



CAUSES, PATHOGENESIS, DIAGNOSIS AND CLINICAL FEATURES OF IRON DEFICIENCY ANEMIA

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Causes of Iron Deficiency Anemia:

The main causes of the development of iron deficiency anemia include:

1. Chronic blood loss from the uterus, esophagus, stomach, intestines, or kidneys; hemorrhagic syndrome; disorders of blood coagulation; liver diseases; overdose of anticoagulants and other factors.
2. Development of malabsorption syndrome, Crohn's disease, intestinal tuberculosis, disaccharidase deficiency, enteropathy, small intestinal tumors, post-parasitic syndrome, pancreatic insufficiency, and impaired iron absorption (chronic atrophic gastritis, gastric cancer, gastrectomy).
3. Increased demand for iron during pregnancy, lactation, periods of intensive growth, and chronic renal insufficiency.
4. Nutritional causes such as anorexia, vegetarianism, and inadequate dietary iron intake.

It is important to emphasize that, in addition to damage to the gastrointestinal tract, impaired absorption of vitamin B12, folic acid, and amino acids may also contribute to the development of iron deficiency anemia (24). Another significant cause of anemia is acute and chronic inflammation of internal organs and oncological diseases in elderly individuals. This type of anemia is known as "anemia of chronic disease" or "anemia of inflammation." In such cases, oral iron preparations are ineffective, and dysfunction of iron-regulating proteins—hepcidin and ferroportin—is observed (25).

Pathogenesis: The main source of iron entering the human body is exogenous iron obtained from food. Iron absorbed in the duodenum is converted into ferritin and stored in macrophages and enterocytes, or transported into the plasma with the help of the protein ferroportin. Heparin, synthesized by the liver, binds to ferroportin receptors, reducing their function and thereby decreasing or completely blocking the release of iron from enterocytes and macrophages into the bloodstream. It is known that cytokines produced during chronic and acute inflammatory diseases, such as TNF- α , IL-6, and IL-10, stimulate the production of hepcidin (25, 27). Excessive production of hepcidin inhibits the function of ferroportin. As a result, iron does not enter the bloodstream, leading to decreased serum iron levels and the development of anemia, while excess iron accumulates in parenchymal organs.



Diagnosis: Iron deficiency anemia consists of two main syndromes: anemia and sideropenia. Anemic syndrome is characterized by tissue hypoxia and presents with symptoms such as weakness, dizziness, fatigue, shortness of breath, irritability, lacrimation, and pallor of the skin. Functional systolic murmurs may be detected during cardiac auscultation. Sideropenic syndrome develops due to iron deficiency in tissues, which reduces the activity of numerous iron-containing enzymes (such as cytochrome oxidase, peroxidase, and others), leading to a variety of clinical manifestations (28). These include altered taste, muscle weakness caused by myoglobin deficiency and impaired oxygen delivery to tissues, dry skin, brittle thinning nails, stomatitis, glossitis, and atrophic changes of the esophageal mucosa (sideropenic dysphagia), as well as atrophic gastritis and enteritis in the gastrointestinal tract (28).

Laboratory Examination: During peripheral blood analysis, a decrease in the number of red blood cells and hemoglobin (Hb) levels is detected.

1. In mild anemia, hemoglobin (Hb) decreases to 110–90 g/L.
2. In moderate anemia, hemoglobin (Hb) decreases to 89–70 g/L.
3. In severe anemia, hemoglobin (Hb) is 69 g/L or lower.

The color index is reduced to <0.85.

The mean hemoglobin content in erythrocytes (MCH) is <26.

The mean corpuscular hemoglobin concentration (MCHC) is <30%.

The mean corpuscular volume (MCV) is <80 fL.

These findings indicate hypochromic and microcytic anemia (24, 29). In most cases, these indicators alone are not sufficient to diagnose iron deficiency anemia, because hypochromic and microcytic anemia is also characteristic of other forms of anemia, such as hemolytic anemia, sideroblastic anemia, thalassemia, and others. A more reliable method for diagnosing iron deficiency anemia is to determine serum iron concentration, transferrin, ferritin levels, as well as serum total iron-binding capacity (TIBC) and transferrin saturation index. Iron deficiency anemia is characterized by a decrease in serum iron levels, compensatory increase in synthesis of transport proteins (normal range 2.50–3.80), and a decrease in ferritin concentration (normal range 44.7–71.6 $\mu\text{mol/L}$) (29).

Conclusion: Today, iron deficiency anemia is one of the most widespread diseases, as confirmed by WHO data. Women of reproductive age, pregnant women, adolescent girls, young children, and elderly people constitute a large percentage of those affected by this disease. Therefore, timely detection, treatment, and prevention of this condition are strongly recommended. To diagnose anemia, it is necessary to monitor MCH, MCHC, and MCV indicators. Not only insufficient iron intake but also diseases of the gastrointestinal tract play an important role in the development of this condition.

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