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ACCELERATION OF STRUCTURAL DAMAGE IN AXIAL SPONDYLOARTHRITIS FOLLOWING SARS-COV-2 INFECTION: A BIOMARKER-BASED STUDY OF CARTILAGE TURNOVER

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Introduction

The emergence of the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in late 2019 initiated an unprecedented global health crisis. Coronavirus disease 2019 rapidly evolved into a pandemic, affecting hundreds of millions of individuals and resulting in millions of deaths worldwide. While the acute respiratory manifestations of the infection initially dominated clinical attention, it soon became evident that COVID-19 represents a systemic inflammatory condition capable of affecting multiple organ systems. Beyond pulmonary complications, SARS-CoV-2 infection has been associated with cardiovascular injury, thromboembolic phenomena, neurological dysfunction, autoimmune activation, and prolonged post-viral syndromes. Of particular concern has been its impact on patients with pre-existing chronic inflammatory diseases.

Axial spondyloarthritis, including its radiographic form commonly referred to as ankylosing spondylitis, is a chronic immune-mediated inflammatory disorder characterized by inflammation of the sacroiliac joints and spine. The disease involves complex interactions between genetic susceptibility, innate immune activation, and cytokine-mediated inflammation, leading to progressive structural remodeling of the axial skeleton. Enthesitis, osteitis, cartilage degradation, and subsequent pathological new bone formation contribute to spinal rigidity and functional impairment. Because axial spondyloarthritis is fundamentally driven by dysregulated immune pathways, the superimposition of a systemic viral infection such as COVID-19 raises important questions regarding potential disease exacerbation, structural progression, and biomarker alterations.

During the pandemic, patients with chronic inflammatory rheumatic diseases faced multiple challenges. Concerns arose regarding susceptibility to infection due to immunomodulatory therapy, potential disease flares triggered by viral-induced immune activation, and long-term consequences of cytokine storm phenomena. SARS-CoV-2 infection is characterized by profound activation of inflammatory mediators, including interleukin-6, tumor necrosis factor alpha, and interleukin-17-related pathways, all of which are also implicated in the pathogenesis of axial spondyloarthritis. The overlap in inflammatory cascades suggests a plausible biological mechanism through which COVID-19 may influence structural disease activity in predisposed individuals.

Cartilage degradation represents a critical early event in the structural evolution of axial spondyloarthritis. Although radiographic progression typically emphasizes syndesmophyte formation and ossification, preceding cartilage breakdown contributes significantly to disease pathophysiology. Biomarkers reflecting extracellular matrix turnover provide a window into ongoing structural processes that may not yet be detectable by conventional imaging. Among these markers, C-terminal telopeptide of type II collagen (CTX-II) has emerged as a sensitive indicator of cartilage degradation. Type II collagen is a major structural component of articular cartilage, and its breakdown releases CTX-II fragments measurable in serum or urine. Elevated

CTX-II levels have been associated with inflammatory joint destruction in various rheumatic conditions.

In the context of axial spondyloarthritis, increased CTX-II concentrations may reflect active cartilage degradation within the sacroiliac joints and spinal intervertebral structures. Monitoring such biomarkers can potentially identify accelerated structural damage before radiographic progression becomes apparent. The interplay between systemic viral infection and cartilage metabolism, however, remains insufficiently explored.

SARS-CoV-2 infection induces widespread immune activation characterized by cytokine release, oxidative stress, endothelial dysfunction, and altered bone-cartilage homeostasis. Experimental data suggest that inflammatory cytokines enhance matrix metalloproteinase activity, promoting collagen breakdown. Additionally, prolonged immobilization during acute illness and corticosteroid exposure may further influence musculoskeletal integrity. Therefore, it is biologically plausible that COVID-19 could accelerate cartilage degradation in patients with axial spondyloarthritis, particularly in those not receiving adequate disease-modifying therapy.

