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## CORRELATIONS BETWEEN HEMOGLOBIN LEVEL AND DISEASE ACTIVITY PARAMETERS IN JUVENILE IDIOPATHIC ARTHRITIS

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Anemia is a frequent extra-articular manifestation of juvenile idiopathic arthritis and may reflect persistent inflammatory activity. This study evaluated associations between hemoglobin level and clinical and laboratory indicators of disease activity in children with juvenile idiopathic arthritis. The study included 80 patients aged 2–18 years and 20 apparently healthy children. Patients with juvenile idiopathic arthritis had higher leukocyte, platelet, erythrocyte sedimentation rate, and C-reactive protein values and lower hemoglobin levels than the control group. Hemoglobin showed statistically significant negative correlations with erythrocyte sedimentation rate, C-reactive protein, platelet count, leukocyte count, neutrophil count, overall disease activity score, and involvement of several upper limb joints. These findings indicate that decreasing hemoglobin level may serve as an additional marker of inflammatory burden and more aggressive joint involvement in juvenile idiopathic arthritis, particularly in patients with polyarticular and systemic patterns of disease.

**Key words:** pediatric rheumatology, inflammatory anemia, disease activity, erythrocyte sedimentation rate, C-reactive protein, joint involvement.

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## КОРЕЛЯЦІЙНІ ЗВЯ'ЗКИ МІЖ РІВНЕМ ГЕМОГЛОБІНУ ТА ПАРАМЕТРАМИ АКТИВНОСТІ ЗАХВОРЮВАННЯ ПРИ ЮВЕНІЛЬНОМУ ІДІОПАТИЧНОМУ АРТРИТІ

Анемія є частим позасуглобовим проявом ювенільного ідіопатичного артриту та може свідчити про збереження запальної активності. Метою дослідження було оцінити зв'язок рівня гемоглобіну з клінічними та лабораторними показниками активності захворювання у дітей з ювенільним ідіопатичним артритом. До дослідження включено 80 пацієнтів віком від 2 до 18 років і 20 практично здорових дітей. У пацієнтів виявлено вищі показники лейкоцитів, тромбоцитів, швидкості осідання еритроцитів та С-реактивного білка, а також нижчий рівень гемоглобіну порівняно з контрольною групою. Рівень гемоглобіну мав статистично значущі негативні кореляції з маркерами запалення, загальною активністю захворювання та ураженням низки суглобів верхніх кінцівок. Отримані дані дозволяють розглядати зниження гемоглобіну як додатковий показник запального навантаження та більш агресивного ураження суглобів у дітей із цією патологією.

**Ключові слова:** дитяча ревматологія, запальна анемія, активність захворювання, швидкість осідання еритроцитів, С-реактивний білок, ураження суглобів.

Over the past decades, the contribution of musculoskeletal and connective tissue diseases to pediatric morbidity has remained substantial. Chronic rheumatic diseases in childhood are clinically important not only because of inflammation and pain, but also because they may impair growth, physical activity, social participation, education, and long-term quality of life. Juvenile idiopathic arthritis (JIA) is the most common chronic inflammatory rheumatic disease of childhood and remains a heterogeneous condition requiring a multidisciplinary diagnostic and therapeutic approach [12, 13, 15].

According to internationally accepted criteria, JIA is defined as arthritis of unknown etiology lasting more than 6 weeks, with onset before the age of 16 years, after exclusion of other causes of joint disease. The clinical spectrum includes several categories with different patterns of joint involvement, systemic manifestations, prognosis, and response to therapy [9, 11]. Destructive joint changes, persistent pain, and limitation of motion may develop in patients with insufficiently controlled inflammation, which explains the importance of objective assessment of disease activity at the earliest possible stage.

Laboratory evaluation of children with JIA includes complete blood count, acute-phase reactants, biochemical and immunological parameters, and instrumental assessment when clinically indicated. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are widely used markers of inflammation, while disease activity indices integrate clinical findings with laboratory results. Although the Juvenile Arthritis Disease Activity Score is frequently used in pediatric rheumatology, the DAS28 index may also be applied in clinical studies to characterize inflammatory burden and joint involvement [1, 14, 17].

