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THE ASSOCIATION BETWEEN RHEUMATOID ARTHRITIS DISEASE ACTIVITY AND THE PREVALENCE OF CARDIOVASCULAR DISEASES IN PATIENTS TREATED AT THE RHEUMATOLOGY CLINIC OF THE INSTITUTE FOR TREATMENT AND REHABILITATION "NIŠKA BANJA"

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Rheumatoid arthritis (RA) is a chronic inflammatory disease that increases the risk of cardiovascular disease (CVD). The relationship between disease activity, therapy and CVD prevalence is still not fully understood.

The paper aims to examine the association between RA activity and therapy type and the frequency of CVD in patients with RA, while accounting for traditional risk factors.

The research includes patients with RA treated at the Rheumatology Clinic of the "Niška Banja" Institute, monitored depending on the activity of the disease, which is shown by the DAS28 index (low/remission, moderate, high) and the type of therapy (DMARDs and biological drugs).

Our findings show that patients with high disease activity have a significantly higher frequency of cardiovascular events compared to patients with moderate disease activity or remission. The therapy had no statistically significant effect on CVD development. Length of illness was not significantly associated with CV outcomes.

It has been shown that inflammation and disease activity play a key role in increasing the risk of CVD, whereas adequate therapy plays a secondary, indirect role. Control of disease activity and monitoring of traditional risk factors are key to CVD prevention in RA patients.

Disease activity, not therapy itself, is the main predictor of cardiovascular events in patients with RA. Achieving remission or low disease activity and controlling traditional risk factors are critical to reducing morbidity and mortality.

Key words: rheumatoid arthritis, cardiovascular diseases, risk factors

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POVEZANOST STEPENA AKTIVNOSTI REUMATOIDNOG ARTRITISA I UČESTALOSTI KARDIOVASKULARNIH BOLESTI KOD BOLESNIKA LEČENIH NA KLINICI ZA REUMATOLOGIJU INSTITUTA ZA LEČENJE I REHABILITACIJU „NIŠKA BANJA"

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Reumatoidni artritis (RA) je hronična inflamatorna bolest koja povećava rizik od kardiovaskularnih bolesti (KVB). Veza između aktivnosti bolesti, terapije i učestalosti KVB još uvek nije u potpunosti razjašnjena.

Cilj rada je da ispita povezanost aktivnosti RA i vrste terapije sa učestalošću KVB kod pacijenata sa RA, uz uvažavanje tradicionalnih faktora rizika.

Istraživanje obuhvata pacijente sa RA lečenih na Klinici za reumatologiju Instituta "Niška Banja", praćene u zavisnosti od aktivnosti bolesti koja je prikazana indeksom DAS28 (nisko/remisija, umereno, visoko) i vrste terapije (DMARDs i biološki lekovi).

Naši nalazi pokazuju da pacijenti sa visokom aktivnošću bolesti imaju značajno veću učestalost kardiovaskularnih događaja u odnosu na pacijente sa umerenom aktivnošću ili remisijom. Terapija nije imala statistički značajan uticaj na razvoj KVB. Dužina trajanja bolesti nije bila značajno povezana sa KV ishodima.

Dokazano je da inflamacija i aktivnost bolesti imaju ključnu ulogu u povećanju rizika od KVB, dok adekvatna terapija ima sekundarnu, indirektnu ulogu. Kontrola aktivnosti bolesti i praćenje tradicionalnih faktora rizika su ključni za prevenciju KVB kod pacijenata sa RA.

Aktivnost bolesti, a ne sama terapija, predstavlja glavni prediktor kardiovaskularnih događaja kod pacijenata sa RA. Postizanje remisije ili niske aktivnosti bolesti i kontrola tradicionalnih faktora rizika su od ključnog značaja za smanjenje morbiditeta i mortaliteta.

