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Original Article

Impaired cardiac and vascular motion in patients with Takayasu's arteritis: A velocity vector imaging-based study

Selen Yurdakul[1], Fatma Alibaz-Öner[2], Haner Direskeneli[2], Saide Aytekin[1]

Abstract

Objective: Takayasu's arteritis (TAK) is a chronic inflammatory vasculitis of the aorta and its major branches. In the present study, we aimed to evaluate the motion of the vascular wall and myocardial contractility by using a novel strain imaging method, velocity vector imaging (VVI), in patients with TAK. We also aimed to compare them with another inflammatory autoimmune disorder, systemic lupus erythematosus (SLE).

Methods: We studied 33 patients with TAK, 18 patients with SLE, and 20 age- and sex-matched controls. All participants were subjected to carotid artery Doppler ultrasonography and transthoracic echocardiographic evaluation. VVI analysis was also performed to assess subclinical left ventricular (LV) systolic dysfunction and to determine tissue motion of the common carotid arteries (CCAs).

Results: Aortic strain and distensibility were significantly impaired in patients with TAK, while the aortic stiffness and carotid artery stiffness indexes were increased. Aortic distensibility was the only parameter that was decreased among SLE patients. The values of CCA peak longitudinal strain, strain rate, and total longitudinal displacement (TLD) were also impaired in patients with TAK. Peak radial velocity was decreased while time-to-peak radial velocity was increased. In the SLE group, peak longitudinal strain, strain rate, TLD, and peak radial velocity were impaired. LV longitudinal peak systolic strain and strain rate were reduced in patients with TAK. Similarly, we revealed impaired subclinical LV systolic function in patients with SLE.

Conclusion: VVI is a novel strain imaging technique with additional value to determine early impairment in vascular and myocardial wall motion in patients with TAK.

Keywords: Takayasu's arteritis, carotid artery, cardiac, strain, velocity vector imaging

ORCID IDs of the authors:
S.Y. 0000-0001-7763-1984;
F.A.Ö. 0000-0002-6653-1758;
H.D. 0000-0003-2598-5806;
S.A. 0000-0003-1968-0889.

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[1] Division of Cardiology, Florence Nightingale Hospital, İstanbul, Turkey
[2] Department of Rheumatology, Marmara University School of Medicine, İstanbul, Turkey

Address for Correspondence:
Fatma Alibaz-Öner, Department of Rheumatology, Marmara University School of Medicine, İstanbul, Turkey

E-mail: falibaz@gmail.com

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Introduction

Takayasu's arteritis (TAK) is a rare chronic inflammatory disease characterized by panarteritis of large arteries (1). It causes occlusive or ectatic changes in the aorta and its major branches (2, 3). Reduced compliance and loss of elasticity in the affected arteries may result in an increased arterial stiffness in TAK. However, there are scarce data evaluating the regional vascular mechanical properties of the major arteries in patients with TAK (4).

A lack of adequate diagnostic tools to identify the disease in its early phase results in delayed diagnosis and treatment in TAK, which might lead to a poor prognosis. Although some imaging modalities such as echocardiography, Positron Emission Tomography (PET) scan, and magnetic resonance imaging are commonly used in routine clinical practice, assessment of disease activity and vascular damage is quite problematic due to the lack of specific laboratory and imaging findings (1).

Velocity vector imaging (VVI) is a two-dimensional strain imaging technique, which provides more accurate data on regional cardiac function (5, 6). Based on the literature, VVI has emerged as a reliable method for the quantification of regional contractile function with the ability of detecting subclinical cardiac systolic dysfunction (7-9). In some recent studies, VVI has also been used to evaluate the motion of the carotid artery wall (10).

In the present study, we aimed to explore this novel approach for the early assessment of the impact of systemic and vascular inflammation on vascular and myocardial injury in patients with TAK. We also aimed to evaluate changes in vascular and myocardial mechanics in a subset of patients with 12 months of follow-up.

