Assessment of myocardial performance index and its association with aortic elasticity in patients with ascending aortic aneurysm

Çıkan aort anevrizması olan hastalarda miyokart performans indeksinin değerlendirilmesi ve aort esnekliği ile ilişkisi

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ABSTRACT

Objective: Ascending aortic aneurysms (AAA) are a leading cause of morbidity and mortality. Nevertheless, their effects on global cardiac functions are yet to be fully understood. Myocardial performance index (MPI) has been widely used to quantitatively assess myocardial functions. The aim of this study was to evaluate left ventricular (LV) functions in patients with AAA using tissue Doppler (TD) echocardiography and MPI in addition to conventional echocardiographic methods.

Methods: Fifty patients with AAA (33 men, 17 woman; mean age 55.5±7.90 years) were included, and 106 patients without aortic dilatation (mean age 54.1±8.18 years) were included as the control group. LV systolic and diastolic functions were analyzed using 2-dimensional, M-mode, and TD echocardiography.

Results: Patients with AAA had significantly higher MPI (0.5±0.04 *vs* 0.4±0.05, p<0.001), TD-MPI (0.5±0.02 *vs* 0.4±0.03, p<0.001), and reduced aortic elasticity, as indicated by reduced aortic distensibility (AD) (1.7±1.27 *vs* 3.1±1.25, p<0.001). Multivariate linear regression analysis showed that TD-MPI was independently correlated with reduced aortic distensibility (B=-0.006, p=0.019, 95% confidence interval,-0.011 to -0.001).

Conclusion: MPI and TD-MPI indicated impairment of global cardiac functions in patients with AAA, which may be attributed to reduced aortic elasticity.

ÖZET

Amaç: Çıkan aort anevrizması (ÇAA) mortalite ve morbiditenin önemli nedenleri arasında yer almaktadır. Bununla birlikte, kalbin genel fonksiyonları üzerine etkisi tamamen bilinmemektedir. Miyokart performans indeksi (MPİ) miyokart fonksiyonlarının değerlendirilmesinde sıklıkla kullanılan bir yöntemdir. Bu çalışmanın temel amacı, ÇAA olan hastalarda, geleneksel ekokardiyografik yöntemlere ek olarak, doku Doppler (dD) ekokardiyografi ve MPİ ile sol ventrikül fonksiyonlarının değerlendirilmesidir.

Yöntemler: Çıkan aort anevrizması tanısı olan 50 hasta (33 erkek, 17 kadın; ortalama yaş 55.5±7.90) ve kontrol grubu olarak aort genişlemesi olmayan 106 hasta (ortalama yaş 54.1±8.18) çalışmaya dahil edildi. Sol ventrikül sistolik ve diyastolik fonksiyonları 2-boyutlu, M-mod ve doku Doppler ekokardiyografi yöntemleri kullanılarak değerlendirildi.

Bulgular: Çıkan aort anevrizması olan hasta grubunda, MPİ (0.5±0.04 ve 0.4±0.05, p<0.001) ve dD-MPİ (0.5±0.02 ve 0.4±0.03, p<0.001) belirgin olarak daha yüksek iken, aort gerilebilme yetisi (1.7±1.27 ve 3.1±1.25, p<0.001) şeklinde tanımlanan aort esnekliğinin azalmış olduğu gözlendi. Çok değişkenli lojistik regresyon analizi sonucuna göre, dD-MPİ'nin azalmış aort gerilebilme yetisi ile bağımsız olarak ilişkili olduğu saptandı (B=-0.006, p=0.019, %95 GA: -0.011 ile -0.001).

Sonuç: Çıkan aort anevrizması olan hastalarda genel kalp fonksiyonlarının belirteçleri olan MPİ ve dD-MPi azalmaktadır. Kalp fonksiyonlarının bozulmasından azalmış aort esnekliği sorumlu olabilir.

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Aortic aneurysms (AA) are a leading cause of morbidity and mortality in individuals >55 years of age.[1] Most are diagnosed in an imaging study performed for an unrelated indication. Although it is a relatively rare pathology when compared with other cardiovascular disorders, the extensive complications of untreated or undiagnosed aneurysms elevate its clinical importance.[2] The aorta has an elastic structure and is generally affected by aging, atherosclerosis, hypertension (HT), and diabetes mellitus (DM).[3,4] Changes in the elastic structure caused by these risk factors are reflected by a decrease in aortic distensibility (AD), reduced aortic elasticity, and increased aortic stiffness.[5] Reduced aortic elasticity causes an increase in aortic pulse pressure (PP), left ventricular (LV) afterload, and LV hypertrophy, which may lead to impairment in cardiac functions.[6] However, to the best of our knowledge, little information is available regarding changes in cardiac functions in patients with AA.