Ključne reči: reumatoidni artritis, kardiovaskularne bolesti, faktori rizika

Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune systemic inflammatory disease characterized by autoantibody production, synovial inflammation and hyperplasia, cartilage and bone destruction, as well as systemic manifestations that significantly increase morbidity and mortality rates [1]. One of the most significant extra-articular complications is cardiovascular disease (CVD), which represents the leading cause of morbidity and mortality in RA patients [2,3].

RA patients have a 1.5–2-fold higher risk of developing CVD compared to the general population, including coronary artery disease (CAD), heart failure, arrhythmias, and peripheral vascular disease [4,5]. Increased CVD risk results from the interaction of chronic inflammation, traditional risk factors, and potential effects of therapy [5,6]. Conventional risk factors such as hypertension, dyslipidemia, diabetes mellitus, obesity, and smoking further contribute to cardiovascular risk in RA patients [7,8].

Chronic inflammation characteristic of RA accelerates atherosclerosis, alters lipid metabolism, and increases thrombogenicity, creating a synergistic effect with classical risk factors [9–13]. Pro-inflammatory cytokines—including TNF-α and IL-6 [14,15]—play key roles in vascular pathology, while therapy with synthetic disease-modifying antirheumatic drugs (DMARDs) and biological agents may partially mitigate these processes. In contrast, long-term corticosteroid therapy may worsen cardiometabolic profiles [16,17].

Several studies have shown that disease activity directly correlates with CVD prevalence, with patients exhibiting high disease activity having a significantly increased risk of cardiovascular (CV) events compared to those with moderate activity or remission [4,6,9]. Understanding this relationship while accounting for standard risk factors is essential for optimizing therapy and preventing CVD in RA patients [18].

Aim

This study aimed to examine the presence of CVD (myocardial infarction (MI) with and without percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), and cerebrovascular insult (CVI)) in RA patients in relation to disease duration, disease activity, and treatment modality.

Materials and Methods

This cross-sectional study included 220 consecutive RA patients (mean age 68.04 ± 6.63 years): 128 men (58.2%) and 92 women (41.8%), treated at the Rheumatology Clinic in “Niška Banja.”

All patients were receiving Methotrexate (MTX), and 88 patients additionally received a JAK inhibitor.

We assessed disease activity using the DAS28 index. Based on disease activity, we categorized patients into three groups:

a) low activity/remission,

b) moderate activity,

c) high activity.

Within these groups, we analyzed the prevalence of CVD (MI with/without PCI, CABG) and the prevalence of all CVDs (CVD+CVI).

Results

Of all RA patients, 71 (32.3%) had low/remission activity, 84 (38.2%) moderate activity, and 65 (29.5%) high disease activity (*Table 1*).

Table 1. Demographic, clinical, and laboratory characteristics of subjects according to the level of disease activity (DAS28 SE)

Tested parameters in patients with RA	All participants: n=220 (100%)	DAS activity			p=
		Low/remission n=71 (32.3%)	Moderate n=84 (38.2%)	High n=65 (29.5%)	
Gender (male)	128 (58.2%)	62 (87.3%)	47 (56%)	19 (29.2%)	0.000
Age (years)	68.04±6.63	68.39±8.64	67.39±5.52	68.49±5.37	0.523
Duration of illness (years)	13.27±6.23	12.04±7.08	13.31±6.38	14.55±4.65	0.042
Sedimentation (ESR)	32.70±18.05	20.15±5.58	28.32±10.94	52.06±18.56	0.000
CRP	10.40±24.42	1.89±2.68	6.74±5.63	24.41±41.17	0.000
Painful joints	8.06±6.63	3.28±2.11	8.27±4.93	13.02±7.97	0.000
Swollen joints	3.04±5.14	0.38±0.74	2.12±1.56	7.14±7.78	0.000
VAS pain scale	37.50±12.55	27.28±9.93	37.36±8.61	48.86±9.39	0.000
MTX (dose in mg)	13.94±3.07	11.69±2.83	14.11±2.56	16.12±2.03	0.000
MTX + biological/JAK Th	88 (40%)	13 (18.3%)	37 (44%)	38 (58.5%)	0.000

CV (MI+PCI+CABG)	57 (25.9%)	3 (4.2%)	19 (22.6 %)	35 (53.8%)	0.000
CV+CVI	7 (3.2%)	1 (1.4%)	3 (3.6%)	3 (4.6%)	0.549

Sex distribution significantly differed between groups, with the highest proportion of men in the remission group (87.3%) and the lowest in the high-activity group (29.2%).

