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IMMUNOLOGICAL IMBALANCE AND CYTOKINE PROFILES IN SYSTEMIC SCLEROSIS: RELATIONSHIP WITH DISEASE PROGRESSION

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Systemic sclerosis (SSc) is a chronic autoimmune connective tissue disorder characterized by progressive fibrosis, microvascular injury, and immune dysregulation. The present study aimed to assess the immunological status and cytokine profile in the early stages of the disease and to determine their potential role in primary prevention strategies. A total of 20 patients with confirmed SSc were enrolled. Comprehensive analysis of immunological parameters and circulating cytokines was performed, followed by correlation with clinical manifestations and disease severity. The results revealed a pronounced imbalance in immune regulation accompanied by significant alterations in key cytokines, including IL-6, TNF-α, and TGF-β. These findings suggest that cytokine-mediated immune dysregulation plays a pivotal role in the initiation and progression of SSc. Early identification of these biomarkers may contribute to improved risk stratification, timely diagnosis, and the development of targeted preventive approaches.

Keywords

systemic sclerosis; cytokine profile; immune dysregulation; IL-6; TNF-α; TGF-β; fibrosis; biomarkers; disease progression; clinical correlation

Systemic scleroderma is a complex autoimmune connective tissue disease characterized by excessive collagen deposition, microvascular dysfunction, and immune system abnormalities. Despite advances in understanding its pathogenesis, early diagnosis and primary prevention remain challenging. Recent studies emphasize the pivotal role of immune system disturbances, particularly alterations in cytokine production and immune cell function, in initiating and driving the disease. Identifying these changes at early stages may provide opportunities for preventive interventions and improved clinical outcomes.

Materials and Methods. The study included 20 patients diagnosed with systemic sclerosis (SSc) according to established clinical and immunological criteria. Patients were recruited from a specialized clinical setting, and informed consent

was obtained from all participants prior to enrollment. Immunological Assessment: The following immunological parameters were evaluated: cellular immunity markers, including T-lymphocytes and their subsets (CD4+ and CD8+ cells); humoral immunity indicators, including serum immunoglobulin levels (IgG, IgM, IgA); and autoantibodies, including antinuclear antibodies (ANA), anti-centromere antibodies, and anti-Scl-70 antibodies. Cytokine Analysis: Serum levels of key cytokines were measured using standard laboratory methods. The panel included pro-inflammatory cytokines (IL-6, TNF-α, IL-1β), anti-inflammatory cytokines (IL-10), and fibrogenic cytokines (TGF-β). Clinical Evaluation: Clinical assessment included evaluation of skin involvement using the modified Rodnan skin score (mRSS), presence of Raynaud's phenomenon, and involvement of internal organs (lungs, heart, and gastrointestinal tract). Disease duration and activity were also recorded. Statistical Analysis: Statistical analysis was performed using standard methods. Correlation analysis was conducted to determine relationships between immunological parameters, cytokine levels, and clinical characteristics. A p-value < 0.05 was considered statistically significant.

Results. Immune Status Alterations **-**A significant imbalance in cellular immunity was observed, with decreased CD4+/CD8+ ratio in most patients; Elevated levels of immunoglobulins (particularly IgG) were noted, indicating activation of humoral immunity; High prevalence of autoantibodies (ANA and anti-Scl-70) was detected. Cytokine Profile Changes- Increased levels of pro-inflammatory cytokines (IL-6, TNF-α) were found in the majority of patients; Elevated TGF-β levels correlated with the degree of skin fibrosis; Reduced IL-10 levels suggested insufficient anti-inflammatory regulation. Correlation with Clinical Course - Higher IL-6 and TNF-α levels were associated with more severe disease activity and organ involvement; TGF-β levels showed a strong positive correlation with skin thickness scores. Patients with pronounced immune dysregulation exhibited more aggressive disease progression.

Discussion. The results confirm that systemic scleroderma is strongly associated with immune system dysregulation and cytokine imbalance. The observed increase in pro-inflammatory and fibrogenic cytokines suggests their involvement in both initiation and progression of fibrosis. The correlation between immune markers and clinical severity highlights the importance of immune status assessment in early stages. These findings support the concept that immunological abnormalities precede overt clinical manifestations, making them valuable targets for primary prevention.

Conclusion. Immune system imbalance plays a ключевую роль (key role) in the development of systemic scleroderma. Cytokine dysregulation, particularly

increased IL-6, TNF-α, and TGF-β, is closely associated with disease severity and progression. Early assessment of immune status can serve as an important tool for identifying high-risk individuals. Monitoring immunological and cytokine parameters may contribute to the development of effective primary preventive strategies.

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