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IRON DEFICIENCY ANEMIA: RELEVANCE AND METHODS OF TREATMENT

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ABSTRACT

The article under discussion depicts iron deficiency anemia, its relevance and methods of treatment. The author of the article considers that iron deficiency anemia (IDA) is one of the most common diseases on the planet, and the damage to many organs and systems associated with it determines the relevance and importance of studying this problem. Modern diagnostic methods, allowing the use of a differential diagnostic algorithm to assess the anemic syndrome, in combination with rational use of iron preparations, can significantly improve the well-being and quality of life of patients with iron deficiency and contribute to the timely prevention of iron deficiency in risk groups to eliminate the adverse effects of iron deficiency on patient's health.

KEYWORDS

Iron deficiency anemia, iron resources, iron-containing drugs, menstrual bleeding, cancer, patient.

INTRODUCTION

Iron deficiency anemia (IDA) is hypochromic microcytic hypo regenerative anemia, occurs due to absolute reduction of iron resources in the body (usually with chronic blood loss or insufficient iron intake).

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) [1].

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. Increasing the body's need for iron: in infants (all types of milk contain very little iron), during pregnancy, and 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 the development of IDA. 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 part of the iron is in a transferrin-bound state 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 deposited as ferritin. Ferritin is a key protein reflecting the body's iron stores. It stores iron in a non-toxic form, which is mobilized when needed. On average, one ferritin molecule contains up to 4,500 iron atoms. Iron is mainly deposited in the liver, bone marrow and spleen [3].

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 IDA, 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 concentration of Hb, this leads to the formation of not only hypochromic but also small-sized 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. The half-life period of erythrocytes is somewhat shortened due to a defect in

the cells themselves, rather than in their surroundings [10].

According to the degree of severity, the following classification of IDA 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. Sideropenia leads to atrophy of the mucous membrane of the tongue, angular stomatitis, dental caries; atrophic changes in the mucous and muscular membranes of the esophagus may manifest as sideropenic dysphagia (Plummer-Vinson-Bechterev symptom), showing difficulty in swallowing dry and solid food and even saliva, which is accompanied by painful 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 flashes before eyes,

noise in 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 IDA patients 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 [3].

A manifestation of IDA is sometimes fever, the body temperature usually does not exceed 37.5°C and disappears after treatment with iron-containing medications.

Diagnosis. The data of laboratory tests of blood, bone marrow and iron metabolism are decisive in making the 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.

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.

Menorrhagia (hypermenorrhea) - uterine bleeding in patients with a preserved rhythm of menstruations, with a duration of bleeding over 7 days. They are recommended to take aminocaproic acid 1 g 4 times a day orally from the first to the fourth 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. Valvular disease therapy should not be stopped after normalization of Hb level, in order to replenish iron stores in depot, and essential increase of Hb in contrast with improvement of well-being will be not fast - in 4-6 weeks; - hemotransfusions in ALS should be carried out 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 of newborns. Red blood cell transfusion is indicated in pregnant women with very low Hb in labor or 1-2 days before delivery [6]. It is not recommended in the earlier term. It is now recognized that iron in the form of gem (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 [5].

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

Modern iron-containing medications can be successfully used for the prevention and treatment of iron deficiency. The choice of the optimal iron drug for the treatment of IDA should be made individually for each patient taking into account anemia severity, iron bioavailability, development of possible adverse reactions and concomitant diseases. Modern diagnostic methods, allowing the use of a differential diagnostic algorithm to assess the anemic syndrome, in combination with rational use of iron preparations, can significantly improve the well-being and quality of life of patients with iron deficiency and contribute to the timely prevention of iron deficiency in risk groups to eliminate the adverse effects of iron deficiency on patient's health.

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