

DIFFERENTIAL DIAGNOSIS AND TREATMENT IRON DEFICIENCY ANEMIA

Khodjayeva Gulnora Abdubannonovna

Fargona Public Health Medical Institute

Assistant of the Department of Propedeutics of Internal Diseases

Anemia is a clinical and hematological syndrome characterized by a decrease in the number of red blood cells and hemoglobin in the blood. A decrease in the hemoglobin content below the level of less than 120 g/l is considered as anemia. However, 80% of all anemia is associated with iron deficiency. Iron in the human body participates in the regulation of metabolism, in the processes of oxygen transfer, in tissue respiration and has a huge impact on the state of immunological resistance. Iron absorption is carried out mainly in the duodenum and upper jejunum. Recommended daily intake of iron with food: 12 mg for men, 15 mg for women, 30 mg for pregnant women.

Classification, clinic and diagnosis of Iron deficiency anemia (further – ZHDA)

In accordance with the International Classification of Diseases of the 10th revision (ICD–10), the following forms of anemia associated with absolute and relative iron deficiency are taken into account.

Clinical classification of IDA based on risk factors:

1. Waiting for posthemorrhagic. This group consists of anemia developing on the basis of repeated small blood loss – metrorrhagia, epistaxis, hematuria, etc.;
2. IDA of pregnant women - an imbalance in the nutrition of pregnant women, deterioration of iron utilization, transfer by the mother's body of a significant amount of it to the developing fetus, iron loss during lactation, etc.;
3. IDA associated with gastrointestinal pathology. These include anemia that occurs after gastrectomy, extensive resections of the small intestine, with various enteropathies. At its core, these are expectations caused by a rough, severe impairment of the function of iron absorption in the proximal duodenum;
4. Secondary IDA, arising from infectious, inflammatory or tumor diseases. Anemia in these cases develops due to large losses of iron in the death of tumor cells, tissue decay, micro– and even macrohemorrhagia, increased need for iron in the foci of inflammation;
5. Essential (idiopathic) IDA;
6. Juvenile IDA – anemia developing in young girls associated with dishormal phenomena;



7. WAITING for a complex genesis. This group includes alimentary anemia.

By stages:

Stage I – loss of iron exceeds its intake, gradual depletion of reserves, absorption in the intestine compensatorily increases;

Stage II – depletion of iron reserves (serum iron level – below 50 mcg / l, transferrin saturation – below 16%) prevents normal erythropoiesis, erythropoiesis begins to fall;

Stage III – the development of mild anemia (100-120 g / l of hemoglobin, compensated), with a slight decrease in the color index and other indices of erythrocyte saturation with hemoglobin;

Stage IV – severe (less than 100 g/l of hemoglobin, subcompensated) anemia with a clear decrease in erythrocyte saturation with hemoglobin;

Stage V – severe anemia (60-80 g / l of hemoglobin) with circulatory disorders and tissue hypoxia.

By severity: light (Hb content - 90-120 g/l); medium (70-90 g/l); heavy (less than 70 g/l).

Clinical picture of IDA. The clinical manifestations of IDA are two major syndromes – anemic and sideropenic.

Anemic syndrome is caused by a decrease in hemoglobin content and a decrease in the number of red blood cells, insufficient oxygen supply to tissues and is represented by nonspecific symptoms. An objective examination reveals pallor of the skin and visible mucous membranes, often some pasty in the area of the shins, feet, and face.

Morning edema – "bags" around the eyes are characteristic. Anemia causes the development of myocardiodystrophy syndrome, which is manifested by shortness of breath, tachycardia, often with arrhythmia, moderate expansion of the boundaries of the heart to the left, deafness of heart tones, low systolic noise at all auscultative points. With severe and prolonged anemia, myocardiodystrophy can lead to severe circulatory insufficiency. IDA develops gradually, so the patient's body adapts to a low level of hemoglobin, and subjective manifestations of anemic syndrome are not always pronounced.

Sideropenic syndrome (hyposiderosis syndrome) is caused by tissue iron deficiency, which leads to a decrease in the activity of many enzymes (cytochrome oxidase, peroxidase, succinate dehydrogenase, etc.).

