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The Effect of Number of Pregnancies on Aortic Stiffness Index, Aortic Velocity Propagation, and Epicardial Fat Thickness

Gebelik Sayısının Aort Sertlik Indeksi, Aort Hızı Yayılımı ve Epikardiyal Yağ Kalınlığı Üzerine Etkisi

ABSTRACT

Objective: Pregnancy causes physiological, hormonal, and hemodynamic changes that affect the aortic wall dimensions and elastic properties. Multiple pregnancies increase the risk of aortic enlargement and reduce aortic elasticity. The aortic stiffness index (ASI) and aortic velocity propagation (AVP) are markers of elasticity. Additionally, epicardial fat thickness (EFT) is associated with cardiovascular risk factors. The impact of multiparity on ASI, AVP, and EFT has not been previously reported in the literature. Therefore, we aimed to investigate the association of these parameters with the number of live pregnancies in this study.

Methods: A total of 410 patients were enrolled in this prospective study. Patients were divided into three groups based on the number of live births: Group 1 (n = 0, 128 patients), Group 2 (4 ≥ n > 0, 157 patients), and Group 3 (n ≥ 5, 125 patients). A linear regression analysis was conducted to investigate trend associations of ASI, AVP, and EFT between the study groups. Multiple linear regression analysis was used to evaluate the independent predictors of continuous parameters.

Results: There were increasing trends in multiparity with variables such as aortic systolic (ASD) and diastolic diameters, pulmonary artery diameters, ASI, and EFT, and a decreasing trend in AVP. The number of pregnancies was strongly and positively correlated with ASI, moderately and positively correlated with EFT and ASD, and moderately and negatively correlated with AVP.

Conclusion: Multiparity was independently associated with ASI, EFT, ASD, and AVP, reflecting decreased elasticity and elevated cardiovascular risk in multiparous women.

Keywords: Aortic stiffness index, aortic velocity propagation, epicardial fat thickness, multiparity

ÖZET

Amaç: Gebelik, aort duvarının boyutlarını ve elastik özelliklerini etkileyen fizyolojik, hormonal ve hemodinamik değişikliklere neden olur. Çoğul gebelikler aort genişlemesi riskini artırır ve aort elastikiyetini azaltır. Aort sertlik indeksi (ASİ) ve aort hız yayılımı (AHY) esnekliğin belirteçleridir. Öte yandan epikardiyal yağ kalınlığı (EYD) kardiyovasküler risk faktörleriyle ilişkilidir. Multiparitenin, ASİ, AHY ve EYD üzerindeki etkisi daha önce literatürde bildirilmemiştir. Bu nedenle bu çalışmada bu parametrelerin canlı gebelik sayısı ile ilişkisini araştırmayı amaçladık.

Yöntem: Bu prospektif çalışmaya toplam 410 hasta dahil edildi. Hastalar canlı doğum sayılarına göre üç gruba ayrıldı [grup 1 (n = 0, 128 hasta), grup 2 (4 ≥ n > 0, 157 hasta) ve grup 3 (n ≥ 5, 125 hasta)]. Çalışma grupları arasında ASİ, AHY ve EYD'nin trend ilişkilerini araştırmak için doğrusal bir regresyon analizi kullanıldı. Sürekli parametrelerin bağımsız belirleyicilerini değerlendirmek için çoklu doğrusal regresyon analizi kullanıldı.

Bulgular: Aortik sistolik (ASÇ) ve diyastolik çaplar, pulmoner arter çapları, ASİ ve EYD gibi değişkenlerde multipariteye doğru artan eğilimler ve AHY'de azalma eğilimi vardı. Gebelik sayısı ASİ ile güçlü ve pozitif, EYD ve ASÇ ile orta ve pozitif, AHY ile orta ve negatif korelasyon gösterdi.

Sonuç: Multiparite, ASİ, EYD, ASÇ ve AHY ile bağımsız olarak ilişkilidir; ve bu, multipar kadınlarda elastikiyetin azalmasını ve kardiyovasküler riskin artmasını yansıtır.