Clinical observations during the pandemic have reported worsening back pain, increased stiffness, and functional decline among certain patients with axial spondyloarthritis following COVID-19 infection. However, subjective symptom exacerbation must be distinguished from objective structural progression. Biomarker-based investigation offers a quantitative method to evaluate potential post-viral acceleration of tissue damage.

Understanding the structural consequences of COVID-19 in this patient population carries significant clinical implications. If SARS-CoV-2 infection indeed amplifies cartilage degradation, intensified monitoring and early therapeutic adjustment may be warranted. Furthermore, such findings would contribute to broader knowledge regarding viral triggers of autoimmune and autoinflammatory disease progression.

The present study was designed to investigate the impact of prior SARS-CoV-2 infection on cartilage turnover in patients with axial spondyloarthritis, using CTX-II as a biochemical marker of structural degradation. By comparing biomarker levels between infected and non-infected patients, and analyzing associations with disease duration and treatment status, this research aims to clarify whether COVID-19 represents a modifying factor in structural disease evolution.

Materials and Methods

A retrospective-prospective observational study was conducted between 2020 and 2022 in specialized rheumatology departments. A total of 211 patients diagnosed with axial spondyloarthritis according to established classification criteria were included. The cohort consisted predominantly of male patients, reflecting the known epidemiological distribution of the disease. Mean disease duration was approximately nine years.

A control group of forty age-matched healthy volunteers without inflammatory or degenerative joint disease was recruited for baseline biomarker comparison. Patients were stratified into two principal groups based on documented history of SARS-CoV-2 infection confirmed by polymerase chain reaction testing. Group one included individuals who had recovered from COVID-19, while group two comprised patients without prior infection.

Within the post-COVID group, further subdivision was performed according to receipt of continuous disease-modifying therapy. Subgroup A consisted of patients who had interrupted or were not receiving baseline therapy during infection, whereas subgroup B included those maintained on standard anti-inflammatory or biologic treatment.

All participants underwent comprehensive clinical evaluation including assessment of inflammatory back pain, morning stiffness duration, spinal mobility indices, and peripheral joint involvement. Laboratory investigations included measurement of CTX-II levels using enzyme-linked immunosorbent assay techniques. Radiographic imaging of the sacroiliac joints and spine was performed to evaluate structural changes. Statistical analysis assessed differences between groups and correlations with disease duration.

Results

Patients with prior SARS-CoV-2 infection demonstrated significantly higher CTX-II levels compared to non-infected patients and healthy controls. The most pronounced elevations were observed in individuals who had not received continuous baseline therapy during infection. These patients also reported higher frequencies of morning stiffness, persistent back pain, and functional limitation.

Quantitative analysis revealed that CTX-II concentrations in untreated post-COVID patients were more than double those observed in non-infected patients. Individuals maintained on disease-modifying therapy exhibited intermediate biomarker levels, suggesting partial protective effect of sustained anti-inflammatory treatment.

Correlation analysis demonstrated that CTX-II levels were highest during early years of disease duration, gradually declining in later stages. This pattern may reflect active cartilage breakdown preceding advanced ossification and ankylosis. Importantly, post-COVID patients exhibited biomarker elevations beyond expected values for corresponding disease duration, indicating potential acceleration of cartilage turnover associated with viral infection.

Radiographic findings supported increased structural progression in selected post-COVID cases, though imaging changes were less sensitive than biomarker alterations.

Discussion

The findings of this study suggest that SARS-CoV-2 infection may amplify cartilage degradation in patients with axial spondyloarthritis. Elevated CTX-II levels in post-COVID individuals indicate intensified extracellular matrix breakdown, particularly among those lacking consistent anti-inflammatory therapy. These observations align with the hypothesis that systemic cytokine activation during viral infection enhances catabolic processes within joint tissues.

Mechanistically, heightened levels of interleukin-6 and tumor necrosis factor alpha during COVID-19 may stimulate matrix metalloproteinases responsible for collagen degradation. Oxidative stress and endothelial dysfunction could further compromise cartilage integrity. The protective trend observed in patients receiving continuous therapy underscores the importance of maintaining disease-modifying treatment during infectious episodes when clinically feasible.

