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CORRELATION ANALYSIS OF IMMUNOLOGICAL MARKERS IN CHILDREN WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract
The study included 97 children aged 7–17 years with a confirmed diagnosis of systemic lupus erythematosus. A correlation analysis of immunological markers was performed, including CRP, lactoferrin, IFNγ, IP-10, C3, C4, and $CD16^+$ cells. Strong significant negative correlations were found between CRP and lactoferrin ($r = –0.83$; $p < 0.001$), CRP and IFNγ ($r = –0.79$; $p < 0.001$), as well as a positive correlation between CRP and $CD16^+$ cells ($r = 0.82$; $p < 0.001$). Lactoferrin was positively correlated with IFNγ ($r = 0.80$; $p < 0.001$) and IP-10 ($r = 0.76$; $p < 0.001$), indicating a coupled activation of the innate immune response. Moderate correlation was established between complement components C3 and C4 ($r = 0.58$; $p < 0.01$), and weak associations between IP-10 and C4 ($r = –0.44$; $p < 0.05$) indicate a complex regulation of inflammation. The results emphasize the importance of CRP, lactoferrin, IFNγ, IP-10 and $CD16^+$ markers in the pathogenesis of SLE in children and can be used to assess disease activity.

Keywords: Systemic lupus erythematosus; children; immunological markers; C-reactive protein; interferon-gamma; IP-10; lactoferrin; $CD16^+$; complement; correlation analysis.

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Immunological markers play a key role in the pathogenesis of systemic lupus erythematosus (SLE), especially in pediatric practice, where the clinical picture may differ in severity and rate of progression [2, 5, 7]. Understanding the interaction between the components of innate and adaptive immunity allows for a more thorough assessment of disease activity, prognosis of its course, and optimization of a personalized approach to therapy [1, 3, 9].

Correlation analysis methods are an important tool in identifying relationships between laboratory parameters reflecting both the inflammatory response and immune system activity. In a pediatric cohort of patients with SLE, it is especially important to track relationships between cytokines, complement components, and cellular markers, since pediatric immunity is in the process of formation and may react in an atypical manner [4, 8, 11].

Despite the accumulated data on the role of individual immunological markers, a comprehensive statistical approach to assessing their interaction in the pediatric population with SLE remains insufficiently studied. This necessitates a multi-level analysis including both humoral and cellular components of the immune response [6, 10, 12].

Purpose of the Study:

To conduct a correlation analysis of immunological markers in children with systemic lupus erythematosus (SLE) to identify relationships between the parameters of innate and adaptive immunity and clinical and laboratory manifestations of the disease.

Materials and Methods:

The study included 97 children aged 7 to 17 years with a verified diagnosis of systemic lupus erythematosus, who were observed in the hospital. All patients underwent a comprehensive clinical and laboratory examination, including assessment of the levels of C-reactive protein (CRP), lactoferrin, complement components C3 and C4, $CD16^+$ cell expression, interferon-gamma (IFNγ) and chemokine IP-10 levels. Determination of the level of CRP, lactoferrin, C3 and

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C4, IFNγ, IP-10 was carried out by ELISA using Vector-Best reagents (Novosibirsk, Russian Federation).

Statistical processing included methods of descriptive statistics and correlation analysis (Pearson coefficient), the level of statistical significance was $p < 0.05$.

Research Results:

In this study, a correlation analysis of immunological parameters in children with a verified diagnosis of systemic lupus erythematosus was performed. The analysis was performed in the overall sample without dividing into subgroups of disease activity, which made it possible to identify universal patterns of interaction between markers. The main statistical tool was the Pearson coefficient (r), with the interpretation of the strength of the relationship according to the following criteria: strong ($|r| > 0.7$), average ($0.4 \leq |r| < 0.7$), weak ($0.2 \leq |r| < 0.4$). Interpretations of the identified relationships are provided below.

