

Cardiovascular Risk Factors in Patients With Inflammatory Arthritis

Esraa E. Othman^{1*}, Asmaa Farouk Enein², Fatma Hamdy³

Rheumatology & Immunology unit, Internal Medicine Department, Faculty of medicine, Mansoura
University

Abstract:

Background: Inflammatory arthritis, involving rheumatoid arthritis (RA) and seronegative spondyloarthropathy (SpA), is associated with elevated cardiovascular (CV) risk, partly due to systemic inflammation and microvascular dysfunction. **Objectives:** To assess the differences in cardiovascular risk factors between patients with RA and SpA, and to examine the relationship between disease activity and cardiovascular risk in individuals with inflammatory arthritis. **Methods:** This study was done on 50 patients (25 RA, 25 SpA) at Mansoura University Hospital. All participants underwent demographic, clinical, and laboratory evaluation, carotid intima-media thickness (CIMT) measurement. CV risk was calculated using the Framingham risk score. **Results:** Patients with RA showed significantly higher body mass index (BMI) and platelet counts compared to those with SpA. Additionally, diabetes was notably more prevalent among RA patients. However, no significant differences were observed between the groups in terms of CIMT or cardiovascular risk score parameters. **Conclusion:** RA and SPA had comparable CV risk profiles despite some differences in demographic and treatment patterns. Higher disease activity was correlated with lower HDL cholesterol in RA patients.

Keywords: Arthritis- Spondyloarthritis - Cardiovascular risk – Rheumatoid – Carotid intimal medial thickness.

Introduction:

Rheumatoid arthritis (RA) is the most common chronic inflammatory condition affecting the joints. It is marked by persistent and damaging inflammation of the synovial membrane, which can result in permanent joint destruction. Beyond joint issues, the widespread inflammation associated with RA can also affect other organs, leading to complications such as interstitial lung disease, vasculitis, and cardiovascular disorders (*Dijkshoorn et al., 2022*). Spondyloarthritis (SpA) encompasses a range of long-term inflammatory disorders, including axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA), along with other peripheral forms such as enteropathic arthritis, reactive arthritis, and undifferentiated SpA. AxSpA mainly targets the sacroiliac joints and spine, where inflammation in the vertebrae, connective tissues, and joints results in ongoing back pain and can eventually cause the vertebrae to fuse together. (*Adhikari et al., 2012*).

Inflammatory arthritis such as RA and SPA are associated with extra-articular morbidity including increased cardiovascular disease (CVD) risk. Patients with RA were found to have a persistently more incidence of atherosclerotic events in comparison to healthy people. Elevated cardiovascular risk is attributed to both systemic inflammation and a higher burden of conventional cardiovascular risk factors (*Dijkshoorn et al., 2022*). Systemic inflammation promotes endothelial activation and dysfunction and contributes to accelerated macrovascular disease in inflammatory arthritis (*Caraba et al., 2024*)

Aim of the study:

The objective of this study is to compare cardiovascular risk factors in patients with RA and SpA, and to investigate the association between disease activity and cardiovascular risk in individuals with inflammatory arthritis.

Patients and methods:

This was a cross-sectional analytic study conducted at the Rheumatology & Immunology outpatient clinics, Internal Medicine Department, Mansoura University Hospital, Egypt. The study was conducted over a 12-month period, from March 2022 to March 2023. Ethical approval was obtained from the Institutional Review Board of the Faculty of Medicine, Mansoura University (IRB code: MS.22.12.2235). All participants provided written informed consent after full clarification of study.

A total of 50 patients with inflammatory arthritis were included in this study. Patients were divided into two groups:

- Group 1: 25 patients diagnosed with RA
- Group 2: 25 patients diagnosed with SPA, comprising 14 patients with axial SPA and 11 patients with PsA.

The study included patients aged over 18 years. RA was diagnosed based on the 2010 ACR/EULAR classification criteria (*Aletaha et al., 2010*). SpA was diagnosed according to the 2009 ASAS classification criteria (*Rudwaleit et al., 2009*) while PsA was identified using the CASPAR criteria (*Taylor et al., 2006*). Patients with current active infections, history of malignancy within the past 5 years, overlap syndromes or patients with coexisting autoimmune diseases were excluded from this study.