Extra-articular manifestations are an important component of JIA. Anemia is among the most common systemic complications and may aggravate fatigue, decrease exercise tolerance, and worsen the overall clinical picture. In patients with chronic inflammatory diseases, anemia is often related to immune-mediated disturbances of iron metabolism, increased hepcidin activity, functional iron deficiency, suppression of erythropoiesis, and shortened erythrocyte survival [6, 10, 18]. In this context, hemoglobin concentration may reflect not only hematological status but also the intensity of inflammation.

The aim of our study was to evaluate the relationships between hemoglobin levels and parameters reflecting the activity of the pathological process in patients with JIA.

**Materials and methods.** The study was conducted at the Department II of Children's Diseases of the Educational-Therapeutic Clinic of the Azerbaijan Medical University from April 2017 to December 2019. A total of 80 children with JIA aged 2–18 years were examined. The control group consisted of 20 apparently healthy children without clinical or laboratory signs of inflammatory joint disease.

The study was performed in accordance with the principles of the Declaration of Helsinki as revised in 2013, the International Ethical Guidelines for Health-related Research Involving Humans of the Council for International Organizations of Medical Sciences (2016), and Good Clinical Practice principles. As the study included minors, written informed consent was obtained from parents or legal representatives before inclusion. Assent from children was obtained when appropriate for age and understanding.

Of the 80 patients with JIA, 39 were boys (48.8 %) and 41 were girls (51.2 %). The control group included 5 boys (25.0 %) and 15 girls (75.0 %). Among patients with JIA, 3 children (3.75 %) were aged 2–3 years, 17 (21.25 %) were aged 4–6 years, 32 (40.0 %) were aged 7–12 years, and 28 (35.0 %) were aged 13–18 years. In the control group, 6 children (30.0 %) were aged 4–6 years, 5 (25.0 %) were aged 7–12 years, and 9 (45.0 %) were aged 13–18 years. The mean age of patients with JIA was 9.6±0.5 years; the mean age of children in the control group was 9.1±0.8 years.

All children underwent a series of clinical, laboratory, and instrumental examinations, including joint assessment, complete blood count, biochemical blood tests, immunological parameters, and radiography of affected joints when clinically indicated. Joint status was evaluated by the presence of tenderness, swelling, limited motion, and arthritis in individual joints.

The degree of disease activity was determined using the DAS28 (Disease Activity Score 28) scale, which includes the number of tender joints, the number of swollen joints, ESR, and the patient's or parent's overall assessment of health using a visual analogue scale. A DAS28 value of <2.6 was interpreted as inactive disease, 2.6–3.1 as low activity, 3.2–5.1 as moderate activity, and >5.2 as high activity.

Quantitative and qualitative data were analyzed using SPSS 26.0. Variation analysis included calculation of the arithmetic mean (M), median (Me), first quartile (Q1), and third quartile (Q3). Between-group differences were assessed using the Mann–Whitney U test. Correlation relationships were determined using Spearman's rank correlation coefficient (ρ). Statistical significance was assessed using a two-sided criterion. The null hypothesis was rejected at $p<0.050$.

**Results of the study.** The comparative analysis demonstrated that children with JIA differed from apparently healthy children not only by clinical manifestations of joint disease, but also by a distinct laboratory profile reflecting active systemic inflammation. The most pronounced between-group differences were observed for acute-phase parameters and complete blood count indicators associated with inflammatory activity. The results of the variation analysis are presented in Table 1.