Anahtar Kelimeler: Aort yayılma hızı, aort sertlik indeksi, epikardiyal yağ kalınlığı, multiparite

ORIGINAL ARTICLE
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During pregnancy, physiological changes affecting the functional and structural status of the aortic and pulmonary arteries occur.[1,2] In addition to hemodynamic alterations, hormonal variations can impact the connective tissue of the vascular media, promoting dilation of the great arteries and decreasing elastic properties.[3] Repeated pregnancies pose a high risk of elevated estrogen and progesterone hormone levels, which accelerate arterial wall deterioration.[4]

The aortic stiffness index (ASI) is a determinant of aortic elastic properties, and an elevated ASI reflects decreased aortic elasticity, identified as a cardiovascular (CV) disease risk factor.[5] The aortic stiffness index has been found to be independently associated with CV morbidity and mortality.[6] In a previous study, ASI was evaluated in pregnant women and was identified as an independent predictor of the development of preeclampsia.[7] Furthermore, women with a history of gestational diabetes were found to have higher ASI after a 5-year follow-up, which is associated with increased CV risk.[8] A review noted that changes in arterial stiffness during pregnancy could occur before clinical signs of gestational complications such as diabetes, hypertension, and kidney disease. The aortic stiffness index was reported as a predictor of these complications as well as CV morbidity and mortality.[9] Although aortic parameters, such as aortic dimensions and aortic compliance, have been evaluated in relation to multiparity, there are no reports in the literature concerning the relationship between ASI and multiparity.[10]

Aortic velocity propagation (AVP) is an easily accessible echocardiographic measurement that reflects aortic stiffness. Aortic velocity propagation has been reported to be inversely related to carotid intima-media thickness and coronary atherosclerosis in previous studies.[11] However, AVP has not been evaluated in pregnancy in the literature, which is the primary focus of this study. Epicardial fat thickness (EFT), a marker of visceral adipose tissue, can be easily measured using standard echocardiography. Epicardial fat thickness has been shown to be associated with CV risk factors, including insulin resistance, high blood pressure, inflammation, metabolic syndrome, and atherosclerosis.[12] Previous research has reported a link between EFT and gestational diabetes as well as pre-eclampsia.[13,14] However, the effect of multiparity on EFT has not been previously evaluated. Therefore, we aimed to investigate the impact of multiparity on the ASI, AVP, and EFT in this study.

ABBREVIATIONS

ASD	Aortic systolic
ASI	Aortic stiffness index
AVP	Aortic velocity propagation
CI	Confidence interval
CIMT	Carotid intima-media thickness
CRP	C-reactive protein
CV	Cardiovascular
EAT	Epicardial adipose tissue
EFT	Epicardial fat thickness
EFT	Epicardial fat thickness
IVS	Interventricular septal wall thicknesses
LAVI	Left atrial volume index
LV	Left ventricular
LVDd	Left ventricular end-diastolic
LVDs	Left ventricular end-systolic
MRI	Magnetic resonance imaging
RVFAC	Right ventricular fractional area change
RVMPI	Right ventricular myocardial performance index
SBP	Systolic blood pressure
TAPSE	Tricuspid annular plane
WBC	White blood cell

Materials and Methods

Patients admitted to the outpatient clinic for routine check-ups were included in this prospective study. Patients with acute or chronic infection, congestive heart failure, diabetes mellitus, coronary artery disease, hypertension, preeclampsia, gestational hypertension, moderate-to-severe valvular heart disease, congenital heart disease, chronic inflammatory disease, rheumatologic disease, malignancy, chronic kidney or liver disease, a history of aortic disease, or chronic obstructive pulmonary disease were excluded from the study. Based on the number of live births, the patients were divided into three groups: Group 1 ($n = 0$), Group 2 ($4 \geq n > 0$), and Group 3 ($n \geq 5$), as reported in previous studies.[15,16] When calculating the number of live births, we did not include birth before 34 weeks to avoid bias due to the duration of gestation. The Van Traning and Research Hospital Clinical Research Ethics Committee approved the trial (Approval Number: 2022/08-06, Date: 13.04.2022). Informed consent was obtained from all patients participating in the study. No artificial intelligence (AI)-assisted technologies (such as Large Language Models [LLMs], chatbots, or image creators) were used in the production of this manuscript. This study was conducted in accordance with the Declaration of Helsinki.

