Koşuyolu Heart Journal

Koşuyolu Heart J 2024;27(2):76–81
DOI: 10.51645/khj.2024.449

Original Article

Evaluation of the Great Artery Mechanics with Carotid-Femoral (Aortic) Pulse Wave Propagation Time in Patients with Rheumatoid Arthritis

Alparslan Şahin,[1] Ersan Oflar,[1] Selçuk Opan,[2] İlker Murat Çağlar[1]

[1]Department of Cardiology, University of Health Sciences, Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul, Türkiye

[2]Department of Cardiology,Sanliurfa Training and Research Hospital, Şanlıurfa, Türkiye

Abstract

Objectives: Rheumatoid arthritis (RA) is the most common chronic, systemic, rheumatic, autoimmune disease in adults, which has unknown etiology and targets primarily synovial tissue. Those patients have increased cardiovascular mortality based on inflammation and atherosclerosis. In our study, we evaluated great artery mechanics in newly diagnosed RA patients with inactive disease. Great artery mechanics, referred as arterial distensibility, elasticity, and compliance can be evaluated by pulse wave propagation time and pulse wave velocity.

Methods: Our study included newly diagnosed and in inactive stage 25 RA patients (mean age: 39.4±9.8 years, 21–63 years, 19 female, 6 male) and 29 healthy subjects (mean age: 36.9±9.9 years, 18–59 years, 13 female, 16 male). Aortic pulse wave propagation time and pulse wave velocitiy were determined using an automatic device, the Complior Colson (France), which allowed online recording and automatic calculation.

Results: Height, waist circumference, and waist/hip ratio values were significantly higher in healthy objects. (p=0.006, p=0.030, p=0.004, respectively) pulse wave propagation time and pulse wave velocity were significantly lower and higher, respectively, in RA patients compared to healthy subjects (p=0.000, p=0.041, respectively).

Conclusion: RA patients have decreased pulse wave propagation time and higher pulse wave velocity.

Keywords: Pulse wave propagation time; pulse wave velocity; rheumatoid arthritis.

Romatoid Artritli Hastlarda Büyük Arter Mekaniklerinin Karotis-Femoral (Aortik) Nabız Dalga İlerleme Zamanı ile Değerlendirilmesi

Özet

Amaç: Romatoid artrit (RA) erişkinde en sık görülen, primer olarak sinovyal dokuları hedef alan, etyolojisi net olarak bilinmeyen, kronik, sistemik, romatizmal, otoimmün bir hastalıktır. Romatoid artritli olgularda kardiyovasküler mortalite temelde inflamasyon ve ateroskleroz nedenli olarak artmıştır. Arteryel distansibilite, kompliyans ve elastisite olarak bilinen büyük arter mekanikleri nabız dalga ilerleme zamanı ve nabız dalga hızı ile değerlendirilebilmektedir. Çalışmamızda yeni tanı, inaktif hastalığı bulunan romatoid artritli hastalarda büyük arter mekaniklerini değerlendirdik. Büyük arter mekaniklerinin değerlendirlmesinde kullanılan nabız dalga ilerleme zamanı ve nabız dalga hızı ölçümleri kullanılabilmektedir.

Gereç ve Yöntem: Bu klinik calışmaya yeni tanı konumuş ve inaktif dönemde hastalığı olan 25 romatoid artrit hastası (ortalama yaş: 39.4±9.8 yıl, 21–63 yaş, 19 kadın, 6 erkek) ile 29 sağlıklı kontrol olgusu (ortalama yaş: 36.9±9.9 yıl, 18–59 yaş, 13 kadın, 16 erkek) dahil edildi. Aortik nabız dalga ilerleme zamanı ve nabız dalga hızı, on-line nabız dalga kaydına izin veren ve otomatik ölçüm yapan Complior Colson (Fransa) cihazı ile belirlendi.

