AMERICAN JOURNAL OF MULTIDISCIPLINARY BULLETIN
ISSN: 2996-511X (online) | ResearchBib (IF) = 9.512 IMPACT FACTOR
Volume-3| Issue-11| 2025 Published: |30-12-2025|

CLINICAL AND IMMUNOLOGICAL CHARACTERISTICS OF SYSTEMIC SCLEROSIS PATIENTS WITH ANTI-FIBRILLARIN ANTIBODIES

https://doi.org/10.5281/zenodo.17988927

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Abstract

Systemic sclerosis is a complex autoimmune disease characterized by immune dysregulation, vascular damage, and progressive fibrosis of the skin and internal organs. Identification of specific autoantibodies is essential for understanding disease heterogeneity and predicting clinical outcomes. Anti-fibrillarin (anti-U3-RNP) antibodies are associated with distinct clinical phenotypes and may indicate a more aggressive disease course. Investigation of their clinical relevance remains important for improving early risk stratification and personalized management of systemic sclerosis.

Keywords

Systemic sclerosis, anti-fibrillarin antibodies, U3-RNP, autoantibodies, fibrosis, disease progression, prognosis

Аннотация

Системная склеродермия является сложным аутоиммунным заболеванием, характеризующимся нарушением иммунной регуляции, сосудистыми повреждениями и прогрессирующим фиброзом кожи и внутренних органов. Идентификация специфических аутоантител имеет ключевое значение для понимания гетерогенности заболевания и прогнозирования клинических исходов. Антифибриллариновые (anti-U3-RNP) антитела ассоциированы с особыми клиническими фенотипами и могут указывать на более агрессивное течение заболевания. Изучение их клинической значимости остается важным для улучшения ранней стратификации риска и персонализированного ведения пациентов с системной склеродермией.

Ключевые слова

Системная склеродермия, антифибриллариновые антитела, U3-RNP, аутоантитела, фиброз, прогрессирование заболевания, прогноз.

Annotatsiya

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Tizimli sklerodermiya — immun regulyatsiyaning buzilishi, tomir shikastlanishi va teri hamda ichki a'zolarning progressiv fibrozlanishi bilan tavsiflanadigan murakkab autoimmun kasallikdir. Maxsus autoantitanalarni aniqlash kasallik geterogenligini tushunish va klinik natijalarni bashorat qilishda muhim ahamiyatga ega. Anti-fibrillarin (anti-U3-RNP) antitanalari ma'lum klinik fenotiplar bilan bog'liq bo'lib, kasallikning yanada agressiv kechishini ko'rsatishi mumkin. Ularning klinik ahamiyatini o'rganish tizimli sklerodermiyada xavfni erta stratifikatsiya qilish va individual yondashuvni takomillashtirish uchun dolzarb hisoblanadi.

Kalit so'zlar

Tizimli sklerodermiya, anti-fibrillarin antitanalari, U3-RNP, autoantitanalar, fibroz, kasallik progressiyasi, prognoz.

Introduction

Systemic sclerosis (SSc), also known as scleroderma, is a complex autoimmune connective tissue disease characterized by progressive fibrosis, immune dysregulation, and widespread microvascular abnormalities. The disease involves the skin and multiple internal organs, leading to significant morbidity and reduced quality of life. Immunological markers, particularly antinuclear antibodies (ANA), play a central role in the diagnosis, classification, and prognostic assessment of SSc. Among these, antibodies directed against U3-ribonucleoprotein (U3-RNP), also known as anti-fibrillarin antibodies (AFA), are of special clinical interest due to their association with distinct disease phenotypes. Systemic sclerosis is a relatively rare condition, with an estimated prevalence ranging from 50 to 300 cases per million population. It predominantly affects adults, with peak incidence between 30 and 50 years of age, although the disease may occur at any stage of life. Women are affected significantly more often than men, with a female-to-male ratio of approximately 5-8:1. Epidemiological studies also demonstrate notable geographic and ethnic variability, with higher prevalence rates reported among Native American and African American populations compared to individuals of European descent. Additionally, regional differences have been observed in the distribution of SSc subtypes and associated autoantibody profiles.

The exact etiology of systemic sclerosis remains incompletely understood. Current evidence suggests that the disease results from a multifactorial interaction between genetic susceptibility, environmental exposures, and immune system abnormalities. Genetic predisposition is supported by familial clustering and the identification of specific genetic markers associated with increased disease risk; however, no single gene has been identified as causative, indicating a polygenic

background. Environmental factors, including exposure to silica, organic solvents, and certain viral infections, have been proposed as potential triggers. The higher prevalence among women further suggests a possible role of hormonal and reproductive factors in disease development. Immune system dysregulation is a fundamental feature of systemic sclerosis. Aberrant activation of innate and adaptive immune responses leads to chronic inflammation, autoantibody production, vascular injury, and excessive extracellular matrix deposition. The development of SSc involves a complex interplay between endothelial dysfunction, immune-mediated tissue injury, and progressive fibrosis of the skin, internal organs, and blood vessels. Although the precise sequence of pathogenic events remains unclear, these mechanisms are recognized as key drivers of disease initiation and progression.

Anti-U3-RNP (anti-fibrillarin) antibodies target the U3 ribonucleoprotein complex, which is involved in ribosomal RNA processing and ribosome assembly. The presence of these antibodies in patients with systemic sclerosis has been associated with specific clinical features and disease patterns. While their exact pathogenic role is not fully elucidated, anti-fibrillarin antibodies are thought to contribute to immune activation and fibrotic processes by promoting the release of pro-inflammatory and pro-fibrotic cytokines. In addition, they may be involved in vascular abnormalities and endothelial dysfunction characteristic of SSc.

