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Original article

Comparable Carotid Vascular Biomechanics in Ankylosing Spondylitis and Psoriatic Arthritis: A Head-To-Head Echo-Tracking Ultrasound Study

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Running title: Carotid vascular biomechanics in AS and PsA

Abstract

Background: Spondyloarthritis, including ankylosing spondylitis (AS) and psoriatic arthritis (PsA), is associated with increased cardiovascular risk that may not be fully captured by conventional risk assessment tools. Arterial stiffness and carotid intima–media thickness (CIMT) represent early markers of vascular ageing; however, direct comparisons of local carotid biomechanical properties between AS and PsA remain limited. **Objectives:** To compare structural and functional carotid vascular parameters derived from one-point echo-tracking ultrasound between patients with AS and PsA and to explore clinical, metabolic and haemodynamic correlates of arterial stiffness. **Methods:** In this cross-sectional study, 108 patients with spondyloarthritis (71 AS, 37 PsA) underwent vascular ultrasound assessment including CIMT, local pulse wave velocity (PWVβ), β-stiffness index, Peterson's elastic modulus (Ep), arterial compliance (AC) and augmentation index (AI). Clinical

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characteristics, inflammatory markers, lipid profile and cardiovascular risk scores were recorded. Between-group comparisons were performed using non-parametric tests. Spearman correlations and multivariable linear regression analyses were applied to identify factors associated with vascular parameters. **Results:** Baseline demographic and metabolic characteristics were comparable between groups, although sex distribution differed ($p = 0.002$). Most structural and functional vascular indices, including CIMT, PWVβ, β-stiffness index, AC and Ep, did not differ significantly between AS and PsA. Augmentation index was higher in PsA compared with AS (median 21.9 vs 12.8, $p = 0.029$). CIMT correlated positively with β-stiffness index ($\rho = 0.404$), PWVβ ($\rho = 0.482$) and Ep ($\rho = 0.451$) (all $p < 0.001$). In multivariable regression models, arterial hypertension emerged as the most consistent independent factor associated with vascular parameters, while inflammatory markers showed limited associations. **Conclusion:** Patients with ankylosing spondylitis and psoriatic arthritis demonstrate broadly comparable local carotid vascular profiles when assessed using echo-tracking ultrasound. Higher augmentation index in psoriatic arthritis suggests differences in haemodynamic wave reflection rather than intrinsic arterial stiffness. Vascular alterations appear to be driven predominantly by haemodynamic and metabolic factors rather than disease phenotype alone.

Keywords: ankylosing spondylitis; psoriatic arthritis; arterial stiffness; echo-tracking; carotid intima-media thickness; augmentation index; spondyloarthritis; vascular ageing

1. Introduction

Spondyloarthritis, including ankylosing spondylitis (AS) and psoriatic arthritis (PsA), is associated with a substantially increased cardiovascular risk that cannot be fully explained by traditional risk factors alone. Chronic systemic inflammation contributes to this excess risk through endothelial dysfunction, oxidative stress and adverse vascular ageing, thereby accelerating atherosclerosis and promoting premature cardiovascular disease [1,2]. Importantly, vascular involvement in spondyloarthritis often remains clinically silent for prolonged periods, and subclinical vascular changes may precede overt cardiovascular events.

Large population-based and longitudinal studies consistently report higher rates of ischemic heart disease, cerebrovascular events and cardiovascular mortality in AS compared with the general population [3-10]. Similarly, PsA

is linked to increased cardiovascular morbidity and mortality, with meta-analyses demonstrating higher incidence of myocardial infarction, stroke and heart failure compared with the general population [10-13]. However, commonly used cardiovascular risk prediction models frequently underestimate risk in inflammatory arthritides, supporting the need for approaches that detect early vascular dysfunction beyond conventional risk assessment.