All participants underwent comprehensive assessment including clinical and laboratory evaluation. Full history was taken including demographic data (age, sex, occupation, habits), disease duration, and current treatment. Clinical examination included general examination, anthropometric measurements (weight, height, BMI), and musculoskeletal examination. Disease activity was assessed. Disease activity in RA patients was assessed using the Disease Activity Score-28 (DAS28) (*Prevoo et al., 1995*). For patients with axial SpA, the Ankylosing Spondylitis Disease Activity Score (ASDAS) was used (*Machado et al., 2011*). In cases of psoriatic arthritis, disease activity was evaluated using the Disease Activity index for Psoriatic Arthritis (DAPSA) score (*Schoels et al., 2016*). The following laboratory tests were done for all the patients including complete blood count, ESR (erythrocyte sedimentation rate), CRP (C reactive protein), liver function tests, fasting lipid profile. Cardiovascular risk was estimated using Framingham risk score (*Wilson et al., 1998*). Cardiovascular risk was classified using the Framingham Risk Score into three categories: low (<10%), moderate (10–20%), and high (>20%).

CIMT was measured bilaterally 2 cm proximal to the carotid bifurcation using a 7.5 MHz linear probe by the same operator. Three measurements were averaged for each side. Atherosclerotic plaques or stenosis were also documented. CIMT was defined as the distance between the luminal–intimal and medial–adventitial interfaces (*Darabian et al., 2013*).

Data were collected, reviewed, verified, and then analyzed using the Statistical Package for the Social Sciences (SPSS) for Windows (SPSS, Inc., Chicago, IL, USA). To assess the normality of variable distributions, the Kolmogorov-Smirnov test was applied. For normally distributed (parametric) data, results were expressed as mean and standard deviation (SD), while non-normally distributed (non-parametric) data were reported using the median and interquartile range. Categorical (qualitative) data were presented as frequencies and percentages. To compare continuous variables, the independent samples t-test was used for data following a normal distribution, and the Mann-Whitney U test was applied for non-normally distributed data. The chi-square test was used for comparing categorical variables. When comparing means across more than two groups, one-way ANOVA was used for normally distributed

variables, and the Kruskal-Wallis test was employed for non-parametric variables. Correlations between continuous variables were assessed using either Pearson's or Spearman's correlation coefficients, depending on the data distribution. A p-value of less than 0.05 was considered statistically significant.

Results:

Demographic and clinical characteristics of the study participants are presented in Table 1. Female patients were significantly more prevalent in the RA group compared to the SpA group (96% vs. 72%, $P = 0.02$). RA patients also had a significantly higher BMI than SpA patients (34.6 vs. 30.5 kg/m², $P = 0.01$). A significant difference in treatment patterns was observed between the two groups. Methotrexate, leflunomide, hydroxychloroquine, corticosteroids, and JAK inhibitors were more commonly prescribed in RA patients ($P = 0.01$, <0.001 , <0.001 , 0.04, and 0.04, respectively), whereas sulfasalazine and anti-TNF agents were more frequently used in SpA patients ($P = 0.007$ and 0.01, respectively).

Laboratory findings are detailed in Table 2. Platelet counts were significantly higher in RA patients compared to SpA patients (272 vs. 233 $\times 10^9/L$, $P = 0.04$). Cardiovascular risk factors are summarized in Table 3. Diabetes was significantly more common in RA patients than in those with SpA (12% vs. 4%, $P = 0.04$). No other significant differences in cardiovascular risk factors were observed between the two groups. The majority of patients in both groups were categorized as having low cardiovascular risk, with 92% of RA patients and 88% of SpA patients falling into this category. Table 4 shows the correlation between disease activity and cardiovascular risk parameters. In RA patients, a significant negative correlation was found between disease activity and HDL cholesterol levels ($R = -0.4$, $P = 0.04$).

Table 1. Clinical and demographic characteristics of patients with rheumatoid arthritis and seronegative spondyloarthritis.

