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Layer-specific strain analysis of left ventricular myocardium after alcohol septal ablation for hypertrophic obstructive cardiomyopathy

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Abstract

Introduction: We aimed to explore the layer-specific systolic strain of left ventricular (LV) myocardium in patients with hypertrophic obstructive cardiomyopathy (HOCM) before and after alcohol septal ablation (ASA).

The routine 2D (frame rate: >50 Hz) data sets were acquired using GE Vivi7 system for 44 consecutive HOCM patients and 21 matched normal subjects. Fifteen of HOCM patients had serial echocardiograms available for speckle tracking analyses before and 1 year after ASA. 2D strain was analyzed by EchoPAC software.

The layer strain from inner to mid-myocardial and outer layers in basal and middle segments in HOCM patients continuously declined. The absolute values of peak systolic strains from the endocardium to mid-myocardium and epicardium in the basal septum of the HOCM group were significantly lower than those of the normal group ($P<.01$). Meanwhile, the layer systolic strain of LV endocardium in the basal septum increased significantly during a 1-year follow-up ($P<.05$).

Conclusions: The layer-specific strains of HOCM patients measured by tissue Doppler echocardiography decreased significantly compared to those of normal individuals. The increased specific layer strain of LV endocardium in the basal septum may be a valid marker of echocardiographic improvement in HOCM patients receiving ASA.

Abbreviations: ASA = alcohol septal ablation, HOCM = hypertrophic obstructive cardiomyopathy, LV = left ventricular, LVEF = left ventricular ejection fraction, PM = pacemaker, STE = speckle-tracking echocardiography.

Keywords: ablation, cardiomyopathy, echocardiography, endocardium

1. Introduction

Two-thirds of patients with hypertrophic cardiomyopathy have left ventricular (LV) outflow tract obstruction, due to basal septal hypertrophy and elongated mitral leaflet. As the optimal therapy, surgical myectomy has usually been performed to relieve obstruction, and alcohol septal ablation (ASA) was introduced 2 decades ago as an alternative percutaneous technique.[1–3] ASA is an effective short and long-term method for eliminating or reducing LV outflow tract obstruction and relieving the symptoms of patients with hypertrophic obstructive cardiomyopathy (HOCM).[4–6]

Moreover, ASA can reduce LV mass, improve LV diastolic function, and change regional and global LV myocardial longitudinal systolic functions. However, there are 3 layers of myocardium, including endocardium, mid-myocardium and epicardium. The function of the 3 layers after ASA in HOCM patients is not well established.[6]

Strain evaluation using serial speckle tracking echocardiography is an excellent tool for assessing regional and global LV functions.[7–9] The layer-specific longitudinal systolic strain can be used to evaluate the deformations of endocardium, mid-myocardium and epicardium respectively in each LV segment by using 2-dimensional speckle-tracking echocardiography (STE).[10]

The objective of this study was to characterize layer-specific strain using STE to assess LV layer-specific longitudinal systolic myocardial function in patients with HOCM before and after ASA. We hypothesized that the endocardial layer-specific strain in the septum was reduced at baseline and then increased after ASA, which may predict the alleviation of HOCM patients who underwent ASA.

2. Methods

2.1. Study population

Between September 2009 and June 2014, 44 consecutive HOCM patients enrolled in Department of Cardiology, Nanjing First Hospital were selected in this retrospective study. HOCM has been defined according to the 2011 ACCF/AHA guideline for the diagnosis and treatment of hypertrophic cardiomyopathy.[11] Indications for the ASA procedure were drug-refractory symptoms of dyspnea [New York Heart Association (NYHA) functional class III–IV], angina pectoris [Canadian Cardiovascular Society class III–IV], or syncope.[4] All patients had an LV outflow tract pressure gradient of >50 mmHg at rest or after

provocation (following ventricular premature beats provoked during cardiac catheterization). Patients with coronary artery disease, prior septal reduction therapy, and myocardial hypertrophy of other causes were excluded. Fifteen patients who completed 1-year follow-up and had adequate echocardiographic images were included for speckle tracking analysis. All subjects gave informed consent before the procedure. In the meantime, 21 healthy individuals with matched age, gender, heart rate, and blood pressure were enrolled as a control group.

The study was complied with the Declaration of Helsinki and approved by Nanjing First Hospital Committee for Medical Research Ethics (Approval No. NFH-20090904A).

2.2. Alcohol ablation of septal hypertrophy

The procedure was performed as previously described.[12] Briefly, an over-the-wire angioplasty balloon was placed into the septal perforator artery through a 6F or 7F left coronary guide catheter by using a standard method. After balloon inflation, angiographic contrast agent was injected through the balloon catheter to identify the perfusion bed of the septal branch. After delineation of the related septal myocardium, 1 to 3 mL of 95% ethanol solution was injected slowly through the balloon catheter over a period of 3 to 5 minutes, followed by normal saline flush. For some patients who had <50% reduction of either the resting or provoked LV outflow tract pressure gradient, ethanol solution was injected in other septal perforator arteries. A temporary pacemaker (PM) lead was placed in patients without permanent PM. Patients with temporary PM were monitored in an intensive care unit for 5 days after septal ablation. If persistent advanced atrioventricular block occurred thereafter, a permanent dual-chamber PM was implanted.