Myocardial performance index (MPI) has been widely used to quantitatively assess myocardial functions.[7] It is likely to be more effective for analysis of global cardiac function than systolic and diastolic measures alone.[8,9] MPI obtained by tissue Doppler (TD) echocardiography may also give a better reflection of global LV function than an isolated evaluation of either ejection or relaxation.[10] The value of TD-MPI to assess LV function in patients with ascending aortic aneurysms (AAA) has not yet been investigated. The aim of this study was to investigate LV function and MPI by TD echocardiography, and to evaluate the relationship of AD with MPI in the assessment of LV function in patients with AAA.

METHODS

Study population was prospectively recruited from consecutive outpatients with AAA diagnosis who underwent coronary angiography with various indications. Of the 268 patients with AAA enrolled, 218 were excluded due to abnormal coronary angiography; only patients with normal angiography were included. The final group consisted of 50 patients with AAA (end-diastolic proximal ascending aorta diameter >40 mm) in the absence of concomitant coronary artery disease (33 men, 17 women; mean age 55.5±7.90 years). A total of 106 patients without aortic dilatation and with normal coronary angiography (mean age 54.1±8.18 years) were selected as the control group. Patients with moderate and severe valvular heart disease, history of myocardial infarction, wall motion abnormalities, heart failure, severe pulmonary disease, major non-cardiovascular diseases, cerebrovascular disease, and renal insufficiency (serum creatinine >1.5 mg/dL in men and >1.4 mg/dL in women) were excluded. Patients with secondary etiologic disorders, such as genetically triggered aneurysms, inflammatory diseases, or trauma-associated aneurysms were excluded, as were those with bicuspid aortic valves and coarctation of the aorta. Only patients with isolated idiopathic or degenerative AAA were included.

Abbreviations:

AA	*Aortic aneurysms*
AAA	*Ascending aortic aneurysms*
AD	*Aortic distensibility*
BMI	*Body mass index*
CFVR	*Coronary flow velocity reserve*
DBP	*Diastolic blood pressure*
DM	*Diabetes mellitus*
ET	*Ejection time*
HT	*Hypertension*
ICT	*Isovolumic contraction time*
IVS	*Interventricular septum*
IVRT	*Isovolumic relaxation time*
LV	*Left ventricular*
LVEF	*Left ventricular ejection fraction*
MPI	*Myocardial performance index*
PP	*Pulse pressure*
PW	*Posterior wall*
SBP	*Systolic blood pressure*
TD	*Tissue Doppler*
TDE	*Tissue Doppler echocardiography*

After detailed medical histories were obtained, each participant underwent complete physical examination and was questioned for major cardiovascular risk factors such as age, sex, DM, smoking status, and HT. In addition, systolic blood pressure (SBP), diastolic blood pressure (DBP), and initial heart rate were recorded. All patients underwent electrocardiography, and comprehensive transthoracic Doppler and TD echocardiography. The study was conducted according to recommendations set forth by the Declaration of Helsinki on medical research involving human subjects. The institutional ethics committee approved the study protocol, and each participant provided written informed consent.

Echocardiography

Standard 2-dimensional, M-mode, and TD echocardiographic examinations were performed using commercially available equipment (Vivid-7; GE Vingmed Ultrasound, Horten, Norway) with a 2.5–3.5 MHz transducer. Simultaneous electrocardiographic recordings were also obtained. An echocardiographer blinded to clinical and laboratory data interpreted each echocardiographic examination independently. LV ejection fraction (LVEF) was determined using Simp-

son's rule, according to the suggestions of the American Society of Echocardiography.[11] LV end-diastolic diameter, LV end-systolic diameter, end-diastolic left atrial diameter, end-diastolic interventricular septum (IVS), and left posterior wall (PW) thickness were measured using M-mode echocardiography. Sample volume of pulsed-wave Doppler was placed between the tips of the mitral leaflets in the apical 4-chamber view. Early (E) and late (A) transmitral flow velocities, as well as the ratio of early to late peak velocities (E/A) were obtained. Mitral pulsed-wave Doppler time intervals were measured from mitral inflow and left ventricular outflow Doppler tracings, as described by Tei and colleagues.[7] Three consecutive beats were measured and averaged for each parameter. Isovolumic relaxation time (IVRT) was measured from the closure of the aortic valve to the opening of the mitral valve. Isovolumic contraction time (ICT) was measured from the closure of the mitral valve to the opening of the aortic valve. Ejection time (ET) was measured from the opening to the closure of the aortic valve on the LV outflow velocity profile (MPI=[IVRT+ICT]÷ET).