Bulgular: Sağlıklı olgularda romatoid artritli hastalara göre boy, bel çevresi ve bel/kalça oranı değerleri anlamlı olarak daha yüksekti (p=0.006, p=0.030, p=0.004). Romatoid artritli hastalarda, sağlıklı olgulara göre nabız dalga hızı anlamlı olarak daha yüksek (p=0.041), nabız dalga ilerleme zamanı ise anlamlı olarak daha düşüktü (p=0.000).

Cite This Article: Şahin A, Oflar E, Opan S, Çağlar İM. Evaluation of the Great Artery Mechanics with Carotid-Femoral (Aortic) Pulse Wave Propagation Time in Patients with Rheumatoid Arthritis. Koşuyolu Heart J 2024;27(2):76–81

Address for Correspondence: Alparslan Şahin

Department of Cardiology, University of Health Sciences, Bakırköy Dr. Sadi Konuk Training and Research Hospital, İstanbul, Türkiye

E-mail: dralpsahin@gmail.com

Submitted: May 14, 2024
Revised: May 23, 2024
Accepted: June 11, 2024
Available Online: August 26, 2024

Sonuç: Sonuç olarak yeni tanı almış inaktif hastalığı bulunan romatoid artritli hastalarda sağlıklı olgularla karşılaştırıldığında nabız dalga ilerleme zamanı daha kısa, nabız dalga hızı daha yüksek bulundu.

Anahtar sözcükler: Nabız dalga ilerleme zamanı; nabız dalga hızı; romatoid artrit.

Introduction

Rheumatoid arthritis (RA) and similar systemic immune and inflammatory diseases are characterized by increased mortality and morbidity.[1,2] A significant portion of this increased mortality occurs due to cardiovascular events.[1,2] Endothelial dysfunction, characterized by decreased nitric oxide capacity, is one of the mechanisms in the initial phase of atherosclerosis pathogenesis.[3] Inflammation basically causes atherosclerosis by causing endothelial dysfunction. This damage to the artery wall due to atherosclerosis causes increased arterial stiffness and deterioration of large artery mechanics.[4,5] These effects on atherosclerosis and the arterial system can be evaluated with non-invasive methods.[4,5] Measurement of pulse wave velocity, such a technique, is one of the important methods in determining the elastic properties of large vessels and is based on the principle of measuring using two ultrasound or pressure-sensitive transducers placed on the skin on the traces of two arteries (such as carotid-femoral, brachial-radial arteries) at a certain distance from each other.[6]

The measured pulse wave velocity is an indicator of arterial wall stiffness; it also shows an inverse proportion to arterial distensibility or relative arterial compliance ([dV/V]/dP, dV: Volume change, dP: Pressure change) calculated with the classical formula of Imura et al.,[7] Like arterial elasticity, pulse wave velocity and the resulting expandability index are also affected by heart rate and blood pressure.[7] Pulse pressure and pulse wave velocity are important cardiovascular risk factor determinants identified in the general population and in patients with hypertension, coronary artery disease, and/or congestive heart failure.[8]

The aim of this study is to evaluate large artery mechanics with pulse wave propagation time and pulse wave velocity in newly diagnosed inactive RA patients and to examine the factors affecting this.

Materials and Methods

This clinical study included 25 newly diagnosed RA patients (mean age: 39.4±9.8 years, 21–63 years, 19 women, 6 men) and 29 healthy control patients (mean age: 36.9±9.9 years, 18 years). Fifty-nine years old (13 women, 16 men) were included in the study. Written permission was obtained from all participants stating that they participated in the study with their own consent. Local Ethical Committee approval was given on June 24, 2013 with issue number: 2013/08/01.

Secondary hypertension, treatment-resistant hypertension, diabetes mellitus (fasting blood sugar >100 mg/dL), hyperlipidemia (total cholesterol <200 mg/dL, triglyceride <150 mg/dL), heart failure, renal failure (plasma creatinine >1.5 mg/dL), those with a history of liver failure, heart valve disease, anti-inflammatory drug use, peripheral vascular disease, cerebrovascular disease and previous myocardial infarction, those with history of atrial fibrillation and/or previous myocardial infarction on twelve-channel surface electrocardiogram, smokers. Moreover, cases with body-mass index <35 kg/m^2 and waist-hip ratio <1 were not included in the study.

