ARAŞTIRMA MAKALESİ / *Rerseasrch Article*

The Relationship Between Cardio-Ankle Vascular Index and Clinical Factors with Aortic Valve Sclerosis in Asymptomatic Patients

Asemptomatik Hastalarda Aort Kapak Sklerozu ile Kalp-Ayak Bileği Damar İndeksi ve Klinik Faktörler Arasındaki İlişki

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Abstract

Introduction	This study aimed to examine the relationship between aortic valve sclerosis (AVS) and arterial stiffness in asymptomatic individuals without known cardiovascular (CV) disease.Both AVS and arterial stiffness are associated with atherosclerosis and have been closely related to CV diseases in previous studies. In this study, we aimed to examine the relationship between arterial stiffness assessed by CAVI and AVS.
Materials and Methods	Patients who applied to the cardiology outpatient clinic were included in the study sequentially. Subjects were analyzed according to exclusion criteria. One hundred sixty-five patients were included in the study, and AVS was detected in 35 (21%) of them. The remaining 130 (79%) patients were included in the control group.AVS was measured with echocardiography, and arterial stiffness was measured with the VaSera VS-1000 CAVI device. A CAVI value of 9 and above was accepted as abnormal. Statistics were made according to the group with and without AVS.
Results	CAVI was statistically different between the AVS and control groups (9.47±1.64 vs. 7.60±1.27 p< 0.001). The Pearson correlation test determined the correlation between AVS and increased CAVI values (p< 0.001). In the multivariable logistic regression analysis model, increased CAVI (OR: 2.048, 95%CI 1.183-3.547, p: 0.010) was an independent predictor for AVS.Others were found as age (p:0.026) and diabetes mellitus (p:0.037).
Conclusion	The relationship between AVS and arterial stiffness is associated with the atherosclerotic process.Careful investigation and regular follow-up of asymptomatic individuals with AVS detected during echocardiography or increased CAVI values are important in other CV diseases.
Keywords	Aortic valve sclerosis; Arterial stiffness; Asymptomatic patients; Cardio-Ankle Vascular Index (CAVI).

Öz

Amaç	*Bu çalışma, bilinen kardiyovasküler (KV) hastalığı olmayan asemptomatik bireylerde aort kapak sklerozu (AVS) ile arteriyel sertlik arasındaki ilişkiyi incelemeyi amaçlamıştır. Hem AVS hem de arteriyel sertlik ateroskleroz ile ilişkilidir ve daha önceki çalışmalarda KV hastalıklarla yakından ilişkili bulunmuştur. . Bu çalışmada, CAVI ile değerlendirilen arteriyel sertlik ile AVS arasındaki ilişkiyi incelemeyi amaçladık.*
Yöntem ve Gereçler	*Kardiyoloji polikliniğine başvuran hastalar sırayla çalışmaya dahil edildi. Olgular dışlama kriterlerine göre analiz edildi. Çalışmaya yüz altmış beş hasta dahil edildi ve bunların 35'inde (%21) AVS tespit edildi. Geriye kalan 130 (%79) hasta kontrol grubuna alındı. Ekokardiyografi ile AVS, VaSera VS-1000 CAVI cihazı ile arter sertliği ölçüldü. 9 ve üzeri CAVI değeri anormal olarak kabul edildi. İstatistikler AVS olan ve olmayan gruba göre*
Bulgular	*CAVI, AVS ve kontrol grupları arasında istatistiksel olarak farklıydı (9,47±1,64 vs. 7,60±1,27 p< 0,001). Pearson korelasyon testi, AVS ile artan CAVI değerleri arasındaki korelasyonu belirledi (p< 0,001). Çok değişkenli lojistik regresyon analizi modelinde, artan CAVI (OR: 2.048, %95 CI 1.183-3.547, p: 0.010) için bağımsız bir öngörücüydü. AVS. Diğerleri yaş (p:0.026) ve diabetes mellitus (p:0.037) olarak bulundu.*
Sonuç	*AVS ile arteriyel sertlik arasındaki ilişki aterosklerotik süreç ile ilişkilidir. Ekokardiyografi sırasında AVS saptanan veya CAVI değerleri yüksek olan asemptomatik bireylerin diğer KV hastalıklarda dikkatli araştırılması ve düzenli takibi önemlidir.*
Anahtar Kelimeler	*C-Reaktif Protein, Kardiyovasküler Risk Skoru, Dislipidemiler*