Age did not differ significantly across activity groups ($p = 0.523$).

Disease duration was significantly longer in the high-activity group (14.55 years) and shortest in remission (12.04 years) ($p = 0.042$).

Inflammatory markers progressively increased with disease activity: ESR rose from remission to moderate and high activity; CRP values were lowest in remission (1.89) and highest in high activity (24.41).

Clinical disease activity indicators were lowest in remission, intermediate in moderate activity, and highest in high activity:

a) tender joints (3.28 vs. 8.27 vs. 13.02),

b) swollen joints (0.38 vs. 2.12 vs. 7.14),

c) VAS pain scale (27.28 vs. 37.36 vs. 48.86).

The MTX dose was highest in the high-activity group (16.12 mg) and lowest in the remission group (11.69 mg). The use of combined MTX+biologic/JAK therapy was most frequent in the high-activity group (58.5%) and least in remission (18.3%).

A total of 57 cardiovascular events (25.9%) were recorded, with a significantly higher prevalence in the high-activity group than in the moderate and low-activity groups (53.8% vs. 22.6% vs. 4.2%).

Combined events with stroke did not differ significantly across groups ($p = 0.549$). Therapy type (MTX vs. MTX + biologic/JAK) was not associated with CVD or combined CV and cerebrovascular events.

To confirm the previously shown differences in the frequency of CV events between groups defined by disease activity level, a binary logistic regression was performed to examine the association between DAS28 categories and the presence of CV events alone and combined with cerebrovascular events (*Table 2*). In both models, we used high disease activity as the reference category, enabling assessment of the relative risk of remission and moderate activity compared with the most active form of the disease.

Results for CV events show that patients in remission had a significantly lower event risk (OR=0.038; p<0.001), as did patients with moderate activity (OR=0.251; p<0.001) compared to patients with high disease activity. These results confirm previous differences and indicate a significant association between the degree of inflammatory activity and the frequency of CV events. Therefore, DAS28 categories may be essential and independent predictors of CV events (*Table 2*).

Table 2. Association of RA activity level with cardiovascular and cerebrovascular events

DAS28 CATEGORY	**CV**			**CV+CVI**		
	OR	95% CI	p	OR	95% CI	p
Remission	0.038	0.011 – 0.133	<0.001	0.295	0.030 – 2.912	0.296
Moderate activity	0.251	0.124 – 0.508	<0.001	0.765	0.149 – 3.923	0.749
High activity	Reference category in both regressions					

In contrast, in the model for CV+CVI events, remission and moderate disease activity did not differ significantly from high activity (OR=0.295; p=0.765), confirming previous results.

We examined the association between treatment modality and CV event frequency using χ^2 analysis, with the treatment variable defined as binary: methotrexate (MTX) monotherapy, which treated all patients, and combination therapy (MTX with a biological drug or a JAK inhibitor), which additionally treated 88 patients.

The results of the χ^2-test (*Table 3*) show a statistically significant association between therapy modality and cardiovascular events, with a higher event rate in monotherapy than in combined therapy (71.9% vs. 28.1%, p<0.05). In contrast, we found no significant difference in the frequency of CV and cerebrovascular events between the treatment groups.