Parameter	Rheumatoid arthritis N=25	Sero-negative arthritis N=25	P-value
Age (years)mean $\pm$ SD	44 $\pm$ 10.23	38.8 $\pm$ 10.6	0.07
Gender			
Females N (%)	24 (96%)	18 (72%)	0.021*
Males N (%)	1 (4%)	7 (28%)	
BMI Kg/m ² mean $\pm$ SD	34.6 $\pm$ 6.9	30.5 $\pm$ 4.7	0.013*
Disease duration (years) median (IQR)	5 (3-7)	4 (1-5)	0.16
Tender joint count median (IQR)	3 (2-10)	2 (2-4)	0.09
Swollen joint count median (IQR)	0 (0-2)	0 (0-2)	0.26
Patient global assessment mean $\pm$ SD	7.68 $\pm$ 0.62	7.4 $\pm$ 0.7	0.11
Morning stiffness >1hour N (%)	12 (48%)	15 (60%)	0.39
Extra-articular manifestation N (%)	2 (8%)	4 (16 %)	0.38
ILD N (%)	0 (0%)	1 (4%)	
Sjogren N (%)	2 (8%)	0 (0%)	

Uveitis N (%)	0 (0%)	2 (8%)	
IBD N (%)	0 (0%)	1 (4 %)	
Methotrexate N (%)	16 (64%)	7 (28%)	0.01*
Leflunomide N (%)	22 (88%)	3 (12%)	<0.001*
Hydroxychloroquine N (%)	18 (72%)	0 (0%)	<0.001*
Sulfasalazine N (%)	4 (16%)	13 (52%)	0.007*
Steroids N (%)	6 (24%)	1 (4%)	0.042*
IL-17 inhibitors N (%)	0 (0%)	5 (20%)	0.018*
JAK-inhibitors N (%)	6 (24%)	1 (4%)	0.042*
Anti-TNF N (%)	0 (0%)	9 (36%)	0.01*
Adalimumab N (%)	0 (0%)	4 (16 %)	
Golimumab N (%)	0(0%)	4 (16%)	
Infliximab N (%)	0(0%)	1 (4%)	

Data are presented as mean $\pm$ standard deviation (SD), median (interquartile range [IQR]), or n (%). P-values are based on Student's t-test or Mann–Whitney U test for continuous variables and χ^2 or Fisher's exact test for categorical variables. $P < 0.05$ considered statistically significant.

BMI: Body mass index, ILD: interstitial lung disease, IBD: Inflammatory bowel disease, Anti –TNF: anti-tumor necrosis factor, IL-17: interleukin 17, JAK inhibitors: janus kinase inhibitors.

Table 2. Laboratory parameters in patients with rheumatoid arthritis and seronegative spondyloarthritis.

Parameter	Rheumatoid Arthritis N=25	Sero negative Arthritis N=25	P value
ESR (mm/hour) median (IQR)	30 (16 -59)	30 (30 -37)	0.28
CRP(mg/dL) median (IQR)	0 (0-1)	0(0-1)	0.624
LDL-cholesterol (mg/dl) mean $\pm$ SD	95 $\pm$ 30	107 $\pm$ 34	0.214
HDL-cholesterol (mg/dl)mean $\pm$ SD	47.5 $\pm$ 9.5	45 $\pm$ 11	0.18
Total Cholesterol(mg/dl) mean $\pm$ SD	181 $\pm$ 37.2	199 $\pm$ 45	0.26
WBCS(cells/ μ L) mean $\pm$ SD	7 $\pm$ 2.1	6.8 $\pm$ 1.7	0.915
HB level (g/dl) mean $\pm$ SD	11 $\pm$ 1.4	11.5 $\pm$ 1.2	0.42
Platelets count (plts / μ l) mean $\pm$ SD	272 $\pm$ 96	233 $\pm$ 60	0.042*

Values are presented as mean $\pm$ SD or median (IQR). P-values calculated using Student's t-test or Mann–Whitney U test as appropriate. $P < 0.05$ considered statistically significant.

ESR: erythrocyte sedimentation rate, CRP: c-reactive protein, LDL: low density lipoprotein, HDL: high density lipoprotein, WBCS: white blood cells count, HB: hemoglobin.

Table 3. Cardiovascular risk factors in patients with rheumatoid arthritis and seronegative spondyloarthritis.