After proper acquisition adjustments were made, left ventricle tissue Doppler echocardiography (TDE) evaluation was performed in apical 4-chamber position by placing the pulsed-wave Doppler beam on section of the mitral annulus close to the left ventricle lateral wall and the IVS. Special attention was paid during recordings to place the Doppler beam in the myocardium, not the endocardium or epicardium. Measurements were made for 3 consecutive heartbeats in all positions, and their average was taken. Doppler measurements were made at a recording rate of 100 mm/sec. Average of the septal and lateral annular mitral early diastolic (e'), late diastolic (a'), and systolic (Sm) spectral TD velocities, as well as the E/e' and e'/a' ratios, were measured. MPI obtained by TDE was defined as (IVRT+ICT)÷ET (Figure 1). All echocardiograms were recorded and interpreted online on hard disks for offline analysis by another observer blinded to patient data. Intra- and interobserver variability of echocardiographic data were evaluated from 15 randomly selected patients with AAA and calculated as the absolute difference divided by the average of the 2 observations. Mean intra- and interobserver variability were 3.3±2.14% and 3.9±2.71%, respectively.

Calculation of aortic root parameters

Aortic root systolic diameter (AoSD) and aortic root diastolic diameter (AoDD) were measured by M-mode at a level of 3–4 cm above the aortic valve from a transthoracic parasternal long-axis view at the time of maximum aortic anterior motion and at the peak of the QRS complex, respectively. Two-dimensional measurements of the aortic root end-diastolic dimensions were made in the parasternal long-axis views at 4 levels: the annulus (defined echocardiographically as the hinge points of the aortic cusps), the sinuses of Valsalva, the supra-aortic ridge, and the proximal ascending aorta. The measurements were made perpendicular to the long axis of the aorta using the leading edge technique in views showing the largest aortic diameters.[12] Diagnosis of AA was determined solely from morphologic analysis of the aorta, and dilation was suspected in the case of an aortic root dimension at the sinuses of Valsalva greater than the upper limit of the 95% confidence interval of the overall distribution.

Blood pressure was measured by arm sphygmomanometer. The indices of aortic root mechanics were calculated as AD=2×(pulsatile change in aortic diameter)/([diastolic aortic diameter]×[PP]).[13] PP was obtained simultaneously by cuff sphygmomanometry of the left brachial artery as (SBP-DBP).

Statistical analysis

Analyses were performed using SPSS software (version 20.0; SPSS Inc., Chicago, IL, USA). Continu-

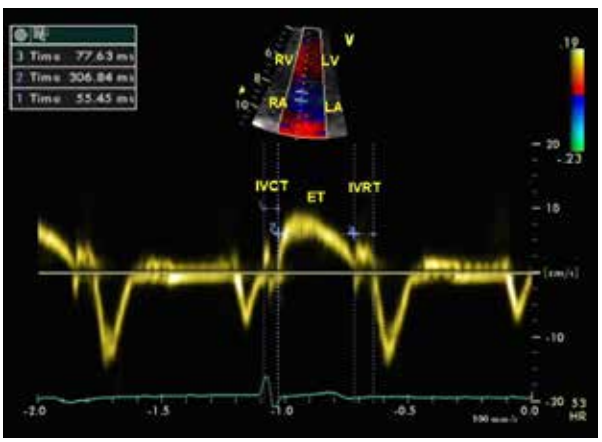

Figure 1. Calculation of tissue Doppler myocardial performance index (TD-MPI). TD-MPI is defined as the sum of isovolumic relaxation time (IVRT) and isovolumic contraction time (ICT) divided by ejection time (ET) in TD echocardiography.

ous variables were expressed as mean±SD or median (min-max) where applicable. Number of cases and percentages were used for categorical data. Continuous variables were tested for normality using the Kolmogorov-Smirnov test. While mean differences between groups were compared using Student's t-test, Mann-Whitney U test was applied for comparisons of medians. Categorical data were analyzed by Pearson's chi-square test. Correlations between aortic distensibility, TD-MPI, laboratory data, hemodynamics, and other echocardiographic parameters were assessed by Spearman correlation analysis. All parameters with $p<0.10$ in univariate analysis were selected in the multivariate model. To avoid overfitting and collinearity in assessing the multivariate model, independent variables were tested for intercorrelation. Collinearity between variables was excluded before modeling. AD, IVS, creatinine, and E/A ratio were selected in multivariate linear regression model, which was used to determine independent predictors