Table 1

**Parameters of examined children with JIA in comparison with the control group**

| Parameter | Control | | | | JIA | | | | pU |
|---|---|---|---|---|---|---|---|---|---|
| | M | Me | Q1 | Q3 | M | Me | Q1 | Q3 | |
| WBC, $10^9$/l | 6.4 | 5.9 | 5.6 | 7.4 | 12.2 | 11.0 | 7.7 | 15.0 | <0.001* |
| RBC, $10^{12}$/l | 4.7 | 4.7 | 4.6 | 4.8 | 4.4 | 4.4 | 4.1 | 4.8 | 0.004* |
| NEUT, $10^9$/l | 5.2 | 5.0 | 4.5 | 6.4 | 7.5 | 6.4 | 4.3 | 9.2 | 0.079 |
| HGB, g/dl | 12.6 | 12.5 | 12.2 | 12.9 | 10.8 | 11.2 | 9.5 | 12.0 | <0.001* |
| PLT, $10^9$/l | 258.3 | 255.0 | 220.0 | 310.0 | 496.0 | 463.5 | 362.5 | 579.0 | <0.001* |
| ESR, mm/hr | 8.9 | 10.5 | 4.5 | 12.0 | 44.3 | 35.0 | 18.0 | 65.0 | <0.001* |
| CRP, mg/l | 3.5 | 3.8 | 2.7 | 4.3 | 43.7 | 26.6 | 12.0 | 60.5 | <0.001* |
| Fe, µg/dl | 36.4 | 35.0 | 22.1 | 39.0 | 32.7 | 29.5 | 21.1 | 39.1 | 0.484 |
| Ferritin, ng/ml | 85.2 | 30.3 | 6.5 | 218.7 | 474.3 | 190.1 | 102.3 | 846.4 | 0.480 |

Notes: M – arithmetic mean, Me – median, Q1 and Q3 – the first and third quartiles of the variation series; pU – statistical significance of the difference according to the Mann–Whitney U criterion; * – statistically significant difference.

In patients with JIA, leukocyte count was almost twice as high as in the control group. The mean leukocyte value was $12.2\times10^9$/l in the JIA group compared with $6.4\times10^9$/l in controls, and the difference was statistically significant ($p<0.001$). The median values showed the same direction of change: $11.0\times10^9$/l in patients versus $5.9\times10^9$/l in controls. This pattern confirms the presence of a systemic inflammatory response in the examined cohort and supports the clinical relevance of complete blood count parameters in assessing disease activity.

The erythrocyte count was lower in children with JIA than in the control group. The mean red blood cell count was $4.4\times10^{12}$/l in patients and $4.7\times10^{12}$/l in controls ($p=0.004$). Although the absolute difference was moderate, it was consistent with the decrease in hemoglobin concentration and suggests that chronic inflammation in JIA may affect several components of erythropoiesis. The reduction in red blood cell parameters should be interpreted together with iron metabolism indicators and acute-phase reactants, because anemia in inflammatory diseases may develop even when iron stores are not truly depleted.

Neutrophil count was higher in patients with JIA than in controls, with mean values of $7.5\times10^9$/l and $5.2\times10^9$/l, respectively. However, the between-group difference did not reach statistical significance ($p=0.079$). This finding indicates a tendency toward neutrophil predominance in the inflammatory process, but also demonstrates variability within the JIA group. Such variability may be related to differences in disease subtype, duration, treatment status, and individual inflammatory response.

Hemoglobin concentration was significantly lower in patients with JIA than in the control group. The mean hemoglobin level was 10.8 g/dl in the patient group and 12.6 g/dl in controls ($p<0.001$). The median hemoglobin value was 11.2 g/dl in patients and 12.5 g/dl in the control group. These data confirm that anemia is a clinically relevant component of the disease profile in the examined children. Because the study group had simultaneously increased inflammatory markers, the decrease in hemoglobin is most consistent with anemia of inflammation or anemia of chronic disease rather than isolated nutritional iron deficiency.

Platelet count was markedly elevated in children with JIA. The mean platelet value reached $496.0\times10^9$/l, whereas in the control group it was $258.3\times10^9$/l ($p<0.001$). The median platelet count also differed substantially: $463.5\times10^9$/l in patients and $255.0\times10^9$/l in controls. Thrombocytosis is a recognized laboratory sign of active inflammation and may be associated with cytokine-driven stimulation of megakaryopoiesis. The simultaneous presence of thrombocytosis and decreased hemoglobin strengthens the assumption that anemia in the examined patients is linked to inflammatory activity.