Arterial stiffening represents a key functional manifestation of adverse vascular ageing and integrates the cumulative impact of haemodynamic load, metabolic factors and chronic inflammation. From a haemodynamic perspective, reduced arterial elasticity alters pulse wave propagation and reflection, increases systolic and pulse pressure and augments left ventricular afterload, providing a mechanistic link between arterial stiffness and adverse cardiovascular outcomes. Although arterial stiffening accompanies physiological ageing, it may be accelerated in chronic inflammatory conditions such as AS and PsA [14-16]. Prior studies in spondyloarthritis have reported heterogeneous results regarding the magnitude and determinants of stiffness and early atherosclerotic changes, likely reflecting differences in study design, populations, and measurement [17-19].

Ultrasound-based assessment enables parallel evaluation of functional and structural vascular alterations. One-point echo-tracking technology provides local carotid biomechanical indices derived from diameter changes throughout the cardiac cycle combined with blood pressure values, including the stiffness index β, Peterson's elastic modulus (Ep), arterial compliance (AC) and augmentation index (AI), as well as local pulse wave velocity (PWVβ) calculated at a single arterial [20-22]. Compared with carotid–femoral PWV, local one-point assessment avoids uncertainties related to external distance estimation and heterogeneous arterial segments, offering a focused and reproducible evaluation of carotid wall mechanics [20,23]. In parallel, carotid intima–media thickness (CIMT) provides a validated marker of early structural arterial remodelling and subclinical atherosclerosis [24,25]. Combining echo-tracking–derived stiffness parameters with CIMT may therefore offer an integrated profile of early vascular damage in inflammatory diseases characterised by accelerated vascular ageing.

Despite the growing evidence of increased cardiovascular risk in both AS and PsA, direct head-to-head comparisons of local carotid biomechanical properties between these two spondyloarthritis phenotypes remain limited. Moreover, augmentation index-reflecting wave reflection and

peripheral vascular tone-may capture haemodynamic differences that are not necessarily mirrored by intrinsic carotid wall stiffness. Accordingly, the present study evaluates carotid arterial stiffness using one-point echo-tracking-derived biomechanical indices in combination with CIMT in patients with AS and PsA. We aimed to characterise subclinical vascular damage across the spondyloarthritis spectrum and to explore clinical and metabolic correlates of functional and structural vascular measures.

2. Materials and Methods

2.1. Study design and study population

This cross-sectional observational study included 108 patients with spondyloarthritis, comprising 71 patients with ankylosing spondylitis (AS) and 37 patients with psoriatic arthritis (PsA). Participants were consecutively recruited from the Departments of Rheumatology and Internal Medicine at University Hospital "St. Marina", Varna, Bulgaria, between January 2023 and December 2024.

Patients included in the present analysis were derived from a larger prospective observational cohort of individuals with spondyloarthritis. To minimise confounding related to age differences between disease groups, frequency matching by age strata was applied during data extraction. Sex distribution was not matched and reflects the underlying epidemiology of the disease populations.

Ankylosing spondylitis was diagnosed according to the Assessment of SpondyloArthritis international Society (ASAS) classification criteria, while psoriatic arthritis was classified based on the Classification Criteria for Psoriatic Arthritis (CASPAR). All diagnoses were established by experienced rheumatologists.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Medical University of Varna (Protocol №133/22.06.2023). Written informed consent was obtained from all participants prior to inclusion.

Predefined exclusion criteria included known ischemic heart disease, cerebrovascular disease, peripheral arterial disease, heart failure, clinically significant arrhythmias, diabetes mellitus, chronic kidney disease stage ≥3, active infection, malignancy, pregnancy, or inability to undergo vascular ultrasonography.

2.2. Clinical and laboratory assessment

Demographic characteristics, smoking status, disease duration (time since diagnosis), and diagnostic delay (time from symptom onset to diagnosis) were recorded. Anthropometric parameters and the presence of arterial hypertension were documented according to current clinical guidelines.

Blood pressure was measured under standardised resting conditions after at least 5 minutes of supine rest using an automated oscillometric device. Systolic and diastolic blood pressure values were recorded, and mean arterial pressure was calculated for descriptive purposes.

Systemic inflammatory activity was assessed by serum C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), determined using routine laboratory methods at the central hospital laboratory. Fasting lipid profile, including total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), was analysed using standardised enzymatic assays.