INTRODUCTION

In echocardiographic evaluation, aortic valve sclerosis (AVS) means focal thickening and calcification of the aortic valve without obstructing blood flow at the exit of the left ventricle[1]. AVS associationwith age, male gender, hypertension, hyperlipidemia, diabetes, and smoking suggested an atherosclerotic process in its pathogenesis[2].AVS prevalence is estimated to be 30% and 40% in 65 and 75 years, respectively[3,4]. Moderate and severe aortic stenosis was detected at a rate of 6% in the average 7-year follow-up of individuals with AVS who did not cause significant stenosis in the left ventricular outflow tract[5]. In addition to being a precursor to aortic stenosis, AVS is also associated with cardiovascular (CV) diseases[6,7]. It was significantly related to CV mortality and all-cause mortality, and diseases such as coronary artery disease and stroke[1].

Arterial stiffness results from structural changes in the arterial system due to atherosclerotic changes[8]. Arterial stiffness was accepted as a CV risk factor, and increased stiffness was a mortality marker[9,10]. Pulse wave velocity (PWV) and cardio-ankle vascular index (CAVI) are frequently used as arterial stiffness measurement methods.PWV is affected by blood pressure, body weight, fasting blood sugar level[11]. CAVI calculates the overall stiffness of the artery from the origin of the aorta to the ankle using the stiffness parameter β and the Bramwell-Hill formula.The most important feature of the CAVI technique is that it is not affected by blood pressure[12]. Operator independence and repeatability of CAVI measurement can be shown among other features that are superior to other methods[13].

This study examined the relationship between arterial stiffness and AVS, which are frequently associated with the atherosclerotic process and CV diseases.

MATERIALS and METHODS

Study Design and Population

This study is a single-center, cross-sectional, prospective study conducted between April 2021 and June 2021.In our study, 540 patients who applied to the cardiology outpatient clinic and underwent routine echocardiography were evaluated sequentially.Exclusion criteria are determined as coronary heart disease, congenital heart disease, aortic stenosis (transaortic flow velocity >2.5 m/s), symptoms of congestive heart failure or ejection fraction less than 50%, chronic kidney disease (glomerular filtration rate (GFR) <30 ml/ min), atrial fibrillation, bicuspid aortic valves, bacterial endocarditis, symptomatic peripheral artery disease, history of stroke or transient ischemic attack, and malignancy. All patients were analyzed according to exclusion criteria, and 165 patients were eligible for the study.Demographic characteristics, blood tests, echocardiographic values , and CAVI results of the patients included in the study were recorded.Body mass index (BMI) was calculated as weight (kg)/height (m2).Smoking was defined as "current smokers" or "non-smokers".Biochemical measurements including kidney function tests, fasting blood glucose, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (TG) were performed.Hematological parameters were measured as part of an automated complete blood count (CBC) using a Mindray BC-5800 automated hematology analyzer (Mindray Medical Electronics Co. Shenzhen, China).Patients with systolic blood pressure > 140 mm Hg and/or diastolic pressure > 90 mm Hg or using antihypertensive medication were considered hypertensive. Patients with fasting glucose of 126 mg/dL and/or using pharmacological treatment were considered diabetic.Total cholesterol > 200 mg/dL or use of medication was defined as hypercholesterolemia.

The study protocol was approved by the local Ethics Committee(Board date , number: 04.04.2021, 2021/57).Following the Declaration of Helsinki and the International Conference on Compliance with Good Clinical Practices, the study was conducted, and written informed consent was obtained from all participants.