Table 1. Baseline clinical and laboratory characteristics

Variables	Patient Group (n=50)				Control Group (n=106)				*p*
	n	%	Mean±SD	Median (Min–Max)	n	%	Mean±SD	Median (Min–Max)	
Baseline characteristics									
Age (years)			55.5±7.90					54.1±8.18	0.301
Body Mass Index (kg/m^2)			24.1±3.94					24.2±2.63	0.805
Male	33	66.0			66	62.3			0.651
Diabetes	6	12.0			14	13.2			0.833
Hypertension	26	52.0			52	49.1			0.732
Hyperlipidemia	14	28.0			29	27.4			0.933
Family history	11	22.0			27	25.5			0.637
Smoking	23	46.0			48	45.3			0.933
Laboratory findings									
Glucose (mg/dl)				95 (65–202)				96 (72–177)	0.587
Total cholesterol (mg/dl)			205.1±33.9				208.6±42.8		0.688
Triglyceride (mg/dl)				177 (67–577)				128.5 (60–395)	0.015
HDL (mg/dl)			39.5±9.8				47.2±11.3		0.003
LDL (mg/dl)			135.6±31.3				142.3±46.7		0.460
Creatinine (mg/dl)				0.9 (0.6–1.3)				0.7 (0.4–1.2)	<0.001
Hemoglobin (mg/dl)			13.9±1.30				14.0±1.31		0.663
Previous medications									
ACE-I/ARB	17	34.0			33	31.1			0.720
Calcium channel blocker	12	24.0			23	21.7			0.748
Oral anti-diabetic	6	12.0			15	14.2			0.713
β Blocker	12	24.0			24	22.6			0.851
Statin	16	32.0			29	27.4			0.550

ACE-I: Angiotensin-converting enzyme inhibitor; ARB: Angiotensin-receptor blocker; HDL: High-density lipoprotein; LDL: Low-density lipoprotein.
Data are n (%) for categorical variables, mean±SD for continuous variables, or median (min-max) for non-normally distributed variables.

of TD-MPI. A two-tailed p<0.05 was considered statistically significant.

RESULTS

Baseline clinical and laboratory characteristics of patients with AAA, as well as those of the control group, are presented in Table 1. The laboratory findings of the 2 groups were similar (p>0.05 for all), except for high-density lipoprotein, triglyceride, and creatinine levels.

Echocardiographic data

Echocardiographic data are presented in Table 2. Patients with AAA, in comparison with controls, had significantly thicker IVS and PW. Additionally, LV end-diastolic diameter and left atrial diameter were larger in the AAA group. Compared with controls, patients with AAA had higher MPI and TD-MPI (0.5±0.04, 0.4±0.05, respectively, p<0.001; 0.5±0.02, 0.4±0.03, respectively, p<0.001). Markers of diastolic function, obtained using both 2D echocardiography and TDE, were significantly impaired in patients with AAA, whereas LVEF remained unchanged (61.9±3.09 *vs* 62.7±2.58, p=0.090; Table 2).

Analysis of pulse pressure and aortic distensibility

Aortic distensibility was significantly lower in patients with AAA than in control subjects (1.7±1.27 *vs* 3.1±1.25, p<0.001; Table 3). Compared with the controls, PP (56.7±10.83 *vs* 49.9±10.50, p<0.001) and SBP (134.6±16.91 *vs* 126.7±13.15, p=0.002) were significantly higher in the patients.

Correlations and multivariate associations of TD-MPI

Correlations of TD-MPI in patients with AAA are presented in Table 4. In patients with AAA, the adjusted TD-MPI had significant inverse relations with aortic distensibility (r=-0.501, p<0.001) and positive corre-

