



7 **CORRECTION**

10 **Correction to “Poster Presentation Abstracts. The 27th**
 11 **Asia-Pacific League of Associations for Rheumatology**
 12 **Congress (APLAR) 2025, 3–7 September 2025, Fukuoka,**
 13 **Japan”**

20 “Poster Presentation Abstracts. The 27th Asia-Pacific League of Associations for Rheumatology Congress (APLAR) 2025, 3–7
 21 September 2025, Fukuoka, Japan,” *International Journal of Rheumatic Diseases* 29, no. S1 (2026): e70440. <https://doi.org/10.1111/1756-185x.70440>.

24 In the abstract “Prognostic value of anti-carp antibodies in early rheumatoid athritis,” Author Mokhinur Ruziboyevna Rakhimova
 25 was added in the author byline.

27 In the abstract “The clinical, diagnostic, and prognostic significance of laboratory and immunologic markers in early rheumatoid
 28 arthritis,” Author Mokhinur Ruziboyevna Rakhimova was added in the author byline.

30 In the abstract “Clinical diagnosis of systemic lupus erythematosus: Cases in Uzbekistan and global experience,” Author Khurshida
 31 Shamuratovna Aybergenova was added in the author byline.

33 The online abstracts have been amended.

35 We apologize for the errors.

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RF- and ACPA-; Group 3 ($n=20$): Healthy controls; Group 4 ($n=20$): Patients with psoriatic arthritis. Participants met the following criteria: Diagnosis of RA according to the 2010 ACR/EULAR classification criteria; Disease duration ≤ 12 months; Age between 18–65 years; No prior immunosuppressive therapy. The following laboratory tests were performed using commercial ELISA kits: Anti-CarP (U/mL); RF (IU/mL); ACPA (IU/mL); ESR (mm/h) CRP (mg/L). Disease activity was assessed using the Disease Activity Score 28 (DAS28). Functional impairment was evaluated using the Health Assessment Questionnaire (HAQ). Radiographic assessment of joint damage was performed using Larsen and Sharp scoring based on standard X-ray images of hands and feet. Statistical analyses were performed using SPSS v.26 (IBM, USA). Normality of data distribution was checked using the Kolmogorov-Smirnov test.

Results: The DAS28 score was significantly higher in RF+ ACPA+ patients (5.8 ± 1.2) compared to RF- ACPA- patients (5.4 ± 1.1 , $p=0.045$), indicating a more active disease state in the seropositive group. Larsen and Sharp scores showed significantly greater joint damage in Anti-CarP positive patients, regardless of RF/ACPA status ($p < 0.01$) (Figure 1). These results suggest that Anti-CarP positivity is associated with increased structural damage in RA patients, reinforcing its potential prognostic value. Further subgroup analysis indicated that Anti-CarP-positive patients had higher levels of inflammatory markers, such as ESR and CRP, compared to Anti-CarP-negative RA patients ($p < 0.05$), suggesting that Anti-CarP may reflect more aggressive disease phenotypes.

Discussion: Anti-CarP antibodies demonstrate prognostic value in assessing disease severity. Their presence is associated with higher inflammatory activity and an increased risk of joint destruction, which aligns with previously published studies. Given the correlation between Anti-CarP and structural joint changes, their detection may be useful for risk stratification in RA patients and identifying those prone to disease progression.

Conclusion: Anti-CarP antibodies can serve as a predictor of joint destruction in RA patients, emphasizing the need for their integration into routine diagnostics and disease management strategies. Further research is required to determine the threshold level of Anti-CarP associated with a high risk of arthritis progression.