Echocardiographic Evaluation

An echocardiographic examination was performed with the Philips IE33 system (Philips Medical Systems, Andover, MA, USA).Echocardiograms were evaluated for routine parameters and AVS by the same experienced cardiologist following the American Society of Echocardiography (ASE) recommendations. Left ventricular (LV) end-systolic volume (ESV), end-diastolic volume (EDV), and ejection fraction (EF) are derived from the apical biplane Simpson's rule.Left ventricular mass index (LVMI) was calculated according to the body surface area of the patients (DuBois formula).Tricuspid lateral annular systolic velocity (S') was measured, as assessed by tissue Doppler imaging, in the lateral segment of the right ventricle from the apical 4-chamber view. AVS was considered increased calcification or thickening in any or all three leaflets of the aortic valve without creating left ventricular stenosis (transaortic flow velocity <2.5 m/s)(Figure 1).

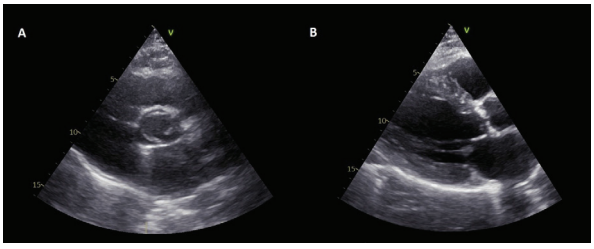

Figure 1: A: Parasternal short axis aortic valve view, B: Parasternal long axis aortic valve view

Measurement of Arterial Stiffness

Arterial stiffness was measured by the CAVI method. CAVI was calculated using the VaSera VS-1000 (Fukuda Denshi Co. Ltd, Tokyo) instrument. Measurements were made after 10 minutes of rest. The subject was placed on his/her back with the head in the middle position.Sleeves were attached to both arms and feet. A microphone was placed on the chest. Electrography, phonocardiography, pressure, and waveforms of brachial and ankle arteries were measured. CAVI was calculated automatically by the instrument. CAVI values were accepted as normal (CAVI < 8), borderline (8 ≤ CAVI < 9), and abnormal (CAVI ≥ 9), following the manufacturer and previous studies.Our study was based on an abnormal CAVI value of 9 and above.

Statistical Analysis

SPSS 21.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for statistical analysis.Kolmogorov-Smirnov and homogeneity of variance tests were performed to select the appropriate analysis technique.Independent samples t-test was used for two-group comparison of normally distributed variables, and Mann–Whitney U-test was used for two-group comparison of non-normally distributed variables. Categorical variables were compared using the Chi-square test. Data were expressed as mean±standard deviation for normally distributed continuous variables and median and interquartile ranges for skewed-distributed continuous variables. Categorical variables were presented as numbers and percentages. All variables were evaluated individually in univariable logistic regression analysis to determine predictive parameters of AVS. Significant variables ($p<0.05$) were considered a potential risk marker and included in the multivariable logistic regression analysis. Amultivariable logistic regression test determined independent predictors. ROC analysis was used to determine the cut-off levels of CAVI that could predict AVS detection. $P < 0.05$ was considered statistically significant.

RESULTS

Five hundred forty patients were evaluated for our study. According to exclusion criteria, 165 patients were included in the study after analysis.Among the patients included in the study, AVS was detected in 35 patients (15 females and 20 males; mean age 66.14±12.03 years).AVS was not detected in 130 patients (61 females and 69 males; mean age 50.69±11.22 years) included in the study and was determined as the control group. When all patients included in the study were evaluated, the mean age was determined as 53.96±13 years and the frequency of AVS was 21%. The basic characteristics of the individuals included in the study are presented in Table-1. There was a statisti-

cal difference between two groups in age (66.14±12.03 vs 50.69±11.22 p<0.001), hypertension (HT) (24 (68%) vs 48 (37%) p:0.001) and diabetes mellitus (DM) (5 (14%) vs. 4(0.03%) p:0.01). There was no difference between the two groups in other demographic characteristics and blood tests.Echocardiographic values and CAVI results of the patients are presented in Table-2. When the two groups were evaluated, left ventricular mass index (LVMI), interventricular septum (IVS), posterior wall (PW), left ventricular outflow tract velocity were found to be higher in the AVS group (101.8 (64.62-177.72) vs 82.13 (51.04-177.56), 12 (9-15) vs 9 (8-16), 12 (9-13) vs 9 (8-13), 104.0±22.85 vs 96.62±17.88 ; p< 0.001, p< 0.001, p< 0.001, p:0.045, respectively). There was no statistical difference between other echocardiographic parameters. CAVI (9.47±1.64 vs. 7.60±1.27 p< 0.001) was higher in the AVS group than in the control group. (Figure 2,3)