The height and weight of the participants were measured with the help of a movable ruler marker and a standard scale with an arm that can measure weight while wearing light clothing and without shoes. Body mass index (kg/m^2) was found by determining the weight in kg and dividing it by the height calculated in m^2. While the participant standing upright with his/her abdominal area relaxed, arms at the sides, and feet together, the waist circumference (cm) was measured in the midline between the last rib and the crista iliaca. Hip circumference (cm) was measured at the level of both trochanter major femoris. Waist-hip ratio was calculated by dividing the waist circumference by the hip circumference. Arterial blood pressure was measured in the supine position with a standard mercury manometer (ERKA, D-83646, Germany) after a 30-min rest. Systolic blood pressure was considered as the point at which Korotkoff sounds were first heard, and diastolic blood pressure was considered as the point at which the sounds disappeared (phase 5).

Pulse and average blood pressure were calculated by the formulas given below;

Pulse pressure=Systolic blood pressure-diastolic blood pressure,

Average blood pressure=(Systolic blood pressure+2 X diastolic blood pressure)/3

Carotid-femoral pulse wave velocity and pulse wave propagation time measurements

Aortic pulse wave velocity was calculated as previously stated using the Complior device (Createch Industrie, France), which allows automatic pulse wave recording and automatic calculation of pulse wave velocity.[4] The correlation coefficient of the automatic measurement method between observers and between measurements of an observer at different times is >0.9. Common carotid artery and femoral artery pressure waveforms were measured non-invasively using a TY-306 Fukuda (Fukuda, Tokyo, Japan) pressure-sensitive transducer. Measurements were repeated in more than 10 different cardiac cycles and the average value was used for result analysis.

Pulse wave velocity was automatically calculated with the formula Pulse Wave Velocity = D/t

(D: Distance [meters] traveled by the pulse wave on the body surface between two recording points, t: Pulse wave transit time (seconds) automatically determined by the Complior device). Pulse wave propagation time was recorded as the transit time in the formula (Fig. 1).

Statistical Analysis

Statistics were calculated using Statistical Package for the Social Sciences version 8.0 (IBM SPSS Statistics for Windows,

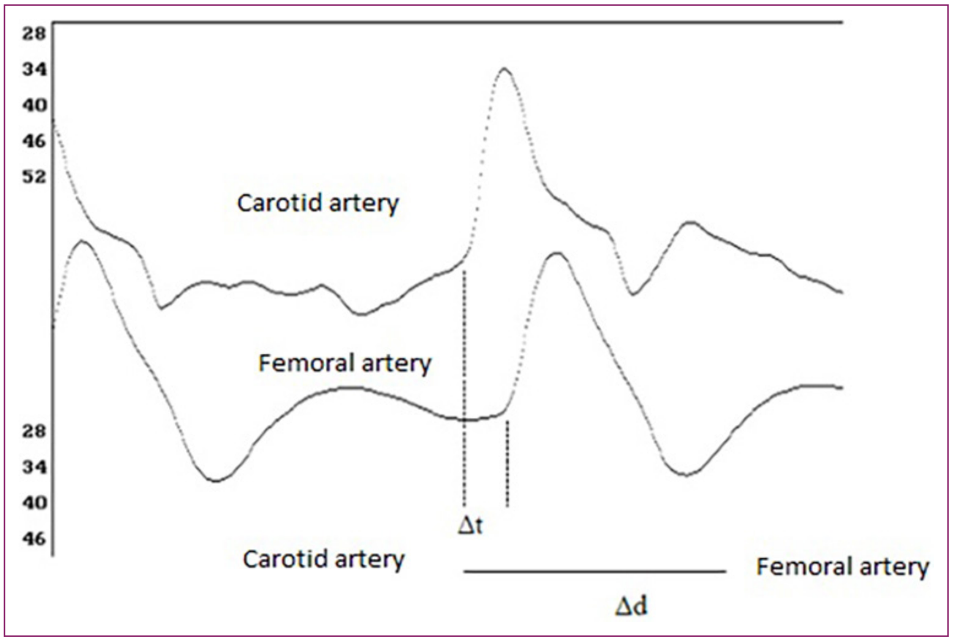

Figure 1. Carotid-femoral pulse wave velocity pulse wave velocity measurement (Δd: distance [m], Δt: transit time [ms], PWV=Δd/Δt).