Table 2. Echocardiographic findings

Variables	Patient Group (n=50)	Control Group (n=106)	*p*
	Mean±SD	Mean±SD	
Left atrial diameter (mm)	38.5±3.38	35.6±2.61	<0.001
Left ventricular end-diastolic diameter (mm)	49.9±4.18	48.1±3.82	0.009
Left ventricular end-systolic diameter (mm)	32.6±3.59	30.4±3.23	<0.001
Interventricular septum (mm)	11.9±1.69	9.9±1.08	<0.001
Posterior wall (mm)	11.6±1.18	9.9±1.11	<0.001
Ejection fraction (%)	61.9±3.09	62.7±2.58	0.090
Aortic root diastolic diameter (mm)	44.0±5.49	28.9±3.27	<0.001
Aortic root systolic diameter (mm)	46.0±5.17	31.0±3.18	<0.001
2-dimensional-Isovolumic relaxation time	86.7±10.76	76.5±11.27	<0.001
2-dimensional-Isovolumetric contraction time	65.0±9.84	61.8±13.02	0.135
2-dimensional-Ejection time	316.5±30.27	315.5±26.78	0.827
2-dimensional-Myocardial performance index	0.5±0.04	0.4±0.05	<0.001
E/A ratio	0.9±0.18	1.2±0.44	<0.001
Tissue Doppler-Isovolumic relaxation time	86.6±10.28	71.8±11.21	<0.001
Tissue Doppler-Isovolumetric contraction time	67.9±7.13	64.4±8.56	0.012
Tissue Doppler-Ejection time	306.8±23.22	317.3±20.47	0.005
Tissue Doppler-Myocardial performance index	0.5±0.02	0.4±0.03	<0.001
e'/a' ratio	0.7±0.18	0.9±0.34	<0.001
E/e' ratio	13.1±4.63	9.1±2.28	<0.001
Sm, cm/sec	8.6±1.95	8.4±1.65	0.366

Table 3. Aortic distensibility and hemodynamic parameters

Variables	Patient Group (n=50)	Control Group (n=106)	*p*
	Mean±SD	Mean±SD	
Systolic blood pressure (mmHg)	134.6±16.91	126.7±13.15	0.002
Diastolic blood pressure (mmHg)	77.8±12.10	76.8±11.76	0.611
Heart Rate (b/m)	72.9±11.33	73.7±10.91	0.679
Pulse Pressure (mmHg)	56.7±10.83	49.9±10.50	<0.001
Aortic distensibility ($cm^2/dyn/10^6$)	1.7±1.27	3.1±1.25	<0.001

Table 4. Correlations and linear regression analysis of TD-MPI in patients with ascending aortic aneurysm

Variables	Spearman Correlation Coefficient	*p*	Regression Coefficient	*p*	95% CI for B	
					Upper	Lower
Aortic distensibility	-0.501	<0.001	-0.006	0.019	-0.011	-0.001
Creatinine	0.260	0.069	-0.003	0.882	-0.038	0.033
E/A ratio	-0.250	0.080	-0.016	0.294	-0.046	0.014
Interventricular septum	0.324	0.022	0.013	0.432	-0.021	0.048
Triglyceride	-0.029	0.839				
High-density lipoprotein	-0.041	0.779				
Left atrial diameter	0.138	0.340				
Left ventricular end-diastolic diameter	-0.057	0.692				
Left ventricular end-systolic diameter	-0.083	0.568				
Posterior wall	0.182	0.205				
Aortic root systolic diameter	0.173	0.230				
2D-Isovolumic relaxation time	0.018	0.903				
2D-Myocardial performance index	0.124	0.390				
Tissue Doppler-Ejection time	-0.065	0.654				
e'/a' ratio	-0.185	0.198				
E/e' ratio	0.024	0.869				
Systolic blood pressure	0.024	0.866				
Pulse pressure	0.099	0.495				

MPI: Myocardial performance index; TD: Tissue Doppler; 2D: 2-dimensional.

lations with AoDD (r=0.288, p=0.043), as well as IVS (r=0.324, p=0.022). A negative correlation between TD-MPI and AD in patients with AAA is shown in Figure 2.

There were increased LV thickness, SBP, and PP in the patient group. When adjusted for these variables, both MPI and TD-MPI were independent of SBP, PP, and LV thickness. All parameters with p<0.10 in univariate analysis were selected in the multivariate model. Aortic distensibility, IVS, creatinine, and E/A ratio were selected in the multivariate linear regression model. Multivariate linear regression analysis showed that TD-MPI was only independently associated with aortic distensibility (B=-0.006; p=0.019; 95% confidence interval, -0.011 to -0.001; Table 4).

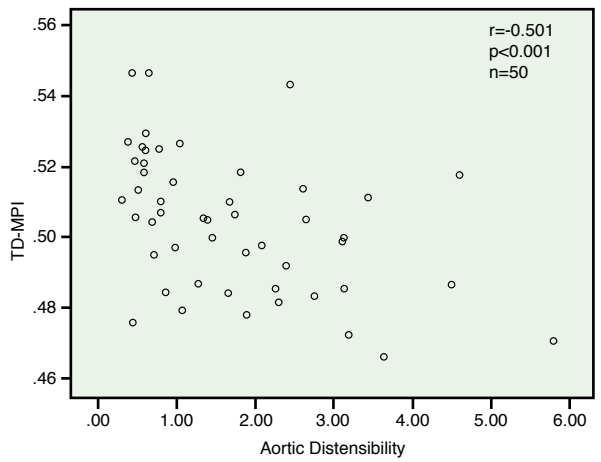

Figure 2. Negative correlation between tissue Doppler myocardial performance index (TD-MPI) and aortic distensibility (AD) in patients with ascending aortic aneurysms (AAA).