The correlation between AVS and increased CAVI values was determined by Pearson correlation test (p< 0.001, Correlation Coefficient: 0.494). In theunivariable regression analysis performed for all variables one by oneto determine the predictors of AVS, age (p< 0.001), HT (p:0.001), DM (p:0.018), IVS (p< 0.001), PW (p< 0.001), LVMI (p< 0.001) and CAVI (p< 0.001) were associated with AVS.Inmultivariate analysis, age (OR: 1.070, 95%CI 1.008-11.365, p: 0.026), DM (OR: 6.633, 95%CI 1.117-39.379, p: 0.037) and CAVI (OR: 2.048, 95%CI 1.183-3.547, p: 0.010) were shown to be independent predictors for AVS(Table 3).In the ROC curve analysis for the estimation of AVS detection, the CAVI cut-off value was 8,475 with a sensitivity of 71.4% and a specificity of 71.5% (area under curve: 0.824 , 95 % Cl, 0.740-0.908, p< 0.001; Figure 4). In the univariable regression analysis performed for all variables one by one to determine the predictors of the increased CAVI

Table 1: Clinical characteristics of the study population

	Aortic Valve Sclerosis n=35	Control Group n=130	p
Age (years)	66.14±12.03	50.69±11.22	<0.001a
Sex (F/M) (n) (%)	15 / 20	61 / 69	0.668b
BMI (kg/m2)	28.8±4.51	28.67±4.58	0.877a
Hypertension (n)	24 (%68)	48 (%37)	0.001b
Diabetes Mellitus (n)	5 (%14)	4 (%0.03)	0.010b
Hyperlipidemia	1 (%0.03)	11 (%0.08)	0.257b
Smoking (n)	5 (%0.14)	18 (%0.14)	0.947b
Hemoglobin (g/dL)	13.51±1.55	13.92±1.41	0.156a
WBC (×109/L)	6.34±1.57	6.66±1.59	0.316a
PLT (×109/L)	211.74±52.06	224.95±49.73	0.193a
MPV (fl)	8.56±0.95	8.67±0.80	0.489a
Creatinine (mg/dL)	0.8 (0.4-1.1)	0.75 (0.5-1.6)	0.384c
Urea (mg/dL)	13 (7-35)	15 (8-31)	0.070c
Glucose (mg/dL)	94 (80-125)	91 (76-424)	0.064c
LDL-C (mg/dL)	128.3±32.36	130.3±33.15	0.767a
HDL-C (mg/dL)	47.53±12.25	47.13±11.39	0.871a
Total Cholesterol (mg/dL)	199.31±50.52	206.84±39.59	0.395a
Triglyceride (mg/dL)	138.48±81.1	157.05±88.13	0.293a

aIndependent t test, bChi-square test, cMann–Whitney U test

value, age (p< 0.001), HT (p< 0.001), PLT (p:0.002), IVS (p< 0.001), PW (p< 0.001), LVMI (p< 0.001), S (p:0.001) and AVS (p< 0.001) were associated with increased CAVI. Multivariate analysis showed that age (OR: 1.101, 95%CI 1.040-1.165, p: 0.001) and AVS (OR: 4.045, 95%CI 1.149-14.242, p: 0.030) were independent predictors of increased CAVI (Table 4).

Table 2: Comparison of the Echocardiographic and CAVI parameters			
	Aortic Valve Sclerosis	Control Group	p
CAVI	9.47±1.64	7.60±1.27	<0.001[a]
LV-EF (%)	60 (55-65)	60 (60-70)	0.240[b]
LV-EDD (mm)	45 (34-50)	45 (40-55)	0.643[b]
LV-ESD (mm)	30 (22-35)	30 (24-38)	0.352[b]
LV-EDV index (mm/m^2)	24.57 (18.78-39.71)	24.94 (17.32-40.09)	0.721[b]
LV-ESV index (mm/m^2)	16.91 (12.52-27.12)	16.95 (11.32-27)	0.652[b]
LVMI gr/m^2	101.8 (64.62-177.72)	82.13 (51.04-177.56)	<0.001[b]
IVS (mm)	12 (9-15)	9 (8-16)	<0.001[b]
PW (mm)	12 (9-13)	9 (8-13)	<0.001[b]
LVOT velocity (cm/s)	104.0±22.85	96.62±17.88	0.045[a]
S (cm/s)	9 (6.1-15)	10 (6-12.5)	0.150[b]
[a]Independent t test, [b]Chi-square test, [c]Mann–Whitney U test			

