Anemia of chronic diseases

Anemia of chronic diseases (often in the abbreviation **ACD**), sometimes also called anemia of inflammation, is the **second most common anemia** after iron deficiency anemia (sideropenic, IDA), from which it is sometimes necessary in differential diagnosis to differentiate.

Basic characteristics

It is usually mild or moderate anemia. It usually has a **normocytic** and **normochromic** character, sometimes it can be microcytic and hypochromic. In order to distinguish it from other anemias, it is therefore necessary to determine other parameters. This anemia accompanies, or participates in the development of anemia especially in the following diseases:

- **chronic inflammatory diseases** (ulcerative colitis, rheumatic diseases),
- **chronic infections** (pneumonia, subacute bacterial endocarditis, osteomyelitis, AIDS),
- **chronic kidney failure** (here it can complement the main cause – insufficient production of erythropoetin),
- **tumors** (carcinomas, lymphomas, leukemia, myeloma),
- **systemic diseases** (RA, SLE, autoimmune vasculitis, scleroderma]]),
- other **microinflammatory conditions** incl. severe obesity, etc.,
- severe **acute conditions** (infection, post-operative or post-traumatic conditions with catabolism) can induce conditions that have certain features of this anemia.

Anemia is present in chronic disease with a probability of 25-30%.

Pathogenesis

Several factors contribute to the formation. In principle, it is considered **hypoproliferative** anemia, i.e. the production of erythrocytes is reduced, but the survival of erythrocytes is also slightly shortened. The main factors are:

- **shortened survival of erythrocytes** due to activation of the macrophage system,
- reduced production of **erythropoetin**,
- reduced sensitivity of erythroid precursors to erythropoietin,
- relative (functional) lack of **iron** for erythropoiesis (cytokines stimulate the formation of ferritin and hepcidin, which leads to the inhibition of the release of iron from cells of the monocytomacrophage system into the extracellular space and a decrease in enteral absorption of Fe).

These changes are caused by the action of **inflammatory cytokines** - interleukins 1, 6, TNF, etc.

Alteration of iron metabolism in anemia of chronic diseases

The condition is referred to as **functional iron deficiency** because iron is poorly available for erythropoiesis, although its total amount is not substantially reduced, but the iron is retained (sequestered) in macrophages. It's in the blood

- reduced **serum iron** concentration (this can sometimes misdiagnose only sideropenic anemia), but iron can be demonstrated in macrophages, e.g. by staining.

In particular, however, other features of sideropenia are not

- on the contrary, there is an increased concentration of **serum ferritin** (this is an acute phase protein),
- on the contrary, the concentration of **serum transferrin** (i.e. the total binding capacity) is reduced,
- **transferrin saturation** is not significantly reduced.

An inflammatory increase (due to IL-6) of the systemic regulator of iron metabolism, hepcidin, is involved in the change in iron metabolism. Hepcidin reduces the amount of the membrane iron exporter - ferroportin - in macrophages, which, together with the inflammatory stimulation of ferritin formation, contributes to the retention of iron in macrophages. The effect of hepcidin is relatively rapid, the serum iron concentration can fall within a few hours after inflammatory stimulation. This is also due to the rapid turnover of iron in the plasma (plasma iron is "exchanged" several times a day). The biological meaning of this event is apparently defense against infection, i.e. retention of iron, which is necessary for invading microorganisms. Another effect of hepcidin is the reduction of iron resorption in the duodenum (by an analogous mechanism), but this manifests itself over a much longer period of time, since the daily resorption is about 1 mg, while the daily turnover of iron is 20-25 mg, and in a healthy person, the iron reserves reach several hundred mg .

Brief clinical notes: diagnosis and treatment

It is not a guide to treatment, just a pathophysiological application of theoretical knowledge.

- This anemia should be considered in the above diseases.

- In addition to the blood count, the examination of parameters of iron metabolism is essential, while serum iron alone is not sufficient, on the contrary, it can be misleading.
- In many cases, inflammatory changes in iron metabolism can be combined with its losses. In such a case, for example, low serum iron does not correspond to serum ferritin concentration.
- In treatment, if possible, the underlying disease (inflammation or infection) should be treated.
- Administration of iron in the anemia of chronic diseases alone does not bring improvement.
- Administration of iron (if there is a current deficiency) due to reduced absorption may not be effective enough orally.
- The administration of erythropoietin (pharmacological doses), sometimes in combination with iron (e.g. in chronic renal failure), can lead to improvement.
- It is possible to give a transfusion of erythrocyte mass in severe anemia.
- A combination of erythropoietin and anti-hepcidin antibody is currently being tested.

Summary video

Differential diagnosis

- Sideropenia

Links

Related articles

- Anemia
- Hepcidin
- Iron

Source

- Author: **doc. MUDr. Martin Vokurka, CSc.** *(Ústav patologické fyziologie 1. LF UK)*

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