DISCUSSION

To the best of our knowledge, this was the first study to investigate LV function and MPI using TDE, and evaluate the relationship between AD and LV function in patients with AAA. Our study demonstrated that MPI- and TD-MPI-indicated global cardiac functions were impaired in patients with AAA. A significant relationship between TD-MPI and AD was also found. In the present study, both systolic and diastolic functions were assessed using TDE in addition to conventional echocardiographic methods. MPI, which was calculated using data obtained from PW Doppler and TD echocardiography, was also used to assess LV functions. Results of our study showed that both MPI and TD-MPI were significantly higher in patients with AAA. An important limitation was the interval between the end and the onset of mitral inflow and ET being measured sequentially, not on the same cardiac cycle, by conventional pulsed-wave Doppler echocardiography. However, TDE as used in the present study could simultaneously record systolic and diastolic mitral annular velocities. Determination of MPI obtained by TDE requires measurement of only 2 simple intervals on the same cardiac cycle.

Results of the present study support the view that diastolic and systolic dysfunction may be more common in patients with AAA by TDE, compared with the control group. The mechanism of this impaired LV dysfunction remains unknown. One interesting finding of our study is the negative and independent correlation between TD-MPI and aortic distensibility. The aorta has an elastic structure and is generally affected by aging, atherosclerosis, HT, and DM.[3,14] Changes in the elastic structure caused by these risk factors are reflected by a decrease in AD, reduced aortic elasticity, and increased aortic stiffness.[15] Aortic stiffening leads to an increase in central SBP, and a decrease in DBP with an increase in PP. An altered SBP may increase LV afterload with an increase in myocardial oxygen demand, LV hypertrophy, fibrosis, and eventually an impairment in cardiac functions.[6] It has been reported in several studies that LV diastolic function was severely reduced in patients with chronic aortic dissection, and aortic stiffness was augmented in patients with aortic aneurysm, which could be a result of elevated LV afterload.[5] It has also been shown that elevated afterload was one of the major causes of LV diastolic dysfunction.[16] In our population, diastolic dysfunction, LV hypertrophy, and decrease in AD were observed in patients with AAA in accordance with these previous reports, whereas systolic functions such as LV ejection fraction and Sm remained unchanged. Therefore, this impairment of global cardiac function in patients with AAA is predominantly the consequence of diastolic dysfunction, which is primarily related to LV hypertrophy and reduced aortic elasticity.

Another explanation for impaired global cardiac function indicated by TD-MPI in patients with AAA may include microvascular dysfunction. Myocardial perfusion depends on the diastolic pressure gradient from epicardium to endocardium and the duration of diastole. A decrease in DBP can compromise myocardial perfusion, resulting in subendocardial ischemia.[17] Other mechanisms of microvascular dysfunction in AAA may involve vascular remodeling, reduced capillary density, and elevated LV end-diastolic pressure, which causes perfusion abnormalities, predominantly in subendocardial layers of myocardium because of increased afterload.[18] Furthermore, arterial distensibility and stiffness are abnormal in aneurysmal arteries, and aortic stiffening is an important complication in patients with AA.[19] Previous studies have shown that coronary flow velocity reserve (CFVR) is impaired in patients with aortic stiffness.[20] In a recent study, we demonstrated that that noninvasive CFVR was significantly impaired in patients with AAA, compared with controls, and that AoSD is the strongest predic-

tor of impaired CFVR.[21] Therefore, reduced CFVR, which reflects microvascular dysfunction, may lead to myocardial ischemia and impaired cardiac functions.

It has been reported that aortic root diameter, especially at the level of the sinuses of Valsalva, may be related to body surface area and body mass index (BMI).[11] Aortic root diameters were not indexed according to BMI, but there were no statistical significant differences between patients and controls. Moreover, after multivariate regression analysis, the relation between TD-MPI and AD was observed to persist, even after adjustment for BMI in patients with AAA.

