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Issue **Article**

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IRON DEFICIENCY ANEMIA AS AN ACTUAL PROBLEM IN MEDICAL PRACTICE

Abstract: *The article discusses iron deficiency anemia as an actual problem in medical practice. The authors of the article believe that the causes of iron deficiency first cause a decrease in its reserves in the macrophages of the liver, spleen and bone marrow, and then the concentration of iron in the blood. A negative iron balance develops, a manifestation of which is ALS. To prevent iron deficiency states it is necessary to effectively and promptly restore iron reserves in the body. Given the predominance of bread in the diet of the majority of the population, it is necessary to ensure the consumption of products (flour, flour products) enriched with iron preparations, combined with C-vitaminization of the diet. In our opinion, this way of primary prevention of IDA is acceptable for city dwellers living in a difficult socio-economic situation.*

Key words: *anemia, iron deficiency, blood, concentration, supply, drug, balance, nutrition, effective.*

Language: *English*

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Introduction

Iron deficiency anemia (IDA) is one of the most common diseases. Until now, there are severe, neglected forms of the disease that are difficult to correct. In this regard, one of the most important tasks of improving the quality of treatment of patients with IDA is the timely detection of iron deficiency, correction of risk factors and adequate therapy.

Main part

Etiology. Chronic blood loss is the main cause of IDA.

Uterine: pregnancy, menorrhagia, and myoma. In healthy women, blood loss during menstruation is 40-50 ml. With constant menstrual blood loss of more than 80 ml of blood, iron reserves are gradually depleted, leading to the development of IDA. Myoma, even in the absence of menstrual bleeding, can lead to iron deficiency.

Gastrointestinal: gastroduodenal erosions and ulcers, esophageal hernia of the diaphragm, portal hypertension with esophageal and rectal varices, ulcerative colitis, gastric and intestinal tumors, diverticula. Donation of blood on a regular basis (5 or more times in a year). Bleeding from the kidneys and

urinary tract, especially in hypernephroma, bladder cancer.

Hematologic diseases: coagulopathies, thrombocytopenia, thrombocytopathies, Randu-Weber-Osler disease. Vasculitis and collagenosis (especially Goodpasture's syndrome).

Alimentary factors: vegetarianism, starvation, malnutrition, monotonous food. Gastrointestinal iron absorption disorders: complete gastrectomy, gastric and/or intestinal resection,

Especially duodenum, where the iron absorption is most intense (90%), atrophic gastritis, celiac disease. Increased iron requirement: in infants (all types of milk contain very little iron), during pregnancy, during adolescence (increased iron consumption during rapid growth).

Other causes: paroxysmal nocturnal hemoglobinuria; isolated hemosiderosis of the lung; hemodialysis in 50 % of cases leads to development of ALS. Pathogenesis. The total amount of iron in the adult body is 3.5-4.0 g. Most of it is a part of hemoglobin (about 2.5 g), a considerable amount (about 0.5-1.0 g) is deposited in ferritin or included in hem and other enzymes (myoglobin, catalase, cytochrome) of the body (about 0.4 g), and a small amount of iron is bound to the hemoglobin in the blood. The balance in the body is maintained by matching the amount of incoming iron with its losses. Transferrin transports iron absorbed into the intestine or obtained after the destruction of old red blood cells to the bone marrow, where it is utilized to synthesize young red blood cells, or to the liver, where iron is is deposited as ferritin. Ferritin is a key protein reflecting the body's iron stores. [1]

It stores iron in a non-toxic form, which is mobilized when necessary. On average, one ferritin molecule contains up to 4,500 iron atoms. Iron is mainly deposited in the liver, bone marrow and spleen. A decrease in serum ferritin levels is a good indicator of an iron deficiency, and an increase in serum ferritin levels indicates an iron overload.