Parameter	Rheumatoid arthritis N=25	Sero-negative arthritis N=25	P value
-----------	------------------------------	---------------------------------	---------

Smoker N (%)	0 (0%)	1 (4%)	0.31
Diabetes N (%)	3 (12%)	1 (4%)	0.029*
Hypertension N (%)	4 (16%)	5 (20%)	0.71
Hypertension medications N (%)	1 (4%)	3 (12%)	0.29
Carotid intimal medial thickness (mm) median (IQR)	0.5 (0.4 - 0.6)	0.5 (0.4 - 0.58)	0.63
Cardiovascular risk score median (IQR)	3.3 (1.7 - 5.9)	2.4 (1.5 - 5.4)	0.7
Cardiovascular risk :			0.83
➤ Low risk N (%)	23 (92%)	22 (88%)	
➤ Moderate risk N (%)	1 (4%)	2 (8%)	
➤ High risk N (%)	1 (4%)	1 (4%)	

Values are presented as numbers (%) or median (IQR). P-values derived from χ^2 /Fisher's exact test for categorical variables and Mann-Whitney U test for continuous variables. $P < 0.05$ considered statistically significant.

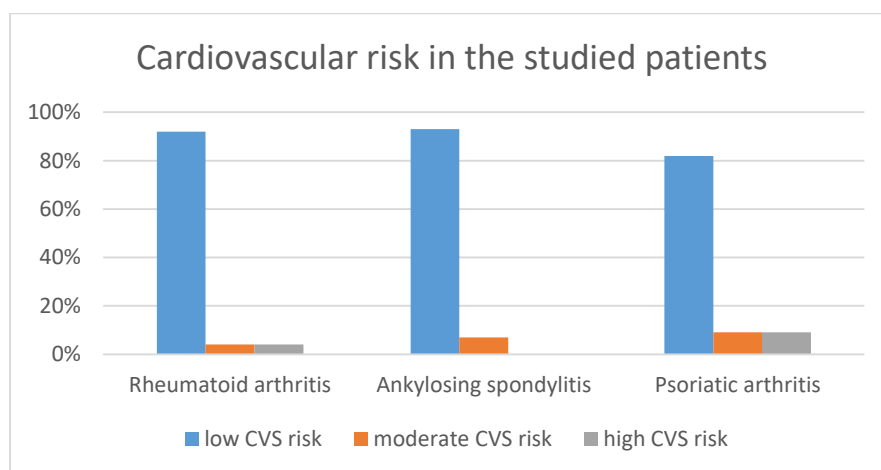


Figure 1. Distribution of cardiovascular risk categories among the studied patients

Table 4. Correlation between disease activity and cardiovascular risk parameters in patients with inflammatory arthritis.

Parameter	Rheumatoid arthritis N=25		Ankylosing spondylitis N=14		Psoriatic arthritis N=11	
	R	P	R	P	R	P
Age	-0.2	0.3	0.04	0.9	0.3	0.4
BMI	-0.04	0.9	-0.01	0.9	0.0	0.3
CIMT	0.02	0.9	-0.4	0.2	-0.1	0.8

Cardiovascular risk score	-0.3	0.2	-0.2	0.6	0.4	0.2
Total cholesterol	-0.2	0.3	-0.007	0.9	0.5	0.1
LDL cholesterol	-0.4	0.07	0.06	0.8	0.3	0.4
HDL cholesterol	-0.4	0.04*	-0.2	0.4	0.2	0.5

Values are Pearson's correlation coefficients (R) with corresponding P-values. $P < 0.05$ considered statistically significant.

BMI: body mass index, CIMT: carotid intimal medial thickness, LDL: low density lipoproteins, HDL: high density lipoprotein

Discussion:

Individuals with autoimmune diseases face a heightened risk of CVD and related mortality. This increased risk is largely driven by immune system mechanisms, making traditional risk factors alone inadequate for accurate risk assessment (*Sivakumaran et al., 2021*). This study was conducted on 25 RA patients and 25 sero negative SPA patients (sero negative group including 11 PsA patients and 14 AS patients) to evaluate cardiovascular risk in patients with inflammatory arthritis. There was no difference in cardiovascular risk score between RA and SPA patients. A significant correlation was observed between disease activity and HDL cholesterol levels in patients with RA.