Sideropenic syndrome is manifested by numerous symptoms, such as: Perversion of taste (pica chlorotica) – an irresistible desire to eat something unusual and inedible (chalk, tooth powder, charcoal, clay, sand, ice), as well as raw dough, minced meat this symptom is more common in children and



adolescents, but is often observed in adult women; diet: addiction to spicy, salty, sour, spicy food; perversion of the sense of smell – addiction to odors that most people perceive as unpleasant (smells of gasoline, acetone, lacquers, paints, shoe polish, etc.); expressed muscle weakness and fatigue, muscle atrophy and decreased muscle strength due to a deficiency of myoglobin and tissue respiration enzymes, dystrophic changes in the skin and its appendages (a symptom of coilonychia – spoon-shaped concavity of nails), angular stomatitis - "jams" in the corners of the mouth (10-15% of patients), glossitis (in 10% of patients) – characterized by sensation of pain and swelling in the tongue, redness of its tip, and later – papillary atrophy ("varnished" tongue); atrophic changes in the gastrointestinal mucosa with pain when swallowing food, especially dry (sideropenic dysphagia); development of atrophic gastritis and enteritis; symptom of "blue sclera" – characterized by bluish coloration or pronounced blueness sclera, imperative urge to urinate, the inability to retain urine when laughing, coughing, sneezing, possibly even bedwetting, due to the weakness of the bladder sphincters, "sideropenic subfebrility" – characterized by a prolonged increase in temperature to subfebrile values; reduction of reparative processes in the skin, mucous membranes.

Diagnostics of the railway.

1. The average hemoglobin content in the erythrocyte in picograms (norm 27-35 pg) reduced;
2. The average concentration of hemoglobin in the erythrocyte is reduced; normally it is 31-36 g / dl;
3. Microcytosis of erythrocytes - reduction of their size;
4. Different form of erythrocytes – poikilocytosis;
5. The number of reticulocytes (in the absence of blood loss and period.

Biochemical blood analysis:

1. Reduction of iron content in blood serum (normally in men 13-30 mmol / l, in women 12-25 mmol / l);
2. OHSS is elevated (reflects the amount of iron that can be bound due to free transferrin; OHSS is normal - 30-86 mmol/L);
3. Latent iron binding capacity of blood serum is increased (it is determined by subtracting the serum iron content from the indicators of the OHSS).

Treatment. The treatment program for IDA includes: treatment of the underlying disease, therapeutic nutrition, treatment with iron-containing drugs (for oral administration), elimination of iron deficiency and anemia, replenishment of iron reserves (saturation therapy).



Patients with iron deficiency anemia are recommended a varied diet, including meat products (veal, liver) and vegetable products (beans, soy, parsley, peas, spinach, dried apricots, prunes, pomegranates, raisins, rice, buckwheat, bread). However, it is impossible to achieve an anti-anemic effect only by diet. Even if the patient eats high-calorie foods containing animal protein, iron salts, vitamins, trace elements, it is possible to achieve iron absorption of no more than 3-5 mg per day. It is necessary to use iron preparations. Currently, the doctor has a large arsenal at his disposal iron preparations characterized by different composition and properties, the amount of iron contained in them, the presence of additional components affecting the pharmacokinetics of the drug, various dosage forms.

According to the recommendations developed by WHO (2009), when prescribing iron preparations, preference is given to preparations containing divalent iron. The daily dose should reach 2 mg / kg of elemental iron in adults. The total duration of treatment is at least three months (sometimes up to 4-6 months). An ideal iron-containing drug should have a minimum number of side effects, have a simple application scheme, the best efficiency / price ratio, optimal iron content, preferably the presence of factors that enhance absorption and stimulate hematopoiesis.

The list of drugs used in the treatment of iron deficiency anemia: Jacktofer (Jacktofer), Maltofer (Maltofer), Sorbifer durules (Sorbifer durules), Tardiferon (Tardiferon), A Feramide (Ferramidum), Ferro-gradumet (Ferro-gradumet), Ferroplex (Ferroplex), Ferroceron (Ferroceronum), Ferrum lek (Ferrum lek), Totem (tothema)

The reasons for the ineffectiveness of pancreatic therapy for oral administration:

- lack of iron deficiency (incorrect interpretation of the nature of hypochromic anemia and erroneous appointment of pancreas);
- insufficient dosage of pancreas (underestimation of the amount of trivalent iron in the preparation);
- insufficient duration of pancreatic treatment;
- violation of the absorption of pancreas administered orally to patients with the corresponding pathology;
- simultaneous administration of drugs that disrupt the absorption of iron;
- the presence of chronic (occult) blood loss, most often from organs Gastrointestinal tract;
- combination of IDA with other anemic syndromes (B12-deficient, folic deficiency).



Prevention of Iron deficiency anemia:

- Periodic monitoring of the blood picture;
- eating foods with a high iron content (meat, liver, etc.);
- preventive intake of iron preparations in risk groups;
- prompt elimination of sources of blood loss.

REFERENCES:

1. Harrison's Principles of Internal Medicine (18th edition) – DanLongo, AnthonyFaucietel – McGraw-HillProfessional – 2011 – 4322 p.;
2. In-house medicine: a textbook of clinical practice – A.S. Svitsky, Kiev VSV "Medicine" 2014r.;
3. Hunt J.R., Roughead Z.K. Adaptation of iron absorption in men consuming diets with high or low iron bioavailability. Amer. J. Clin. Nutr. 2000; 71: 94—102.