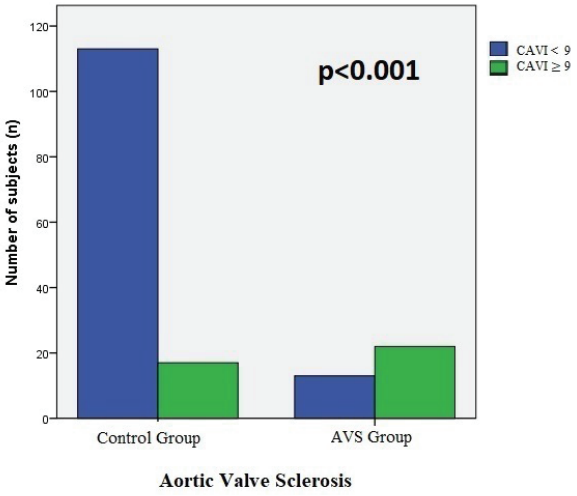

Figure 2: Frequency of aortic valve sclerosis in different cut off value of cardio-ankle vascular index

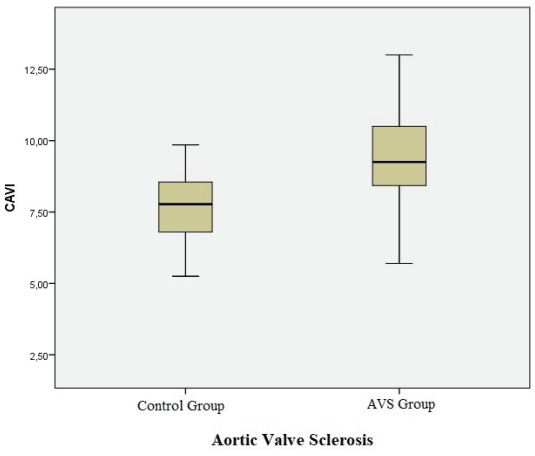

Figure 3: Cardio-ankle vascular index was increased in aortic valve sclerosis (AVS) patients, compared to the control subjects.

Table 3: Univariable and Multivariable analysis showing the association between parameters and Aortic Valve Sclerosis

	Univariable Analysis				Multivariable analysis			
	OR	95 %Cl		p	OR	95 %Cl		p
		Lower	Upper			Lower	Upper	
Age	1.113	1.070	1.157	<0.001	1.070	1.008	11.365	**0.026**
Gender	1.179	0.555	2.503	0.669				
BMI	1.007	0.928	1.092	0.876				
HT	3.727	1.679	8.275	0.001	0.806	0.252	2.583	0.717
DM	5.250	1.329	20.738	0.018	6.633	1.117	39.379	**0.037**
Smoker	1.037	0.356	3.022	0.947				
Creatinine	1.229	0.134	11.230	0.855				
LDL	0.998	0.986	1.011	0.765				
HG	0.820	0.623	1.079	0.157				
WBC	0.874	0.672	1.136	0.315				
PLT	0.995	0.987	1.003	0.193				
LV-EF	0.854	0.674	1.082	0.190				
LV-EDD	1.003	0.904	1.114	0.950				
LV-ESD	1.036	0.902	1.190	0.620				
IVS	1.794	1.393	2.310	<0.001	0.670	0.335	1.338	0.256
PW	2.386	1.716	3.319	<0.001	2.777	1.232	6.257	0.014
LVMI	1.031	1.016	1.046	<0.001	0.998	0.975	1.021	0.861
S	0.901	0.766	1.059	0.206				
CAVI	3.150	2.012	4.929	<0.001	2.048	1.183	3.547	**0.010**

BMI; Body mass index, CAVI; Cardio-ankle vascular index, DM; Diabetes Mellitus, HG; Hemoglobin, HT; Hypertension, IVS; İnterventricular septum, LDL; Low density lipoprotein cholesterol, LV-EDD; Left ventricular end-diastolic diameter, LV-EDV; Left ventricular end-diastolic volume, LV-EF; Left ventricular ejection fraction, LVMI; Left ventricular mass index, PLT; Platelets, PW; Posterior wall , S; Tricuspid lateral annular systolic velocity, WBC; White blood cell.