2.3. Vascular ultrasound assessment

Vascular ultrasound examinations were performed using a high-resolution ultrasound system (Aloka Hitachi Prosound α7) equipped with a high-frequency linear transducer. All measurements were obtained at the level of the right common carotid artery approximately 2 cm proximal to the carotid bifurcation. The right carotid artery was selected to ensure methodological consistency and minimise examination time variability. Participants were examined in the supine position after a short resting period.

Carotid intima–media thickness (CIMT) was measured at the far wall of plaque-free segments of the common carotid artery using B-mode imaging. Three measurements obtained during separate cardiac cycles were averaged for analysis. Atherosclerotic plaque was defined as focal wall thickening ≥1.5 mm or ≥50% greater than the surrounding intima–media thickness, and such segments were excluded from CIMT assessment.

Arterial stiffness was evaluated using one-point echo-tracking technology, allowing continuous assessment of arterial diameter changes synchronised with the electrocardiogram. Brachial blood pressure values measured immediately prior to the examination were entered into the system to enable automated calculation of carotid biomechanical parameters. The analysed indices included local pulse wave velocity (PWVβ), β-stiffness index, pressure–strain elastic modulus (Peterson's modulus, Ep), arterial compliance (AC), and augmentation index (AI).

All examinations were performed by a single experienced operator blinded to laboratory results. To assess measurement consistency, repeated measurements were performed in a random subset of participants, demonstrating good intra-observer reproducibility.

2.4. Cardiovascular risk assessment

Ten-year cardiovascular risk was estimated using validated prediction models. The Framingham Risk Score and SCORE2 were calculated in accordance with current European Society of Cardiology recommendations. For participants aged ≥70 years, the SCORE2-OP algorithm was applied when appropriate.

2.5. Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 27.0. Continuous variables were assessed for normality using visual inspection and Shapiro–Wilk testing and are presented as mean ± standard deviation or median with interquartile range, as appropriate. Comparisons between AS and PsA groups were conducted using the Mann–Whitney U test for continuous variables and Pearson's chi-square test for categorical variables.

Associations between vascular ultrasound parameters, inflammatory markers, lipid profile components, and cardiovascular risk scores were explored using Spearman's rank correlation analysis. Given the exploratory nature of correlation analyses, results were interpreted with caution and considered hypothesis-generating.

Multivariable linear regression models were constructed to assess factors associated with arterial stiffness indices and carotid intima–media thickness. Covariates were selected a priori based on clinical relevance and included disease type, age, sex, body mass index (BMI), arterial hypertension, and treatment status (targeted therapy vs biologic-naïve). Model assumptions were evaluated using residual diagnostics and multicollinearity assessment. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics

A total of 108 patients with spondyloarthritis were included, comprising 71 patients with ankylosing spondylitis (AS) and 37 with psoriatic arthritis (PsA). Age distribution was comparable between groups (49.4 ± 9.7 vs 52.7

± 10.2 years, p = 0.083). Disease duration was also similar (9.7 ± 6.2 vs 9.0 ± 7.0 years, p = 0.400).

Sex distribution differed significantly, with male predominance in AS and female predominance in PsA (66.2% vs 35.1% male, p = 0.002). The prevalence of current smoking and arterial hypertension did not differ significantly between disease entities. Body mass index was comparable between groups.

Inflammatory markers showed similar erythrocyte sedimentation rate values, whereas C-reactive protein levels were higher in AS compared with PsA (p = 0.022). Lipid profile parameters did not differ significantly (Table 1).

Table 1. Baseline demographic, clinical, laboratory and vascular characteristics of patients with ankylosing spondylitis and psoriatic arthritis