Table 3. Association of therapeutic modality with frequency of cardiovascular events

Cardiovascular events			Therapy		Total	Pearson X^2	p
			Monotherapy	Combined			
MI+Stent+CABG	no	number	91	72	163	4.562	0.041
		%	55.8%	44.2%	100%		
	yes	number	41	16	57		
		%	71.9%	28.1%	100%		
MI+Stent+CABG +CVI	no	number	127	86	213	0.393	0.705
		%	59.6%	40.4%	100%		
	yes	number	5	2	7		
		%	71.4%	28.6%	100%		
In total		number	132	88	220		
		%	60%	40%	100%		

Based on the results, the therapy modality shows a significant association with CV event frequency, suggesting substantial predictive value. In contrast, no significant association was observed for the combined outcomes.

To examine the association between different levels and durations of RA and the implemented therapy modality with CV and cerebrovascular events, we conducted a binary logistic regression analysis. The methotrexate dose was excluded from the study because it did not reach statistical significance in the preliminary models.

For the outcome MI+Stent+CABG (*Table 4, first model*), the results show that combined therapy (MTX+biological/JAK) is significantly negatively associated with the presence of CV events (B = –2.062; OR = 0.127; $p < 0.001$), which means that the risk in this group is reduced by 87.3% (1 - OR) compared to monotherapy, that is, combined therapy minimizes the probability of events 7.9 times (1 / OR). At the same time, a higher DAS28 shows a strong positive association with the risk (B = 2.091; OR = 8.089; $p < 0.001$), which means that the risk increases more than eight times with a higher level of RA activity. Since both predictors are significant ($p < 0.001$), the Wald test shows that DAS28 disease activity is more important than therapeutic modality (40,497 vs. 20,639). Therefore, disease activity and therapeutic modality may be significant and independent predictors of CV events, whereas disease duration is not.

Table 4. Association between activity level and duration of rheumatoid arthritis and therapy modality with cardiovascular and cerebrovascular events

Cardiovascular events	Parameter	B	S.E.	Wald	Sig.	Exp(B)	95% CI for EXP(B)	
							Lower	Upper
MI+Stent+CABG	MTX+Biological therapy	-2.062	0.454	20.639	0.000	0.127	0.052	0,310
	DAS28	2.091	0.329	40.497	0.000	8.089	4.249	15,401
	RA duration	0.042	0.032	1.763	0.184	1.043	0.980	1,110
	Constant	-5.423	0.834	42.227	0.000	0.004		
MI+Stent+CABG +CVI	MTX+Biological therapy	-0.826	0.883	0.875	0.350	0.438	0.077	2,472
	DAS28	0.757	0.546	1.926	0.165	2.132	0.732	6,212
	RA duration	-0.044	0.068	0.423	0.516	0.957	0.837	1,093
	Constant	-4.178	1.337	9.765	0.002	0.015		

† B – Regression coefficient, SE – Standard error, Wald – Significance of the Regression Coefficient B, Sig. – Significance (p-value), OR (Exp(B) – Odds ratio, CI – Confidence interval.

For the extended outcome (MI+Stent+CABG+CVI), none of the predictors were statistically significant, most likely due to the small sample size (n = 7), which limited the model's statistical power.

These results confirmed the conclusion of the previous analysis (*Table 3*) that the frequency of CV events is 3 times lower in the combined therapy group than in the monotherapy group (28.1% < 71.9%; p = 0.041) and that there is no statistically significant difference in CV and CVI.

Figure 1 shows the ROC curve of significant predictors of CV events. DAS28 disease activity has a negative and more significant discriminative ability (AUC = 0.774, p = 0.000), while combined therapy (MTX + Biological therapy) shows a weaker and positive predictive value (AUC = 0.419, p = 0.041), which confirms that the therapeutic effect comes to the fore only in a multivariate context, when taken into account together with other clinical parameters.