IBM Corp., Armonk, New York, USA) statistics program. All variables were expressed as mean ± standard deviation. Mann–Whitney U-test was used to compare the groups. The relationship between pulse wave propagation time and variables was evaluated by linear regression test. A value of $p<0.05$ was considered statistically significant.

Results

Our study included 25 patients with newly diagnosed and inactive RA (mean age: 39.4±9.8 years, 21–63 years, 19 women, 6 men) and 29 healthy control cases (mean age: 36.9±9.9 years, 18–59 years, 13 women, 16 men) were included in the study. None of the patients had an additional cardiovascular disease such as hypertension, diabetes mellitus, hyperlipidemia, and coronary artery disease. The patients were not using any medication for the treatment of RA because it was in the inactive period.

There was no significant difference between the groups in terms of age, body weight, hip circumference, systolic blood pressure, diastolic blood pressure, average blood pressure, pulse pressure, and pulse rate (p=0.301, p=0.064, p=0.557, p=0.141, p=0.122, p=0.085, p=0.289, p=0.172, respectively). Height, waist circumference and waist/hip ratio values were significantly higher in healthy cases than in patients with RA (p=0.006, p=0.030, p=0.004), as shown in Figure 2. In patients with rheumatoid arthritis, the pulse wave velocity was significantly higher (p=0.041) and the pulse wave propagation time was significantly lower (p=0.000) compared to healthy subjects, as shown in Figure 3. A comparison of the features of RA and healthy cases is shown in Table 1. A comparison of the average values of patients with RA and the healthy control group is shown in Table 2.

When all participants were examined, there was a positive correlation between waist circumference pulse pressure, and pulse

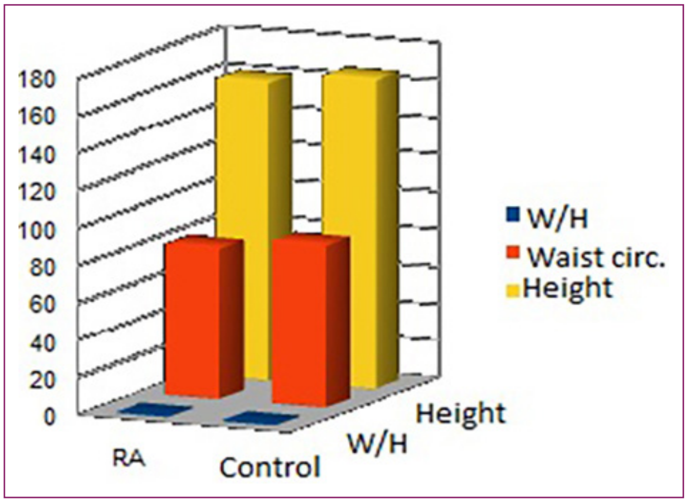

Figure 2. Height, waist circumference, and waist/hip ratio were significantly higher in healthy subjects when compared to patients with rheumatoid arthritis (p=0.006, p=0.030, p=0.004).

wave propagation time (p=0.039, p=0.003, respectively), while there was a negative correlation between systolic blood pressure and pulse wave velocity (p=0.003, p=0.27, p=0.000).

