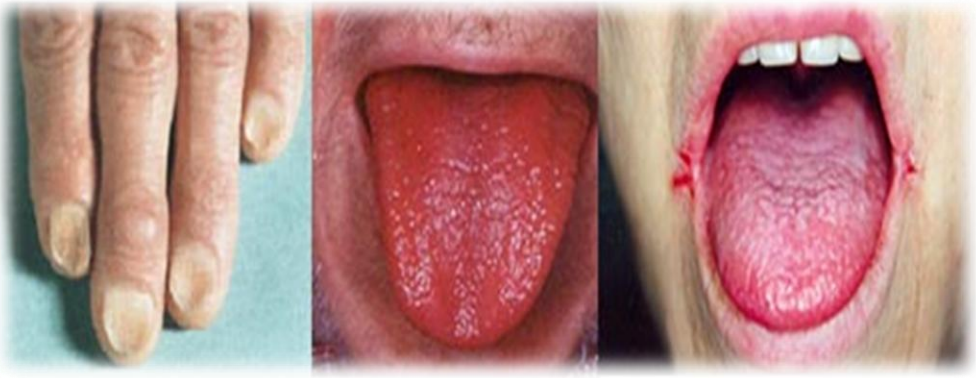
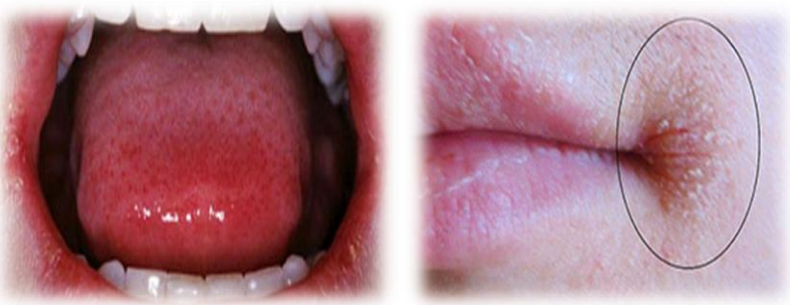


Fig. 1 *Clinical case*

The main symptoms of anemia, such as shortness of breath and tachycardia, are caused by a decrease in blood oxygen levels and peripheral hypoxia. Compensatory displacement of blood from the mesenteric arteries may disrupt perfusion of the intestinal mucosa. Dysmotilia, nausea, anorexia and even malabsorption are associated with anemia. Decreased metabolic and energy efficiency during exercise also contributes to weight loss in anemia.

Central hypoxia can cause symptoms such as headache, dizziness, vertigo, or tinnitus. A number of studies have confirmed that treatment of anemia improves cognitive function. Thus, anemia negatively affects almost all areas of daily life of patients with IBD. ***(Fig. 2)***



A **B**

Fig. 2 Iron deficiency anemia in the oral cavity

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Men with iron deficiency anemia (IDA) may experience impotence. Loss of libido contributes to a deterioration in quality of life in both sexes. In addition, latent iron deficiency can be the cause of "non-hematologic" symptoms such as hair loss, paresthesia of the arms and legs, and cognitive decline, and can also be strongly associated with restless legs syndrome.

Oral iron preparations are available and used in patients with mild disease activity. These drugs are inexpensive and convenient, but can cause gastrointestinal side effects such as abdominal pain and diarrhea, limiting their use and patient compliance. These drugs are partially absorbed due to inflammation. Unabsorbed iron can be toxic and impair IBD activity. Although cost-effective intravenous iron preparations are widely available and have improved safety profiles, clinicians are reluctant to use them. We present an overview of the pathophysiological mechanisms of IDA in IBD, improved diagnostic and therapeutic strategies, and the efficacy and safety of iron replacement therapy in IBD. Cases of iron deficiency are often secondary. In general, it is possible to distinguish two groups of situations that lead to it. The first group includes physiological and pathological conditions associated with high need for iron in the body. These include periods of strong growth in children, pregnancy, breastfeeding, acute and chronic blood loss, and more. The second group of reasons is cases of insufficient intake of iron in the body: "gem" iron deficiency in the diet, starvation, impaired intestinal absorption, and others. Among the reasons for the development of iron deficiency condition, the following pathological conditions are also important.

For example, infectious diseases, mainly chronic and recurrent forms, impair iron absorption. Some parasitic diseases: nematodes, trichocephaly, amebiasis, schistosomiasis can lead to chronic blood loss, which leads to iron deficiency. Injury or complications can lead to acute and chronic blood loss, followed by the development of iron deficiency status and anemia. The nutritional factor has a major negative impact on the occurrence of iron deficiency. A diet low in iron or a diet low in bioavailability, lack of nutrients needed for blood formation - folic acid, vitamins A, C, protein, copper, etc., often leads to iron deficiency is coming. It is the alimentary factor that is the most common cause of iron deficiency among low-income groups.

