Arterial Stiffness Is Significantly Associated With Left Ventricular Diastolic Dysfunction in Patients With Cardiovascular Disease

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SUMMARY

Left ventricular (LV) diastolic dysfunction is considered the main cause of heart failure with preserved ejection fraction (HFpEF). There have been few reports on the correlation between LV diastolic dysfunction and arterial stiffness in patients with clinical cardiovascular disease.

This cross-sectional study enrolled 100 patients (67 men, 33 women; mean age, 70 years). All participants were diagnosed with cardiovascular disease. A total of 89 (89%) patients had coronary artery disease or HF. Patients with reduced EF and valvular disease were excluded. Arterial stiffness was assessed by the cardio-ankle vascular index (CAVI), and LV diastolic dysfunction was estimated using echocardiography. The patients were divided into two groups based on the median value of CAVI. In all patients the ratio of early diastolic transmitral flow velocity to early diastolic mitral annular velocity (E/e') was significantly higher in the high CAVI group than in the low CAVI group (15.5 ± 6.4 versus 12.5 ± 2.9 , $P = 0.003$). In the HF subgroup, E/e' was also significantly higher in the high CAVI group than in the low CAVI group (17.2 ± 5.9 versus 13.0 ± 3.1 , $P = 0.026$). In univariate regression analysis, CAVI was significantly associated with E/e' in all patients ($\beta = 0.28$, $P = 0.004$) and in HF patients ($\beta = 0.4$, $P = 0.028$). Also in multivariate analysis, CAVI remained as an independent predictive factor of E/e' ($\beta = 0.252$, $P = 0.037$).

A high CAVI was independently associated with LV diastolic dysfunction in patients with clinical cardiovascular disease. These results suggested that arterial stiffness contributed to the development of LV diastolic dysfunction. (Int Heart J 2016; 57: 729-735)

Key words: Cardio-ankle vascular index, Heart failure with preserved ejection fraction

In Japan and other countries, approximately 40–50% of patients with heart failure (HF) have preserved left ventricular ejection fraction (LVEF). This is called HF with preserved ejection fraction (HFpEF) and is thought to be mainly caused by LV diastolic dysfunction.¹⁻⁴⁾ Various factors, including aging, hypertension, diabetes mellitus, chronic kidney disease, metabolic syndrome, and arterial stiffness are considered causes of LV diastolic dysfunction and can be important in the development of HFpEF.^{5,6)} The development of arterial stiffness in HF patients was reported to correlate with poor prognosis; therefore, assessment and management of arterial stiffness are important.⁷⁾ Pulse wave velocity (PWV) has been widely used as a non-invasive index of arterial stiffness.⁸⁾ Especially in Japan, brachial-ankle PWV (baPWV) is widely measured as a simple index of arterial stiffness.^{9,10)}

Cardio-ankle vascular index (CAVI), which was developed in Japan and has less evidence than baPWV, also provides a reproducible index of arterial stiffness, independent of blood pressure (BP).^{11,12)} A correlation between LV diastolic

dysfunction and arterial stiffness has been reported, mainly in patients with subclinical cardiovascular disease such as metabolic syndrome.¹³⁻¹⁹⁾ However, there have been few reports on this correlation in patients with clinical cardiovascular disease and LV diastolic dysfunction.

We examined the correlation between CAVI and multiple parameters, including echocardiographic and laboratory measurements of LV diastolic function and heart failure. We divided patients with clinical cardiovascular diseases, such as coronary artery disease (CAD), HF, and arrhythmia, into two groups based on the median value of CAVI.

METHODS

Study design and population: In this cross-sectional study, we enrolled 100 patients (67 men, 33 women; mean age, 70 years) diagnosed with clinical cardiovascular disease at the National Defense Medical College Hospital (Tokorozawa, Japan) from

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2013 to 2015. The study protocol complied with the Declaration of Helsinki and the institutional ethics policies.

Clinical cardiovascular diseases included CAD, HF, and arrhythmia. CAD or HF was present in 89% (89/100) of patients. Continuous medical treatment for clinical cardiovascular disease was required for inclusion in the analysis. Exclusion criteria were as follows: 1) LVEF < 50%; 2) more than mild valvular disease including aortic valve disease; 3) persistent atrial fibrillation (AF) during measurements; 4) peripheral artery disease (PAD) of the lower limbs, defined by an ankle-brachial index < 0.9; and 5) estimated glomerular filtration rate (eGFR) < 30 mL/minute.