Discussion

In our study, we did not detect any differences in age, body weight, hip circumference, systolic blood pressure, diastolic blood pressure, mean blood pressure, pulse pressure, and pulse rate in the comparison of newly diagnosed inactive RA patients and healthy subjects. Height, waist circumference, and waist/hip ratio were significantly higher in healthy subjects. Pulse wave propagation time and pulse wave velocity, which were the ultimate aim of the study, were found to be significantly lower

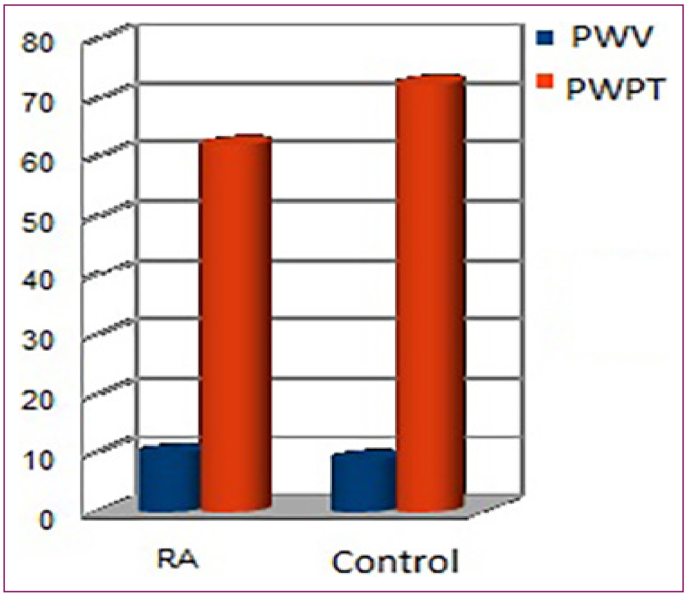

Figure 3. In patients with rheumatoid arthritis pulse wave velocity was significantly increased (p=0.041) and pulse wave propagation time was significantly lower (p=0.000) when compared with the healthy control.

and higher, respectively, in patients with rheumatoid arthritis. While there was a positive correlation between waist circumference and pulse pressure and pulse wave propagation time in all participants, there was a negative correlation between systolic blood pressure and pulse wave speed (in the classical formula, pulse wave speed and time are inversely proportional; Pulse Wave Speed=Distance/Time).

Determination of pulse wave velocity and pulse wave propagation time is one of the most important methods to evaluate the elastic properties of arteries. Pulse wave velocity can be measured using two ultrasound or pressure-sensitive transducers fixed transcutaneously on the trace of a pair of arteries (such as carotid-femoral arteries) separated by a certain distance, as stated in the current study.[6] The measured pulse wave velocity is an index of arterial wall stiffness

Table 1. Comparison of demographic properties of rheumatoid arthritis and control group

	Rheumatoid arthritis group (n=25)		Control group (n=29)	
	n	%	n	%
Male gender	6	24	16	55.1
Female gender	19	76	13	44.8
Avarage age	39.4±9.8		36.9±9.9	

and is also inversely proportional to arterial distensibility or relative arterial compliance ([dV/V]/dP) calculated by the classical formula of Imura et al.,[7]

Like arterial compliance, pulse wave velocity and the resulting distensibility index are also dependent on blood pressure.[7] After ventricular ejection, a pulse wave velocity is generated along the arterial tree (depending on the elastic, geometric properties and blood density of the arterial tree). Arterial wall thickness and lumen diameter changes are the main elements in pulse wave velocity measurement. This issue can be expressed as a mathematical formula: Pulse wave velocity according to the Moens–Korteweg equation = $\sqrt{Eh/2\delta R}$ or pulse wave velocity according to the Bramwell-Hill equation = $\sqrt{\Delta P.V/\Delta V}$ is δ. Here E: Young's modulus of the arterial wall ($E=\Delta P.D/h.\Delta D$ (cm^3. mmHg-1)), h: Wall thickness, R: Arterial radius, δ: Blood density, ΔP: Pressure change, ΔV: Volume change, ΔD: Represents Cap change.