In case of iron excess in the body, ferritin turns into hemosiderin. At the same time remember that ferritin belongs to the proteins of the acute phase of inflammation, therefore the increase of ferritin in the blood may be due to an active inflammatory process and not only to iron excess. Some malignant tumors also have the ability to synthesize and secrete large amounts of ferritin into the blood (as part of the paraneoplastic syndrome). Its normal serum content is 30-300 ng/ml. The causes of iron deficiency first cause a decrease in iron stores in the macrophages of the liver, spleen and bone marrow, followed by a concentration of iron in the blood. A negative balance of iron develops, a manifestation of which is an iron deficiency disease. In ID, erythron cells are unable to synthesize normal Hb, and the cytoplasmic hemoglobinization decreases in normoblasts. The concentration of Hb in mature erythrocytes decreases, morphologically manifested by hypochromia. Since the cell size partly depends on the critical Hb concentration, this leads to the formation of not only hypochromic but also small in size erythrocytes (microcytes). Consequently, WBC will be hypochromic and microcytic, and Hb and hematocrit levels will be disproportionately lower than the number of erythrocytes. Anisocytosis, poikilocytosis are observed and occasionally normoblasts appear in the blood [3]. The half-life period of erythrocytes is somewhat shortened due to a defect in the cells themselves, rather than in their surroundings.

According to the degree of severity, the following classification of GAD is accepted:

- mild - Hb concentration in blood from 110 to 90 g/l;
- of moderate degree - blood Hb concentration in blood from 90 to 70 g/l;
- severe degree - blood Hb concentration in blood from 70 to 55 g/l;
- extremely severe - blood Hb concentration <55 g/l.

Clinical picture. Iron deficiency in the body is manifested by sideropenic and anemic syndromes. Sideropenic (hyposiderosis) symptoms: pronounced changes in the skin, mucous membranes, nails, hair; muscle weakness, perversion of taste (pathophagy - addiction to chalk, clay, charcoal, toothpaste, ice) and smell (pathoosmia - like the smell of gasoline, paints, shoe polish, exhaust fumes).

On examination, attention is drawn to the pallor of the skin, sometimes with a slight greenish hue ("chlorosis"); the skin is dry, flaky, cracks form easily on the hands and feet; no pigmentation from tanning. Hair loses its shine, becomes thin, split, breaks easily, thinning and graying early. Nails become thin, matte, flattened, break easily, and sometimes become concave [12]. Sideropenia leads to atrophy mucous membrane of the tongue, angular stomatitis, tooth decay; atrophic changes in the mucous and muscular membranes of the esophagus may manifest as sideropenic dysphagia (Plummer-Vinson-Bechterev's symptom), manifested by difficulty in swallowing dry and solid food and even saliva, which is accompanied by excruciating spasms, especially at night. Esophagoscopy and X-ray examination reveals spastic narrowing of the initial part of the esophagus Anemic symptoms: complaints of patients about weakness, fatigue, chronic fatigue, brokenness, decreased capacity for work, headaches, dizziness, flickering of flies in front of the eyes, noise in the head. These symptoms are due to a decrease in iron-dependent and iron-containing enzymes in the muscles and the development of intracellular hypoxia.

Common symptoms for patients with IDA may include shortness of breath, palpitations, chest pain, irritability, arterial hypotension. Tachycardia, expansion of the boundaries of relative dullness of the heart to the left, anemic systolic murmur at the apex

and pulmonary artery are defined. The jugular and femoral arteries exhibit a wave-like murmur, which is explained by hydraemia and increased velocity of blood flow. The murmurs have a functional character and disappear along with elimination of anemia. In severe IDA, elderly patients may develop cardiovascular insufficiency.

A manifestation of IDA is sometimes fever, the body temperature usually does not exceed 37,5ºC and disappears after treatment with iron-containing medications.

Diagnosis. Laboratory tests of blood, bone marrow, and iron metabolism are decisive for diagnosis. The blood picture is characterized by the presence of signs of hypochromic microcytic anemia. Hb concentration decreases. The number of erythrocytes also decreases, but to a lesser extent than the Hb level. Small hypochromic erythrocytes, erythrocytes of unequal size and shape (anisocytosis, poikilocytosis) prevail in blood smears. In severe anemia, individual erythroblasts may appear. The number of reticulocytes is unchanged [13]. Specific signs of IDA

Low serum iron content (normal 12.5-30.4 μmol/L). Increased total serum iron-binding capacity (the norm 30.6-84.6 μmol/L). Low serum ferritin concentration: a reflection of low levels of iron deposited in the bone marrow. Low levels of MCV (mean red blood cell volume) <80 fl and MSN (mean hemoglobin content of the erythrocyte) <27 pg.