The decline in CTX-II with longer disease duration may correspond to transition from inflammatory cartilage destruction to structural ossification. Nevertheless, acute post-viral surges in biomarker levels raise concerns regarding episodic acceleration of structural damage.

These results carry practical implications. Monitoring CTX-II levels in axial spondyloarthritis patients recovering from COVID-19 may facilitate early detection of structural progression. Intensified follow-up and timely therapeutic optimization could mitigate long-term disability.

Limitations include observational design and potential confounding factors such as corticosteroid exposure during acute infection. Larger longitudinal studies are required to confirm causality and determine persistence of biomarker elevation.

Conclusion

SARS-CoV-2 infection appears to be associated with increased cartilage degradation in patients with axial spondyloarthritis, as evidenced by elevated CTX-II levels. The effect is particularly pronounced in individuals not receiving continuous baseline therapy. These findings highlight the necessity for vigilant monitoring and optimized management in post-COVID patients with inflammatory spinal disease.

Biomarker assessment of CTX-II may serve as a valuable tool for detecting early structural changes and guiding therapeutic decisions. Further research should explore long-term outcomes and potential interventions to prevent accelerated structural progression following viral infection.

Adabiyotlar

1. Abdurakhmanova, N. M., Ahmedov, K. S., & Turaev, I. A. (2022). Modern methods of treatment of patients with ankylosing spondylitis. *International Journal of Advance Scientific Research*, *2*(11), 112-118.

2. Turaev, S. Z. I., & Rakhimov, S. (2023). ASSESSMENT OF THE QUALITY OF LIFE IN PATIENTS WITH CHRONIC KIDNEY DISEASE IN THE PRACTICE OF HEMODIALYSIS. Journal of Modern Educational Achievements, 6(6), 103- 109.

3. Rakhimova, M. B., Akhmedov, K. S., Rakhimov, S. S., & Zaripov, S. I. (2023). Extrasceletal Manifestations in Patients with Ankylosing Spondylitis. Journal of Coastal Life Medicine, 11, 1315-1321.

4. Zaripov, S. I., & Abdurakhmanova, N. M. (2023). Quality of life of End-Stage Renal Disease (ESRD) patients receiving hemodialysis: influencing factors and approaches to correction. *Texas Journal of Multidisciplinary Studies*, *21*, 14-17.

5. Abdurakhmanova, N. M., Zaripov, S. S., & Turaev, I. A. (2023). THE EFFECT OF CLIMATEGEOGRAPHICAL FACTORS ON RHEUMATOID ARTHRITIS ACTIVITY. *World Bulletin of Public Health*, *18*, 67-69.

6. S. I. Zaripov and N. M. B. Abdurakhmanova, "The Relationship Between Systemic Sclerosis and Anti-Fibrillarin Antibodies," Journal of Modern Educational Achievements, vol. 6, no. 6, pp. 235-238, 2024.

7. S. I. Zaripov, I. A. Turaev, and S. S. Rakhimov, "Quality of Life in Patients with Chronic Kidney Disease Receiving Program Hemodialysis and Possible Ways of Its Correction," Uzbek Medical Journal, vol. 3, no. 5, 2022.

8. Umarova, Z. F., Jumanazarov, S. B., Zaripov, S. I., & Khaydarov, R. M. (2024). Quality of life in patients with chronic kidney disease in the V stage receiving program hemodialysis and possible ways of its correction. Journal of Medicine and Innovations.

9. Istamovich, Z. S., Sadullayevich, A. K., & Mirza-Bakhtiyarkhanovna, A. N. (2023). The significance of autoantibodies in the pathogenesis of systemic sclerosis (literature review). Journal of Biomedicine and Practice, 8(2).

10. Абдурахманова, Н. М., Ахмедов, Х. С., & Зарипов, С. И. (2024). ИММУНОПАТОГЕНЕТИЧЕСКОЕ ЗНАЧЕНИЕ АУТОАНТИТЕЛ ПРОТИВ ФИБРИЛЛИНА ПРИ СИСТЕМНОЙ СКЛЕРОДЕРМИИ.