CAD was defined as the presence of > 75% stenosis in at least one coronary vessel on an angiogram or a past history of myocardial infarction (MI) or any coronary artery revascularization. HF was defined as the presence of multiple signs and symptoms that were consistent with cardiac decompensation or a past history of hospitalization for HF. All patients with HF had preserved EF and no valvular disease according to exclusion criteria. Arrhythmia was defined as documentation of paroxysmal AF or ventricular tachycardia. Hypertension was diagnosed by BP above 140/90 mmHg or the use of BP-lowering medications. Hypercholesterolemia was diagnosed by low-density cholesterol (LDL-C) > 140 mg/dL or the use of LDL-lowering medications. Diabetes mellitus (DM) was diagnosed by fasting blood glucose > 126 mg/dL or the use of anti-diabetes medications.

Plasma LDL-C, triglycerides, high-density cholesterol (HDL-C), glycated hemoglobin, creatinine, and B-type natriuretic peptide (BNP) were measured by standard laboratory tests. The Modification of Diet in Renal Disease equation adjusted for a Japanese population was used to calculate eGFR.²⁰⁾

Cardio-ankle vascular index: CAVI was obtained automati-

cally using a vascular screening system (Vasera VS-1000, Fukuda Denshi; Tokyo) based on BP and heart-ankle PWV (haPWV), monitoring of heart sounds, and electrocardiogram. haPWV was calculated by dividing the distance from the aortic valve to the ankle artery by the sum of the time intervals between aortic valve closure sound and notch of the brachial pulse wave and between the rise of the brachial pulse wave and the ankle pulse wave. CAVI was determined using the following formula,

$$\text{CAVI} = a [2\rho/(\text{Ps}-\text{Pd})^* \ln(\text{Ps}/\text{Pd})^* \text{haPWV}^2] + b,$$

where Ps and Pd are systolic and diastolic BP, respectively; ρ is blood density; and a and b are constants. CAVI was taken as the average of the right and left CAVI values. BP and heart rate (HR) were measured at the time of CAVI measurement.

Echocardiographic assessment: Transthoracic echocardiography was performed at nearly the same time as the CAVI measurement by trained sonographers following a standard protocol. LV dimension, wall thickness, and left atrial diameter (LAD) were measured and LV diastolic dysfunction was assessed by recording LV diastolic inflow on pulsed Doppler echocardiography. Peak early diastolic transmitral flow velocity (E) and peak late diastolic transmitral flow velocity (A) phases of mitral inflow were sampled at the mitral valve leaflet tips from an apical 4-chamber view; from these, the ratio of E to A (E/A) was calculated. LV myocardial velocity by tissue-Doppler imaging was evaluated at the septal mitral valve annulus. The peak early diastolic annular velocity (e') was measured and the ratio of E to e' (E/e') was calculated as an index of LV diastolic dysfunction.

Statistical analysis: Continuous data are expressed as the mean $\pm$ standard deviation (SD) with the 95% confidential interval for a normal distribution. Comparison of variables in the two groups was performed by Student's *t*-test. Cross-table

Table I. Baseline Characteristics of Patients With Cardiovascular Disease

	Total (n = 100)	High CAVI (n = 50)	Low CAVI (n = 50)	P
Age (years)	70 $\pm$ 8	74 $\pm$ 6	67 $\pm$ 9	< 0.001*
Sex (male/female)	67 (67%) / 33 (33%)	32 (64%) / 18 (36%)	35 (70%) / 15 (30%)	0.528
Height (cm)	161 $\pm$ 9.2	159 $\pm$ 8.5	162 $\pm$ 9.7	0.196
Body weight (kg)	62 $\pm$ 12	59 $\pm$ 12	64 $\pm$ 12	0.074
BMI (kg/m ²)	23.8 $\pm$ 3.6	23.3 $\pm$ 3.4	24.2 $\pm$ 3.7	0.179
Hypertension (%)	80	80	78	0.835
Hyperlipidemia (%)	73	78	68	0.265
Diabetes mellitus (%)	21	28	14	0.087
Number of RF	1.7	1.6	1.8	0.127
CAD (%)	61	66	56	0.310
1VD (%)	35	32	38	0.534
2VD (%)	11	16	6	0.112
3VD (%)	15	18	12	0.406
Prior MI (%)	19	20	18	0.801
HF (%)	30	30	30	1.000
Arrhythmia (%)	16	14	18	0.590
Medication (%)				
Beta blockers	51	53	48	0.615
ACE-I/ARB	59	57	61	0.716
CCB	61	63	59	0.652
Statins	76	84	67	0.065
Antidiabetic agents	23	27	20	0.427