Methods

Study design and patient population

The study included 33 patients (mean age: 39.06±11.44 years; female (F)/male (M): 31/2) with TAK, fulfilling the 1990 classification criteria of the American College of Rheumatology (ACR); 18 patients (mean age: 42.05±10.42 years; F/M: 17/1) with systemic lupus erythematosus (SLE), classified according to ACR criteria for SLE; and 20 age- and sex-matched control subjects (mean age: 36.7±11.9 years; F/M: 17/3) (11, 12). Inclusion criteria for the study were functional capacity of class I according to the New York Heart Association and sinus rhythm (1, 2). Patients with left ventricular (LV) EF <60%, coexistence of mitral and/or aortic valve disease above than a mild degree, known or suspected coronary artery disease, low quality echocardiographic image for VVI analysis, having atrioventricular conduction abnormalities and atrial fibrillation were excluded from the study. We also excluded patients with cardiovascular risk parameters defined as hyperlipidemia, hypertension, diabetes, and smoking.

At the end of 12 months, 10 of 33 patients with TAK were reevaluated regarding carotid artery wall motion and LV systolic function, based on VVI-derived strain imaging.

Echocardiographic and carotid artery ultrasound examinations were performed by one operator who was blinded to the diagnoses of the study population.

The study protocol was approved by local ethics committee, and a written informed consent was obtained from each patient. The study was performed according to the Declaration of Helsinki.

Conventional echocardiographic and aortic stiffness measurements

Patients underwent transthoracic echocardiography (Siemens, Sequoia, C256; Mountain View, CA, USA) by using a 2.3-3.5 MHz transducer. Left ventricular end-diastolic and end-systolic diameters were measured from the parasternal long-axis view by using M-mode echocardiography (13). From the apical four-chamber view, LV EF was calculated using the modified Simpson's method (13).

Systolic (AoS) and diastolic (AoD) diameters of the ascending aorta were calculated at the area of 3 cm above the aortic valve in the parasternal long-axis view of the LV, using M-mode echocardiography, and defined as the average of measurements of three cardiac consecutive cycles. Aortic stiffness parameters were calculated from the formulas identified before, as mentioned below (14). Blood pressure (BP) was measured using conventional sphygmomanometry (15). Measurements were performed on the left arm with the subject sitting for at least five minutes. The first Korotkoff sound was recorded as the systolic blood pressure (SBP) and the fifth as the diastolic blood pressure (DBP). Three measurements were taken for each subject with one-minute intervals, and the average values were calculated.

Aortic strain (%): AoS-AoD/AoD×100

Aortic stiffness: logarithm (SBP/DBP)/aortic strain

Aortic distensibility (cm^2 $dyn.-1.10^{-3}$):2×aortic strain/SBP-DBP

Velocity vector imaging of the left ventricle

The four-chamber, two-chamber, and long-axis views of the left ventricle were recorded for the VVI analysis in a high frame rate. Recorded images were analyzed offline using the VVI software (Syngo VVI, Siemens Medical Solutions; Mountain View, CA, USA). The frame rate was between 70 and 100 frames per second. The endocardial borders were defined manually by the echocardiographer, and then the VVI software automatically determined the sampling points for all of the segments according to 16-segment LV model of the American Society of Echocardiography (16). Following this procedure, images were analyzed using the VVI software. Strain (%) and strain rate (1/s) were defined as the change in the relative distance between localized tracked trace points. Strain (S) was defined as the instantaneous lengthening or shortening, and the strain rate (SR) was defined as the rate of lengthening or shortening (11).

Carotid artery ultrasound examinations

Carotid artery B-mode USG was performed by an experienced sonographer (SY). The carotid intima media thickness (CIMT) and plaques were measured in the left and right common carotid arteries (CCA) by using high-resolution B-mode USG (Siemens, Sequoia, C256; Mountain View, CA, USA) with a linear transducer having the frequency of 11 MHz. Both CCAs were on the near and far walls of the distal 2 cm of the CCA proximal to its bifurcation. Intima media thickness measurements were performed at 3 points of the far walls of both distal CCAs. The distance between the leading edge of the first echogenic line and the leading edge of the other echogenic line was measured, as defined by Pignoli et al. (17) above Three measurements were obtained for each side of the CCAs. Separate mean values were calculated and recorded as the mean CIMT of the left and right CCA. Plaque was defined as a focal structure encroaching into the arterial lumen of at least 0.5 mm or 50% of the surrounding IMT value or a thickness of ≥1.5 mm as measured from the media-adventitia interface to the intima-lumen interface (18). Systolic and diastolic diameters of the CCA were defined as the radial diameter change during a heart cycle. Luminal stenosis was calculated as the percent ratio of internal diameter to external diameter at peak diastole (19). Carotid artery stiffness index was calculated using the following formula (20):