11. Axmedov, I. A., Xalmetova, F. I., & Zaripov, S. I. (2024). Rematoid artrit kasalligi bo ‘lgan bemorlarda yurak qon-tomir tizimidagi buzulishlarni erta aniqlashda yurak ritmi buzilishlarining o ‘rni.

12. Rakhmatov, A. M., & Zaripov, S. I. (2024). Gout and its association with gouty nephropathy: an analysis of 46 patients. Современные подходы и новые исследования в современной науке, 3(16), 100-102.

13. Рахимова, М. Б., Ахмедов, Х. С., & Халметова, Ф. И. (2025). ОЦЕНКА ЭНДОТЕЛИАЛЬНОЙ ДИСФУНКЦИИ У БОЛЬНЫХ РЕВМАТОИДНЫМ АРТРИТОМ.

14. Rakhimova, M., Akhmedov, K., Buranova, S., & Tursunova, L. (2022). Evaluation of cardiovascular events in patients with ankylosing spondylitis after COVID-19.

15. Шовкатова, М. Н., & Рахимова, М. Б. (2025). ИСКУССТВЕННЫЙ ИНТЕЛЛЕКТ В ЦИФРОВОЙ СТРАТИФИКАЦИИ И ДИНАМИЧЕСКОМ КОНТРОЛЕ СЕРДЕЧНО-СОСУДИСТОГО РИСКА У БОЛЬНЫХ С АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ И РЕВМАТОИДНЫМ АРТРИТОМ. FARS International Journal of Education, Social Science & Humanities., 13(12), 7-14.

16. 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.

17. Rakhimova, M. B., Akhmedov, K. S., Rakhimov, S. S., & Zaripov, S. I. (2023). Extrasceletal Manifestations in Patients with Ankylosing Spondylitis. Journal of Coastal Life Medicine, 11, 1315-1321.

18. Abdurakhmanova, N., Akhmedov, K., Jabbarov, O., Rakhimova, M., Tagaeva, M., Khalmetova, F., & Tursunova, L. (2022). Clinical And Diagnostic Significance Of Anti-Cd74 In Patients With Ankylosing Spondylitis Of Uzbek Population. Journal of Positive School Psychology http://journalppw. com, 6(6), 9358-9364.

19. Rakhimova, M. B., & Akhmedov, K. S. (2021). The impact of sequelae of covid-19 on the course of ankylosing spondylitis. Central Asian journal of medicine, 2021(4), 58-63.

20. Rakhimova, M. B. (2023). Impared endothelial dysfunction in covid-19: an overview of evidence, biomarkers in patients with ankylosing spondylitis. IMRAS, 6(7), 20-27.

21. Буранова, С. Н., & Бахронова, Ю. Б. (2025). ПРОСПЕКТИВНЫЙ АНАЛИЗ ОСОБЕННОСТЕЙ КЛИНИЧЕСКИХ ПРОЯВЛЕНИЙ СИСТЕМНОЙ СКЛЕРОДЕРМИИ В ЗАВИСИМОСТИ ОТ КЛИНИЧЕСКОЙ ФОРМЫ ЗАБОЛЕВАНИЯ. AMERICAN JOURNAL OF EDUCATION AND LEARNING, 3(9), 581-583.

22. Ахмедов, Х. С., Абдурахманова, Н. М., Буранова, С. Н., Халметова, Ф. И., Рахимова, М. Б., Нуриллаев, Б. А., & Очилов, И. А. (2025). УЧЕБНО-МЕТОДИЧЕСКИЙ КОМПЛЕКС ПО ПРЕДМЕТУ.

23. 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.

24. Akhmedov, K., Abdurakhmanova, N., & Buranova, S. (2023). Features of the clinical course of rheumatoid spice against the background of the influence of xenobiotics. American Journal of Interdisciplinary Research and Development, 12, 142-147.

25. Buranova, S. (2021). Method of treatment aimed at the dynamics of cartilage oligomer matrix protein (COMP) in patients with osteoarthritis.