Variable	AS (n = 71)	PsA (n = 37)	p-value
Age, years	49.4 ± 9.7	52.7 ± 10.2	0.083
Male sex, n (%)	47 (66.2)	13 (35.1)	0.002
Disease duration, years	9.7 ± 6.2	9.0 ± 7.0	0.400
Current smoker, n (%)	42 (59.2)	23 (62.2)	0.762
Hypertension, n (%)	26 (36.6)	10 (27.0)	0.316
BMI, kg/m^2	28.3 ± 5.8	27.1 ± 4.6	0.447
ESR, mm/h	36.7 ± 26.1	29.8 ± 16.9	0.356
CRP, mg/L	9.5 ± 17.9	3.9 ± 5.7	0.022
Total cholesterol, mmol/L	5.37 ± 1.24	5.51 ± 1.20	0.482
Triglycerides, mmol/L	1.49 ± 1.38	1.48 ± 1.25	0.821
LDL-cholesterol, mmol/L	3.28 ± 1.04	3.23 ± 1.14	0.854
HDL-cholesterol, mmol/L	1.39 ± 0.33	1.57 ± 0.52	0.119
CIMT, mm	0.60 ± 0.17	0.63 ± 0.25	0.510
β-stiffness index	7.64 ± 2.59	8.01 ± 3.42	0.985
PWV, m/s	6.06 ± 0.99	6.16 ± 1.15	0.724
Augmentation index, %	15.8 ± 13.5	21.6 ± 15.3	0.029
Arterial compliance	0.89 ± 0.26	1.09 ± 1.66	0.224
Peterson's elastic modulus	100.5 ± 35.8	105.0 ± 42.6	0.653
Framingham risk score, %	5.8 ± 5.0	5.4 ± 4.4	0.776
SCORE, %	9.4 ± 6.9	11.6 ± 7.1	0.121

3.2. Vascular ultrasound findings

Structural vascular assessment demonstrated comparable carotid intima–media thickness between AS and PsA [0.54 (IQR 0.22) vs 0.61 (IQR 0.22), p = 0.510].

Functional arterial stiffness indices were largely similar between groups. The β-stiffness index showed identical median values [7.0 (IQR 3.0) vs 7.0 (IQR 3.9), p = 0.985], and local pulse wave velocity (PWVβ) was also comparable [5.8 (IQR 1.2) vs 5.8 (IQR 1.5), p = 0.724]. Arterial compliance demonstrated overlapping distributions [0.86 (IQR 0.32) vs 0.79 (IQR 0.40), p = 0.224], as did Peterson's elastic modulus [88 (IQR 41) vs 93 (IQR 57), p = 0.653].

In contrast, augmentation index (AI) was significantly higher in PsA compared with AS [21.9 (IQR 19.65) vs 12.8 (IQR 19.0), p = 0.029] (Figure 1).

