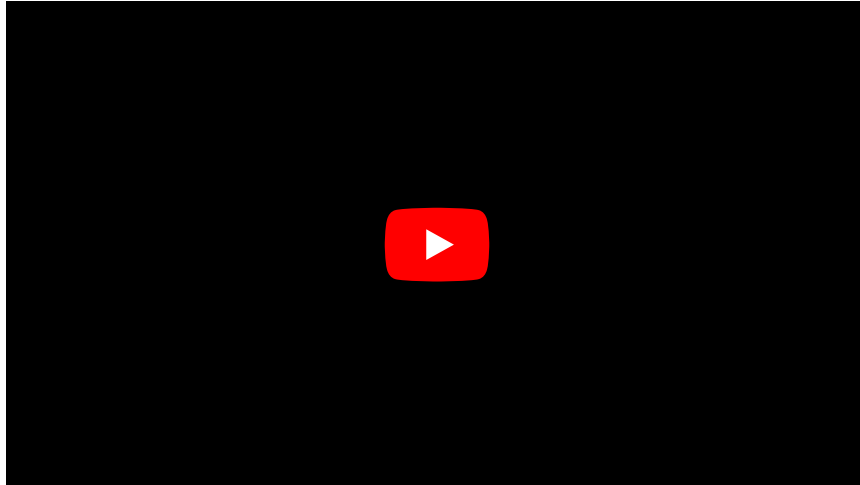


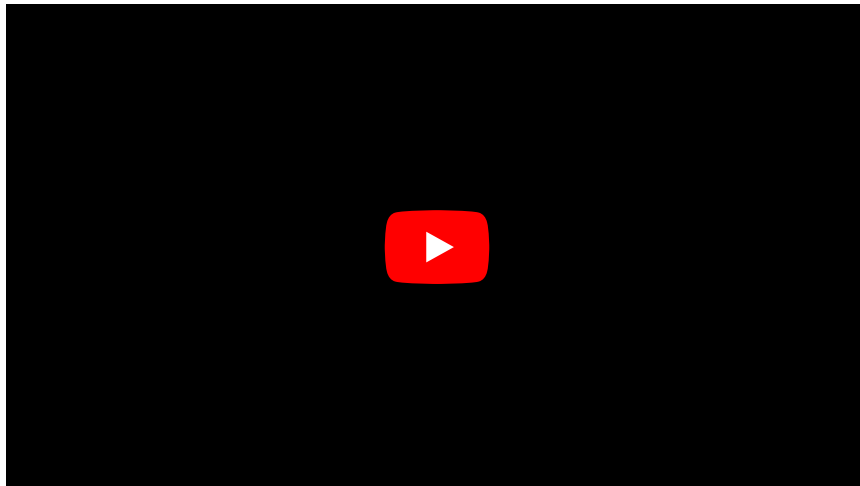
# Anemia of chronic disease

**Anemia of chronic diseases** (sometimes also called inflammatory anemia) is the second most common anemia after iron deficiency anemia (IDA - iron deficiency anemia), from which it is sometimes necessary to distinguish it in differential diagnosis.

## Anemia of chronic disease:



## Anemia of chronic disease 2:



## Basic characteristics

It is usually mild or moderate anemia. It is usually normocytic and normochromic in nature, sometimes it can be microcytic and hypochromic. Therefore, additional parameters need to be established to distinguish it from other anaemias. This anemia accompanies, or is involved 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 renal failure** (here it may supplement the main cause - insufficient production of erythropoietin),
- **tumors** (carcinomas, lymphomas, leukemia, myeloma),
- systemic diseases (RA, SLE, autoimmune vasculitis, scleroderma),
- other micro-inflammatory conditions incl. severe obesity, etc.,
- severe **acute conditions** (infections, postoperative or posttraumatic conditions with catabolism) may induce conditions that have certain features of this anemia.

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

## Patogenesis

Several factors contribute to the emergence. In principle, it is considered **hypoproliferative** anemia, ie. erythrocyte production is reduced, but erythrocyte survival is also slightly shortened. The main factors are:

- **shortened erythrocyte survival** due to activation of the macrophage system,
- reduced erythropoietin production
- decreased sensitivity of erythroid precursors to erythropoietin,
- relative (functional) iron deficiency for erythropoiesis (cytokines stimulate the production of ferritin and hepcidin, leading to inhibition of iron release from cells of the monocytemacrophage system into the extracellular space and a decrease in enteral absorption of Fe).

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

## Change in iron metabolism in anemia of chronic diseases

The condition is referred to as functional iron deficiency because iron is little available for erythropoiesis, although its total amount is not significantly reduced, but iron is retained (sequestered) in macrophages.

- decreased **serum iron** levels (sometimes only sideropenic anemia may be misdiagnosed), but iron can be detected in macrophages, eg by staining.

In particular, however, there are no other features of sideropenia,

- on the contrary, the concentration of serum ferritin is increased (it is an acute phase protein),
- on the contrary, the serum transferrin concentration (ie 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, contributes to 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 production contributes to the retention of iron in macrophages. The effect of hepcidin is relatively rapid, serum iron levels may decrease 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 purpose of this process is probably to defend against infection, ie the retention of iron, which is necessary for invading microorganisms. Another effect of hepcidin is to reduce the **resorption of iron** in the duodenum (analogous mechanism), but this is manifested in a much longer time, because the daily resorption is about 1 mg, while the daily turnover of iron is 20-25 mg and in a healthy person there are iron reserves reaching several hundred mg .

## Brief clinical notes: diagnosis and treatment

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

- This anemia should be **considered** in the above-mentioned diseases.
- In addition to the blood count, it is essential to examine the parameters of iron metabolism, and serum iron alone is not enough; on the contrary, it can be misleading.
- In many cases, inflammatory changes in iron metabolism can be combined with iron loss. In this case, for example, low serum iron does not correspond to the serum ferritin concentration.
- If possible, the underlying disease (inflammation or infection) should be treated.
- **Administration of iron** in chronic disease anemia alone does not bring improvement.
- Administration of iron (if present deficient) may not be effective enough orally due to reduced absorption.
- Administration of **erythropoietin** (pharmacological doses), sometimes in combination with iron (eg in chronic renal failure), may lead to improvement.
- It is possible to transfuse the erythrocyte mass in severe anemia.
- The combination of erythropoietin with an anti-hepcidin antibody is currently being tested.

## Summary video



## Diferencial diagnosis

- Sideropenia

## Odkazy

### Related articles

- Anemia
- Heparidin
- Iron

### Source

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### Literature

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