Echocardiography

All echocardiograms were performed in accordance with the American Society of Echocardiography recommendations, using a 2.5 MHz phased array (FPA) probe with the Philips Epiq 7 equipment (Philips Epiq 7 Ultrasound AS).[17] Left ventricular (LV) ejection fraction was measured using the modified Simpson method. The interventricular septal wall thicknesses (IVS) and the left ventricular end-diastolic (LVDd) and end-systolic (LVDs) chambers dimensions were recorded. The left atrial volume index (LAVI) was calculated by dividing the left atrial volume by the body surface area. Tissue Doppler imaging of the left ventricle was performed from the apical four-chamber view at a frame rate exceeding 80 frames per second. All measurements were taken from frozen images captured over three to five cardiac cycles. Standard Doppler values were used to determine early (E) and late (A) diastolic wave velocities based on mitral inflow. Pulsed wave tissue Doppler velocities (S, e', a') were recorded by positioning the sample volume at the LV lateral myocardial segment. The right ventricular myocardial performance index (RVMPI) was calculated using the formula: RVMPI = (isovolumetric contraction time + isovolumetric relaxation time) / ejection time. Right ventricular fractional area change (RVFAC) was calculated using the following formula: RVFAC = [(right ventricular end-diastolic area - right ventricular end-systolic area) / right ventricular end-diastolic area] x 100. The systolic excursion of the tricuspid annular plane (TAPSE) was measured using M-mode echocardiography, which quantifies the systolic longitudinal displacement of the lateral tricuspid annulus toward the apex. Pulmonary artery velocity was measured using pulsed wave Doppler, with the sample volume placed in the main trunk of

the pulmonary arteries beyond the pulmonary valve. Pulmonary artery acceleration time was assessed in the parasternal short-axis view by placing the sample volume of pulsed wave Doppler in the main pulmonary trunk, and it was defined as the time from the beginning of right ventricular (RV) ejection to the peak velocity. Carotid intima-media thickness (CIMT) was measured during diastole as the distance between the media-adventitia interface and the leading edge of the lumen-intima interface.

The aortic stiffness index was calculated using the following formula:

ASI = log [systolic blood pressure / diastolic blood pressure] / [(aortic systolic diameter - aortic diastolic diameter) / aortic diastolic diameter)] (Figure 1A).

Epicardial fat thickness was defined as the echo-free region between the outer edge of the myocardium and the visceral layer of the pericardium in end-diastole over the course of three cardiac cycles, measured from the RV parasternal long-axis view (Figure 1B). The aortic annulus was used as a reference point in the measurement of EFT, and the average of the maximum calculations was accepted.