Many studies have shown that arterial stiffness is an independent predictor of hypertension, stroke, atherosclerosis, cardiovascular events, and mortality.[9] Although there are slightly different values for each patient group, a meta-analysis of 17 parallel studies (including 15,877 patients) found that every 1 m/s increase in aortic pulse wave velocity increased the risk of cardiovascular disease and death by 10%, and one standard deviation increase increased the risk of cardiovascular disease and death by 40%. In addition, as a result

Table 2. Comparison of average parameter values between rheumatoid arthritis and control group

	RA	Control	p
Age	39.4±9.78	36.9±9.94	0.3
Weight	66.1±9.9	71.5±12.5	0.06
Height	160.2±7.6	166.8±8.5	0.006
Waist circumference	81.9±11.2	87.8±10.2	0.03
Waist/hip ratio	0.82±7.2	0.88±8.02	0.004
Hip circumference	100.6±13.8	98.8±5.9	0.55
Systolic blood pressure	119.6±13.9	113.6±12.09	0.14
Diastolic blood pressure	76.4±8.1	72.7±7.6	0.12
Average blood pressure	90.4±8.8	86.1±7.4	0.08
Pulse pressure	43.2±11.8	40.1±10.6	0.28
Pulse rate	80.1±9.2	76.3±10.1	0.17
Pulse wave velocity	9.79±1.51	8.76±1.09	0.04
Pulse wave propagation time	61.9±9.2	72.44±9.02	0.0

RA: Rheumatoid arthritis.

of this meta-analysis, it was once again seen that aortic pulse wave velocity has a predictive value independent of classical cardiovascular risk factors.[10]

As stated in this study, age, different blood pressures, heart rate, and arteriosclerosis affect the arterial tree to varying degrees; it has also more prominent effects on the central and elastic arteries. Increased resting heart rate increases arterial stiffening and cardiovascular mortality.[11] Albaladejo et al.[12] found a statistically non-significant positive relationship between pulse wave velocity and heart rate in 11 individuals with pacemakers. In our study, no significant difference was found between the two groups in terms of pulse rate.

With age, the elastic tissue of the aorta gradually decreases and is replaced by collagen tissue; meanwhile, the structure of elastic fibers also changes. Aortic pulse wave velocity (carotid-femoral or trunk pulse wave velocity) levels increase with age in both genders.[9] In our study, there was no significant difference in age between the two groups, and since the average ages for both groups were relatively low (39.4±9.8 years, 36.9±9.9 years, respectively), the effect of age on pulse wave velocity and pulse wave propagation time was not significant in our study group.

The aortic wall is not completely elastic. Due to the existing viscous elements, the faster the blood is thrown the more resistance will occur for tension and accordingly the pulse pressure will increase more. It is known that pulse wave speed increases with increasing intra-arterial pressure.[13] Potential stiffening mechanisms associated with increased blood pressure; includes thinning in the medial layer, smooth muscle cell hypertrophy and hyperplasia, expansion of the extracellular matrix, and fusion of collagen and elastin layers.[14] In our study group, there was no significant difference in blood pressure between the two groups. In the group with rheumatoid arthritis, systolic blood pressure, diastolic blood pressure, mean blood pressure, and pulse pressure had a statistically insignificant increase. In addition, the chance of hardened vessels being exposed to atherosclerosis is high.[15]

RA is a systemic immune and inflammatory disease and is associated with accelerated atherosclerosis and increased cardiovascular mortality and morbidity.[16] This inflammation can impair endothelial function, arterial compliance, and arterial elasticity, and may be an initiating or accelerating factor for atherosclerosis.[17] Some studies have shown that arterial elasticity is reduced in patients with RA.[18]

The cases with RA included in our study are far from the active period and the effects of drug use because they are newly diagnosed and are in the inactive period, and currently do not use medication for rheumatoid arthritis. Some studies show that in newly diagnosed and inactive patients, endothelial dysfunction and the negative effects of the disease on the cardiovascular system begin long before the onset of inflammation.[19]

Endothelial dysfunction is an important cause of atherosclerosis, coronary vasoconstriction, and myocardial ischemia, especially in connective tissue diseases.[20] In our study, height, waist circumference, and waist/hip ratios were significantly higher in the healthy control group than in the RA patient group. Although a clear relationship between height and pulse wave velocity and pulse wave propagation time has not been demonstrated in healthy subjects, in a study conducted by London et al.,[20] it was shown that short height values were associated with increased arterial stiffness in patients with end-stage renal failure.