Compared with the healthy population, the value of MPI and TD-MPI were slightly higher in the control group, while AD was slightly lower. A direct comparison with the present study is difficult because the control group consisted of patients with DM, HT, and hyperlipidemia, rather than healthy subjects, to provide clinical match with AAA patients. Several previous studies demonstrated that in patients with DM, HT, and hyperlipidemia, MPI and AD might be impaired, even with a normal coronary angiography.[22,23] Moreover, Sahin et al. demonstrated that MPI and AD were strongly associated with LV geometry in patients with HT.[24] Hence, our results are consistent with previous reports.

Clinical implications

AAA may lead to global cardiac dysfunction as indicated by TD-MPI before LVEF impairment. According to results of the present study, this cardiac dysfunction is predominantly the consequence of decreased aortic distensibility and reduced aortic elasticity. Aortic distensibility and stiffness have been demonstrated to be reduced by several medications, such as certain antihypertensive agents, statins, and thiazolidinediones, as well as by lifestyle modification.[25] Therefore, long-term medical treatment and lifestyle modification in patients with AAA may prevent cardiac dysfunction and the enlargement of the aorta by reducing aortic distensibility.

Study limitations

Several limitations of the present study should be mentioned. Conventional coronary angiography was used to exclude coronary artery disease instead of more accurate methods, such as intravascular ultrasonography or optic coherence tomography, which have been shown to provide valuable information on cross-sectional coronary vascular structure with high spatial resolution.[26] Therefore, in this group of patients, vulnerable coronary plaques cannot be ruled out completely. Secondly, although the study population had medical conditions that may affect myocardial performance such as DM, HT, and hyperlipidemia, or were taking medications, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, β-receptor blockers, and statins, there were no differences in the proportion of patients with these medical conditions or on these medications between groups. Thirdly, TD-MPI was used to determine global cardiac functions. Although TD-MPI was validated in several previous reports, there are more accurate and reliable methods, such as Speckle tracking or 3-dimensional echocardiography. Therefore, future studies using Speckle tracking and 3-dimensional echocardiography would be more informative in assessing the relationship between aortic aneurysms, cardiac functions, and aortic stiffness. Lastly, variables such as MPI and AD may be influenced by aortic regurgitation. As patients with moderate and severe valvular heart disease were excluded, we think it is unlikely that mild aortic disease affected the results of the present study.

Conclusion

The present study demonstrated that MPI- and TD-MPI-indicated global cardiac functions are significantly impaired in patients with AAA. This impairment of cardiac function is predominantly the consequence of decreased aortic distensibility and reduced aortic elasticity. As a consequence, our data indicate that parameters describing LV workload, LV hypertrophy, diastolic dysfunction, and AD are related to key mechanisms responsible for impaired TD-MPI in patients with AAA.