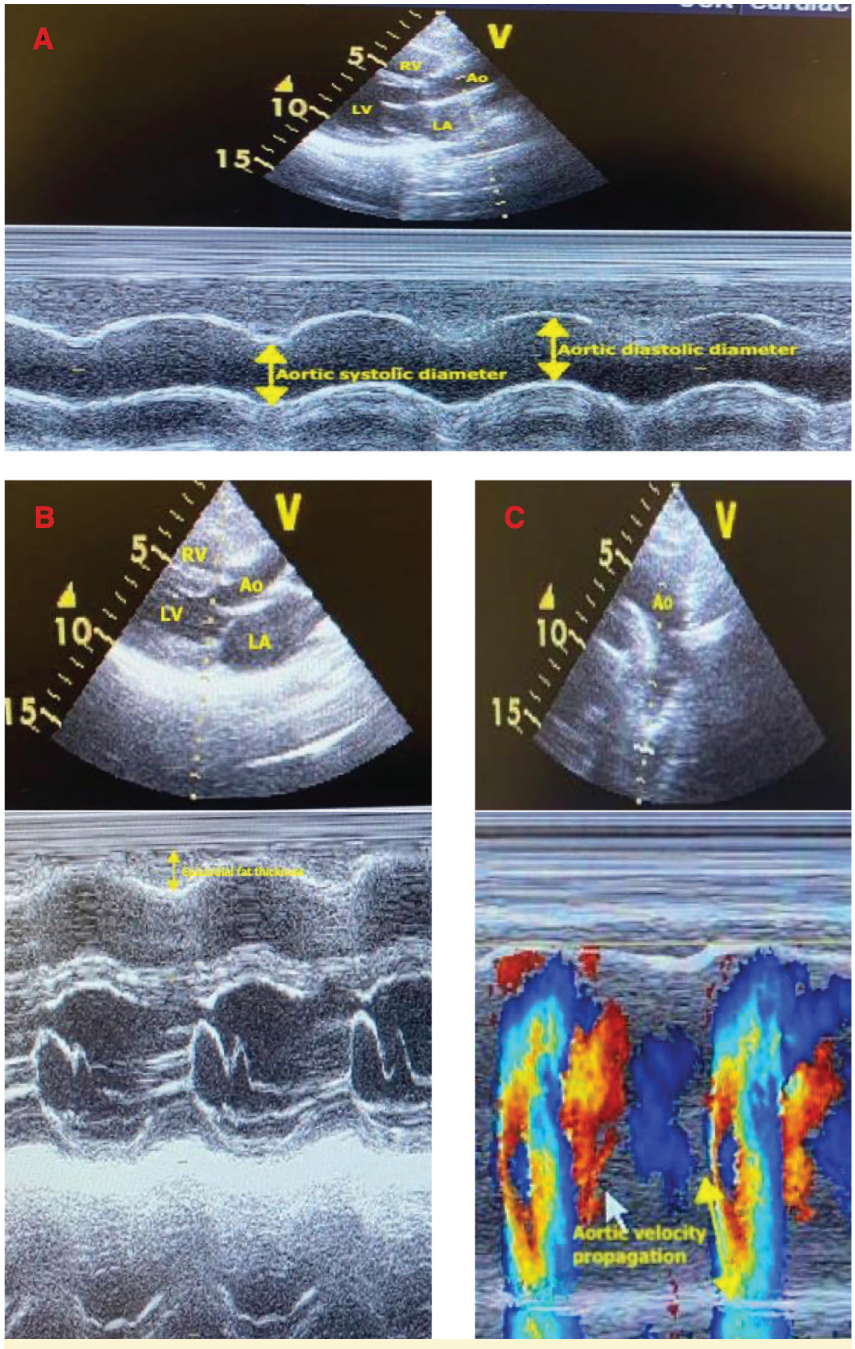

Figure 1. Measurements of aortic stiffness index (A), epicardial fat thickness (B), and aortic velocity propagation (C).

Table 1. Comparisons of Baseline Characteristics Between Study Groups

	Group 1 (n = 0) *n = 128*	Group 2 (4 ≥ n > 0) *n = 157*	Group 3 (n ≥ 5) *n = 125*	*P* for Trend
Age, years	42.5 (27.0-56.0)	40.0 (34.0-52.0)	52.0 (45.0-60.0)	<0.001
BMI, kg/m^2	27.3 (24.8-30.6)	27.3 (24.2-31.6)	29.3 (26.2-33.3)	<0.001
SBP, mmHg	105 (100-120)	110 (100-120)	120 (115-130)	<0.001
White blood cell count, x10^3/μL	7.84 (6.64-9.02)	7.84 (6.58-9.44)	7.84 (6.88-9.51)	0.026
Hemoglobin, g/dL	13.9 (13.2-14.6)	13.6 (12.8-14.8)	13.9 (13.2-14.9)	0.416
Platelets, x10^3/μL	255 (222-301)	248 (215-296)	257 (230-298)	0.481
MPV, fL	9.60 (9.00-10.6)	9.90 (9.20-10.7)	9.50 (8.80-10.3)	0.388
RDW, fL	41.2 (39.6-43.5)	42.4 (40.4-44.7)	41.7 (39.6-44.6)	0.127
Glucose, mg/dL	90.0 (83.8-99.5)	88.0 (76.0-100)	92.0 (82.4-100)	0.460
Creatinine clearance, mL/min	101 (84.1-127)	110 (89.5-134)	107 (85.3-125)	0.880
CRP, mg/dL	3.25 (1.85-6.34)	4.65 (2.00-6.90)	5.38 (2.20-8.60)	0.005
Uric acid, mg/dL	5.00 (4.20-6.03)	5.00 (4.00-6.40)	5.50 (4.20-8.09)	0.002
Sodium, mEq/L	139 (138-140)	139 (137-140)	139 (135-140)	0.011
Calcium, mEq/L	9.11 (9.00-9.39)	9.18 (8.90-9.55)	9.30 (8.90-9.50)	0.082
Albumin, g/dL	4.40 (4.20-4.60)	4.41 (4.05-4.63)	4.40 (4.00-4.60)	0.460
Triglycerides, mg/dL	145 (95.0-179)	151 (113-192)	160 (136-214)	<0.001
HDL-cholesterol, mg/dL	42.0 (37.8-50.5)	39.3 (38.0-49.0)	39.3 (36.0-47.0)	0.061
LDL-cholesterol, mg/dL	90.8 (74.0-119)	93.4 (86.0-119)	102 (87.0-123)	0.059
Heart rate, beats/min	77.0 (71.8-85.8)	79.0 (70.0-85.0)	79.5 (72.0-86.0)	0.641

BMI, Body Mass Index; CRP, C-Reactive Protein; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; MPV, Mean Platelet Volume; RDW, Red Cell Distribution Width; SBP, Systolic Blood Pressure.