The complete blood count (CBC) revealed a significantly elevated platelet count in the RA group compared to the SPA group. This finding aligns with the results of Khan et al. who also observed a statistically significant difference in platelet levels between patients with rheumatoid arthritis (RA) and ankylosing spondylitis (AS). Their study suggested that elevated platelet counts in RA are associated with both clinical and radiological disease progression. (*Khan et al., 2024*). Platelets contribute to inflammation in RA through various mechanisms. When activated, they release microparticles that interact with neutrophils, largely through the expression of platelet-type lipoxygenase and activation of the eicosanoid pathway (*Duchez et al., 2019*). In RA, synovial neutrophils are often found coated with these platelet-derived microparticles, which can exacerbate synovial inflammation (*Li et al., 2021*). Moreover, platelets influence neutrophil activity by modulating their chemotactic and cytotoxic responses via specific surface receptors. The formation of platelet-neutrophil complexes, especially in the presence of anti-citrullinated protein antibodies (ACPA), can activate inflammatory pathways that contribute to the development of inflammatory polyarthritis (*Catrina et al., 2021*).

No statistically significant differences were found between the RA and SpA groups in terms of hypertension and cardiovascular risk scores, consistent with findings from a previous study involving 98 RA and 41 SpA patients. However, in the present study, diabetes mellitus (DM) was significantly more common among RA patients compared to those with SpA. This discrepancy may be related to the more frequent use of steroids in RA treatment compared to SpA. In contrast, the earlier study reported no significant difference in DM prevalence between the two groups. Additionally, patients in the previous study had lower BMI values compared to those in the current study (*Ağadayı et al., 2019*).

No statistically significant difference in carotid intima-media thickness (CIMT) was observed between the RA and SpA groups in this study. However, a previous study by Ali et al reported significantly higher CIMT values in SpA patients compared to those with RA, which may be attributed to a longer disease duration in their patient cohort (*Ali et al., 2020*). Similarly, Skare et al. found that SpA patients exhibited significantly increased CIMT, with a positive correlation between CIMT and disease duration also longer than that observed in the current study (*Skare et al., 2013*).

In this study, no association was found between disease activity and age, BMI, CIMT, total cholesterol, or LDL cholesterol in RA patients. However, a significant negative correlation was observed between disease activity and HDL cholesterol levels ($p = 0.04$). Similar to these results, Ngoc et al reported no correlation between CIMT and DAS28 score (*Ngoc et al., 2023*). Also, another study showed no correlation between CIMT and RF, anti-CCP, CRP concentrations, and DAS28-CRP (*Rojas-Giménez et al. 2022*). Additionally, Ozisler et al reported a significant correlation between CIMT and age but not with RF, anti-CCP, and CRP concentrations (*Ozisler et al., 2018*). Conversely, another study by Rajabzadeh et al reported a significant increase in CIMT corresponding with higher disease activity, as measured by DAS28, in RA patients. (*Rajabzadeh et al., 2023*). Similarly, previous research has also shown that CIMT tends to increase as disease severity worsens in RA (*Targońska-Stepniak et al., 2011*) (*van Breukelen et al., 2015*).

Consistent with the findings of the current study, Chang et al reported a negative correlation between inflammation indicated by CRP levels and levels of atheroprotective small-sized HDLs (*Chang et al., 2023*). Similarly, Shah et al found a statistically significant inverse relationship between CRP and HDL cholesterol (*Shah et al., 2017*). Furthermore, it has been noted that patients with high disease activity ($\text{DAS28} > 5.1$) exhibit an atherogenic lipid profile, which tends to improve following treatment (*Gantait et al., 2020*).

In the spondyloarthropathy group, no significant correlation was observed between disease activity and age, BMI, CIMT, or lipid profile. This aligns with previous studies where no association was found between CIMT and ESR, CRP, or the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) in ankylosing spondylitis patients (*Skare et al., 2013*) (*Peters et al., 2010*). Similarly, Talari et al reported no significant correlation between CIMT and disease activity (*Talari et al., 2021*). Additionally, two other studies confirmed the absence of a relationship between CIMT and disease activity in patients with PsA (*Mazlan et al., 2009*) (*Lin et al., 2014*).

Conclusion:

No difference in cardiovascular risk was observed between RA and SpA patients. In RA patients, higher disease activity was linked to lower HDL cholesterol levels. However, disease activity showed no significant correlation with other cardiovascular risk factors in the studied population.