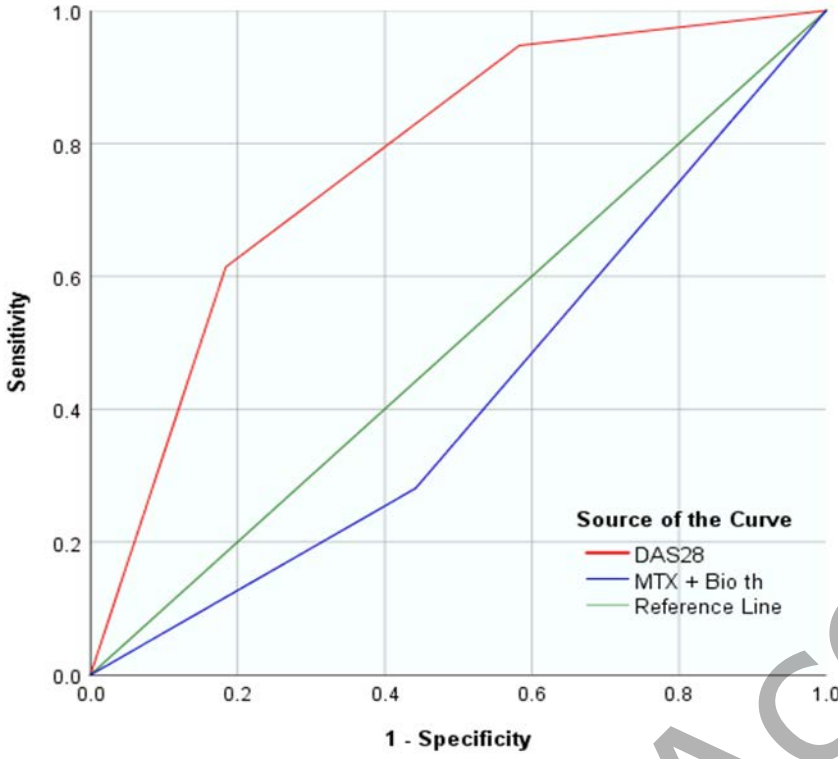

Figure 1. ROC curve for DAS28 and therapy modality in predicting CV events

Discussion

Based on the results presented, we conclude that there is a significant association between rheumatoid arthritis severity and the frequency of cardiovascular disease. Subjects with high disease activity had a significantly higher frequency of MI, PCI, and CABG than patients with moderate activity or remission, suggesting that increased inflammatory activity may contribute to the development of atherosclerotic changes and increased CV risk in patients with RA.

In a meta-analysis that included over 80,000 patients with RA, Aviña-Zubieta et al. [3] showed that the overall risk of MI in patients with RA is 1.5-2 times higher than in the general population. These researchers emphasized that disease activity, as measured by CRP levels and the DAS28 index, directly correlates with the incidence of CV events. Myasoedova et al. [7] in a longitudinal cohort of 600 patients monitored the frequency of CV events according to disease activity. Their analysis showed that patients with high disease activity have almost twice the risk of MI and CVI compared to patients in remission, which is consistent with our findings, which showed that high disease activity was the main predictor of CVD.

Ruscitti et al. [5] conducted a study of 350 patients and compared the incidence of CVD among patients receiving DMARDs, biologic therapy, or corticosteroids. Their analysis showed no significant difference in CVD incidence between the treatment groups, confirming our findings: the therapy itself did not significantly affect the risk.

Del Rincón et al [8] investigated the correlation of inflammation and CVD in a cohort of 575 patients. They found that chronic inflammation, especially elevated levels of CRP and ESR, increases the risk of MI and angina pectoris, independently of classical risk factors, underscoring the role of inflammation and disease activity. In contrast, classical therapy plays a secondary role.

Giles et al [10] studied subclinical atherosclerosis in patients with RA using ultrasound measurement of carotid artery intima-media thickness. The results showed that patients with high disease activity have significantly greater intima-media thickness, which is an indicator of increased CV risk. These findings are in direct agreement with our analysis. Goodson et al [19] in a study with 1,000 patients showed that patients with high RA activity had an increased incidence of CVD, and the therapeutic regimen did not significantly affect the outcome. Peters et al [20] in their cohort of over 1,500 patients also confirm that a high level of disease activity is associated with a higher risk of MI. At the same time, DMARDs or biological drugs did not show a statistically significant effect. Crowson et al. [21], analyzing longitudinal data, found that early, aggressive control of inflammation significantly reduces the risk of CV events, underscoring the importance of achieving remission or low disease activity as a key CVD prevention strategy. In a study of 855 patients with RA, Eleke E et al. [22] showed that the temporal course of risk did not indicate a change in CVD risk over the course of RA.