The clinical, diagnostic, and prognostic significance of laboratory and immunologic markers in early rheumatoid arthritis

Khilola Mirakhmedova; *Khilola Mirakhmedova*
Tashkent Medical Academy

Introduction: Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting approximately 0.5–1% of the global population. RA leads to progressive joint destruction, systemic inflammation, and disability, significantly impacting patients' quality of life. Early diagnosis is critical to initiate timely treatment and prevent irreversible damage. However, early-stage RA diagnosis remains challenging, particularly in seronegative patients (RF- and ACPA-), where conventional serological markers fail to provide a clear diagnosis. Anti-carbamylated protein (Anti-CarP) antibodies have emerged as a potential biomarker that may improve RA detection, especially in seronegative cases.

Studies suggest that Anti-CarP is associated with more severe joint damage and increased disease progression risk.

Aim of the Work: This study aims to assess the diagnostic value of anti-CarP in early RA by evaluating its correlation with disease activity scores (DAS28), inflammatory markers (ESR, CRP), and radiographic damage (Larsen and Sharp scores)

Materials and Methods: This prospective observational study included 160 participants at the Multidisciplinary Clinical Hospital of Tashkent Medical Academy. The participants were categorized into four groups: Group 1 ($n=80$): Early RA patients, RF+ and ACPA+; Group 2 ($n=40$): Early RA patients, RF- and ACPA-; Group 3 ($n=20$): Healthy controls; Group 4 ($n=20$): Patients with psoriatic arthritis. Participants met the following criteria: Diagnosis of RA according to the 2010 ACR/EULAR classification criteria; Disease duration ≤ 12 months; Age between 18–65 years; No prior immunosuppressive therapy. The following laboratory tests were performed using commercial ELISA kits: Anti-CarP (U/mL); RF (IU/mL); ACPA (IU/mL); ESR (mm/h) CRP (mg/L). Disease activity was assessed using the Disease Activity Score 28 (DAS28). Functional impairment was evaluated using the Health Assessment Questionnaire (HAQ). Radiographic assessment of joint damage was performed using Larsen and Sharp scoring based on standard X-ray images of hands and feet. Statistical analyses were performed using SPSS v.26 (IBM, USA). Normality of data distribution was checked using the Kolmogorov-Smirnov test.

Results: Serum biomarker levels significantly varied across the groups ($p < 0.001$ for all comparisons). Anti-CarP levels were significantly higher in RF- and ACPA-negative RA patients compared to healthy controls (30.7 ± 9.3 IU/mL vs. 2.5 ± 1.1 IU/mL, $p < 0.001$). This suggests that Anti-CarP may serve as a key diagnostic and prognostic marker, particularly in seronegative RA cases. Further subgroup analysis indicated that Anti-CarP-positive patients had higher levels of inflammatory markers, such as ESR and CRP, compared to Anti-CarP-negative RA patients ($p < 0.05$), suggesting that Anti-CarP may reflect more aggressive disease phenotypes.

Discussion: Our results confirm the role of Anti-CarP antibodies in the diagnosis and prognosis of RA, particularly in seronegative patients. ROC analysis demonstrated that Anti-CarP had a sensitivity of 82.5% and specificity of 76.8%, indicating high diagnostic significance. Incorporating Anti-CarP into diagnostic panels may enhance the detection of seronegative RA and improve disease prognosis.

Conclusion: Anti-CarP antibodies can serve as an additional diagnostic biomarker in seronegative RA patients and as a marker of risk for joint damage progression. Integrating Anti-CarP into routine diagnostics may facilitate earlier treatment initiation and improved disease prognosis.

Heterogeneity of the pathogenesis of spondyloarthritis: Role of type 1 interferon in axial involvement

Sotaro Nakajima¹; Haruka Tsuchiya¹;
Risa Yoshihara¹; Kazuyoshi Ishigaki^{1,2,3}; Haruka Takahashi¹;
Tomohisa Okamura⁴; Kazuhiko Yamamoto⁵; Hiroko Kanda^{1,6};
Hirofumi Shoda¹; Tetsuya Tomita⁷; Keishi Fujio¹
¹Department of Allergy and Rheumatology, Graduate School of Medicine, The University of Tokyo; ²Laboratory for Human Immunogenetics, RIKEN Center for Integrative Medical Sciences;