List of Abbreviations:

- ACR – American College of Rheumatology

- **AS** – Ankylosing spondylitis
- **ASAS** – Assessment of SpondyloArthritis international Society
- **BMI** – Body mass index
- **CASPAR** – Classification Criteria for Psoriatic Arthritis
- **CIMT** – Carotid intima-media thickness
- **CRP** – C-reactive protein
- **CV** – Cardiovascular
- **DAS28** – Disease Activity Score-28
- **DAPSA** – Disease Activity in Psoriatic Arthritis
- **DM** – Diabetes mellitus
- **ESR** – Erythrocyte sedimentation rate
- **HDL** – High-density lipoprotein
- **IBD** – Inflammatory bowel disease
- **ILD** – Interstitial lung disease
- **JAK** – Janus kinase
- **LDL** – Low-density lipoprotein
- **PsA** – Psoriatic arthritis
- **RA** – Rheumatoid arthritis
- **SD** – Standard deviation
- **SpA** – Spondyloarthritis
- **TNF** – Tumor necrosis factor

References:

Adhikari MC, Guin A, Chakraborty S, et al. (2012): Subclinical atherosclerosis and endothelial dysfunction in patients with early rheumatoid arthritis as evidenced by measurement of carotid intima-media thickness and flow-mediated vasodilatation: an observational study. *Seminars in Arthritis and Rheumatism*, 41(5), 669–675.

Ağadayı E., Yurdakul F.G., Ağadayı S., et al., (2019): Cardiovascular risk factors and metabolic syndrome in rheumatoid arthritis and spondyloarthritis: Correlation with uric acid levels. *Journal of Clinical Practice and Research*, 41(3), p.300

Aletaha D., Neogi T., Silman A.J., et al., (2010): 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Arthritis & rheumatism*, 62(9), pp.2569-2581.

Ali M.A.M., AbdelKareem M.I.M., Doma, M.A.E., et al., (2020): Metabolic Syndrome and Cardiovascular Diseases in Seronegative Spondyloarthritis and Rheumatoid Arthritis Patients: A Comparative Study. *The Egyptian Journal of Hospital Medicine*, 80(3), pp.1086-1091.

Caraba A, Stancu O, Crişan V, Georgescu D (2024): Anti TNF-alpha treatment improves microvascular endothelial dysfunction in rheumatoid arthritis patients. *International Journal of Molecular Sciences*, 25(18), 9925.

Catrina A., Krishnamurthy A. and Rethi, B (2021): Current view on the pathogenic role of anti-citrullinated protein antibodies in rheumatoid arthritis. *RMD open*, 7(1), p.e001228.

Chang, C.K., Chiang, E.P.I., Chang, K.H., Tang, K.T., Chen, P.K., Yip, H.T., Chen, C.H. and Chen, D.Y., 2023. The sizes and composition of HDL-cholesterol are significantly associated with inflammation in rheumatoid arthritis patients. *International Journal of Molecular Sciences*, 24(13), p.10645

Darabian S., Hormuz M., Latif M.A., et al (2013): The role of carotid intimal thickness testing and risk prediction in the development of coronary atherosclerosis. *Current atherosclerosis reports*, 15, pp.1-4.

Dijkshoorn B., Raadsen R. and Nurmohamed M.T (2022): Cardiovascular disease risk in rheumatoid arthritis anno 2022. *Journal of clinical medicine*, 11(10), p.2704.

Duchez A.C., Boudreau L.H., Naika G.S., et al., (2019): Respective contribution of cytosolic phospholipase A2 α and secreted phospholipase A2 IIA to inflammation and eicosanoid production in arthritis. *Prostaglandins & Other Lipid Mediators*, 143, p.106340.

Gantait K., Barman H. and Gantait I. (2020): A comparative study on different fraction of lipid and das28 score in patients of early arthritis. *J West Bengal Univ Health Sci*, 1(1), pp.41-46.

Khan S., Yousaf M.J., Rashid A. et al., (2024): Comparison of Platelets-to-Lymphocyte and Neutrophil-to-Lymphocyte Ratios in Rheumatoid Arthritis and Ankylosing Spondylitis. *Age (years)*, 34(6.4), pp.46-6.

Li M. and Xie L. (2021): [Retracted] Correlation between NLR, PLR, and LMR and Disease Activity, Efficacy Assessment in Rheumatoid Arthritis. *Evidence-Based Complementary and Alternative Medicine*, (1), p.4433141.

Lin Y.C., Dalal D., Churton S., et al., (2014): Relationship between metabolic syndrome and carotid intima-media thickness: cross-sectional comparison between psoriasis and psoriatic arthritis. *Arthritis care & research*, 66(1), pp.97-103.