Treatment. The aim of iron deficiency therapy is the elimination of iron deficiency and restoration of iron reserves in the body. This is possible only by eliminating the underlying cause of ALS and at the same time compensating for iron deficiency in the body.

The most common causes of ALS in women are menorrhagia and myoma. Menorrhagias (hypermenorrhea) are uterine Bleeding in patients with a preserved menstruation rhythm, when the duration of bleeding is more than 7 days. They are recommended to take Aminocaproic acid 1 g four times a day from the 1st to the 4th day of menstruation, which reduces the blood loss by 50%. Non-steroidal anti-inflammatory drugs (ibuprofen, nimesulide) reduce the production of prostaglandins and thromboxanes in the endometrium, reducing the volume of blood loss during menstruation by 30-38%. Ibuprofen is prescribed 400 mg 4 times, nimesulide 50 mg 3 times a day on the days of menorrhagia. Their effect is comparable to that of aminocaproic acid. Immediate laparotomic hysterectomy is recommended in the presence of myoma.

IDA therapy should not be stopped after normalization of Hb levels to replenish iron stores in the depot, and a significant increase in Hb in contrast to the improvement of the well-being will not be fast - after 4-6 weeks; hemotransfusions in WDAS should be performed strictly on vital indications. Real danger of infection with viruses of serum hepatitis, mononucleosis and HIV during hemotransfusions is high. In addition, transfusion of red blood cells immunizes the recipient with antigens that he or she does not have. This can affect the course of the pregnancy later in life. In these cases, miscarriages are possible, stillbirths, hemolytic disease in newborns. Transfusion of red blood cells is indicated in pregnant women with very low Hb in labor or 1-2 days before delivery. It is not recommended in the earlier term . It is now recognized that iron in the form of heme (blood sausage, liver, meat, fish) is better absorbed in the body than from plant foods.

It should also be noted that even a diet balanced by the basic ingredients allows only to "cover" the physiological need of the body for iron, but not to eliminate its deficiency.

Two groups of iron preparations are used: those containing divalent and trivalent iron. A daily dose of iron should be 100-200 mg. Maximum absorption occurs when tablets are taken on an empty stomach; intake during or after meals reduces it by 50-60%. In the presence of marked adverse events associated with irritation of the upper gastrointestinal tract, it is possible to take the drug during or after meals. The bioavailability (absorption) of iron increases in the presence of ascorbic acid (approximately 2-3 times).

Divalent iron preparations in tablet form: tardiferon (200 mg of elemental iron), feospan (200 mg), ferrogradumet (100 mg), sorbifer durules (100 mg), cheferol (100 mg), actiferin (100 mg), iron fumarate (65 mg), totem (50 mg), ferratab (50 mg), fenuls (45 mg), hemopher (44 mg), ferroplex (10 mg)., catalase, glutathione, tocopherols, B-carotene. Trivalent iron preparations for oral administration: ferrum lek (100 mg), Maltofer (100 mg), Maltofer Fol (100 mg), ferlatum, ferro-III, biofer.

Iron preparations for parenteral administration: ectofer, venofer, ferrumlek, ferinject, ferrovir, cosmofer, lickferr. The duration of treatment of clinically expressed IDA is 3-5 months until normalization of hemoglobin levels, and for pregnant women - at least until the onset of childbirth to restore iron reserves.

Conclusion

To prevent iron deficiency states, it is necessary to effectively and promptly restore iron reserves in the body. Given the predominance of bread in the diet of the main part of the population, it is necessary to ensure the consumption of products (flour, flour products) enriched with iron preparations, combined with C-vitaminization of the diet. In our opinion, this way of primary prevention of IDA is acceptable for city dwellers living in a difficult socio-economic situation.

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