Our findings further confirm these data and show that patients with high disease activity have an increased risk of all CV events. At the same time, the type of therapy did not significantly affect the incidence of CVD. The results indicate that controlling inflammation and disease activity are primary in preventing CVD, while the therapeutic regimen plays a secondary, indirect role.

Also, our results emphasize the need for a comprehensive approach: controlling traditional risk factors (hypertension, dyslipidemia, diabetes, smoking), patient education, regular cardiovascular status monitoring, and achieving remission or low disease activity are key steps in reducing morbidity and mortality in RA patients.

Overall, our analysis is consistent with previous studies and confirms that disease activity directly affects CVD incidence, whereas therapy alone does not significantly alter risk. Furthermore, it

emphasizes the need for intensive control of disease activity, especially in patients with high inflammatory markers and clinical signs of active disease.

Conclusion

Finally, we can conclude that:

• Patients with RA have a multiple increased risk of CVD, especially those with high disease activity;

• Control of inflammation, optimization of therapy, and monitoring of traditional risk factors are key to the prevention of CV complications and improvement of patients' quality of life;

• The therapy itself did not significantly affect the development of coronary and other CVDs, which emphasizes the importance of achieving remission or low disease activity.

References:

1. Nagy G, Roodenrijs NMT, Welsing PM, Kedves M, Hamar A, van der Goes MC, et al. EULAR definition of difficult-to-treat rheumatoid arthritis. Ann Rheum Dis. 2021 Jan;80(1):31-35. https://doi.org/10.1136/annrheumdis-2020-217344
2. Nishant J, Agarwal A, Mahajan V, Singh S, Arora R. Association of cardiovascular risks in rheumatoid arthritis patients: Management, treatment, and future perspectives. Health Sci Rev. 2023;80:100–108. https://doi.org/10.1016/j.hsr.2023.100108
3. Aviña-Zubieta JA, Choi HK, Sadatsafavi M, Etminan M, Esdaile JM, Lacaille D. Risk of cardiovascular mortality in rheumatoid arthritis: a meta-analysis. Arthritis Rheum. 2008;59:1690–1697. https://doi.org/10.1002/art.24092
4. Kitas GD, Gabriel SE. Cardiovascular disease in rheumatoid arthritis: state of the art and future perspectives. Ann Rheum Dis. 2011;70:8–14. https://doi.org/10.1136/ard.2010.142133
5. Ruscitti P, Cipriani P, Liakouli V, Iacono D, Pantano I, Caso F, et al. Increased cardiovascular events and subclinical atherosclerosis in rheumatoid arthritis patients: one-year prospective study. PLoS One. 2017;12:e0170005. https://doi.org/10.1371/journal.pone.0170108
6. Fragoulis GE, McInnes IB. Cardiovascular risk and rheumatoid arthritis: pathogenesis, evidence and implications. Nat Rev Cardiol. 2021;18:233–244. https://doi.org/10.2174/1570161117666190619143842
7. Myasoedova E, Davis J, Roger L, Sara J. Crowson C. The Journal of Rheumatology September 2021, 48 (9) 1379-1387. https://doi.org/10.3899/jrheum.200842
8. Del Rincón I, Williams K, Stern MP, Freeman GL, Escalante A. High incidence of cardiovascular events in rheumatoid arthritis cohort not explained by traditional risk factors. Arthritis Rheum. 2001;44:2737–2745. https://doi.org/10.1002/1529-0131(200112)44:12<2737::AID-ART460>3.0.CO;2-%23
9. Provan SA, Lillegraven S, Sexton J, Uhlig M. Inflammatory burden and CV events in RA: a prospective registry analysis. Arthritis Res Ther. 2022;24:84 https://doi.org/10.1093/rheumatology/kez371