Aortic velocity propagation was assessed using M-mode Doppler from a suprasternal position by dividing the distance between the propagation slope's onset and termination by the corresponding time (Figure 1C). All echocardiographic measurements for ASI, AVP, and EFT were performed by a second cardiologist, with inter- and intra-observer variability of 4% and 2%, respectively.

Statistical Analysis

All statistical analyses were performed using R statistical software, version 4.2.2 (Institute for Statistics and Mathematics, Vienna, Austria). The Kolmogorov-Smirnov test was used to assess whether the variables followed a normal distribution. The mean (standard deviation, SD) was used to indicate continuous variables with normally distributed data, and the median (Q1-Q3) was used for those without normal distribution. Categorical variables were presented as numbers and percentages. Linear regression for continuous variables was used to determine the p values for linear trends in baseline and echocardiographic features comparisons. Multivariable models were created by adjusting for age, body mass index, systolic blood pressure (SBP), white blood cell (WBC) count, triglycerides, left ventricular end-diastolic diameter (LVDd), uric acid, serum calcium, IVS, left atrial volume index (LAVI), and C-reactive protein (CRP). Variables that were clinically relevant or unbalanced between parity groups were included in the multiple linear regression models for detecting outcomes. The models did not contain variables identified as having multicollinearity using thresholds of a variance inflation factor > 3 or tolerance < 0.1. Spearman correlation analysis was conducted to detect the correlations between ASI, EFT, aortic systolic diameter, and AVP with the number of pregnancies. The 95% confidence interval (CI) was used to analyze the data, and the significance level was set at a two-tailed p-value of 0.05.

Results

A total of 410 patients [128 patients in Group 1 (median age = 42.5 years), 157 patients in Group 2 (median age = 40 years), and 125 patients in Group 3 (median age = 52 years)] were divided into three categories according to the number of pregnancies as described above. There were increasing trends in age, body mass index (BMI), SBP, WBC count, CRP, uric acid, and triglyceride levels, and a decreasing trend in sodium with increasing multiparity (Table 1).

Table 2 shows the linear trend of echocardiographic measurements with multiparity. There were increasing trends in IVS, LVDd, LVDs, LAVI, A wave, RV basal diameter, pulmonary artery diameter, aortic systolic and diastolic diameters, ASI, CIMT, and EFT, along with a decreasing trend in AVP with increasing multiparity. Multiple linear regression models demonstrated that Group 2 and Group 3 were independently associated with higher levels of ASI, EFT, and aortic systolic diameter, and lower levels of AVP compared to the reference Group 1 (Table 3). Correlation