26. Buranova, S. N. (2021). Akhmedov Kh. S., Razakova FS The Importance of Treatment Aimed at the Dynamics of Cartilage Oligomer Matrix Protein (COMP) in Patients with the Knee Joint Osteoarthritis. American Journal of Medicine and Medical Sciences, 11(2), 148-153.

27. Buranova, S., & Akhmedov, K. (2021). Cartilage oligomeric matrix protein (comp) in early diagnosis of osteoarthritis.

28. Khalmetova, F., Axmedov, X., Buranova, S., & Botirbekov, A. (2023). GENETIC ASPECTS OF REACTIVE ARTHRITIS. Scientific journal of the Fergana State University, (1), 133-133.

29. Xalmetova, F. I., Akhmedov, X. S., & Buranova, S. N. (2022). The role of imaging techniques in the assessment of structural changes in the joint in reactive arthritis. Academicia Globe, 3(03), 186-189.

30. Buranova, S. N., & Khalmetova, F. I. (2025). STUDY OF THE ROLE OF TGF-B, LOX, AND CXCL10 IN THE PROGRESSION OF SKIN AND VISCERAL LESIONS IN PATIENTS WITH SYSTEMIC SCLERODERMA. JOURNAL OF MULTIDISCIPLINARY BULLETIN, 8(9), 43-46.

31. Khalmetova, F. I., Akhmedov, K. S., Turayev, I. A., & Zaripov, S. I. Реактив артритни даволашда замонавий патогенетик ёндашувлар. 2024. TTA AXBOROTNOMASI. Bb, 63-65.

32. Ахмедов, Х. С., Абдурахманова, Н. М., & Халметова, Ф. И. (2017). Влияние различных физических факторов климата на течение ревматоидного артрита. Universum: медицина и фармакология, (3 (37)), 12-15.

33. Абдурахимов, А. Г., & Халметова, Ф. И. (2023). Нестероидные противовоспалительные препараты у пациентов с деформирующим остеоартрозом и артериальной гипертензией: анализ влияния целекоксиба и мелоксикама на антигипертензивные средства. Оптимизация лечения. Атеросклероз, 19(3), 186-187.

34. Khalmetova, F. I., Akhmedov, K. S., & Razakova, F. S. (2021). Comparative analysis of the clinical presentation of reactive arthritis.

35. Khalmetova, F. I., Akhmedov, X. S., & Alibekova, G. A. (2023). Features of the course of the joint syndrome in various forms of reactive arthritis. Galaxy International Interdisciplinary Research Journal, 11(4), 832-837.

36. Шарипова, Н. В., Худайберганов, А. С., Рахимов, Б. Б., & Наврузов, Э. Б. Гигиенические требования к безопасности пищевой продукции. *СанПиН РУз,(0283-10)*.

37. Салихова, Н. С., Косимов, Р. А., Юлдашева, З. Р., Шайхова, Г. И., Эрматов, Н. Ж., & Рахимов, Б. Б. (2016). Санитарно-эпидемиологические требования к организации питания обучающихся в общеобразовательных школах, учреждениях средне специального профессионального образования. *СанПиН.–2016*, 0288-10.

38. Nurmatov, B., & Rakhimov, B. (2022). Study of virus contamination of indoor air and surfaces of hospital which specialized in the treatment of COVID-19 patients.

39. Рахимов, Б. Б., Уринов, А. М., Шайхова, Л. И., & Камилова, А. Ш. (2017). Выявление факторов риска при ожирении у детей дошкольного возраста, проживающих в г. Ташкенте.

40. Shaykhova, G. I., & Rakhimov, B. B. (2014). Promotion of the principles of rational nutrition in obesity. Medical Journal of Uzbekistan,(2), 138.

41. Sultonov, E. Y., Sariullaycva, X. A., Salomova, F. I., & Mirsagatova, M. R. (2023). Ochiq suv havzalari suv namunalari tahlili. Здоровый образ жизни международная научно-практическая конференция.