10. Giles JT, Post WS, Blumenthal RS, Polak JF, Petri M, Gelber AC, et al. Longitudinal predictors of progression of carotid atherosclerosis in rheumatoid arthritis. Arthritis Rheum. 2011;63:3216–3225. https://doi.org/10.1002/art.30542
11. Wu CY, Yang HY, Luo SF, Chen YH, Chen DY. Impact of disease activity on cardiovascular outcomes in rheumatoid arthritis: a nationwide cohort study. Rheumatology. 2023;62:1128–1137. https://doi.org/10.3390/jcm11226812
12. Özkan U, Kakilli N, Gürdoğan M, Taştekin N, Birtane M. Rheumatoid arthritis and cardiovascular comorbidities. Explor Musculoskeletal Dis. 2023;1:264–88. doi.org/10.37349/emd.2023.00028
13. Van Halm VP, Nielen MMJ, Nurmohamed MT, van Schaardenburg D, Reesink HW, Voskuyl AE, et al. Lipids and inflammation: serial measurements in donors who later developed RA. Ann Rheum Dis. 2007;66:184–188. https://doi.org/10.1136/ard.2006.051672
14. Luciano, N., Barone, E., Timilsina, S. et al. Tumor Necrosis Factor Alpha Inhibitors and Cardiovascular Risk in Rheumatoid Arthritis. Clinic Rev Allerg Immunol 65, 403–419 (2023). https://doi.org/10.1007/s12016-023-08975-z
15. Roghani SA, Shamsi A, Jalili C, Jalili F, Lotfi R, Garman N, Rostampour R, Taghadosi M. Interleukin-6 positively correlates with cardiovascular disease predictor algorithms and biomarker in rheumatoid arthritis patients. J Cell Mol Med. 2024 Aug;28(16):e70028. https://doi.org/10.1111/jcmm.70028
16. Liu, D., Yu, G., Yuan, N., & Nie, D. (2025). The efficacy and safety of biologic or targeted synthetic DMARDs in rheumatoid arthritis treatment: one year of review 2024. Allergologia Et Immunopathologia, 53(3), 140-162. https://doi.org/10.15586/aei.v53i3.1278
17. Atzeni F, Sarzi-Puttini P, et al. Cardiovascular effects of methotrexate and biologics in RA: systematic review. Autoimmun Rev. 2021;20:102736. https://doi.org/10.1038/s41584-021-00593-3
18. Semb, A.G., Ikdahl, E., Wibetoe. Prevention of atherosclerotic cardiovascular disease in rheumatoid arthritis. Nat Rev Rheumatol 16, 361–379 (2020). https://doi.org/10.1038/s41584-020-0428-y
19. Goodson NJ et al. Baseline levels of C-reactive protein and prediction of death from cardiovascular disease in patients with inflammatory polyarthritis: a ten-year followup study of a primary care-based inception cohort.Arhritis Rheum.2005;52:2293-2299. https://doi.org/10.1002/art.21204

20. Peters MJ et al. Does rheumatoid arthritis equal diabetes mellitus as an independent risk factor for cardiovascular disease? A prospective study.Arthritis Rheum. 2009;61:1571-1579. https://doi.org/10.1002/art.24836
21. Crowson CS, Rollefstad S, Kitas GD, van Riel P, Gabriel SE, Semb AG. Risk scores for CV disease in RA. Am J Cardiol. 2012;110:420–424. https://doi.org/10.1093/rheumatology/kex038
22. Eleke E et al. The effect of disease duration and disease activity on the risk of cardiovascular disease in rheumatoid arthritis patients 2015;74:998-1003. https://api.semanticscholar.org/CorpusID:31581977