ESR and CRP showed the most evident inflammatory differences between groups. The mean ESR was 44.3 mm/hr in the JIA group and 8.9 mm/hr in the control group ($p<0.001$). The mean CRP level was 43.7 mg/l in patients and 3.5 mg/l in controls ($p<0.001$). The wide interquartile ranges in the JIA group reflect heterogeneity of inflammatory activity, but the overall direction of change was clear and statistically robust. These findings are consistent with the known role of ESR and CRP as markers of disease activity in inflammatory arthritis [1, 17].

Serum iron was slightly lower in patients with JIA than in controls, but the difference was not statistically significant (32.7 µg/dl versus 36.4 µg/dl, $p=0.484$). Ferritin values were higher in the JIA group, with a mean of 474.3 ng/ml compared with 85.2 ng/ml in controls; however, this difference also did not reach statistical significance ($p=0.480$), most likely due to high variability. The combination of relatively low serum iron, increased ferritin, and active inflammation corresponds to the typical pattern of functional iron deficiency in anemia of chronic disease. In this condition, iron may be retained in macrophage stores and become unavailable for effective erythropoiesis despite adequate or elevated ferritin levels [6, 9, 18].

The mean DAS28 value in patients with JIA was $4.55\pm0.10$, indicating predominantly moderate disease activity in the cohort. This result is important because it provides an integrated clinical and laboratory measure against which hemoglobin level can be interpreted. The subsequent correlation analysis demonstrated that hemoglobin was not an isolated hematological parameter; rather, it was significantly related to several indicators of inflammation and disease burden.

Hemoglobin level showed statistically significant negative correlations with leukocyte count ($\rho=-0.298$, $p=0.008$), neutrophil count ($\rho=-0.295$, $p=0.009$), platelet count ($\rho=-0.441$, $p<0.001$), CRP ($\rho=-0.387$, $p=0.001$), ESR ($\rho=-0.505$, $p<0.001$), and DAS28 ($\rho=-0.471$, $p<0.001$). The strongest correlation was observed between hemoglobin and ESR, suggesting that the severity of anemia increases as systemic inflammatory activity becomes more pronounced. The negative correlation with DAS28 indicates that lower hemoglobin values are associated with higher overall disease activity.

The graphical analysis confirmed these relationships. Scatter plots demonstrated a downward trend between hemoglobin and ESR, hemoglobin and CRP, and hemoglobin and DAS28. Although individual values varied, the general distribution of points supported the statistical findings and showed that patients with lower hemoglobin more frequently had higher acute-phase reactants and higher disease activity scores. This strengthens the interpretation of hemoglobin as an additional indicator of inflammatory burden.

Joint-level analysis revealed that lower hemoglobin values were associated with lesions of several upper limb joints. Negative correlations were found with hand tenderness (ρ=-0.363, p=0.001), hand swelling (ρ=-0.234, p=0.038), wrist tenderness (ρ=-0.302, p=0.007), elbow tenderness (ρ=-0.233, p=0.039), elbow swelling (ρ=-0.234, p=0.038), shoulder swelling (ρ=-0.285, p=0.011), and shoulder arthritis (ρ=-0.238, p=0.034). These associations are clinically meaningful because involvement of multiple upper limb joints is more often observed in polyarticular and systemic patterns of JIA, which are usually characterized by more aggressive inflammation and a higher systemic burden.

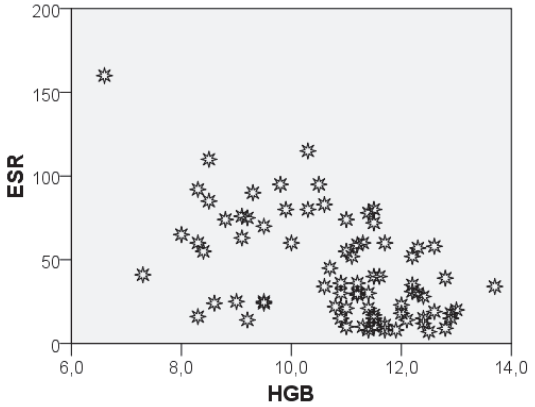


Fig. 1. The results of the correlation analysis. A – Hemoglobin and ESR; B – Hemoglobin and CRP; C – Hemoglobin and DAS28.