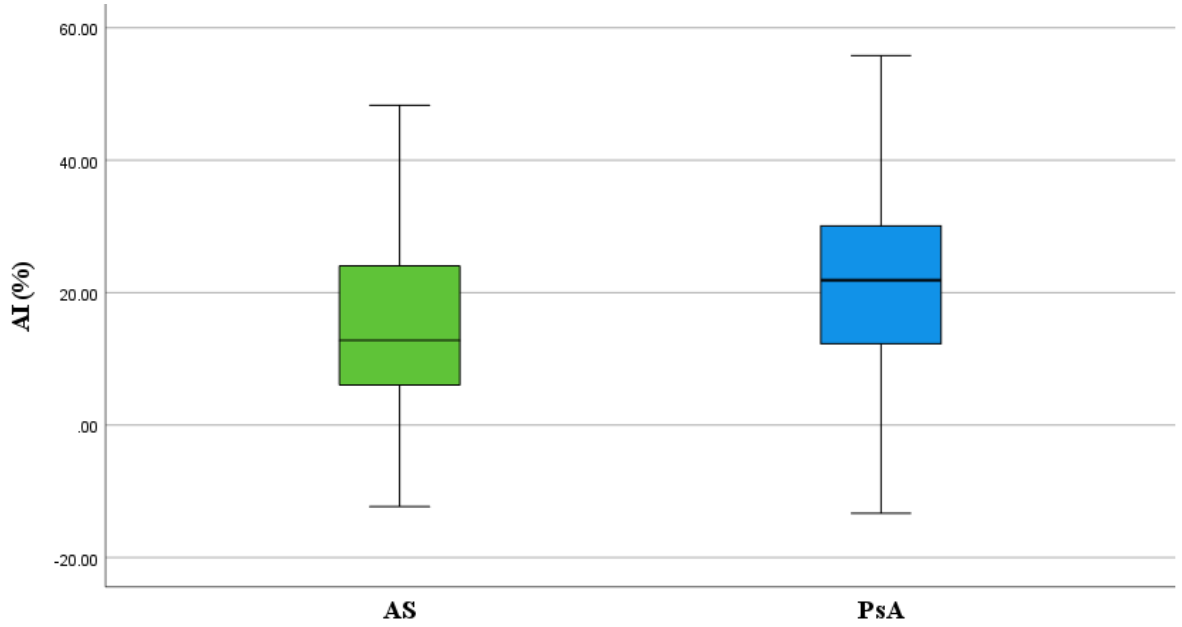


Figure 1. Augmentation index (AI) in patients with ankylosing spondylitis and psoriatic arthritis

3.3. Cardiovascular risk scores

Estimated 10-year cardiovascular risk did not differ significantly between disease groups. Framingham risk score values were comparable (p = 0.776), as were SCORE2 estimates (p = 0.121).

3.4. Treatment distribution

Targeted antirheumatic therapy distribution differed significantly between AS and PsA (p < 0.001). Among patients with ankylosing spondylitis, anti-TNF therapy was administered in 63.4%, anti-IL-17 agents in 15.5%, biologic-naïve status in 15.5%, and JAK inhibitors in 5.6%. In contrast, patients with psoriatic arthritis were more frequently treated with anti-IL-

17 agents (48.6%) and JAK inhibitors (18.9%), while anti-TNF therapy was used in 21.6% and biologic-naïve status was present in 10.8%.

3.5. Correlation analysis

Spearman rank correlation analysis was performed in the overall cohort (n = 108). Age correlated positively with cardiovascular risk scores, including the Framingham risk score ($\rho = 0.449$, $p < 0.001$) and SCORE2 ($\rho = 0.558$, $p < 0.001$). Carotid intima–media thickness correlated positively with β-stiffness index ($\rho = 0.404$, $p < 0.001$), PWVβ ($\rho = 0.482$, $p < 0.001$) and Peterson's elastic modulus ($\rho = 0.451$, $p < 0.001$). Triglyceride levels demonstrated modest positive correlations with functional stiffness parameters (all $p < 0.05$), while low-density lipoprotein cholesterol correlated positively with augmentation index and cardiovascular risk estimates.

3.6. Multivariable regression analysis

Multivariable linear regression analyses were performed using augmentation index (AI), PWVβ, β-stiffness index and carotid intima–media thickness (CIMT) as dependent variables. Covariates included disease type, age, sex, body mass index, arterial hypertension and treatment status.

The regression model for AI was statistically significant ($F = 2.75$, $p = 0.016$; $R^2 = 0.140$), with arterial hypertension independently associated with higher AI values ($p = 0.030$). Disease type showed a borderline association ($p = 0.085$).

For PWVβ, the model reached statistical significance ($F = 2.47$, $p = 0.028$; $R^2 = 0.128$), with arterial hypertension and body mass index positively associated with PWVβ ($p = 0.040$ and $p = 0.046$, respectively).

The β-stiffness model demonstrated overall significance ($F = 3.25$, $p = 0.006$; $R^2 = 0.162$), with arterial hypertension emerging as the only independent associated factor ($p = 0.009$). The CIMT model showed borderline overall significance ($F = 1.97$, $p = 0.066$; $R^2 = 0.121$), with arterial hypertension independently associated with higher CIMT values ($p = 0.038$).

4. Discussion

In this head-to-head analysis, patients with ankylosing spondylitis and psoriatic arthritis demonstrated broadly comparable structural and functional vascular characteristics when assessed using one-point echo-tracking ultrasound. Most stiffness indices and carotid intima–media thickness values did not differ significantly between the two diseases despite differences in inflammatory burden and treatment exposure. In contrast, augmentation index was higher in psoriatic arthritis, suggesting differences in wave reflection and peripheral vascular tone rather than intrinsic carotid wall stiffness. Arterial hypertension consistently emerged as the strongest determinant of vascular parameters, whereas inflammatory markers showed limited associations with stiffness indices.