Table 2. Comparisons of Echocardiographic Parameters Between Study Groups

	Group 1 (n = 0) *n = 128*	Group 2 (4 ≥ n ≥ 1) *n = 157*	Group 3 (n ≥ 5) *n = 125*	*P* for Trend
LVEF, %	60.0 (60.0-62.0)	60.0 (60.0-62.0)	60.0 (60.0-60.0)	0.923
IVS, cm	1.00 (0.90-1.20)	1.10 (0.90-1.20)	1.10 (1.00-1.20)	0.002
LVDd, cm	4.20 (3.90-4.60)	4.30 (4.00-4.70)	4.60 (4.10-5.00)	<0.001
LVDs, cm	2.80 (2.50-3.12)	2.80 (2.50-3.30)	2.90 (2.50-3.30)	0.024
LAVI, mL/m^2	29.0 (25.8-32.0)	30.0 (27.0-34.0)	31.0 (28.0-34.0)	<0.001
E wave, cm/s	0.75 (0.60-0.88)	0.73 (0.62-0.91)	0.67 (0.58-0.88)	0.121
A wave, cm/s	0.65 (0.53-0.76)	0.67 (0.55-0.79)	0.69 (0.60-0.85)	0.005
LV S wave, m/s	0.08 (0.07-0.10)	0.08 (0.07-0.10)	0.08 (0.07-0.10)	0.205
LV e', m/s	0.13 (0.10-0.16)	0.12 (0.09-0.15)	0.12 (0.09-0.15)	0.191
LV a', m/s	0.08 (0.07-0.11)	0.08 (0.07-0.10)	0.09 (0.08-0.11)	0.112
RV basal diameter, cm	3.10 (2.70-3.60)	3.30 (2.80-3.80)	3.40 (2.90-3.90)	0.027
RV longitudinal diameter, cm	5.80 (5.00-6.20)	5.70 (5.00-6.10)	5.70 (5.00-6.30)	0.965
RVFAC, %	0.35 (0.28-0.40)	0.34 (0.26-0.38)	0.35 (0.25-0.41)	0.812
sPAP, mmHg	14.0 (6.00-20.0)	15.0 (6.00-22.0)	18.0 (6.00-24.0)	0.089
RVMPI	0.62 (0.49-0.82)	0.63 (0.50-0.81)	0.68 (0.47-0.83)	0.853
TAPSE, cm	2.00 (1.80-2.50)	2.00 (1.60-2.50)	2.00 (1.70-2.50)	0.981
Pulmonary artery velocity, m/s	0.90 (0.81-0.97)	0.90 (0.82-1.00)	0.88 (0.80-0.96)	0.518
Pulmonary artery acceleration time, cm/s	96.0 (74.0-112)	96.0 (80.0-111)	98.0 (80.0-111)	0.745
Pulmonary artery diameter, cm	2.07 (1.75-2.35)	2.10 (1.90-2.40)	2.30 (2.00-2.60)	<0.001
Aortic systolic diameter, mm	28.6 (4.2)	30.1 (3.7)	31.6 (3.7)	<0.001
Aortic diastolic diameter, mm	21 (19-24)	23 (20-26)	26 (23-29)	<0.001
AVP, m/s	0.58 (0.44-0.73)	0.46 (0.39-0.70)	0.40 (0.33-0.48)	<0.001
Aortic stiffness index	1.17 (0.81-1.88)	1.78 (1.39-2.54)	2.91 (2.12-3.96)	<0.001
PAR	0.73 (0.64-0.82)	0.69 (0.64-0.79)	0.74 (0.67-0.79)	0.996
CIMT, cm	0.06 (0.05-0.09)	0.08 (0.05-0.10)	0.09 (0.08-0.11)	0.043
Epicardial fat thickness, cm	0.40 (0.30-0.50)	0.40 (0.30-0.70)	0.70 (0.60-0.90)	<0.001

AVP, Aortic Velocity Propagation; CIMT, Carotid Intima-Media Thickness; IVS, Interventricular Septum; LAVI, Left Ventricular Volume Index; LVDd, Left Ventricular Diastolic Diameter; LVDs, Left Ventricular Systolic Diameter; LVEF, Left Ventricular Ejection Fraction; LV, Left Ventricular; PAR, Pulmonary-to-Aortic Diameter Ratio; RV, Right Ventricular; RVFAC, Right Ventricular Fractional Area Change; RVMPI, Right Ventricular Myocardial Performance Index; sPAP, Pulmonary Artery Pressure; TAPSE, Tricuspid Annular Plane Systolic Excursion.

analyses revealed a strong positive correlation between the number of pregnancies and ASI (r = 0.5, P < 0.001), a moderate positive correlation with aortic systolic diameter (r = 0.3, P < 0.001), and epicardial fat thickness (r = 0.45, P < 0.001), and a moderate negative correlation with AVP (r = -0.34, P < 0.001) (Figure 2).

Discussion

The primary findings of this investigation demonstrated that multiparous patients had larger pulmonary and aortic diameters, increased CIMT, EFT, ASI, along with a lower AVP. Additionally, multiparity was independently associated with ASI, EFT, aortic systolic diameter, and AVP. Significant correlations were observed between ASI, EFT, AVP, aortic diameters, and the number of pregnancies.

Estrogen and progesterone levels increase throughout the gestational period, leading to altered hemodynamics during pregnancy.[4] The rise in cardiac output and blood volume during pregnancy predisposes women to an elevated risk of CV diseases.[18] It is well established that hormonal changes in pregnancy can result in t the structural degradation of elastic fibers and cause hyperplasia and hypertrophy of the aortic smooth muscle cells.[4] This process increases the laxity of the aortic wall, leading to aortic enlargement and thinning of the aortic wall. Consequently, the expansion of the aortic diameter can increase the tension on the aortic wall.[2,4] According to previous research, the changes in aortic diameter that occur during pregnancy might persist long after delivery and may not return to pregestational values.[19]

Previous studies have explored the relationship between aortic dimensions and multiparity. Gutin et al.[20] found that women

Table 3. Multiple Linear Regression Analysis for Detecting Independent Predictors of Aortic Stiffness Index