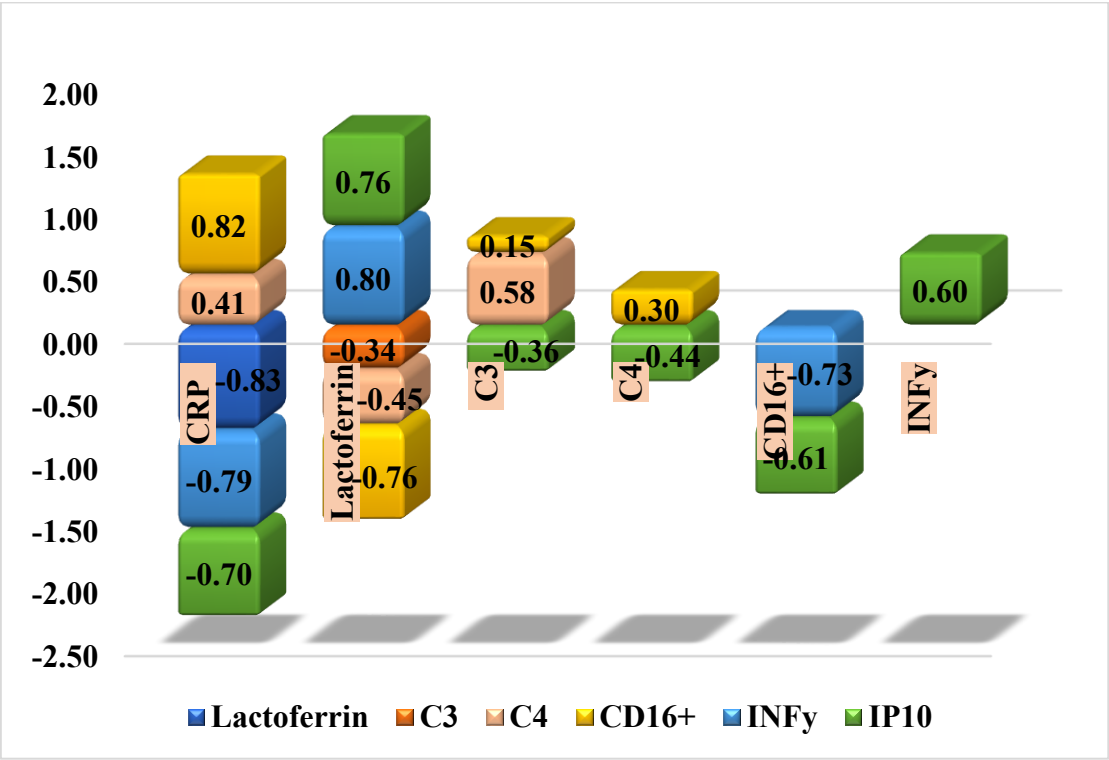

Fig. 1. Correlation relationships in the general sample of children with SLE

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Among the strong correlations, significant negative associations were found between the levels of C-reactive protein (CRP) and lactoferrin (r = –0.83), as well as CRP and INFγ (r = –0.79), which may reflect the multivector regulation of the humoral innate response (CRP is a classic acute phase protein synthesized by hepatocytes under the influence of IL-6) and the production of proinflammatory cytokines, including γ-interferon. Perhaps, a reduced level of INFγ against the background of an increase in CRP reflects the activation of type 1 T-helpers (Th1), while an increase in the acute phase response can reduce the production of cellular cytokines. A similar relationship is observed between $CD16^+$ cells and INFγ (r = –0.73), which may indicate depletion or functional inactivation of NK cells with a dominant Th1 response.

Lactoferrin showed a strong positive correlation with INFγ (r = 0.80) and IP10 (r = 0.76), reflecting the coupled activation of the innate antimicrobial response and the production of proinflammatory cytokines. IP10 (CXCL10) is a chemokine induced by INFγ, and its high correlation with lactoferrin may indicate a common activation axis involving monocytes and epithelial cells [2].

Also, a strong positive correlation is observed between CRP and $CD16^+$ cells (r = 0.819), which may be associated with the active recruitment and activation of effector cells of the innate immune system (NK, monocytes), which is typical for phases of high disease activity.

The moderate correlations include those between C3 and C4 (r = 0.58), which confirms their functional coupling within the classical complement activation pathway. The moderate correlation between CRP and C4 (r = 0.41) may also reflect the relationship between the inflammatory response and complement activation characteristic of SLE exacerbations. Of interest is the relationship between $CD16^+$ and C4 (r = 0.30), as well as $CD16^+$ and C3 (r = 0.15), which may indicate the involvement of complement-dependent cytotoxicity in the pathogenesis of lupus, although the degree of involvement is variable.

Among the weak correlations were: CRP–C3 (r = 0.26), INFγ–C3 (r = –0.12), INFγ–C4 (r = –0.28), which may indicate a limited association of the classical inflammatory axis with complement components in the overall sample. Negative weak associations between IP10 and complement components (IP10–C3: r = –

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0.36; IP10–C4: r = –0.44) may indicate an independent role of chemokines in pathogenesis or reflect compensatory mechanisms with a decrease in complement activity.

Conclusion:

Thus, the obtained data demonstrate that in the cohort of children with SLE, the interactions between CRP, lactoferrin, INFγ and IP10, as well as coupled reactions between cellular markers ($CD16^+$) and humoral inflammatory mediators, are of the greatest significance in the formation of pathophysiological cascades. These data may be useful in developing new diagnostic panels of disease activity and refining the individual prognosis.

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