Machado P., Landewé R., Lie E., et al., (2011): Ankylosing Spondylitis Disease Activity Score (ASDAS): defining cut-off values for disease activity states and improvement scores. *Annals of the rheumatic diseases*, 70(1), pp.47-53.

Mazlan S.A., bin Mohamed Said M.S., Hussein H., et al., (2009): A study of intima media thickness and their cardiovascular risk factors in patients with psoriatic arthritis. *Acta Medica (Hradec Kralove)*, 52(3), pp.107-16.

Ngoc, C.T. and Son, D.N., Characteristics of the carotid intima-media thickness and atherosclerotic plaques of carotid arteries in elderly people with rheumatoid arthritis at University Medical Center Ho Chi Minh City

Ozisler C, Ates A, Karaaslan Y, Elalmis OU, Parlak IS, Dortbas F, et al. Clinical significance of aortic stiffness, carotid intima-media thickness and serum osteoprotegerin level in rheumatoid arthritis patients. *The Egyptian Rheumatologist*. 2019;41(2):111-5.

Peters M.J., Smulders Y.M., Serne E., et al., (2010): Signs of accelerated preclinical atherosclerosis in patients with ankylosing spondylitis. *The Journal of Rheumatology*, 37(1), pp.161-166.

Prevoo M.L.L., Van'T Hof M., Kuper H.H., et al., (1995): Modified disease activity scores that include twenty-eight-joint counts development and validation in a prospective longitudinal study of patients with rheumatoid arthritis. *Arthritis & Rheumatism: Official Journal of the American College of Rheumatology*, 38(1), pp.44-48.

Rajabzadeh F., Akhlaghipour I., Moosavi S.S., (2023): Comparison of the intima-media thickness of the common carotid artery in patients with rheumatoid arthritis: A single-center cross-sectional case-control study, and a brief review of the literature. *Health Science Reports*, 6(11), p.e1718.

Rojas-Giménez, M.; López-Medina, C.; Ladehesa-Pineda, M.L.; et al., (2022): Subclinical Atherosclerosis Measure by Carotid Ultrasound and Inflammatory Activity in Patients with Rheumatoid Arthritis and Spondylarthritis. *J. Clin. Med.* 2022, 11, 662. <https://doi.org/10.3390/jcm11030662>

Rudwaleit M.V., Van De Heijde D., Landewé R., et al., (2009): The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part II): validation and final selection. *Annals of the rheumatic diseases*, 68(6), pp.777-783.

Schoels M.M., Aletaha D., Alasti F. et al., (2016): Disease activity in psoriatic arthritis (PsA): defining remission and treatment success using the DAPSA score. *Annals of the rheumatic diseases*, 75(5), pp.811-818.

Shah S.J. (2017): Lipid profile in rheumatoid arthritis and its relation with inflammatory markers. *Archivos de Medicina*, 2(1), p.6.

Sivakumaran J., Harvey P., Omar A., et al., (2021): Assessment of cardiovascular risk tools as predictors of cardiovascular disease events in systemic lupus erythematosus. *Lupus Science & Medicine*, 8(1), p.e000448

Skare T.L. Verceze G.C., Oliveira A.A.D. et al., (2013): Carotid intima-media thickness in spondyloarthritis patients. *Sao Paulo Medical Journal*, 131(2), pp.100-105.

Talari H., Sehat M., Shayestehpour M., (2021): The association of psoriatic arthritis with carotid intima-media thickness. *Journal of Kerman University of Medical Sciences*, 28(2), pp.173-178.

Targonska-Stepniak B., Drelich-Zbroja A. and Majdan M. (2011): The relationship between carotid intima-media thickness and the activity of rheumatoid arthritis. *JCR: Journal of Clinical Rheumatology*, 17(5), pp.249-255.

Taylor W, Gladman D, Helliwell P, et al., (2006): Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis and Rheumatism*, 54(8), 2665–2673.

van Breukelen—van der Stoep D.F., van Zeben D., et al., (2015): Association of cardiovascular risk factors with carotid intima media thickness in patients with rheumatoid arthritis with low disease activity compared to controls: a cross-sectional study. *PLoS One*, 10(10), p.e0140844.

Wilson PW, D'Agostino RB, Levy D, et al., (1998): Prediction of coronary heart disease using risk factor categories. *Circulation*, 97(18), 1837–1847.