	Coefficient	SE	*t*	*P*
Aortic Stiffness Index				
Group 1	Ref	Ref	Ref	Ref
Group 2	0.7560	0.2651	2.85	0.0046
Group 3	1.7431	0.3136	5.56	<0.0001
AVP				
Group 1	Ref	Ref	Ref	Ref
Group 2	-0.0568	0.0219	-2.59	0.0099
Group 3	-0.1695	0.0259	-6.54	<0.0001
Epicardial Fat Thickness				
Group 1	Ref	Ref	Ref	Ref
Group 2	0.0637	0.0258	2.47	0.0138
Group 3	0.2746	0.0305	9.01	<0.0001
CIMT				
Group 1	Ref	Ref	Ref	Ref
Group 2	0.0084	0.0125	0.67	0.5019
Group 3	0.0154	0.0148	1.04	0.2987
Aortic Systolic Diameter				
Group 1	Ref	Ref	Ref	Ref
Group 2	1.1094	0.4391	2.53	0.0119
Group 3	1.3289	0.5229	2.54	0.0114

AVP, Aortic Velocity Propagation; CIMT, Carotid Intima-Media Thickness; SE, Standard Error.
*Multivariable models were adjusted for age, body mass index, white blood cell count, systolic blood pressure, C-reactive protein, triglycerides, left ventricular diastolic diameter, uric acid, serum calcium, interventricular septum diameter, and left atrial volume index.

with multiparity had larger ascending and descending aortas compared to nulliparous women. Similarly, Pourafkari et al.[19] reported that multiparity was a significant predictor of aortic enlargement. A magnetic resonance imaging (MRI)-based study conducted by Singh et al.[10] also demonstrated that aortic enlargement was independently associated with multiparity. However, Zoet et al.[21] did not find any differences in aortic dimensions and pulse wave Doppler parameters between parous and nulliparous women. The small sample size in their study may explain this contradictory finding. In accordance with previous studies, multiparous women had larger aortic systolic and diastolic dimensions, and multiparity was independently associated with aortic systolic diameter in our study.

Hormonal changes during pregnancy affect connective tissue, altering the structure of the medial layer of the aorta. Histological analysis of the tunica media has shown fragmentation of reticulin, the protein that normally encircles elastic fibers, along with enlargement and smooth muscle cell hyperplasia.[2] Eventually, elastic fibers lose their organized structure, reducing elasticity. The aortic stiffness index is a non-invasive measurement of arterial elasticity; however, the effect of multiparity on ASI has not been evaluated in the literature. Mersich et al.[22] demonstrated that an increased number of trimesters of pregnancy was associated with higher carotid artery stiffness index values. Conversely, Ulusoy et al.[23] reported that young pregnant women had lower ASI than non-pregnant women. We believe the results of that study should be interpreted with caution, as higher ASI values have been widely recognized as indicators of reduced aortic elastic properties in previous reports.[24-27] Our study might be the first to evaluate ASI in relation to a trend of increasing multiparity. There have been no reports in the literature comparing EFT and/or AVP between parous and nulliparous women.

It has been reported that EFT is associated with gestational diabetes and preeclampsia in previous studies.[28,29] Our study revealed higher EFT values in multiparous women, with multiparity being independently associated with EFT. A possible explanation for this finding is that epicardial adipose tissue (EAT) functions as brown adipose tissue, and higher estrogen levels may increase the expression of genes responsible for EAT production, resulting in higher EFT in multiparous women compared to nulliparous women.[30,31] On the other hand, AVP is a well-established marker of aortic elasticity and is associated with increased CV risk.[11] The relationship between AVP and the number of pregnancies has not been explored in previous research. This study revealed the effect of the number of pregnancies on AVP in multiparous women.

Based on our study results, we suggest that these echocardiographic parameters might reflect increased CV risk and could be used for risk stratification in these patients. Multiparous women may be at higher risk for aortic pathologies

