



EVALUATION OF THE ROLE OF ANGIOGENIC AND ANGIOSTATIC FACTORS IN SYSTEMIC SCLEROSIS

Abdakimova Baxtigul Ilhomiddin qizi
Tashkent State Medical University

Introduction:

Systemic sclerosis (SSc) is a chronic autoimmune connective tissue disorder characterized by progressive fibrosis, vascular abnormalities, and immune dysregulation. Endothelial dysfunction and impaired vascular remodeling are central to its pathogenesis. Angiogenesis, regulated by proangiogenic and antiangiogenic factors, plays a critical role in tissue repair and vascular homeostasis. Platelet-derived growth factor (PDGF) promotes vascular proliferation, while thrombospondin-1 (TSP-1) is a potent angiostatic factor. Understanding the balance between these factors can provide insights into vascular pathology in SSc.

Keywords: Systemic sclerosis, angiogenesis, angiostasis, PDGF, TSP-1, biomarkers

Methodology:

A case-control study was conducted involving 80 patients diagnosed with systemic sclerosis (according to the ACR/EULAR criteria) and 20 age- and sex-matched healthy controls. Serum PDGF and TSP-1 levels were measured using enzyme-linked immunosorbent assay (ELISA). Statistical analysis was performed using Student's t-test with $p < 0.05$ considered significant.

Results:

The mean PDGF level in SSc patients was significantly higher compared to the control group ($p < 0.05$), while TSP-1 levels were significantly lower ($p < 0.05$). These findings indicate a shift towards angiogenic dominance in the disease process.



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Discussion:

The elevated PDGF levels may reflect an attempt to stimulate angiogenesis in response to vascular injury, while the reduced TSP-1 suggests a loss of angiostatic regulation. This imbalance may contribute to the abnormal vascular remodeling and fibrosis seen in SSc. Previous studies support the role of angiogenic dysregulation as a driver of disease progression.

Conclusion:

Angiogenic and angiostatic imbalance plays a pivotal role in systemic sclerosis pathogenesis. PDGF and TSP-1 could serve as potential biomarkers for disease severity and therapeutic targets in vascular-related complications.