Although we did not include any cases with a waist/hip ratio >1 in our study, the difference between the patient and control group in terms of waist circumference and waist/hip ratios is important. Many studies have shown that there is a strong positive correlation between waist circumference and waist/hip ratio and pulse wave velocity time.[21] In our study, although the waist circumference and waist/hip ratio were significantly higher in healthy subjects, the pulse wave velocity was found to be significantly higher and the pulse wave progression time was significantly lower in cases with rheumatoid arthritis, which increased the importance of the results of the study.

Conclusion

RA is a disease that is most common in adults, primarily targets synovial tissues, causes complications in other systems, and is characterized by endothelial dysfunction and therefore increased and accelerated atherosclerosis, similar to other connective tissue diseases. Therefore, it is important to detect and monitor cardiovascular risk status in patients with RA.

In our study, to reveal this situation, the comparison between newly diagnosed RA patients in the inactive period and healthy cases was made by evaluating the pulse wave propagation time and pulse wave speed; it was determined that carotid-femoral pulse wave velocity was statistically significantly higher and pulse wave propagation time was lower in patients with rheumatoid arthritis. This is especially important when considering the patient population, as the patients are newly diagnosed, young, in the inactive phase, and have no additional risk factors. As a result, in this patient group, the cardiovascular risk is increased due to impaired large artery mechanical properties (reduced elasticity of the arteries) and therefore the risks of cardiovascular mortality and morbidity are high.

Disclosures

Ethics Committee Approval: The study was approved by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Clinical Research Ethics Committee (no: 2013/08/01, date: 24/06/2013).

Authorship Contributions: Concept – A.Ş., E.O., S.O., İ.M.Ç.; Design – A.Ş., E.O., S.O., İ.M.Ç.; Supervision – A.Ş., E.O., İ.M.Ç.; Funding – A.Ş., S.O.; Materials – A.Ş., S.O., E.O.; Data collection and/or processing – A.Ş., S.O., E.O.; Data analysis and/or interpretation – S.O., E.O.; Literature search – A.Ş., S.O.; Writing – A.Ş., E.O.; Critical review – A.Ş., E.O., S.O., İ.M.Ç.

Conflict of Interest: All authors declared no conflict of interest.

Use of AI for Writing Assistance: Not declared.

Financial Disclosure: The authors declared that this study received no financial support.

Peer-review: Externally peer-reviewed.