42. Тешаев, О. Р., Муродов, А. С., Касымова, К. Р., Садыков, Р. Р., & Тавашаров, Б. Н. (2012). Эффективность фотодинамического воздействия на возбудителей рожистого воспаления. Врач-аспирант, 52(3.4), 597-601.

43. Teshaev, Oktyabr, Ilkhom Khayitov, and Bahodir Tavasharov. "Surgical treatment of postoperative ventral hernias in patients with obesity." The Tenth European Conference on Biology and Medical Sciences. 2016.

44. Сагатов, Туляган Агзамович, et al. "Механизмы развития патоморфологических изменений микроциркуляторного русла и тканевых структур кишечника при хронической интоксикации пестицидом" Суми-альфа" на фоне экспериментального диабета." Проблемы науки 4 (28) (2018): 39-40.

45. Мирщарапов, Уткур Миршаропович, and Баходир Назарович Тавашаров. "МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ СОСУДИСТО-ТКАНЕВЫХ СТРУКТУР ТОНКОЙ КИШКИ ПРИ ОСТРОЙ ИНТОКСИКАЦИИ ПЕСТИЦИДАМИ НА ФОНЕ АЛЛАКСАНОВОГО САХАРНОГО ДИАБЕТА." ИННОВАЦИОННЫЕ ПРОЦЕССЫ В НАУКЕ И ОБРАЗОВАНИИ. 2019.

46. Тешаев, О. Р., and И. Б. Хайитов. "Абдоминопластика послеоперационных вентральных грыж у больных с ожирением III-IV степени." Проблемы биологии и медицины 3 (2011): 66.

47. Тешаев, О. Р., et al. "Особенности лечебной тактики при острых гастродуоденальных язвенных кровотечениях." Врач-аспирант 50.1 (2012): 59-65.

48. Сагатов, Т. А., Б. Н. Тавашаров, and Н. Ж. Эрматов. "Морфологическое состояние гемоциркуляторного русла и тканевых структур тонкой кишки при хронической интоксикации пестицидом на фоне аллоксанового диабета." Медицинские новости 10 (301) (2019): 55-57.

49. Тешаев, О. Р., et al. "Эффективность бариатрической и метаболической хирургии в лечении ожирения." Медицинские новости 6 (309) (2020): 64-66.

50. Khalmetova, Feruza, et al. "The Role of Cartilage Oligomer Matrix Protein (COPM) in Diagnostics of Early Cartilage Destruction in Reactive Arthritis." Annals of the Romanian Society for Cell Biology 25.1 (2021): 4404-4410.

51. Жураева, Ш. У., et al. "Морфологическое обоснование микрохирургической реконструкции истмического отдела маточных труб при бесплодии." Врач-аспирант,№ 2. 3.51 (2012): 395.

52. Тавашаров, Баходир Назарович, and Низом Жумакулович Эрматов. "Влияние пестицида" омайт-57э" на состояние гемоциркуляторного русла и тканевых структур тонкой кишки на фоне аллоксанового диабета." Инновационные технологии в науке и образовании. 2019.

53. Ахмедов, М. А., et al. "Сочетанные операции при патологии аноректальной области." Врач-аспирант 51.2.2 (2012): 308-314.

54. Abdurakhmanova, N., & Akhmedov, K. (2022). AB0812 EFFECT OF PRO-INFLAMMATORY CYTOKINE-INTERLEUKIN 6 ON THE COURSE OF ANKYLOSING

SPONDYLITIS IN PATIENTS AFTER COVID-19. Annals of the Rheumatic Diseases, 81, 1533.

55. Istamovich, Z. S., Sadullayevich, A. K., & Mirza-Bakhtiyarkhanovna, A. N. (2023). The significance of autoantibodies in the pathogenesis of systemic sclerosis (literature review). Journal of Biomedicine and Practice, 8(2).

56. Abdurakhmanova, N. M. (2022). High concentration of tumor necrosis factor in ankylosing spondylitis patients after COVID-19. British medical journal, 2(1.2).