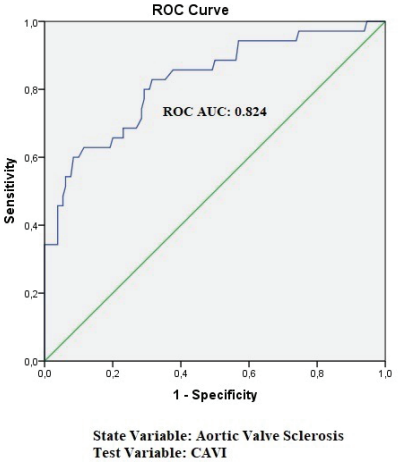

Figure 4: Receiver operating characteristic curve of CAVI to predict aortic valve sclerosis. The area under curve was : 0.824 , 95 % Cl, 0.740-0.908, p< 0.001). The optimal cutoff level of CAVI was 8.475 with 71.5% specificity and 71.4% sensitivity.

Table 3: Univariable and Multivariable analysis showing the association between parameters and Aortic Valve Sclerosis

	Univariable Analysis				Multivariable analysis			
	OR	95 %Cl		p	OR	95 %Cl		p
		Lower	Upper			Lower	Upper	
Age	1.140	1.091	1.191	<0.001	1.101	1.040	1.165	**0.001**
Gender	0.760	0.370	1.561	0.455				
BMI	1.005	0.929	1.087	0.909				
HT	5.598	2.496	12.553	<0.001	1.313	0.430	4.011	0.633
DM	1.667	0.397	7.000	0.485				
Smoker	0.882	0.305	2.555	0.818				
Creatinine	1.348	0.159	11.438	0.785				
LDL	0.993	0.981	1.006	0.288				
HG	0.845	0.650	1.098	0.207				
WBC	0.976	0.767	1.240	0.840				
PLT	0.986	0.978	0.995	0.002	0.988	0.976	1.000	0.058
LV-EF	1.061	0.890	1.265	0.506				
LV-EDD	1.044	0.943	1.154	0.407				
LV-ESD	1.034	0.905	1.182	0.620				
IVS	1.872	1.451	2.416	<0.001	1.307	0.667	2.560	0.435
PW	2.074	1.530	2.812	<0.001	0.764	0.325	1.799	0.538
LVMI	1.037	1.021	1.053	<0.001	1.003	0.978	1.029	0.809
S	0.733	0.608	0.883	0.001	0.769	0.581	1.020	0.068
AVS	11.249	4.787	26.436	<0.001	4.045	1.149	14.242	**0.030**

BMI; Body mass index, CAVI; Cardio-ankle vascular index, DM; Diabetes Mellitus, HG; Hemoglobin, HT; Hypertension, IVS; İnterventricular septum, LDL; Low density lipoprotein cholesterol, LV-EDD; Left ventricular end-diastolic diameter, LV-EDV; Left ventricular end-diastolic volume, LV-EF; Left ventricular ejection fraction, LVMI; Left ventricular mass index, PLT; Platelets, PW; Posterior wall , S; Tricuspid lateral annular systolic velocity, WBC; White blood cell.

DISCUSSION

AVS has been associated with CV diseases and mortality in many previous studies. However, after adjusting for factors that increase CV risks, such as age and gender, in some studies, the presence of AVS is associated with reduced CV risks[14]. However, recent evidence has clearly shown that AVS is associated with CAD, stroke, and CV mortality, according to the meta-analysis result published by Mateo et al. in 2018[2]. At the same time, AVS can be considered a potential prognostic factor in patients without clear evidence of coronary artery disease[15]. Since AVS is closely associated with CV risk factors such as age, male gender, hypertension, hyperlipidemia, diabetes mellitus, and smoking, it is thought that atherosclerosis plays a role in the pathophysiology of AVS[16]. Studies have revealed the role of other cells and factors, especially fibroblasts, in this atherosclerotic process[17].

