

Disorders of iron metabolism

Iron deficiency (sideropenia)

Lack of iron in the body is usually caused by insufficient absorption from the intestine or chronic blood loss. It can result in **sideropenic anemia (hypochromic microcytic anemia)**, which is among the **most common hematological diseases**'. However, anemia is usually a late symptom of gradually developing sideropenia. It will show up in the blood count only after the iron has almost completely disappeared. Therefore, it is necessary to detect iron deficiency at an early stage, which is not yet accompanied by anemia.

Based on the determination of the basic parameters of iron metabolism, we distinguish three degrees of deficiency:

- **Prelatent iron deficiency** is the term for a condition where there is a gradual decrease in stores, but the delivery of iron to the erythroblasts bone marrow is not yet affected. About half of the patients have serum ferritin levels below 12 µg/l.
- With '*latent iron deficiency*', its reserves are basically exhausted. Ferritin is reduced below the lower limit of the norm and is already accompanied at this stage by a decrease in the level of iron in the serum and a reduced supply to the erythroblasts of the bone marrow. The binding capacity for iron increases. A sensitive indicator of latent iron deficiency is a drop in transferrin saturation below 15%. However, anemia does not yet develop.
- With '*manifest iron deficiency*', anemia develops with a decrease in hemoglobin values below the lower limit of the norm. Iron deficiency anemia is characterized by low serum iron and ferritin, and an increased concentration of transferrin (binding capacity for iron). In hemolytic anemias or iron overload, on the other hand, serum iron is increased, while the total binding capacity for iron is reduced.

| Prelatent iron deficiency | Latent iron deficiency | Manifest iron deficiency |
|--|--|--|
| decrease in iron stores - decrease in ferritin | lack of iron stores - decrease in ferritin | lack of iron stores - decrease in ferritin |
| | decrease in serum iron | decrease in serum iron |
| | drop in transferrin saturation below 15% | drop in transferrin below 10% |
| | increasing the total binding capacity for iron | increasing the total binding capacity for iron |
| | increase in sTfR | increase in sTfR |
| | | decrease in hemoglobin concentration - anemia |

Laboratory finding in iron deficiency

Excess iron

The organism is not equipped with an excretory pathway for iron, and therefore, under certain circumstances, excess iron can accumulate in the tissues. Early diagnosis can prevent tissue damage from excess iron. Iron overload usually develops very slowly. We distinguish 3 stages:

- In the stage of *prelatent iron excess*, its content in the organs increases, but without exceeding their storage capacity.
- In the period of the *latent stage of iron overload*, *the storage capacity of the cells is exceeded, but the function of the organs is not yet damaged, the level of ferritin and the level of iron in the serum increase and the saturation of transferrin rises above 55%*.
- In the stage of *manifest iron excess*, some organs are already damaged.

| Prelatent iron overload | Latent Iron Excess | Manifest iron excess |
|---|---|---|
| increase in iron stores - increase in <u>ferritin</u> | increase in iron stores - increase in ferritin above 300 µg/l | increase in iron stores - increase in ferritin (in case of severe impairment above 2000 µg/l) |
| | increase in serum iron | significant increase in serum iron |
| | increase in transferrin saturation above 55% | increase in transferrin saturation (can exceed 90% in severe disease) |

Laboratory finding in excess of iron

Hemochromatosis'

Accumulation of iron in tissues is related to a disease we refer to as hemochromatosis.

- **Primary hemochromatosis** is a hereditary disease caused by increased absorption of iron from the intestine.

Excess iron is stored in parenchymatous organs such as the liver, heart, pancreas, and adrenal glands. It has a toxic effect on affected organs and disrupts their function by catalyzing chronic reactions leading to the formation of free radicals. The main clinical manifestations are hyperpigmentation of the skin, [hepatosplenomegaly](#) and [\[\[diabetes mellitus\]](#).

- **Secondary hemochromatosis** can develop as a result of, for example, repeated transfusions, excessive intake of iron-containing preparations or hemolytic anemia. In the biochemical picture, we find increasing levels of ferritin and iron in the serum, the saturation of transferrin increases while it simultaneously decreases.

Iron poisoning

Iron poisoning

Links

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Template:Pathobiochemistry of metabolic pathways (Masopust)

Taken from

- SHROVETIDE, Jaroslav – PRUSHA, Richard. *Pathobiochemistry of metabolic pathways*. 2. edition. Charles University, 2004. 208 pp. pp. 119–120. ISBN 80-238-4589-6.

References

- [wikipedia: Human iron metabolism](#)
- [wikipedia: Heparin](#)
- [wikipedia: Ferroportin](#)
- [wikipedia: Iron overload](#)

References