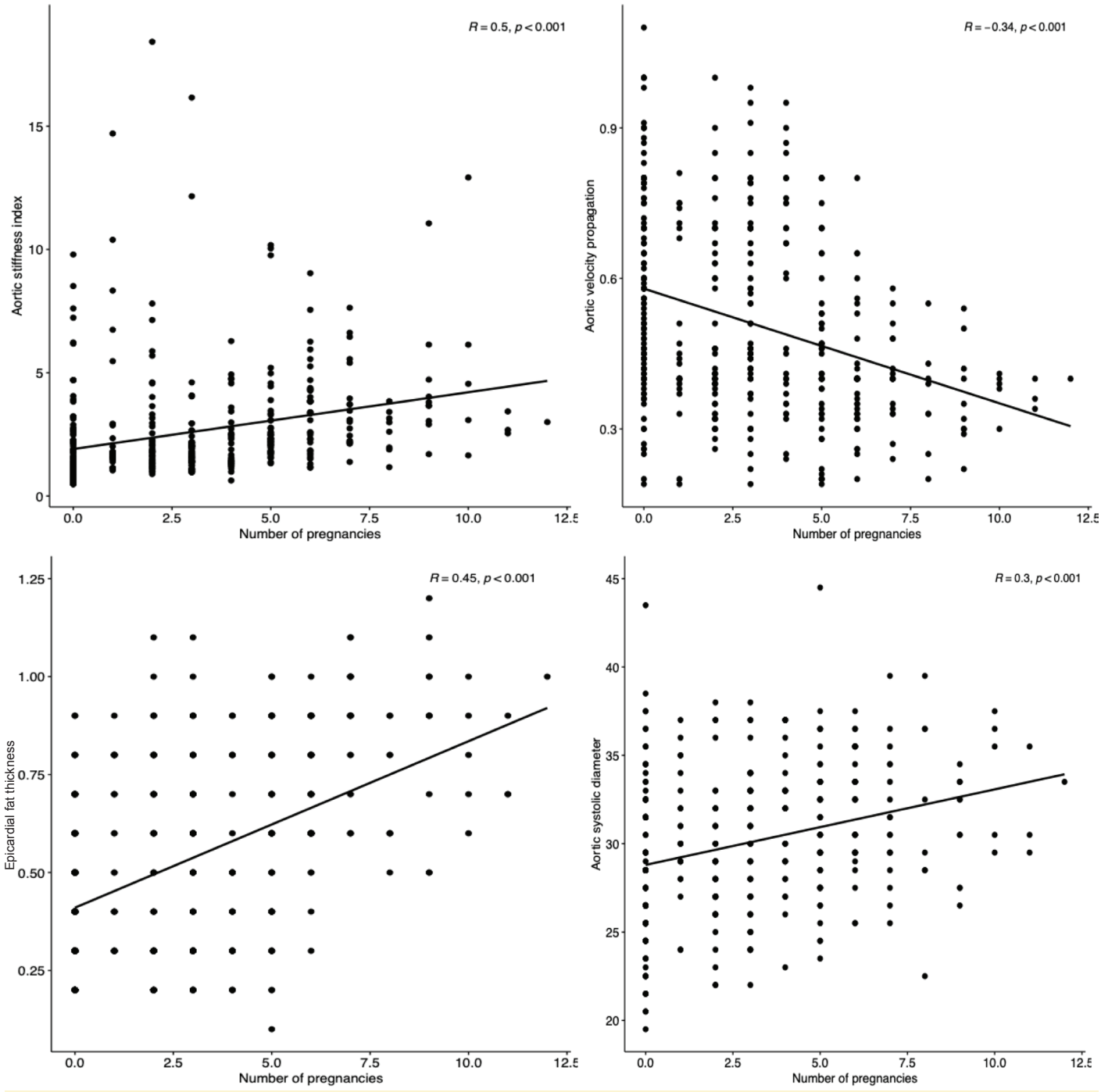

Figure 2. Correlations between the number of pregnancies and aortic stiffness index, aortic velocity propagation, epicardial fat thickness, and systolic aortic diameter.

and CV disease. Therefore, this patient population should be monitored closely, and strategies to prevent adverse events should be implemented early.

Limitations

The primary limitation of this research was that it was conducted in a single geographic region. Thus, the results might not be generalizable to other geographical areas. Multiparous women in the study were older, and although age was included as a confounding factor in the multivariable model, there may still be unknown confounding factors related to age that were not accounted for. Additionally, the use of MRI for assessing aortic diameters might provide more accurate results.

Conclusion

Multiparous women had higher ASI and EFT and lower AVP values compared to nulliparous women. Multiparity was independently associated with these parameters, which reflect decreased aortic elasticity and increased CV risk in these patients. However, we

believe that further research is necessary to fully clarify the relationship between multiparity and elevated cardiovascular risk.

Ethics Committee Approval: Ethics committee approval was obtained from The Van Traning and Research Hospital Clinical Research Ethics Committee (Approval Number: 2022/08-06, Date: 13.04.2022).

Informed Consent: Informed consent was obtained from all patients participating in the study.

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