*Significant *P* values are reported. CAVI indicates cardio-ankle vascular index; BMI, body mass index; RF, cardiovascular risk factors; CAD, coronary artery disease; 1VD, 2VD, and 3VD, 1-, 2-, and 3-vessel disease, respectively; MI, myocardial infarction; HF, heart failure; ACE-I/ARB, angiotensin-converting enzyme inhibitors and/or angiotensin 2 receptor blockers; and CCB, calcium channel blockers.

analyses were performed using the chi-square test. Univariate and multivariate regression analyses were performed to identify independent variables from baseline characteristics and laboratory measurements to explain E/e' . In the multivariate analysis, all the independent factors correlating with E/e' ($P < 0.1$) in the univariate analysis were included in a crude model. In all analyses, $P < 0.05$ was considered statistically significant. All statistical analyses were performed using JMP version 10.0 (SAS Institute Inc.; Cary, NC, USA).

RESULTS

Baseline characteristics of the study population: Table I shows the baseline characteristics of the study population. No significant differences were found between the two groups in percentage of males; body mass index; prevalence of hypertension, hyperlipidemia, diabetes mellitus, and number of these cardiovascular risk factors; prevalence of CAD, number of diseased coronary vessels, prior MI, HF and arrhythmia; or medication including angiotensin-converting enzyme inhibitors and/or angiotensin 2 receptor blockers (ACE-I/ARB) and calcium channel blockers (CCB). However, age was significantly high-

Table II. Laboratory Measurements in Patients With Cardiovascular Disease

	Total ($n = 100$)	High CAVI ($n = 50$)	Low CAVI ($n = 50$)	P
CAVI	8.9 ± 1.1	9.7 ± 0.6	8.0 ± 0.8	$< 0.001^*$
haPWV (cm/s)	1602 ± 322	1700 ± 197	1465 ± 242	$< 0.001^*$
sBP (mmHg)	137 ± 20	139 ± 21	136 ± 17	0.465
dBp (mmHg)	81 ± 11	79 ± 10	83 ± 11	0.120
mBP (mmHg)	100 ± 12	99 ± 13	100 ± 12	0.589
PP (mmHg)	56 ± 16	59 ± 17	53 ± 14	0.043^*
HR (bpm)	64 ± 12	62 ± 12	67 ± 12	0.053
LVDd (cm)	4.7 ± 0.6	4.7 ± 0.5	4.7 ± 0.6	0.812
IVS (cm)	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.2	0.331
PW (cm)	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.2	0.618
LVEF (%)	69 ± 8	70 ± 8	68 ± 9	0.116
LAD (cm)	3.8 ± 0.5	3.8 ± 0.5	3.8 ± 0.5	0.747
E/A	0.8 ± 0.3	0.8 ± 0.3	0.8 ± 0.3	0.946
e' (cm/s)	5.5 ± 1.7	5.3 ± 1.7	5.8 ± 1.7	0.127
E/e'	14 ± 5.2	15.5 ± 6.4	12.5 ± 2.9	0.003^*
LDL-C (mg/dL)	93 ± 25	90 ± 24	96 ± 25	0.215
TG (mg/dL)	137 ± 76	135 ± 65	138 ± 85	0.853
HDL-C (mg/dL)	53 ± 12	55 ± 16	58 ± 16	0.355
HbA1c (%)	6.3 ± 0.8	6.3 ± 0.9	6.0 ± 0.7	0.073
eGFR (mL/minute)	63 ± 17	60 ± 15	67 ± 18	0.040^*
BNP (pg/mL)	70 ± 112	102 ± 150	44 ± 51	0.013^*

*Significant P values are reported. CAVI indicates cardio-ankle vascular index; haPWV, heart-ankle pulse wave velocity; sBP, systolic blood pressure; dBp, diastolic blood pressure; mBP, mean blood pressure; PP, pulse pressure; HR, heart rate; LVDd, left ventricular diastolic diameter; IVS, interventricular septum wall thickness; PW, posterior wall thickness; LVEF, left ventricular ejection fraction; LAD, left atrial diameter; E, peak early diastolic transmitral flow velocity; A, peak late diastolic transmitral flow velocity; e' , peak early diastolic annular velocity; E/A, ratio of E to A; E/e' , ratio of E to e' ; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; eGFR, estimated glomerular filtration rate; and BNP, B-type natriuretic peptide.