Iron homeostasis is strongly provided by the absorption of iron from duodenal enterocytes and is tightly regulated by hepsidine Hepcidin is a 25-amino acid peptide hormone that has specific antimicrobial activity and is an acute phase protein mainly synthesized by hepatocytes. For hexidine and iron-exporting cells, cell targets are villi enterocytes, reticuloendothelial macrophages, and hepatocytes. Hepcidin binds to the basolateral iron carrier and the exporting ferroportin 1, which leads to its phosphorylation, internalization by binding to JAK 2, and lysosomal degradation, which prevents the release of iron into plasma.

High levels of hepcidin suppress ferroportin, thereby reducing the release of iron from enterocytes and macrophages, leading to hypopheremia. Increased iron content in enterocytes reduces the expression of enterocyte brush boundary reductase (Dcytb) and divalent metal carrier

1 (DMT1) in villo enterocytes, inhibits iron absorption from food, and causes iron deficiency anemia. Therefore, by regulating the expression of DMT1 and ferroportin, hepsidin acts as a negative regulator of iron absorption in the duodenum and iron excretion from enterocytes and macrophages.

Women of childbearing age are at risk for developing iron deficiency because of their high need

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for iron. Most of them have chronic latent (latent) iron deficiency and are not diagnosed for a long time because there are no obvious clinical signs.

Serum ferritin is a measure of the amount of iron stored in the reticuloendothelial system; with absolute iron deficiency, serum ferritin concentration is <15 µg/l. Serum ferritin - acute phase reactive; normal or high levels can be found in inflammatory conditions despite iron deficiency. Thus, guidelines recommend defining serum iron deficiency as serum ferritin <30 g/L or transferrin saturation (TSAT) <16% in patients with still IBD without biochemical or clinical signs of inflammation. If inflammatory active IBD is present, as evidenced by high CRP levels, the guidelines specify a cut-off serum ferritin <100 g/L to increase sensitivity and specificity.

Microcytosis (low mean corpuscular volume, MCV) and hypochromia (low mean corpuscular hemoglobin, MCH) are present on the CBC and are indicators of absolute iron deficiency. High MCV is due to vitamin B12 and folic acid deficiency, certain medications (thiopurines, azathioprine, or 6-mercaptopurine), alcoholism, and hypothyroidism [24]. Therefore, a normal or high MCV does not rule out IDA as an option. The MCV may be low or normal in patients with ACD.

Serum transferrin carries Fe3+ into plasma and delivers iron from absorption sites (duodenal enterocytes and macrophages) to all tissues. Therefore, its level is higher with IDA. However, because it is an acute phase protein, levels may decrease during inflammation despite normal or low iron stores.

Transferrin saturation (TSAT), which is an indicator of circulating transferrin iron load, provides an indirect measure of iron utilization. TSAT is the ratio of serum iron to total iron-binding capacity times 100. It is also reduced in IDA and AKD. Pregnancy and oral contraceptives increase plasma transferrin levels; therefore, despite normal iron stores, TSAT may be low in these patients. Hepcidin levels increase with inflammation and decrease with IDA. This prevents iron absorption, causes iron retention in macrophages, and inhibits erythropoiesis.

Soluble transferrin receptors (sTfR) are a measure of erythropoietic activity. This is directly proportional to erythropoietic activity and inversely proportional to the presence of iron in tissues [28]. sTfR is used to differentiate iron deficiency (elevated sTfR and low serum ferritin) from inflammation (normal sTfR and low serum ferritin) and to diagnose the combination of iron deficiency and inflammation (due to elevated sTfR and limited serum ferritin). its cost and is not available in many laboratories.

The sTfR/log ferritin ratio (sTfR-ferritin index) may be the first indicator of iron depletion. A relative <1 indicates ACD and excludes iron deficiency, while a ratio of 2 indicates IDA or mixed IDA and ACD. Functional iron deficiency is an imbalance between the erythroid bone marrow's iron demand and its iron stores, in which the body cannot deliver iron fast enough to increase the rate of erythropoiesis. This leads to a decrease in the amount of cellular hemoglobin (Hb) in reticulocytes and erythrocytes [30]. The amount of reticulocyte hemoglobin (CHr) and the proportion of hypochromic erythrocytes are indicators of erythrocyte hemoglobinization and, therefore, functional iron deficiency, regardless of inflammatory conditions. With IDA, CHr > 40 and hypochromic erythrocytes > 5%. Iron binds to protoporphyrin IX to form the gemstone. In the absence of iron, zinc binds to protoporphyrin to form zinc protoporphyrin (ZPP). ZPP measures bone marrow iron levels during erythropoiesis and is independent of ACD or inflammation.

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