Logarithm (SBP/DBP)×CCA diastolic diameter/CCA systolic diameter-CCA diastolic diameter

Velocity vector imaging of the common carotid artery

Cine-looped images from short- and long-axis views of the CCA in three consecutive cardiac cycles were obtained and stored for offline analysis. The frame rate was kept between 70-100 frames per second. Novel software (Syngo VVI, Siemens Medical Solutions; Mountain View, CA, USA) VVI was performed for the offline analysis from the near and the far wall of the right and the left CCA in the B-mode images. In order to evaluate the systolic wall motion of the CCA, the media-adventitia border was manually traced from a still-frame image by the researcher at the region approximately 1 cm proximal to the carotid bulb. For the longitudinal measurements, a segment 6 mm in length was traced and five guiding points, each 0.25 cm apart, were marked. In the transverse images, the arterial wall was divided into six segments. The border was then automatically tracked by the software. Gray-scale recorded images were analyzed, and the velocity vectors in the two-dimensional plane were obtained. Longitudinal S, SR, total longitudinal displacement (TLD), peak radial velocity (Pv), and time-to-peak radial velocity (Tv) parameters were calculated from three consecutive cardiac cycles, and the average value was recorded.

Reproducibility

All VVI measurements were performed by a single observer. A sample of 10 VVI measurements was randomly selected and examined by the same observer in two different days to determine intraobserver variability. Intraclass correlation coefficients for the same observer were calculated (21).

Statistical analysis

Statistical data were performed with the Statistical Package for the Social Sciences 16.0 (SPSS Inc.; Chicago, IL, USA) program. Results were expressed as means and standard deviations. One-way ANOVA and the Bonferroni correc-

tion were used for comparisons of each group. We used the paired t-test to evaluate changes in VVI measurements at the end of 12 months. The results were considered significant when the p-value was less than 0.05.

Results

Clinical characteristics and conventional echocardiographic data

Patients with TAK and SLE were similar to the control group in terms of age (p=0.32) and gender (p=0.72). The duration of disease among TAK patients was 5.9 years, and 35.5% of patients were in the active phase. According to the TAK angiographic classification, 41.9% of the study group had type 1, 58.1% had type 5, and 1 patient had type 2 of the disease (22). Clinical and demographic characteristics are presented in Table 1. SBP and DBP indices were increased in the TAK group (p=0.0001), whereas they were similar in the SLE group compared with the controls (p=0.94 for SBP; p=0.45 for DBP).

Table 1. Clinical and demographic characteristics

Variable	Patients with TAK (n=33)	Control group (n=20)	p
Age (years)	39.06±11.44	36.7±11.9	0.74
Male gender (n)	2	3	0.57
SBP (mmHg)	148±5.70	115±6.04	0.0001
DBP (mmHg)	91.93±4.77	70.50±6.66	0.0001
Disease duration (years)	5.9		
Sedimentation rate (mm/h)	32.9		
CRP (mg/L)	40.02		
Active disease (%)	35.48%		
Angiographic type-1 disease (%)	41.93%		
Angiographic type-5 disease (%)	58.06%		
Medical therapy:			
Azathiopurine	52.9% (n=18)		
Methotrexate	32.3% (n=11)		
Leflunomide	5.8% (n=2)		
Azathiopurine + TNF inhibitor	5.8% (n=2)		
Corticosteroid	2.9% (n=1)		

TAK: Takayasu's arteritis; SBP: systolic blood pressure; DBP: diastolic blood pressure; CRP: C-reactive protein; TNF: tumor necrosis factor

Carotid artery doppler ultrasonography measurements

Among patients with TAK, 2 of 33 (6.06%) had luminal stenosis of the CCA>50%, and the mean value of stenosis, based on carotid B-mode USG evaluation, was 27.75%. In the SLE group, none of the patients had luminal stenosis in the CCA.

The mean IMT of the CCA in patients with TAK was significantly higher than HC (p=0.0001), while it was similar between SLE patients and patients in the control group (p=0.05). Carotid artery stiffness index was increased in patients with TAK (p=0.0001), whereas it was similar between SLE patients and HC (p=0.94) (Table 2).