Table III. Baseline Characteristics of Patients With HF

	Total ($n = 30$)	High CAVI ($n = 15$)	Low CAVI ($n = 15$)	P
Age (years)	71 ± 7	72 ± 5	69 ± 9	0.256
Sex (male/female)	14 (47%) / 16 (53%)	7 (47%) / 8 (53%)	7 (47%) / 8 (53%)	1.000
Height (cm)	158 ± 10	158 ± 8	157 ± 11	0.801
Body weight (kg)	58 ± 12	57 ± 11	59 ± 13	0.702
BMI (kg/m^2)	23.2 ± 3.7	22.6 ± 2.8	23.6 ± 4.3	0.438
Hypertension (%)	83	87	80	0.764
Hyperlipidemia (%)	63	67	60	0.716
Diabetes mellitus (%)	23	40	7	0.072
Medication (%)				
Beta blockers	52	33	71	0.041^*
ACE-I/ARB	69	60	79	0.297
CCB	52	60	43	0.374
Statins	69	80	57	0.197
Antidiabetic agents	24	40	7	0.031^*

*Significant P values are reported. HF indicates heart failure; CAVI, cardio-ankle vascular index; BMI, body mass index; ACE-I/ARB, angiotensin-converting enzyme inhibitors and/or angiotensin 2 receptor blockers; and CCB, calcium channel blockers.

Table IV. Laboratory Measurements in Patients With HF

	Total (n = 30)	High CAVI (n = 15)	Low CAVI (n = 15)	P
CAVI	8.9 ± 1.2	9.9 ± 0.7	7.9 ± 0.9	< 0.001*
haPWV (cm/s)	1622 ± 268	1787 ± 253	1501 ± 279	0.012*
sBP (mmHg)	141 ± 22	142 ± 25	140 ± 17	0.830
dBp (mmHg)	78 ± 10	79 ± 10	78 ± 9	0.929
mBP (mmHg)	99 ± 13	100 ± 14	99 ± 11	0.865
PP (mmHg)	62 ± 17	63 ± 19	62 ± 14	0.824
HR (bpm)	61 ± 8	61 ± 8	62 ± 9	0.425
LVDd (cm)	4.9 ± 0.7	4.7 ± 0.5	5.1 ± 0.8	0.104
IVS (cm)	1.0 ± 0.3	1.0 ± 0.2	1.1 ± 0.3	0.372
PW (cm)	1.0 ± 0.1	1.0 ± 0.1	1.0 ± 0.2	1.000
LVEF (%)	66 ± 9	70 ± 7	62 ± 10	0.014*
LAD (cm)	4.0 ± 0.6	4.0 ± 0.7	4.0 ± 0.5	0.921
E/A	0.9 ± 0.3	0.9 ± 0.3	0.8 ± 0.3	0.360
e' (cm/s)	5.2 ± 1.6	5.0 ± 1.4	5.4 ± 1.8	0.501
E/e'	15.1 ± 5.1	16.7 ± 6.0	13.0 ± 3.1	0.026*
LDL-C (mg/dL)	104 ± 25	99 ± 25	109 ± 22	0.317
TG (mg/dL)	126 ± 50	132 ± 43	117 ± 54	0.327
HDL-C (mg/dL)	60 ± 17	53 ± 12	67 ± 17	0.017*
HbA1c (%)	6.1 ± 0.7	6.4 ± 0.8	5.8 ± 0.6	0.114
eGFR (mL/minute)	61 ± 20	58 ± 18	62 ± 20	0.769
BNP (pg/mL)	138 ± 172	205 ± 217	71 ± 57	0.027*

*Significant *P* values are reported. HF indicates heart failure; CAVI, cardio-ankle vascular index; haPWV, heart-ankle pulse wave velocity; sBP, systolic blood pressure; dBp, diastolic blood pressure; mBP, mean blood pressure; PP, pulse pressure; HR, heart rate; LVDd, left ventricular diastolic diameter; IVS, interventricular septum wall thickness; PW, posterior wall thickness; LVEF, left ventricular ejection fraction; LAD, left atrial diameter; E, peak early diastolic transmitral flow velocity; A, peak late diastolic transmitral flow velocity; e', peak early diastolic annular velocity; E/A, ratio of E to A; E/e', ratio of E to e'; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; eGFR, estimated glomerular filtration rate; and BNP, B-type natriuretic peptide.

