

PATHOGENETIC FEATURES OF THE COURSE OF ANEMIA IN JUVENILE RHEUMATOID ARTHRITIS

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Relevance of the problem

Anemia is one of the most common extra-articular manifestations in patients suffering from juvenile rheumatoid arthritis (JRA), with a reported prevalence ranging from 15 to 60%. It is well known that anemia in JRA is associated with higher disease activity, worse outcome parameters, and increased mortality [3]. The opinion of most researchers agrees that anemia of chronic disease (ACD) most often prevails in patients with JRA, with the combination of ACD and iron deficiency anemia (IDA) in second place, with the overall prevalence of anemia ranging from 30 to 70% [4]. In the absence of inflammation, serum ferritin, as an indicator of total body iron stores, is the most useful parameter for differentiating ACD from IDA [1,2,5]. Despite the huge amount of research in this direction, today the question of the pathogenesis and course of anemia in patients with juvenile rheumatoid arthritis remains open.

Purpose of the study. To study the pathogenetic features of the clinical course of anemia in patients with juvenile rheumatoid arthritis

Material and methods. 129 patients with JRA aged 3 to 18 years were examined, of which 119 (92.2%) had the articular form and 10 (7.8%) had the articular-visceral form of the disease. The duration of the disease ranged from 3 months to 8 years. The duration of the disease ranged from 3 months to 8 years. All patients were divided into two groups: a main group of 99 children diagnosed with JRA and with anemic syndrome and a comparison group of 30 children diagnosed with JRA without signs of anemia. In addition to traditional clinical and laboratory studies, a blood test was performed for rheumatoid factor, C - reactive protein, erythropoietin, ferroportin, ferritin, serum iron, hemoglobin equivalent in reticulocytes RET- He, IL 6, radiography of joints. The activity of the JRA was assessed based on a clinical examination, the nature of the articular syndrome and the JADAS 10 index.



Results and discussion. Of the 129 JRA patients examined, 99 (76.7%) were diagnosed with anemia. According to the criteria for diagnosing anemia and its severity based on the hemoglobin concentration, all patients were divided into groups with mild - 63.6% and moderate - 36.4% severity. There were no patients with severe profound anemia with hemoglobin less than 60 g/l. Among the examined patients, 36 were diagnosed with a moderate degree of anemia and 63 with a mild degree of the disease. The number of leukocytes, as a distinctive sign of an active inflammatory process, in sick children was slightly higher than that of the comparison group, in children with mild anemia it was higher - $11.42 \times 10^9 / l$ (with a range of M 14.5 - m 9.9), and in the group with a moderate degree of anemia - $14.01 \times 10^9 / l$ (with a range of M 17.3 - m 11.2) ($P = 0.05$), with the age norm - from 4 to 9 * $10^9 / l$, with a shift The leukoformula in sick children is observed more to the left, as evidenced by the greater detection of band neutrophils relative to the comparison group. Among immature granulocytes, there were such young forms as metamyelocytes and myelocytes. In children with JRA, the number of lymphocytes did not differ significantly from the group of children without anemic syndrome, however, an increase in this indicator took place, but it was not possible to isolate their increase relative to the comparison group, since due to the peculiarities of age norms $24 - 60 \times 10^9 / l$, the indicators are within normal limits values.

Analysis of EPO values in the peripheral blood of patients with JRA showed that, with fluctuations from 12.0 to 25.8 mIU / ml, in none of the patients, both with anemia and without existing signs of anemia, the content of the factor turned out to be not lower than normal values . This can be explained by the normal functioning of the kidneys, which are not affected by ongoing inflammatory processes in the body of a child with JRA. Consequently, patients with JRA generally have a normal level of EPO as their distinguishing feature, which in turn indicates erythropoietin independence of the development of anemia in JRA. However, when studying EPO in relation to the degree of anemia, a decrease in EPO content in the group of patients with moderate anemia can be observed Upon detailed study, it becomes clear that EPO decreases due to the activation of IL6, an inflammatory cytokine that blocks EPO. Thus, it can be argued that there is a cytokine-mediated decrease in the concentration of EPO in the blood serum. And the development of anemia is accompanied by its own etiopathogenesis, which is not typical for known anemias. By studying the indicator of transport-protein function, we discovered patterns that helped reveal the pathogenesis of the development of anemia in children with JRA, as well as what happens to the proteins responsible for the transfer of iron molecules



immediately after absorption. Ferritin as an acute phase indicator increased in patients in groups 1 and 2 to 104.6 ± 39.4 and 212.7 ± 14.8 ng / ml, however, it did not in any way reflect the state of iron depot in the body $p < 0.001$ in all comparison groups and among themselves, i.e. The greater the clinical manifestations of juvenile rheumatoid arthritis, the higher ferritin became. Ferroportin in patients with mild anemia was 0.21 ± 0.04 ng / ml, which indicates its decrease, but with an increase in the degree of anemia, it began to decrease significantly $p < 0.001$ and in the group with moderate anemia it was 0.16 ± 0.01 ng / ml. Based on this, we can conclude that the more ferritin increases, ferroportin decreases in direct proportion to it. Another transport protein, transferrin, had a high degree of confidence in its increase in the blood, which means the body's compensatory work to increase the uptake of iron molecules, so in patients with mild anemia it was 290.3 ± 21.1 mg/ dl, while in the comparison group it was 230.6 ± 15.7 mg/ dl ($p < 0.001$). In patients who developed moderate anemia, transferrin was 315.7 ± 10.4 mg/ dl, a high level of significance ($p < 0.001$) between both study groups.

Conclusions

Thus, thanks to the results of our research, we can come to the main conclusion that the development of anemia in children with JRA is characterized by both qualitative and quantitative changes in red blood cells, and gross violations of the protein transport function for the absorption of iron in the child's body.

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