Table 2. Carotid artery doppler ultrasonography and velocity vector imaging evaluation in patients with TAK and SLE

Variable	TAK (n=33)	SLE (n=18)	Control group (n=20)	p
CIMT (cm)	0.11±0.03	0.09±0.01	0.07±0.009	0.0001
Carotid artery stiffness index	4.97±3.3	2.21±0.85	1.96±0.72	0.0001
Peak longitudinal S (%)	1.22±0.70	2.09±1.28	4.33±2.45	0.0001
Peak longitudinal SR (1/s)	0.17±0.10	0.23±0.11	0.68±0.3	0.0001
TLD (ms)	0.22±0.10	0.14±0.01	0.27±0.12	0.003
Peak radial velocity (mm/s)	0.10±0.01	0.07±0.02	0.27±0.11	0.0001
Time-to-peak radial velocity (ms)	221±67.8	177±39.9	139±33.8	0.0001

TAK: Takayasu's arteritis; CIMT: carotid intima media thickness; S: strain; SR: strain rate; TLD: total longitudinal displacement; SLE: systemic lupus erythematosus

Considering VVI analysis, peak longitudinal S and SR and TLD values were significantly impaired in patients with TAK (p=0.002, p=0.0001, and p=0.003, respectively). Peak radial velocity was decreased (p=0.0001), while Tv was markedly increased (p=0.0001) in TAK patients (Table 2). In the SLE group, peak longitudinal S (p=0.0001), SR (p=0.0001), TLD (p=0.04), and Pv (p=0.0001) were significantly impaired. Time-to-peak radial velocity of the CCA was decreased; however, the difference did not reach statistical significance (p=0.09). Based on VVI results, we observed significant impairment in longitudinal and radial motion of the CCA in patients with both TAK and SLE.

When we compared longitudinal and radial wall motion of the CCA among patients with active and inactive TAK no significant difference was obtained regarding regional wall motion of the CCA. We also evaluated longitudinal and radial deformation of the CCA to compare patients with disease type 1 and type 5. However, no marked difference was demonstrated. We also grouped patients with TAK according to the degree of luminal narrowing of the CCA, based on B-mode carotid artery USG evaluation. Seven of 33 patients had CCA stenosis ≥40%, and the remainder had stenosis <40%. There was no significant difference between the two groups regarding longitudinal S (p=0.07), longitudinal SR (p=0.45), TLD (p=0.86), and time-to-peak radial velocity (p=0.35). Such a result provides important data about the impaired deformation of the CCA wall, even in the pre-stenotic phase. Additionally, patients with TAK were grouped according to CRP values. Among all patients, 14 had CRP values >5 mg/L, and the remainder had CRP values <5 mg/L. Considering the wall mechanics of the CCA, longitudinal S (p=0.29), SR (p=0.58), and TLD (p=0.15) measurements were slightly more impaired in patients with CRP values >5 mg/L, but the difference was not statistically significant. Radial deformation parameters were similar between the groups.

At the end of 12 months, 10 of 33 TAK patients were reevaluated. There was no change in the clinical status and the medical treatment of

Table 3. Aortic stiffness measurements in patients with TAK and SLE

Variable	TAK (n=33)	SLE (n=18)	Control group (n=20)	p
Aortic strain (%)	5.77±3.2	11.97±6.7	13.91±4.77	0.0001
Aortic stiffness	6.17±5.1	2.17±1.3	2.6±1.3	0.003
Aortic distensibility (cm^2 dyn. - 1. 10^{-3})	0.47±0.3	0.61±0.3	1.64±0.77	0.0001

TAK: Takayasu's arteritis; SLE: systemic lupus erythematosus

Table 4. Left ventricular deformation parameters

	TAK (n=33)	SLE (n=18)	Control group (n=20)	p
LV strain (%)	13.20±3.5	14±4.53	20.97±4.5	0.0001
LV strain rate (1/s)	0.23±0.18	0.23±0.12	4.92±0.55	0.0001

LV: Left ventricle; TAK: Takayasu's arteritis; SLE: systemic lupus erythematosus

these patients. Peak longitudinal S (1.43±1.1% to 1.3±1; p=0.0001), SR (0.17±0.11 to 0.10±0.05; p=0.0001), and peak radial velocity (0.11±0.04 mm/s to 0.03±0.02 mm/s; p=0.0001) measurements were significantly more impaired, while time-to-peak radial velocity was increased (227±48.9 ms to 247±109.4 ms; p=0.003), indicating deteriorated CCA wall motion even in the short-term follow-up.