A central observation of the present study is the consistent association between structural and functional vascular measures across the cohort. Positive correlations between carotid intima–media thickness and local stiffness indices indicate that arterial remodelling and functional stiffening may evolve in parallel within the spondyloarthritis spectrum. However, the absence of between-disease differences in most local stiffness parameters suggests that disease phenotype alone may not represent the primary driver of carotid biomechanical alterations. The higher augmentation index observed in psoriatic arthritis likely reflects haemodynamic influences related to peripheral vascular resistance rather than intrinsic arterial rigidity, consistent with the known physiological determinants of augmentation index [26]

Across multivariable models, arterial hypertension was the most consistent independent factor associated with vascular alterations. Given the markedly different distribution of targeted therapies between ankylosing spondylitis and psoriatic arthritis, treatment exposure was considered a potential confounder and included as a covariate in multivariable analyses; however, no independent association between treatment status and vascular ultrasound parameters was observed after adjustment. These findings align with evidence demonstrating the central role of haemodynamic stress in elastin fragmentation, collagen accumulation and progressive arterial stiffening [27,28]. Modest associations between metabolic parameters, particularly triglycerides and body mass index, and stiffness indices further suggest a potential contribution of metabolic burden to vascular ageing in inflammatory arthritis, consistent with previous rheumatologic studies exploring vascular and metabolic biomarkers [29].

Inflammatory activity demonstrated limited direct relationships with ultrasound-derived vascular parameters in the present analysis. Previous studies have reported heterogeneous findings, with some cohorts showing associations between disease activity and structural vascular measures but weaker relationships with functional stiffness indices [17,18]. Prospective data in psoriatic arthritis suggest that cumulative inflammatory exposure rather than single time-point inflammatory markers may be more relevant for vascular remodelling [30]. The lack of strong correlations in our cohort may therefore reflect the chronic and cumulative nature of vascular adaptation rather than current inflammatory status.

Most earlier investigations evaluated ankylosing spondylitis or psoriatic arthritis primarily in comparison with healthy controls and reported increased arterial stiffness or carotid intima–media thickness [19,31,32]. In contrast, the present head-to-head comparison indicates that vascular profiles may appear broadly similar when the two spondyloarthritis phenotypes are evaluated directly under comparable clinical conditions. Direct comparisons between spondyloarthritis phenotypes using local echo-tracking–derived stiffness parameters remain limited, highlighting the relevance of the current findings. Previous reports have described variable associations between disease activity and vascular stiffness, including impaired aortic elasticity related to disease burden [33], whereas other studies observed minimal changes in stiffness parameters despite anti-inflammatory treatment [34, 35]. These discrepancies underscore the multifactorial nature of vascular involvement and the influence of study design and measurement methodology.

The use of one-point echo-tracking represents an important methodological aspect of the present study. Local carotid assessment allows precise evaluation of arterial wall motion and avoids uncertainties related to external distance estimation inherent to carotid–femoral pulse wave velocity measurements [22,23]. This regional approach may be particularly sensitive for detecting early functional vascular alterations that are not fully captured by global stiffness indices.

Several limitations should be acknowledged. The cross-sectional design precludes causal interpretation of vascular remodelling processes. Participants were selected from a larger observational cohort to minimise demographic imbalance between groups, which may influence generalisability. Sex distribution differed between ankylosing spondylitis and psoriatic arthritis, reflecting the underlying epidemiology of the diseases, and may have influenced vascular haemodynamic parameters

despite adjustment in multivariable analyses. The absence of a healthy control group limits conclusions regarding absolute cardiovascular risk; however, the primary aim was to compare vascular characteristics between ankylosing spondylitis and psoriatic arthritis. In addition, local carotid measurements may not fully reflect systemic arterial stiffness, and the smaller psoriatic arthritis subgroup may have limited statistical power to detect subtle disease-specific differences.

5. Conclusions

In conclusion, ankylosing spondylitis and psoriatic arthritis demonstrate broadly comparable structural and functional vascular profiles when evaluated directly using echo-tracking ultrasound. The higher augmentation index observed in psoriatic arthritis suggests differences in haemodynamic wave reflection rather than intrinsic arterial stiffness. Arterial hypertension and metabolic burden appear to represent key contributors to vascular alterations, whereas current inflammatory activity shows limited association with stiffness indices. These findings support the concept that vascular ageing in spondyloarthritis may be influenced more strongly by haemodynamic and metabolic factors than by disease phenotype alone.

