SYSTEMIC LUPUS ERYTHEMATOSUS AND ITS IMMUNOLOGICAL ASPECTS

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Relevance of the Study.

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by the production of autoantibodies against nuclear and cytoplasmic antigens, leading to widespread tissue and organ damage. In recent years, the incidence of SLE has increased globally, particularly among young women of reproductive age. The variability of clinical manifestations and the complexity of immunopathogenesis make SLE one of the most difficult autoimmune diseases to diagnose and treat. Understanding the immunological mechanisms underlying the disease—especially the role of B and T lymphocyte dysfunction, cytokine imbalance, and complement activation—is crucial for improving diagnostic accuracy and developing targeted therapies.

Purpose of the Study.

The purpose of this study is to analyze the main immunological mechanisms involved in the pathogenesis of systemic lupus erythematosus and to identify the key biomarkers associated with disease activity and progression. The study also aims to assess how these immunological parameters can be used for predicting disease flares and monitoring treatment effectiveness.

Materials and Methods.

The study was based on a comprehensive analysis of clinical and laboratory data from patients diagnosed with SLE according to the ACR/EULAR criteria. Immunological parameters such as antinuclear antibodies (ANA), anti–double-stranded DNA (anti-dsDNA) antibodies, complement components (C3, C4), and cytokine profiles (IL-6, IL-10, TNF-α, IFN-γ) were evaluated. Standard immunoassay techniques, enzyme-linked immunosorbent assay (ELISA), and flow cytometry were used for quantitative and qualitative assessment of immune markers. Statistical analysis was performed to determine correlations between immunological changes and disease activity indices.

Results.

The findings demonstrated that SLE patients exhibited a significant increase in autoantibody titers, especially ANA and anti-dsDNA, accompanied by decreased complement levels. Elevated serum concentrations of pro-inflammatory cytokines (IL-6, TNF-α, IFN-γ) were observed during disease exacerbations, whereas regulatory cytokines (IL-10) were elevated in chronic phases, suggesting their compensatory role. The imbalance between T-helper (Th1/Th2) responses and abnormal B-cell hyperactivity were identified as key mechanisms in sustaining autoimmune inflammation. Correlation analysis confirmed a strong association between cytokine levels, complement consumption, and clinical activity scores (SLEDAI).

Conclusion.

The study confirms that systemic lupus erythematosus is primarily driven by immune dysregulation, characterized by autoantibody production, cytokine imbalance, and complement activation. Immunological markers play a decisive role in diagnosing disease activity, predicting relapses, and assessing treatment response. Early identification of immunological abnormalities allows for the timely implementation of targeted immunomodulatory therapy, which can reduce disease activity, prevent organ damage, and improve patient prognosis. Further research should focus on developing personalized immunotherapeutic strategies based on individual immune profiles.