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Deficiency Anemia in Children. Etiopathogenesis, Clinical Course of Hemopoietic Factor Deficiency According to Severity Levels

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Abstract: *In this article Deficiency anemia in children and Etiopathogenesis, clinical course of hemopoietic factor deficiency according to severity levels is described in detail.*

Keywords: *anemia, children, hematopoiesis, a general blood test, pregnancy, laboratory and etc.*

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Often, tests reveal signs of iron deficiency anemia in children. The reason for the wide spread of this pathology is that the mechanism of hematopoiesis in children has not yet been fixed, and even seemingly insignificant factors can affect it. Another cause of anemia in children is rapid growth, which requires large amounts of nutrients. Iron deficiency anemia is not harmful. Children suffering from this pathology have poor appetite and weak immunity, which often leads to diseases. Such children are not active, they gradually gain weight. In addition, with iron deficiency anemia, children often feel restless and tearful. The main way to detect anemia in children is laboratory tests, in particular, a general blood test. The presence of iron deficiency anemia can be indicated by indicators such as low hemoglobin (the lower limit depends on the child's age), a small number of red blood cells and a color index of 0.85 or below. Anemia in newborns can occur due to problems that occur during pregnancy - bleeding, placental abruption, endangerment of the fetus, infectious diseases in the mother. The period from 28 to 32 weeks is especially dangerous. Anemia in the expectant mother is also an important risk factor. Often, anemia is diagnosed in children born prematurely, as well as with multiple pregnancies. Improper, unbalanced or monotonous diet at any age is a common cause of anemia. With nutritional errors, the body does not receive enough iron and vitamins necessary for its absorption - primarily vitamins C and B12. Anemia is often caused by regular bleeding. Allergy, exudative diathesis and neurodermatitis can sometimes cause iron deficiency anemia. Infectious diseases (tuberculosis, pyelonephritis, etc.), helminthic infections and fungal infections lead to absorption of iron - the child can get enough of it, but the trace element is simply not absorbed. One of the most common diseases in pediatrics is anemia. Anemia occurs in 40% of cases in children under 3 years of age and 30% in adulthood. Often, the occurrence of anemia is associated with the rapid growth of the child's body, the formation, development and maturation of blood cells in children. First, with anemia, it is necessary to organize the correct daily routine and proper nutrition of the child, as well as drug therapy and general strengthening

measures. Children are recommended to spend enough time in the fresh air, additional sleep; gymnastics is assigned. Breastfed anemic children should be fed on time. It is also necessary to adjust the diet of a nursing woman, to add iron and multivitamin preparations. Infants receiving formula feeding are prescribed iron-enriched customized infant formula. The diet of older children should include liver, beef, legumes, herbs, seafood, fruit and vegetable juices. Anemia - or a low level of hemoglobin in the blood - is not an independent disease, but a frequent symptom. About 2 billion people worldwide suffer from anemia. In addition, the symptom is often diagnosed in children. According to the WHO, anemia occurs to some degree or another in 47.4% of preschool children and 25.4% of school-aged children. Hemoglobin is a very important iron-containing protein whose function is to carry oxygen to the organs and tissues of the body. With a lack of hemoglobin, the whole body is starved of oxygen. Therefore, the symptoms of anemia in children are very different. The normal level of hemoglobin in children is different from that of adults. The hemoglobin norm in newborns is 200–240 g/l, in children six months old - 115–175 g/l, from six months to 5 years old - 110–140 g/l, from 5 to 12 years old - 110-145 g / l, 12 from to 15 years old - 115-150 g/l.

Next to the disease cause, disease severity and patient's age influence treatment choices. In symptomatic acquired AA, supportive treatment with erythrocyte and platelet transfusions and infection prevention is provided. For severe cases, the first-line treatment is allo-HCT from a matched sibling donor in young patients and IST in older patients without a well-matched donor. Inherited BMF are usually not responsive to IST, and besides supportive therapy, allo-HCT is the mainstay of the treatment. The high overall response rate of about 70–80% observed in patients with acquired AA treated with IST suggests that indeed in most cases the primary mechanism inducing BM hypoplasia is of auto-immune nature (e.g., cytotoxic T cells triggering apoptosis in BM cells). Alternative mechanisms include exposure to radiation or toxic agents such as pesticides or benzol, treatment with antineoplastic drugs, antibiotics, non-steroidal anti-inflammatory drugs, as well as active infections (e.g., with viruses such as Epstein Barr, hepatitis virus, human immunodeficiency virus, and parvovirus). Rarely, AA is associated with lymphoproliferative neoplasms. In these cases, common denominators like particular (immuno-) genetic background or exposure to viruses and toxic environmental factors may in fact increase the risk for both diseases. On the other hand, treatment of lymphoproliferative processes may trigger auto-immunity. Possibly in a HLA-DR restricted manner, AA can co-occur as a “collateral damage” of an auto-immune process directed against the malignant lymphoid clone.

The cause of acquired AA was not clear for many years. While initially toxic effects were postulated as the reason of a quantitative HSC defect, nowadays autoimmune processes are considered mainly responsible for acquired AA occurring in the absence of a positive medical history of predisposing drugs, toxic agents or infections. In fact, while several pathomechanisms have been proposed, the greatest proportion of cases is likely due to uniform T-cell mediated auto-immunity and marrow destruction leading to defective, nearly absent hematopoiesis. Consistently, activated T lymphocytes were observed to induce apoptosis in HSCs and oligoclonal expansion of dysregulated CD8+ T-cell populations demonstrated in ex vivo BM models of AA patients. Furthermore, increases in T-helper 17 (Th17) cells, the effector cells which produce the pro-inflammatory cytokine interleukin-17 (IL-17), were found in peripheral (PB) and BM of AA patients. Disease activity associated positively with enhanced numbers of Th17 and interferon (IFN)-γ-producing cells, and negatively with regulatory T cells (Treg) populations known to suppress auto-reactivity of other T-cell populations to normal tissue including the BM environment and HSCs. Indeed, especially Tregs from the BM of patients with AA were found to show pronounced quantitative as well as qualitative defects. A parent who notices signs of anemia in a child should first of all do blood tests. The diagnosis is made by the indicators of the number of erythrocytes and hemoglobin in the blood. Anemia is diagnosed when the amount of hemoglobin is

less than 110 g/l and erythrocytes are less than 3.5 x 10^{12} g/l. This indicator refers to the mild level of anemia. Moderate anemia is determined by the following indicators: hemoglobin is less than 90 g/l, erythrocytes up to 2.5x10^{12} g/l, and severe anemia is less than 70 g/l, erythrocytes are less than 2.5x10^{12} g/l. When anemia is detected in a child, there are cases of referral not only to hematologists, but also to gastroenterologists, nephrologists and pediatricians. The stages of treatment of anemia should be carried out under the full supervision of a doctor. If the child is mildly anemic, iron-rich foods and additional vitamins are given. It is necessary not to stop this process in order to avoid recurrent anemia. The diet should include beef liver, legumes, seafood, fresh vegetables and fruits, greens. The doctor prescribes iron supplements and vitamins.

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