Aim of the Study

The aim of this study was to evaluate the immunological and pathogenetic significance of anti-fibrillarin antibodies in patients with systemic sclerosis.

Materials and Methods

The study included 70 patients diagnosed with systemic sclerosis who were treated in the Rheumatology Department of the Tashkent Clinical Hospital. Diagnosis was established according to the 2013 EULAR/EUSTAR classification criteria. The mean age of the patients was 47.6 ± 3.8 years, and the mean disease duration was 6.5 ± 2.1 years. The study population consisted of 62 women (88.6%) and 8 men (11.4%). All patients underwent comprehensive clinical evaluation, laboratory testing, instrumental investigations, and detailed immunological assessment, including detection of anti-fibrillarin antibodies.

Results

Cutaneous manifestations were common among the study participants. Skin edema was observed in 26 patients (37,1%), skin thickening in 29 (44,3%), digital ulcers in 13 (20%), and sclerodactyly in 28 patients (42,9%). Raynaud's phenomenon was present in all examined individuals. Among internal organ manifestations,

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dysphagia was the most frequently reported symptom, occurring in 90% of patients (Figure 1).

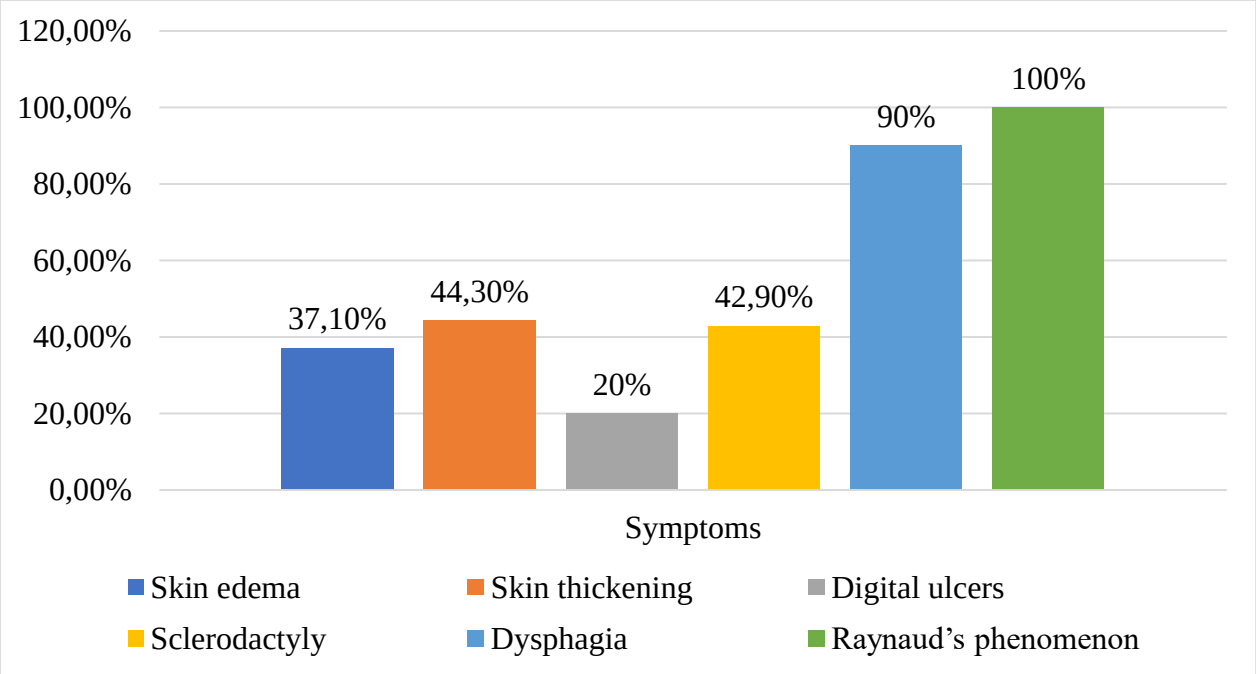

Figure 1. Main clinical manifestations of systemic sclerosis among patients

Anti-fibrillarin antibodies were detected in 17 patients (24,3%). In this subgroup, disease onset occurred at a younger age, with a mean age of 34,2 years. Limited cutaneous systemic sclerosis (lcSSc) was diagnosed in 2 patients (11,7%), while 15 patients (88,2%) had the diffuse cutaneous form (dcSSc). Comparative analysis revealed that patients positive for anti-fibrillarin antibodies tended to have earlier disease onset, a more aggressive disease course, and more rapid progression of pulmonary and cutaneous fibrosis.

Discussion

The findings of this study highlight the distinct clinical and immunological characteristics associated with anti-fibrillarin antibodies in systemic sclerosis. The high frequency of skin involvement, Raynaud's phenomenon, and internal organ manifestations such as dysphagia corresponds to the classical presentation of SSc. The identification of anti-fibrillarin antibodies in a subset of patients, particularly those with early disease onset, suggests their potential involvement in disease pathogenesis and their value as prognostic markers.

The association between anti-fibrillarin antibodies and a more severe disease course, characterized by rapid progression of lung and skin fibrosis, underscores the importance of detailed autoantibody profiling in patients with systemic

sclerosis. Recognition of specific autoantibody patterns may assist clinicians in risk stratification, treatment planning, and close monitoring of disease progression.

Conclusion

In conclusion, the presence of anti-fibrillarin antibodies in patients with systemic sclerosis is associated with early disease onset and a more aggressive fibrotic phenotype involving the skin and lungs. Early detection of these antibodies may help identify patients at higher risk of rapid disease progression who could benefit from timely immunomodulatory and anti-fibrotic therapeutic strategies. Incorporating autoantibody profiling into routine clinical practice may improve individualized treatment approaches and overall disease management outcomes.

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