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REFERENCES

1. Paterick TE, Humphries JA, Ammar KA, Jan MF, Loberg R, Bush M, et al. Aortopathies: etiologies, genetics, dif-

ferential diagnosis, prognosis and management. Am J Med 2013;126:670–8. CrossRef
2. Sanon S, Lee VV, Elayda MA, Gondi S, Livesay JJ, Reul GJ, et al. Predicting early death after cardiovascular surgery by using the Texas Heart Institute Risk Scoring Technique (THIRST). Tex Heart Inst J 2013;40:156–62.
3. Mitchell GF, Parise H, Benjamin EJ, Larson MG, Keyes MJ, Vita JA, et al. Changes in arterial stiffness and wave reflection with advancing age in healthy men and women: the Framingham Heart Study. Hypertension 2004;43:1239–45. CrossRef
4. Tok D, Özcan F, Kadife İ, Turak O, Çağlı K, Başar N, et al. Impaired coronary flow reserve evaluated by echocardiography is associated with increased aortic stiffness in patients with metabolic syndrome: an observational study. Anadolu Kardiyol Derg 2013;13:227–34. CrossRef
5. Shingu Y, Shiiya N, Ooka T, Tachibana T, Kubota S, Morita S, et al. Augmentation index is elevated in aortic aneurysm and dissection. Ann Thorac Surg 2009;87:1373–7. CrossRef
6. Nemes A, Geleijnse ML, Forster T, Soliman OI, Ten Cate FJ, Csanády M. Echocardiographic evaluation and clinical implications of aortic stiffness and coronary flow reserve and their relation. Clin Cardiol 2008;31:304–9. CrossRef
7. Tei C, Ling LH, Hodge DO, Bailey KR, Oh JK, Rodeheffer RJ, et al. New index of combined systolic and diastolic myocardial performance: a simple and reproducible measure of cardiac function-a study in normals and dilated cardiomyopathy. J Cardiol 1995;26:357–66.
8. Tei C, Dujardin KS, Hodge DO, Bailey KR, McGoon MD, Tajik AJ, et al. Doppler echocardiographic index for assessment of global right ventricular function. J Am Soc Echocardiogr 1996;9:838–47. CrossRef
9. Koç F, Tokaç M, Kaya C, Kayrak M, Yazıcı M, Karabağ T, et al. Diastolic functions and myocardial performance index in obese patients with or without metabolic syndrome: a tissue Doppler study. Turk Kardiyol Dern Ars 2010;38:400–4.
10. Uluçay A, Tatli E. Myocardial performance index. [Article in Turkish] Anadolu Kardiyol Derg 2008;8:143–8.
11. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification. Eur J Echocardiogr 2006;7:79–108. CrossRef
12. Roman MJ, Devereux RB, Kramer-Fox R, O'Loughlin J. Two-dimensional echocardiographic aortic root dimensions in normal children and adults. Am J Cardiol 1989;64:507–12.
13. Stefanadis C, Stratos C, Boudoulas H, Vlachopoulos C, Kallikazaros I, Toutouzas P. Distensibility of the ascending aorta in coronary artery disease and changes after nifedipine administration. Chest 1994;105:1017–23. CrossRef
14. Mackey RH, Sutton-Tyrrell K, Vaitkevicius PV, Sakkinen PA, Lyles MF, Spurgeon HA, et al. Correlates of aortic stiffness in elderly individuals: a subgroup of the Cardiovascular Health Study. Am J Hypertens 2002;15(1 Pt 1):16–23. CrossRef
15. Grotenhuis HB, Ottenkamp J, Westenberg JJ, Bax JJ, Kroft LJ, de Roos A. Reduced aortic elasticity and dilatation are associated with aortic regurgitation and left ventricular hypertrophy in nonstenotic bicuspid aortic valve patients. J Am Coll Cardiol 2007;49:1660–5. CrossRef
16. Mottram PM, Haluska BA, Leano R, Carlier S, Case C, Marwick TH. Relation of arterial stiffness to diastolic dysfunction in hypertensive heart disease. Heart 2005;91:1551–6. CrossRef
17. Tesic M, Djordjevic-Dikic A, Beleslin B, Trifunovic D, Giga V, Marinkovic J, Petrovic O, et al. Regional difference of microcirculation in patients with asymmetric hypertrophic cardiomyopathy: transthoracic Doppler coronary flow velocity reserve analysis. J Am Soc Echocardiogr 2013;26:775–82.
18. Moltzer E, Essers J, van Esch JH, Roos-Hesselink JW, Danser AH. The role of the renin-angiotensin system in thoracic aortic aneurysms: clinical implications. Pharmacol Ther 2011;131:50–60. CrossRef
19. Nollen GJ, Groenink M, Tijssen JG, Van Der Wall EE, Mulder BJ. Aortic stiffness and diameter predict progressive aortic dilatation in patients with Marfan syndrome. Eur Heart J 2004;25:1146–52. CrossRef
20. Nemes A, Forster T, Csanády M. Reduction of coronary flow reserve in patients with increased aortic stiffness. Can J Physiol Pharmacol 2007;85:818–22. CrossRef
21. Baykan AO, Yüksel Kalkan G, Gür M, Uçar H, Acele A, Şeker T, et al. Coronary flow velocity reserve in patients with ascending aorta aneurysm. Echocardiography 2015;32:975–82. CrossRef
22. Mishra RK, Kizer JR, Palmieri V, Roman MJ, Galloway JM, Fabsitz RR, et al. Utility of the myocardial performance index in a population with high prevalences of obesity, diabetes, and hypertension: the strong heart study. Echocardiography 2007;24:340–7. CrossRef
23. Kalkan GY, Gür M, Şahin DY, Baykan AO, Elbasan Z, Kuloğlu O, et al. Coronary flow reserve and myocardial performance index in newly diagnosed diabetic patients. Echocardiography 2013;30:1164–71. CrossRef
24. Sahin DY, Gür M, Elbasan Z, Kalkan GY, Ozdoğru I, Kivrak A, et al. Myocardial performance index and aortic distensibility in patients with different left ventricle geometry in newly diagnosed essential hypertension. Blood Press 2013;22:329–35. CrossRef
25. Zieman SJ, Melenovsky V, Kass DA. Mechanisms, pathophysiology, and therapy of arterial stiffness. Arterioscler Thromb Vasc Biol 2005;25:932–43. CrossRef
26. Nicholls SJ, Tuzcu EM, Sipahi I, Schoenhagen P, Nissen SE. Intravascular ultrasound in cardiovascular medicine. Circulation 2006;114:55–9. CrossRef

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