Figure 1. Correlation between cardio-ankle vascular index (CAVI) and the ratio of early diastolic transmitral flow velocity to early diastolic mitral annular velocity (E/e') in all patients.

er in the high CAVI group than in the low CAVI group.

Laboratory measurements and echocardiographic characteristics: Table II shows the laboratory measurements and echocardiographic characteristics of the patients according to CAVI value. E/e', pulse pressure, and BNP were significantly higher in the high CAVI group than in the low CAVI group. The high CAVI group had a lower eGFR. Systolic and diastolic BP, HR, LV diastolic diameter, LV wall thickness, E/A, and LVEF were similar between the two groups.

Subgroup analysis of HF patients: There were 30 patients with HF. The percentage of HF patients who were hypertensive was 83%. As in the overall analysis, no significant differences were found between the high and low CAVI groups in

Figure 2. Correlation between CAVI and E/e' in HF patients.

percentage of males; body mass index; or prevalence of hypertension, hyperlipidemia, and diabetes mellitus. In this subgroup analysis, age was not significantly different between the two groups (Table III). Table IV shows the laboratory measurements and echocardiographic characteristics of HF patients according to the CAVI value. Compared with the low CAVI group, the high CAVI group had significantly higher E/e', LVEF, and BNP. HDL-C was lower in the high CAVI group. Systolic and diastolic BP, HR, LV diastolic diameter, LV wall thickness, and LAD were not different between the two groups.

Regression analysis: In univariate analysis, the CAVI was significantly associated with E/e' in all patients ($\beta = 0.28$, $P = 0.004$) (Figure 1) and in HF patients ($\beta = 0.4$, $P = 0.03$) (Figure 2). Table V shows the results of regression analysis for predic-

Table V. Regression Analysis for Prediction of E/e' in Patients With Cardiovascular Disease

Total (n = 100)	Univariate		Multivariate	
	β	P	β	P
Age (years)	0.268	0.007*	0.087	0.499
Sex (male = 1, female = 0)	-0.148	0.143		
BMI (kg/m ²)	-0.03	0.773		
Hypertension (yes = 1, no = 0)	0.203	0.043*	0.143	0.196
Hyperlipidemia (yes = 1, no = 0)	-0.004	0.972		
Diabetes mellitus (yes = 1, no = 0)	0.126	0.212		
CAD (yes = 1, no = 0)	-0.004	0.968		
Prior MI (yes = 1, no = 0)	0.006	0.952		
HF (yes = 1, no = 0)	0.139	0.168		
Arrhythmia (yes = 1, no = 0)	-0.004	0.965		
Medication				
Beta blockers (yes = 1, no = 0)	0.037	0.723		
ACE-I/ARB (yes = 1, no = 0)	0.037	0.723		
CCB (yes = 1, no = 0)	0.127	0.219		
Statins (yes = 1, no = 0)	-0.008	0.938		
Antidiabetic agents (yes = 1, no = 0)	0.077	0.458		
CAVI	0.284	0.004*	0.252	0.037*
sBP (mmHg)	0.19	0.058	0.241	0.211
dBp (mmHg)	0.07	0.487		
PP (mmHg)	0.186	0.064	-0.157	0.448
HR (bpm)	-0.083	0.411		
LVEF (%)	0.14	0.163		
LDL-C (mg/dL)	0.111	0.278		
TG (mg/dL)	-0.068	0.513		
HDL-C (mg/dL)	0.176	0.088	0.211	0.050
HbA1c (%)	0.076	0.491		
eGFR (mL/minute)	-0.116	0.254		
BNP (pg/mL)	0.198	0.062	0.110	0.345

*Significant P values are reported. BMI indicates body mass index; CAD, coronary artery disease; MI, myocardial infarction; HF, heart failure; ACE-I/ARB, angiotensin-converting enzyme inhibitors and/or angiotensin 2 receptor blockers; CCB, calcium channel blockers; CAVI, cardio-ankle vascular index; sBP, systolic blood pressure; dBp, diastolic blood pressure; PP, pulse pressure; HR, heart rate; LVEF, left ventricular ejection fraction; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; eGFR, estimated glomerular filtration rate; and BNP, B-type natriuretic peptide.

tion of E/e' in patients with cardiovascular disease. The factors other than CAVI that correlated with E/e' were age, prevalence of HT, systolic BP, pulse pressure, HDL-C, and BNP. In a crude model, CAVI remained as an independent predictive factor of E/e' ($\beta = 0.252$, $P = 0.037$), but age and other factors did not.