References

1. Wållberg-Jonsson S, Johansson H, Ohman ML, Rantapää-Dahlqvist S. Extent of inflammation predicts cardiovascular disease and overall mortality in seropositive rheumatoid arthritis: A retrospective cohort study from disease onset. J Rheumatol 1999;26:2562–71. DOI:10.1371/journal.pone.0220531.
2. Manzi S, Meilahn EN, Rairie JE, Conte CG, Medsger TA, Jansen-McWilliams L, et al. Age-specific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: Comparison with the Framingham study. Am J Epidemiol 1997;145:408–15. DOI: 10.1093/oxfordjournals.aje.a009122.
3. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43. DOI: 10.1161/hc0902.104353.
4. Asmar R, Benetos A, Topouchian J, Laurent P, Pannier B, Brisac AM, et al. Assessment of arterial distensibility by automatic pulse wave velocity measurement. Validation and clinical application studies. Hypertension 1995;26:485–90. DOI: 10.1161/01.hyp.26.3.485.
5. Yildiz M. Commentary on viewpoint: The human cutaneous circulation as a model of generalized microvascular function. J Appl Physiol (1985) 2008;105:384, author reply 389. DOI: 10.1152/japplphysiol.90316.2006.
6. Smulyan H, Vardon S, Griffiths A, Gribbin B. Forearm arterial distensibility in systolic hypertension. J Am Coll Cardiol 1984;3:387–93. DOI: 10.1016/s0735-1097(84)80024-8.
7. Imura R, Yamamoto K, Kanamori K, Mikami T, Yasuda H. Non-invasive ultrasonic measurement of the elastic properties of the human abdominal aorta. Cardiovasc Res 1986;20:208–14. DOI:10.1093/cvr/20.3.208.
8. Darne B, Girerd X, Safar M, Cambien F, Guize L. Pulsatile versus steady component of blood pressure: A cross-sectional analysis and a prospective analysis on cardiovascular mortality. Hypertension 1989;13:392–400. DOI: 10.1161/01.hyp.13.4.392.
9. Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension 2001;37:1236–41. DOI: 10.1161/01.hyp.37.5.1236.
10. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: A systematic review and meta-analysis. J Am Coll Cardiol 2010;55:1318–27. DOI: 10.1016/j.jacc.2009.10.061.
11. Mangoni AA, Mircoli L, Giannattasio C, Ferrari AU, Mancia G. Heart rate-dependence of arterial distensibility *in vivo*. J Hypertens 1996;14:897–901. DOI: 10.1097/00004872-199607000-00013.
12. Albaladejo P, Copie X, Boutouyrie P, Laloux B, Declere AD, Smulyan H, et al. Heart rate, arterial stiffness, and wave reflections in paced patients. Hypertension 2001;38:949–52. DOI: 10.1161/hy1001.096210.
13. Asmar R, editor. Factors influencing pulse wave velocity. In: Arterial Stiffness and Pulse Wave Velocity. France: Elsevier; 1999. p. 57–88.
14. Glasser SP, Arnett DK, McVeigh GE, Finkelstein SM, Bank AJ, Morgan DJ, et al:Vascular compliance and cardiovascular disease: A risk factor or a marker? Am J Hypertens 1997;10:1175–18. DOI: 10.1016/s0895-7061(97)00311-7.
15. Glagov S, Grande JP, Xu CP, Giddens DP, Zarins CK. Limited effects of hyperlipidemia on the arterial smooth muscle response to mechanical stress. J Cardiovasc Pharmacol 1989;14 Suppl 6:S90–7.
16. Van Doornum S, McColl G, Wicks IP. Accelerated atherosclerosis: An extraarticular feature of rheumatoid arthritis? Arthritis Rheum 2002;46:862–73. DOI: 10.1002/art.10089.
17. Wong M, Toh L, Wilson A, Rowley K, Karschimkus C, Prior D, et al. Reduced arterial elasticity in rheumatoid arthritis and the relationship to vascular disease risk factors and inflammation. Arthritis Rheum 2003;48:81–9. DOI: 10.1002/art.10748.
18. Mahley RW, Mahley LL, Bersot TP, Pepin GM, Palaoglu KE. The Turkish lipid problem: Low levels of high-density lipoproteins. Turk J Endocr Metab 2002;1:1–12.
19. Kocabay G, Hasdemir H, Yildiz M. Evaluation of pulse wave velocity in systemic lupus erythematosus, rheumatoid arthritis and Behçet's disease. J Cardiol 2012;59:72–7. DOI: 10.1016/j.jjcc.2011.09.004.
20. London GM, Guerrin AP, Pannier B, Marchais SJ, Stimpel M. Influence of sex on arterial hemodynamics and blood pressure. Role of body height. Hypertension 1995;26:514–9. DOI: 10.1161/01.hyp.26.3.514.
21. Johansen NB, Vistisen D, Brunner EJ, Tabak AG, Shipley MJ, Wilkonson IB, et al. Determinants of aortic stiffness: 16-year follow-up of the whitehall II study. PLoS One 2012;7:e37165. DOI:10.1371/journal.pone.0037165.