Author Contributions

Author Contributions: Conceptualization: M.M., T.G.; Methodology: M.M., T.G., M.D.; Formal analysis: M.M.; Investigation: M.M., M.D.; Writing – original draft: M.M.; Writing – review & editing: T.G., M.K., M.D.

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Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Medical University of Varna (Protocol №133/22.06.2023). Written informed consent was obtained from all participants prior to inclusion.

Informed Consent Statement

Written informed consent was obtained from all subjects involved in the study.

Data Availability Statement

The data presented in this study are available on reasonable request from the corresponding author.

Conflicts of Interest

The authors declare no conflict of interest.

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Сравними каротидни съдови биомеханични характеристики при анкилозиращ спондилит и псориатичен артрит: директно сравнително ехографско изследване чрез echo-tracking

Резюме

Въведение: Спондилоартритите, включително анкилозиращият спондилит (АС) и псориатичният артрит (ПсА), са свързани с повишен сърдечно-съдов риск, който невинаги се отразява адекватно чрез конвенционалните методи за оценка на риска. Артериалната ригидност и дебелината на интима–медия комплекса на каротидните артерии (CIMT) представляват ранни маркери на съдово стареене. Въпреки това, директните сравнения на локалните каротидни биомеханични свойства между пациенти с АС и ПсА остават ограничени. **Цел:** Да се сравнят структурните и функционалните съдови параметри на каротидните артерии, получени чрез едноточков echo-tracking ултразвук, между пациенти с АС и ПсА, както и да се изследват клиничните, метаболитните и хемодинамичните фактори, свързани с артериалната ригидност. **Методи:** В това напречно (cross-sectional) проучване 108 пациенти със спондилоартрит (71 с АС и 37 с ПсА) бяха подложени на съдов ултразвуков анализ, включващ измерване на CIMT, локална пулсово-вълнова скорост (PWVβ), индекс на β-ригидност, модул на еластичност на Peterson (Ep), артериален комплайънс (AC) и индекс на аугментация (AI). Регистрирани бяха клинични характеристики, възпалителни маркери, липиден профил и сърдечно-съдови рискови скорове. Сравненията между групите бяха извършени чрез непараметрични тестове. За определяне на факторите, свързани със съдовите параметри, бяха използвани корелационен анализ на Spearman и мултивариантни линейни регресионни модели. **Резултати:** Базовите демографски и метаболитни характеристики бяха сходни между групите, въпреки че разпределението по пол се различаваше статистически значимо ($p = 0.002$). Повечето структурни и функционални съдови показатели, включително CIMT, PWVβ, индекс на β-ригидност, AC и Ep, не показаха съществени разлики между АС и ПсА. Индексът на аугментация беше по-висок при ПсА в сравнение с АС (медиана 21.9 срещу 12.8, $p = 0.029$). CIMT корелира положително с индекса на β-ригидност ($\rho = 0.404$), PWVβ ($\rho = 0.482$) и Ep ($\rho = 0.451$) (всички $p < 0.001$). В мултивариантните регресионни модели артериалната хипертония се очерта като най-

последователния независим фактор, асоцииран със съдовите параметри, докато възпалителните маркери показаха ограничени асоциации. **Заключение:** Пациентите с анкилозиращ спондилит и псориатичен артрит демонстрират в голяма степен сходни локални каротидни съдови характеристики, оценени чрез echo-tracking ултразвук. По-високият индекс на аугментация при псориатичен артрит предполага различия в хемодинамичното отражение на пулсовата вълна, а не в самата вътрешна артериална ригидност. Съдовите промени изглежда се определят предимно от хемодинамични и метаболитни фактори, а не единствено от фенотипа на заболяването.

Ключови думи: анкилозиращ спондилит; псориатичен артрит; артериална ригидност; echo-tracking; дебелина на интима–медия комплекса; индекс на аугментация; спондилоартрит; съдово стареене