Thus, the results indicate that anemia in children with JIA is closely related to the severity of the inflammatory process. The decrease in hemoglobin level was accompanied by higher platelet count, ESR, CRP, and DAS28 values, as well as by more extensive joint involvement. These findings support the inclusion of hemoglobin assessment in the routine interpretation of disease activity, especially when evaluating children with suspected anemia of chronic disease and active polyarticular involvement.

**Discussion.** The obtained results support the concept that anemia in JIA is closely linked to the inflammatory process. High disease activity is accompanied by increased production of pro-inflammatory cytokines, especially interleukin-6, which stimulates hepatic hepcidin synthesis. Hepcidin inhibits iron export through ferroportin degradation, reduces intestinal iron absorption, and promotes retention of iron in macrophages. As a result, iron becomes less available for erythropoiesis even when total body iron stores are sufficient or increased [9, 18].

The laboratory profile observed in the present study corresponds to this mechanism. Children with JIA had lower hemoglobin values, increased platelet count, high ESR and CRP levels, and higher ferritin values. Although ferritin did not differ significantly due to variability, its elevation in many patients is biologically plausible because ferritin behaves as an acute-phase protein and may increase in inflammatory and autoinflammatory conditions. This explains why interpretation of iron metabolism in JIA should not rely on serum iron or ferritin alone [9, 10, 18].

The negative correlation between hemoglobin and ESR was the strongest among the analyzed associations. This finding is clinically important because ESR is influenced by both inflammation and blood composition. A decrease in hemoglobin may therefore reflect the combined effect of systemic inflammatory activity and altered erythrocyte parameters. The significant correlations with CRP and platelet count provide additional evidence that reduced hemoglobin is associated with inflammatory activation rather than being an isolated hematological abnormality [1, 17].

The association between low hemoglobin level and upper limb joint involvement suggests that anemia may be more pronounced in patients with more extensive articular disease. Hand, wrist, elbow, and shoulder involvement often indicates polyarticular distribution and greater functional burden. These patients may have persistent inflammatory stimulation and higher cytokine activity, which can intensify disturbances of iron metabolism and erythropoiesis. Therefore, hemoglobin level may be useful as an additional parameter when evaluating the overall severity of disease [7, 16].

The present study has several limitations. The sample size was moderate, and the cohort was heterogeneous in age and clinical presentation. The study was cross-sectional in its main analytical design, which limits conclusions about causality. In addition, specific markers of iron metabolism such as

transferrin saturation, soluble transferrin receptor, and hepcidin were not included in the present analysis. Nevertheless, the observed correlations provide clinically relevant evidence that hemoglobin should be interpreted in connection with disease activity parameters in children with JIA.

## Conclusion

The study demonstrated that children with juvenile idiopathic arthritis had significantly lower hemoglobin levels and higher inflammatory parameters compared with apparently healthy children. Hemoglobin concentration was negatively correlated with leukocyte count, neutrophil count, platelet count, erythrocyte sedimentation rate, C-reactive protein, and the disease activity score. The strongest relationship was observed with erythrocyte sedimentation rate, indicating that anemia becomes more pronounced as systemic inflammatory activity increases. Negative correlations between hemoglobin level and lesions of hand, wrist, elbow, and shoulder joints suggest that anemia is more severe in patients with more extensive joint involvement, particularly in polyarticular and systemic patterns of disease. These findings confirm that hemoglobin is not only a hematological indicator, but also a clinically meaningful marker associated with inflammatory burden and disease severity in juvenile idiopathic arthritis.

*Prospects for further research. Further studies should include longitudinal follow-up and assessment of hepcidin, transferrin saturation, soluble transferrin receptor, and treatment response to clarify the mechanisms and prognostic significance of anemia in juvenile idiopathic arthritis.*

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