Aortic stiffness measurements

Our data for aortic stiffness parameters confirmed that aortic elasticity indices were impaired in patients with TAK (Table 3). Aortic strain and distensibility were decreased (p=0.0001), whereas aortic stiffness was markedly increased in patients with TAK (p=0.003). There was no difference in aortic strain (p=0.43) and stiffness (p=0.93) measurements between the SLE group and the control group, while aortic distensibility was impaired in both groups (p=0.0001).

Conventional echocardiographic and velocity vector-derived strain imaging of the left ventricle

Regarding conventional echocardiographic parameters, LV EDD, ESD, and EF were similar in patient groups, compared to HC (for the TAK group, p=0.97, p=0.26, and p=0.99 respectively; for the SLE group, p=0.83, p=0.74, and p=0.99 respectively) (Table 4). Strain and SR data were obtained and analyzed from all patients and controls. Base, mid, and apical segments of the LV were analyzed from apical four-chamber, two-chamber, and long-axis views. The mean values of S and SR of each LV wall were taken into consideration for the assessment of LV longitudinal systolic functions.

Longitudinal peak systolic S and SR measurements of the LV were markedly decreased in patients with TAK compared with the control group, indicating subclinical LV systolic dysfunction (p=0.0001) (Table 4). Supporting impairment of LV longitudinal deformation in patients with TAK, LV peak systolic S and SR parameters of patients with SLE were also decreased (p=0.0001) (Table 4).

In the short-term follow-up assessment, we also demonstrated marked impairment of LV subclinical systolic function (S: 15±4.07% to 12.75±1.03%; p=0.0001; SR: 0.32±0.30 to 0.22±0.12; p=0.0001).

Reproducibility

Spearman's test was performed for the correlations of intraobserver variability. The intraobserver correlations were good for the VVI-derived parameters (r=0,68 and p=0.04 for peak longitudinal S of the CCA; r=0.98 and p=0.0001 for peak longitudinal SR of the CCA; r=0.74 and p=0.021 for the TLD of the CCA; r=0.99 and p=0.0001 for peak radial velocity of the CCA; p=0.69 and p=0.027 for time-to-peak velocity of the CCA; r=0.86 and p=0.002 for LV longitudinal S; r=0.98 and p=0.0001 for LV longitudinal SR).

Discussion

In the current study, we analyzed the mechanical properties of carotid arteries and myocardium in patients with TAK and compared those parameters of patients with TAK with SLE as another inflammatory disease and HC. We were able to demonstrate reduced arterial wall motion in the pre-stenotic phase and impaired systolic function of the LV with preserved EF in TAK patients. We also demonstrated significant progressive impairment in both the carotid artery and cardiac mechanics at the end of a 12-month period. We suggest that vascular and myocardial damage in the preclinical phase is a consequence of a generalized inflammatory process in TAK. Identification of an inflammatory injury may be an important approach to provide early therapy in these patients.

The early stage of TAK presents with non-specific inflammatory features (2). Then progressive thickening of the vessel lumen predisposes to arterial occlusions (23). The CCA is frequently involved in TAK (24). Morphological characteristics of the CCA have been extensively studied before. Conventional B-mode USG is a widely used tool that provides accurate measurements of CIMT and visualizations of carotid artery wall morphology. However, few data investigated the wall mechanics of the CCA (4).

Currently, strain imaging is considered to be superior to conventional echocardiographic parameters because of its ability to provide quantitative endocardial deformation analysis (25-27). VVI is a two-dimensional strain imaging method that evaluates regional myocardial motion providing the quantification of S, SR, and velocity (28). In previous studies, VVI has been used to demonstrate subclinical LV dysfunction (29-31). Recently, advances in novel strain imaging have allowed quantification of vessel wall motion, offering a detailed analysis of longitudinal and radial deformation.[4] In a few studies, VVI-based strain imaging has been presented as a novel method to quantitatively assess CCA wall motion (32). Svedlund et al. (10) presented VVI as a feasible technique in assessing longitudinal CCA wall motion. In their study, they demonstrated decreased longitudinal motion of the CCA in patients with coronary artery disease.

The most commonly affected vessels are the subclavian and the CCA in TAK (33). Stenosis of the vessel lumen is reported in >90% of patients (30). Vascular lesions are characterized by leukocyte infiltration into tunica media, intimal hyperplasia, and adventitial thickening (1). Fibrosis of the media and intima, followed by arterial occlusion, develops as a consequence of the inflammatory process. Additionally, loss of elasticity in the vessel wall prevents systolic expansion of the artery.

