

THE ROLE OF ANGIOGENIC (PDGF) AND ANGIOSTATIC (TSP-1) VASCULAR FACTORS IN THE DEVELOPMENT OF SYSTEMIC SCLERODERMA

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Abstract

Systemic scleroderma (SSc) is a severe autoimmune disease that leads to progressive fibrosis and vascular abnormalities. This study investigates the role of Platelet-Derived Growth Factor (PDGF) and Thrombospondin-1 (TSP-1) as key angiogenic and angiostatic factors in SSc progression. We examined 100 participants, including 80 patients with SSc and 20 healthy controls. Results show significantly elevated PDGF and TSP-1 levels in SSc patients, correlating with the degree of fibrosis and internal organ involvement.

Keywords

Systemic scleroderma, PDGF, TSP-1, angiogenesis, fibrosis, vascular factors

Introduction

Systemic scleroderma is a progressive autoimmune disease that involves vascular damage, immune activation, and excessive fibrosis. Angiogenic and angiostatic factors such as PDGF and TSP-1 play critical roles in the pathological mechanisms of the disease. This study aims to evaluate the significance of these markers in the development and severity of systemic scleroderma.

Materials and Methods

A total of 100 individuals were included in this study: 80 patients diagnosed with systemic scleroderma and 20 healthy controls. Participants were examined at the Tashkent Medical Academy's multidisciplinary clinic (Building 12 - Internal Diseases, and Building 9 - Rheumatology).

PDGF and TSP-1 levels were measured using the ELISA method. Clinical and laboratory assessments included ultrasound of internal organs and complete blood count tests. Patients' age ranged from 25 to 50 years, with 80% females and 20% males. Statistical analysis was performed using Student's t-test.

Results

Patients with systemic scleroderma showed significantly higher levels of PDGF and TSP-1 compared to the control group ($p < 0.01$). Elevated PDGF levels were associated with increased skin and lung fibrosis. Higher TSP-1 levels were observed in patients with more severe vascular symptoms and Raynaud's phenomenon. Ultrasound revealed internal organ involvement in 62.5% of patients, including hepatic fibrosis and renal changes.

Discussion

Our findings confirm the important role of PDGF and TSP-1 in the pathogenesis of systemic scleroderma. PDGF promotes fibroblast activation and collagen production, while TSP-1 inhibits angiogenesis and activates profibrotic pathways via TGF-beta. The imbalance between these factors contributes to persistent vascular injury and fibrosis.

These results are consistent with studies by Kahaleh et al. and Distler et al., which demonstrated elevated PDGF and TSP-1 levels in systemic sclerosis.

Conclusion

PDGF and TSP-1 are critical contributors to the progression of systemic scleroderma. Their evaluation may serve as useful diagnostic and prognostic biomarkers. Targeted therapies that regulate their activity could improve treatment outcomes and reduce fibrosis severity in affected patients.

Practical Recommendations

- Include PDGF and TSP-1 testing in routine assessment of SSc patients
- Monitor their levels to evaluate fibrosis risk
- Consider targeted therapy in patients with high biomarker expression

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