DIAGNOSIS AND MODERN METHODS OF TREATMENT OF IRON-DEFICIENCY ANEMIA

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Конец формы**Keywords:** *ferritin, transferrin, serum iron, iron-deficiency anemia.*

Abstract

Iron-deficiency anemia (IDA) is one of the most common diseases worldwide and ranks first in prevalence among women of reproductive age [3,5]. The prevalence of anemia, including IDA, depends on many factors, such as socioeconomic living conditions, nutrition, bleeding of various origins, parasitic diseases, and others. According to experts from the World Health Organization (WHO), anemia is more common in developing countries, with young children and pregnant women being the two population groups most vulnerable to anemia. Iron deficiency may cause symptoms both in the presence and absence of anemia or may remain asymptomatic. Common symptoms and signs include fatigue and lethargy, impaired concentration, dizziness, tinnitus, pallor, and headache. In susceptible individuals, iron deficiency August contribute to the development of restless leg iron deficiency may contribute to the development of restless legs syndrome. Other manifestations include alopecia, dry hair or skin, koilonychia, and ***atrophic glossitis. Iron deficiency and anemia Avgust also aggravate symptoms and*** *worsen the prognosis of diseases such as heart failure and ischemic heart disease.*

Materials and Methods. The diagnosis is based on medical history and laboratory findings, including a complete blood count (CBC), assessment of hematocrit (Hct), measurement of red blood cell count and reticulocyte count, determination of the mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC), as well as assessment of red blood cell size in patients with anemic syndrome for the diagnosis of IDA. In IDA, decreased levels

of hemoglobin and hematocrit are observed, along with reduced mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration (MCH and MCHC, respectively) and decreased mean corpuscular volume (MCV). The red blood cell count is usually within the normal range. Reticulocytosis is not characteristic of IDA but may occur in patients with bleeding. Morphological features of IDA include erythrocyte hypochromia and anisocytosis with a tendency toward microcytosis [1,2]. These morphological characteristics do not allow iron-deficiency anemia to be distinguished from so-called **anemia of chronic disease (ACD)**, which is associated with functional iron deficiency caused by the redistribution of iron in the presence of inflammation, infection, or malignancy. All patients with suspected IDA should undergo assessment of serum iron metabolism parameters, including serum ferritin, serum transferrin, total iron-binding capacity (TIBC), serum iron concentration, and transferrin saturation (TSAT), in order to confirm the presence of absolute iron deficiency [4,5].

Results and Discussion. The distinguishing features of true IDA are a low serum ferritin level, reflecting depletion of tissue iron stores, and increased TIBC and transferrin levels (Table 1). Serum iron and TSAT are typically decreased. However, normal or even elevated values do not exclude the diagnosis of IDA, since the intake of iron-containing medications shortly before testing, a meat-rich diet, or a red blood cell transfusion performed within the preceding 10–14 days may significantly distort the serum iron level and, consequently, the TSAT. These factors should therefore be taken into account when interpreting laboratory results. The development of IDA is preceded by a stage of **latent iron deficiency**, the laboratory criteria of which include low serum iron and ferritin levels in the presence of a normal hemoglobin concentration. Microcytic hypochromic anemia is a characteristic morphological feature of **β-thalassemia**, severe forms of which are associated with profound anemia and pronounced signs of iron overload, including increased serum ferritin and TSAT and decreased transferrin and TIBC levels.

However, mild subclinical forms of thalassemia, presenting with mild microcytic hypochromic anemia, are often misdiagnosed as iron-deficiency anemia when serum iron metabolism parameters are not assessed. This may result in inappropriate iron therapy, potentially accelerating the development of tissue iron overload.

All patients with IDA should undergo a comprehensive evaluation to determine the underlying cause of iron deficiency and to identify concomitant pathology [3]. This includes a general biochemical blood test assessing total protein, albumin, total bilirubin, direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine, urea, alkaline phosphatase, and gamma-glutamyl transferase (GGT), as well as a urinalysis. The results of these basic investigations may help determine the direction of further diagnostic evaluation. Assessment of biochemical markers of liver function is necessary for the correct interpretation of iron metabolism parameters, as impaired hepatic protein synthesis may lead to decreased transferrin production [1]. Renal dysfunction may contribute to both relative and absolute iron deficiency. Other biochemical parameters are used in the differential diagnosis of anemia of various etiologies. Bone marrow aspiration and trephine biopsy may be indicated in cases of persistent, treatment-resistant anemia and/or in the presence of other cytopenias, such as leukopenia or thrombocytopenia. In IDA, no specific pathological changes are usually detected in the bone marrow; cellularity is generally normal, although moderate hyperplasia may occasionally be observed. A characteristic feature of the bone marrow in IDA is a reduction in the number of sideroblasts—erythroblasts containing iron granules (normally 20–40%). IDA should be differentiated from anemia associated with chronic inflammatory and malignant diseases (anemia of chronic disease), as well as from hypochromic anemias associated with iron overload, including α- and β-thalassemia, porphyrias, and lead intoxication.

Начало формы**Конец формыTreatment of Iron-Deficiency Anemia. The goal of IDA treatment is to administer an amount of iron sufficient to normalize hemoglobin levels (120–140 g/L in women and 130–160 g/L in men) and replenish tissue iron stores (serum ferritin >40–60 µg/L). For the treatment and prevention of iron deficiency, oral preparations of ferrous iron (ATC code B03AA) or ferric iron (ATC code B03AB) are used, with ferrous sulfate being among the most commonly prescribed preparations. The quantitative and qualitative composition of iron-containing medications varies considerably and includes high- and low-dose, single-component, and combination preparations. According to WHO recommendations, the optimal dose of elemental iron for the treatment of IDA is 120 mg/day, while the recommended dose for the prevention of iron deficiency is 60 mg/day. In children, the dosage of oral iron salt preparations depends on age and is approximately 3 mg/kg/day in children under three years of age, 45–60 mg/day in children over three years of age, and up to 120 mg/day in adolescents. Approximately 20% of patients develop diarrhea or constipation during treatment; these adverse effects can generally be managed with symptomatic therapy. Gastrointestinal irritation, including nausea and epigastric discomfort, can be minimized by taking iron preparations with meals or reducing the dose. The use of high-dose iron preparations is associated with an increased frequency of gastrointestinal adverse effects.**

The duration of treatment is determined by the severity of the initial iron deficiency and may range from 1 to 3 months**.** Treatment may be supplemented with multivitamin preparations to ensure adequate nutritional support for normal metabolic and tissue-building processes.

References

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