In the present study, we observed, based on conventional B-mode USG, diffuse thickening of the intima media in patients with TAK. We also demonstrated an increase in arterial stiffness, documented by the measurement of the central aorta and CCA. According to our data, VVI-derived strain imaging presented significant impairment in the longitudinal and radial wall motion of the CCA. We also established a marked decrease in peak longitudinal S and SR, as well as TLD of the CCA. Peak radial velocity was also impaired and time-to-peak radial velocity was increased, suggesting a disturbance of the systolic expansion of the CCA in the radial axis. We also observed disruption of the

systolic expansion of the CCA in the SLE group. Disturbance of elastic deformation of the CCA in patients with TAK and SLE supports widespread inflammatory damage as result of both disorders. Our detailed analysis showed that impairment of longitudinal and radial wall motion of the CCA without significant luminal stenosis. We assume that VVI can been presented as a new approach for the early assessment of uninvolved vessels in the pre-stenotic phase in patients with TAK. Supporting our study, Yang et al. evaluated CCA wall motion in patients with Marfan syndrome (34). Consistent with our findings, Cho et al. detected impairment in radial and circumferential wall motion of the CCA in patients with TAK (4). Additionally, progressive impairment of carotid artery and cardiac mechanics, even in a short-term period, emphasizes the importance of closer follow-up of patients with TAK.

Cardiac involvement and ventricular dysfunction due to connective tissue diseases have been reported previously (35). However, there is limited data evaluating LV systolic dysfunction in TAK (36). Keenan et al. (37) suggested that cardiac complications associated with the disease can be attributed to increased arterial stiffness. They performed cardiac magnetic resonance (CMR) imaging to identify a previously silent myocardial infarction and myocardial scar and postulated that mid-wall fibrosis based on CMR imaging can be observed as evidence of primary inflammatory involvement of the myocardium. Our analysis revealed that subtle changes in LV contractility develop in patients with TAK without any cardiac disease. The possible underlying mechanism of LV deterioration in TAK is not clarified yet; however, impairment of myocardial tissue deformation may be attributed to persistent inflammatory damage. In the present study, we also collected observational data reflecting the subclinical LV systolic dysfunction in patients with SLE. Impaired cardiac function with a higher mortality and morbidity is related to the presence of accelerated atherogenesis that is mostly associated with the systemic inflammation in SLE. The relevance of ventricular deterioration in patients with SLE has been emphasized by several studies using different imaging modalities (38).

Despite optimal medical treatment, the long-term clinical outcomes in patients with TAK is still unsatisfactory. Complex clinical presentation of the disease and a lack of controlled data result in delayed diagnosis and treatment. In order to overcome the challenges associated with the early assessment of TAK, VVI can be used to identify early mechanical changes of the arterial wall as a result of inflammation. Identification of vascular injury in the pre-stenotic phase may help to have a chance for earlier treatment before development of irreversible damage in the vessel wall. VVI-based strain imaging has emerged as a novel method that provides quantitative data for detecting myocardial dysfunction before clinical signs develop.

It may not be practical to assess vascular and myocardial wall motion in all patients with TAK or inflammation of unknown origin. However, in order to identify subtle changes in vascular and myocardial motion in patients with early-phase TAK, we suppose that the VVI method can be used especially for detection of early inflammatory changes in the follow-up of patients.

In the current study, we combined a simultaneous evaluation of morphological and functional properties of CCA wall motion. We were able to demonstrate impaired longitudinal and radial deformation of the artery in patients with TAK. VVI-based strain imaging provides detailed data for the preclinical evaluation of myocardial and carotid artery wall motion. In the current study, we performed this novel method with its additional value to identify subclinical systolic dysfunction of the LV. In order to assess disease activity, especially in uninvolved vessels early, VVI can be used as a quantitative approach to identify preclinical changes of vascular and myocardial motion.

Limitations and strengths

The strength of this study is its detailed evaluations of CCA wall motion and LV systolic function using a noninvasive imaging technique (VVI) that is known to be a reliable predictor of subclinical cardiac dysfunction.

Image quality is an important determining factor for the correct measurement of VVI parameters. In the CCA, the media-adventitia border should be defined with great caution. There is currently limited evidence-based data on VVI analysis of arterial wall motion to determine the validity of VVI. Further studies are warranted to determine the efficiency of strain imaging modalities in this group of patients.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Marmara University, School of Medicine.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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