DISCUSSION

The incidence of patients with HFpEF is constantly increasing, but therapeutic strategies are limited. Therefore, identification of the pathophysiologic factors in HFpEF is necessary to formulate new therapeutic strategies. CAVI is a typical parameter of arterial stiffness that is independent of BP. In this study, we investigated the relationship between CAVI and echocardiographic parameters in patients with clinical cardiovascular disease. The results showed that CAVI was positively correlated with E/e' in all patients and in the HF subgroup. These data suggested a strong correlation between arterial stiffness and LV diastolic dysfunction, independent of BP.

The contribution of elevated LV afterload to the development of LV diastolic dysfunction has been considered.²¹⁾ From what is known as the Windkessel effect, when blood is ejected from the heart to the aorta, the transient expansion of the aorta transmits pulse waves to peripheral arteries. This pulse wave is

reflected at the bifurcation of the common iliac artery, whereas the retrograde reflection pulse wave amplifies arterial BP when it returns to the ascending aorta. The return of the reflection pulse wave to the ascending aorta happens during diastole when PWV is slow and during late systole when PWV is fast due to arterial stiffness. This change in loading sequence amplifies systolic BP and increases pulse pressure.^{22,23)} As a result, LV afterload is increased. In addition, this change reduces the time constant τ , an indicator of LV relaxation, which is a component of LV diastolic function and significantly correlates with the development of LV diastolic dysfunction.²⁴⁾ When PWV is fast, the arrival of the reflection pulse wave during late systole causes a reduction in diastolic pressure and may correlate with LV diastolic dysfunction due to endomyocardial ischemia secondary to a reduction in coronary perfusion pressure.²⁵⁾ In the present study, we employed CAVI calculated by inserting haPWV, because it reflects stiffness of the whole arterial system from the aortic root to the lower extremities and might be more appropriate to estimate the part of LV afterload.

Systemic vascular resistance (SVR) is widely used as an index of LV afterload. Thus, vasodilator treatment is administered to reduce SVR in cases of acute decompensated HF and to prevent elevation of SVR and LV afterload mismatch in chronic HF. However, as a component of LV afterload, arterial stiffness is important along with SVR, especially in elderly patients.^{5,6,26-29)} Therefore, it may not be appropriate to prevent the

development of LV diastolic dysfunction by reduction of SVR alone.

Some reports have discussed the correlation between arterial stiffness and LV diastolic dysfunction, mainly in subclinical patients with cardiovascular risk factors, and in patients with clinical cardiovascular diseases including CAD or HF.¹³⁻¹⁹⁾ In the present study, all patients had clinical cardiovascular diseases. In particular, approximately 90% of the patients enrolled had CAD or HF and required continuous medical treatment. Many of the patients already had stiff arteries and LV diastolic dysfunction, so the average values of CAVI (8.9) and E/e' (14.0) were high. Based on our results, patients with possible LV diastolic dysfunction may require therapeutic intervention for arterial stiffness. However, a medical intervention for arterial stiffness has not been established. There have been several reports on possible effective pharmacologic therapies, such as ACE-I/ARB, CCB, statins, and sarpogrelate hydrochloride, and non-pharmacologic therapies, such as exercise and continuous positive airway pressure.³⁰⁻³⁹⁾ Further studies are needed to establish indicators and therapeutic methods for arterial stiffness.

There are several limitations in our study. First, the sample size was small. Second, patients with reduced EF (LVEF < 50%), more than mild valvular disease, ankle-brachial index < 0.9, persistent AF, and eGFR < 30 mL/minute were excluded, so the applicability of our results to such patients is unknown. Third, the study did not focus on patients with HF because we aimed to ascertain the general relationship between CAVI and echocardiographic data as an initial study.

In conclusion, a high CAVI value was independently associated with LV diastolic dysfunction in patients with clinical cardiovascular disease. The results suggest that arterial stiffness contributed to the development of LV diastolic dysfunction. The measurement of CAVI might be an important application for the risk of HFpEF.

DISCLOSURE

None.

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