Arterial stiffness was thought to occur due to atherosclerotic changes.It has been determined that the pathophysiology of arterial stiffness is an active process driven by vascular smooth muscle cell (VSMC) transdifferentiation[18]. Isolated systolic hypertension may develop as a result of the development of stiffness in the large arteries[19]. As the artery hardens, it increases left ventricular afterload and lowers coronary perfusion pressure, resulting in LV remodeling, LV dysfunction, valve dysfunction, and aortic root enlargement even in the absence of coronary artery disease[20]. Arterial stiffness was associated with CV mortality and morbidity regardless of organ damage that it

causes[21].

The frequency of AVS in our study population was found to be 21%. Our mean age is 53.96±13, and in a meta-analysis with a similar mean age (mean age: 54%), the prevalence of AVS was found to be 9%[14]. In the same meta-analysis, a study with a mean age of 57 years reported a 27% prevalence of AVS. In our population, a prevalence was found above the average in the literature, but in a range consistent with known studies. The link between AVS and arterial stiffness may be associated with the atherosclerotic process and inflammation[22,23]. In our study, we examined the relationship between arterial stiffness and AVS. We found a significant relationship between increased CAVI values and the presence of AVS. We also found that arterial stiffness was an independent predictor of AVS.According to the multivariable regression analysis, apart from arterial stiffness, age and diabetes mellitus were also predictors of AVS. As the patients admitted to the cardiology outpatient clinic were recruited sequentially due to the design of our study, our study about the prevalence of AVS in the population will provide a small idea. Also, the control group was taken as sequential patients, and a natural comparison environment was formed.

The relationship between AVS and age has been confirmed in all prevalence studies[3,4,14].

For the relationship between AVS and diabetes mellitus, there are studies in the literature showing that diabetes mellitus is an independent predictor of AVS, which strongly supports our data[24,25].

Since arterial stiffness is not a procedure that cardiologists can easily look at in a busy hospital working environment, regression analysis was performed in our study to determine independent predictors of increased CAVI values.In the created model, we found that AVS, which we can easily detect visually during routine echocardiography, is an independent predictor of increased CAVI values. According to multivariable regression analysis, age was found to be an independent predictor for increased CAVI values, apart from AVS.

Celik et al. examined the relationship between arterial stiffness and AVS previouslystudy[26]. In their study, arterial stiffness was calculated by measuring the carotid-femoral pulse wave velocity (PWV) which showed no relationship between arterial stiffness and AVS. In systematic reviews, PWV measurement is found to be affected by age and blood pressure factors[27]. The fact that the blood pressure effect was not evaluated in their study is the main limitation ofthe study.

In the study of Korkmaz et al., the relationship between the CAVI method and arterial stiffness and AVS was investigated[28]. In their study, a relationship was found between arterial stiffness and AVS. The patient population was randomly selected during the study design, and similarity was expressed between the two groups. In our study, the patients were included in the study sequentially, and the differences in demographic characteristics between the two groups were revealed.

During routine echocardiography, cardiologists should perform a visual inspection for AVS. The relationship between AVS and arterial stiffness should be considered depending on the common pathophysiological processes and common causes. Patients with AVS or arterial stiffness should be approached carefully regarding atherosclerotic diseases and should be called for control intermittently. Also, it should be noted that arterial stiffness can directly cause coronary artery disease with the rupture of atherosclerotic plaques, and AVS can cause aortic stenosis over the years[29,5].

Study Limitations

The most important limitation of our study is the relatively small study population after exclusion criteria.In addition, as in previous studies, some borderline CAVI values

are statistically in the normal category since values of 9, and above are taken as abnormal CAVI values. Finally, our study is a cross-sectional study and will be limited in demonstrating the pathophysiological mechanisms.

CONCLUSION

In this study, we showed the relationship between arterial stiffness measured using the CAVI technique and AVS. Both parameters may be markers of the atherosclerotic process in individuals without known CV disease.

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Conflicts of interest

None declared

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