AMERICAN JOURNAL OF EDUCATION AND LEARNING
ISSN: 2996-5128 (online) | ResearchBib (IF) = 10.91 IMPACT FACTOR
Volume-4| Issue-4| 2026 Published: |30-04-2026|

UDC: 616.72-002.772:616.11-002.77

ASSOCIATION BETWEEN ADAMTS7 LEVELS, INFLAMMATORY MARKERS, AND STRUCTURAL JOINT CHANGES IN RHEUMATOID ARTHRITIS

https://doi.org/10.5281/zenodo.19869591

Buranova S.N., Khalmetova F.I.
Tashkent State Medical University, Tashkent, Uzbekistan
Bekmirzayev A.E.
Tashkent State Medical University Termez Branch

Abstract

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent inflammation and progressive joint destruction. Identification of biomarkers reflecting both inflammatory activity and structural damage remains a key challenge. This study aimed to evaluate the association between ADAMTS7 levels, inflammatory markers, and structural joint changes in RA patients. A cross-sectional study included 46 patients aged 21–48 years, predominantly female, diagnosed according to ACR/EULAR criteria. Disease activity was assessed using DAS28. Serum levels of ADAMTS7, TNF-α, and IL-6 were measured by ELISA. Structural joint changes were evaluated using imaging methods. Elevated ADAMTS7 levels were observed in patients with higher disease activity. Significant positive correlations were found between ADAMTS7 and TNF-α, IL-6, as well as with structural joint damage. ADAMTS7 may serve as a promising biomarker reflecting both inflammation and joint destruction in RA.

Keywords

rheumatoid arthritis, ADAMTS7, TNF-α, IL-6, biomarkers, joint damage, inflammation.

Rheumatoid arthritis is a systemic autoimmune disease characterized by chronic synovial inflammation, cartilage destruction, and bone erosion, leading to disability and reduced quality of life. Despite advances in treatment, early identification of patients at risk of severe disease progression remains challenging. Increasing attention has been paid to extracellular matrix-degrading enzymes involved in joint destruction. ADAMTS7, a member of the metalloproteinase family, plays a role in tissue remodeling and has been studied mainly in cardiovascular pathology. However, its role in rheumatoid arthritis remains insufficiently explored. Pro-inflammatory cytokines such as TNF-α and IL-6 are

central mediators of RA pathogenesis and are directly involved in synovial inflammation and joint damage. The interaction between ADAMTS7 and these cytokines, as well as its relationship with structural joint changes, requires further investigation. Therefore, this study aims to assess the association between ADAMTS7 levels, inflammatory activity, and structural joint damage in RA patients.

Materials and Methods. A cross-sectional study was conducted including 46 patients diagnosed with rheumatoid arthritis according to the 2010 ACR/EULAR classification criteria. The age of participants ranged from 21 to 48 years, and the majority were female. Disease activity was evaluated using the Disease Activity Score (DAS28). Serum levels of ADAMTS7, TNF-α, and IL-6 were measured using enzyme-linked immunosorbent assay (ELISA) according to standard protocols. Structural joint changes were assessed using imaging techniques, including evaluation of joint space narrowing, cartilage damage, and erosive changes, with particular attention to the knee joint. Statistical analysis was performed using SPSS software. Correlations between variables were analyzed using Pearson or Spearman correlation coefficients, with statistical significance set at $p < 0.05$.

Results and Discussion. The study included 46 patients aged 21–48 years, with a predominance of women, consistent with the epidemiology of rheumatoid arthritis. Patients with higher disease activity demonstrated significantly elevated levels of ADAMTS7 compared to those with lower activity. A statistically significant positive correlation was found between ADAMTS7 levels and pro-inflammatory cytokines TNF-α and IL-6 ($p < 0.05$), indicating a close relationship between ADAMTS7 and systemic inflammation. Furthermore, ADAMTS7 levels were significantly associated with the severity of structural joint damage, particularly in the knee joint, where higher levels corresponded to more pronounced cartilage degradation and joint space narrowing. These findings suggest that ADAMTS7 may play a key role in linking inflammatory processes with structural joint destruction. As a matrix-degrading enzyme, ADAMTS7 contributes to extracellular matrix remodeling, which may explain its association with cartilage damage. Its correlation with TNF-α and IL-6 further supports its involvement in inflammatory pathways underlying RA progression. These results are consistent with emerging evidence highlighting the importance of metalloproteinases in joint damage and their potential as biomarkers and therapeutic targets.

Conclusion. ADAMTS7 represents a promising biomarker in rheumatoid arthritis, reflecting both inflammatory activity and structural joint damage. Its significant association with pro-inflammatory cytokines and joint structural alterations suggests its involvement in disease progression. Assessment of

ADAMTS7 levels may improve early detection of aggressive disease forms and support the development of personalized treatment and preventive strategies.

REFERENCES:

1. Colige A., et al. ADAMTS metalloproteinases in cartilage biology. *Biochim Biophys Acta*. 2010;1803(1):120–134.
2. Feldmann M., Maini R.N. TNF defined as a therapeutic target. *Nat Med*. 2003;9(10):1245–1250.
3. Firestein G.S., McInnes I.B. Immunopathogenesis of rheumatoid arthritis. *Immunity*. 2017;46(2):183–196.
4. Khalmetova, F. I., Akhmedov, K. S., Buranova, S. N., Rakhimova, M. B., Rakhimov, S. S., & Abdurakhimova, L. A. (2023). Immunological Features of Reactive Arthritis of Various Etiologies. Journal of Coastal Life Medicine, 11, 1322-1325.
5. Murphy G., Nagase H. Reappraising metalloproteinases in rheumatoid arthritis. *Nat Rev Rheumatol*. 2008;4(3):128–135.
6. Rakhimova, M. B., Akhmedov, K. S., & Turaev, Y. A. (2021). Endothelial dysfunction as a link in the pathogenesis of ankylosing spondylitis against the background of a new coronavirus infection. ACADEMICIA: An International Multidisciplinary Research Journal, 11(3), 2493-2498.
7. Rakhimova, M., Khalmurad Akhmedov, Buranova, S., & Tursunova, L. (2022). Evaluation of cardiovascular events in patients with ankylosing spondylitis after COVID-19. International Journal of Early Childhood Special Education, 14(5), 7171–7175.
8. Schett G., Gravallese E. Bone erosion in rheumatoid arthritis. *Nat Rev Rheumatol*. 2012;8(11):656–664.
9. Smolen J.S., Aletaha D., McInnes I.B. Rheumatoid arthritis. *Lancet*. 2016;388(10055):2023–2038.
10. Tanaka T., Narazaki M., Kishimoto T. IL-6 in inflammation and disease. *Cold Spring Harb Perspect Biol*. 2014;6(10):a016295.
11. Миррахимова, М. Х., and Н. Ю. Нишанбаева. "РОЛЬ TOLL-ПОДОБНЫХ РЕЦЕПТОРОВ В ПАТОГЕНЕЗЕ АТОПИЧЕСКОГО ДЕРМАТИТА." *AMERICAN JOURNAL OF EDUCATION AND LEARNING* 2.5 (2024): 234-236.
12. Mirraximova, Maktuba, and Nilufar Nishonboeva. "Genining polimorfizmi atopik dermatitda." Scientific journal of the Fergana State University 1 (2023): 137-137.

13. Nishanbayeva N. Y. et al. CAUSES OF ATOPIC DERMATITIS AND MEASURES FOR ITS PREVENTION //AMERICAN JOURNAL OF EDUCATION AND LEARNING. – 2025. – Т. 3. – №. 2. – С. 566-573.

14.