2.3. Echocardiography

Echocardiographic studies were performed using the criteria of American Society of Echocardiography and a Vivid 7 ultrasound machine (General Electric Co., Milwaukee, WI) with a 2.5 MHz probe before ASA and during 1-year follow-up. Three consecutive beats were registered, and means were used for further analysis. Offline echocardiography data were reanalyzed independently by 2 senior echocardiographers in a blinded fashion using EchoPAC PC software.[13] LV parasternal short-axis views at basal, mid-ventricular, and apical levels were acquired (frame rate: 56~92 frames/s). Maximal wall thickness was measured from all LV segments in the parasternal short-axis view. Asymmetric septal hypertrophy was defined as septal to posterior free wall ratio of >1.4. LV end-diastolic diameter and LV end-systolic diameter were evaluated by M-mode or 2D imaging. LV ejection fraction (LVEF) was determined using apical 4- and 2-chamber views by the modified Simpson's biplane method. LV diastolic function was calculated by trans-mitral pulsed Doppler and average e' from septal and lateral tissue Doppler images. Left atrial diameter was determined by M-mode or 2D echocardiography in the parasternal long-axis plane and left atrial area or volume was calculated as the average end-systolic area or volume from apical 4- and 2-chamber views. LV outflow tract pressure gradients were assessed only at rest, and a pressure gradient of ≥30 mmHg was defined as significant obstruction.

2.4. Strain and strain rate analysis

A dedicated software package (EchoPAC Dimension, GE Healthcare, Horten, Norway) was applied for strain and strain rate analysis by tracing the LV endocardial contour in end-systolic frames.[14] Myocardial markers (acoustic speckles) were identified and followed frame-to-frame within consecutive 2-dimensional echocardiographic images and optimized by manual adjustment in accordance with the surrounding tissue motion. Segments that could not be tracked were excluded. Region of interest was adjusted to fit the average of myocardial thicknesses. Subsequently, the software automatically defined the myocardium, processed all frames of the loop and demonstrated the results. LV global longitudinal strain was defined as the average of peak longitudinal strains in a 16-segment mode.

Based on the automatic definition of endocardial and epicardial contours, 3 layers (an endocardial, a mid-myocardial, and an epicardial layer) were automatically defined with the system by dividing the wall thickness into 3 layers of similar thicknesses.[15] Layer-by-layer longitudinal strains were automatically obtained from the apical long-axis slices (2- and 4-chamber long-axis views). The peak systolic strain and strain rate within 3 separate myocardial layers of each LV segment were automatically calculated using a medium degree of spatial and temporal smoothing. All segmental values were averaged to produce a GLS for each myocardial layer and the whole myocardium. End-systole was defined as aortic valve closure in the apical axis view and transferred to all other views.

2.5. Statistical analysis

Continuous quantitative variables were represented as mean ± standard deviation, and categorical data were expressed as frequency and percentage. In the case of normal distribution, differences between groups were compared using the Student t test. Otherwise, a non-parametric test (Mann–Whitney) was used. Differences between groups were compared with the Kruskal–Wallis test for continuous data and the Chi-square test for categorical data. Categorical variables were analyzed using the Fisher exact test. Data analysis was performed using SPSS 19.0 software (IBM, Armonk, NY). Statistical significance was set at a level of $P < .05$.

3. Results

3.1. Baseline characteristics and echocardiographic findings of normal and HOCM patients

The 44 HOCM patients included 18 females and 26 males with a mean age of (40 ± 9) years old, and the normal individuals comprised 7 females and 14 males with a mean age of (36 ± 8) years old (Table 1). All subjects had normal LVEF, and LVEF of the HOCM group was significantly higher than that of the normal group [(65 ± 15) vs $(72 \pm 6)\%$, $P < .05$]. The interventricular septum thickness, maximal wall thickness, maximum and minimum left atrial areas, as well as maximum and minimum, left atrial volumes of the HOCM group exceeded those of the normal group.

3.2. Layer-specific strain analysis by speckle tracking echocardiography

The baseline segmental and average LV longitudinal systolic 2D myocardial strains from apical 4-chamber, 2-chamber and long-axis views are presented in Table 2. For the HOCM group, the global strain and layer strain continuously declined from inner to mid-myocardial and outer layer in basal and middle segments,

Baseline characteristics and conventional echocardiographic findings of normal and HOCM subjects.

	Normal (n=21)	HOCM (n=44)	P value
Age, year	36±8	40±9	.132
Male gender, %	14 (66.7)	26 (65.0)	.896
HR, bpm	69.48±9.99	71.62±11.99	.484
LV end-diastolic diameter, mm	45.25±3.7	45.02±6.77	.89
LV end-systolic diameter, mm	27.15±3.23	26.36±4.02	.444
LVEF, %	65.15±14.78	71.83±6.25	<.05
Interventricular septum thickness, mm	8.57±1.16	18.9±7.5	<.01
Posterior wall thickness, mm	9.14±1.42	11.83±3.42	<.01
Interventricular septum/posterior wall ratio	0.95±0.08	1.66±0.7	.162
Maximal wall thickness, mm	9.76±0.94	23.96±8.25	<.01
Left atrial diameter, mm	29.82±3.89	44.55±7.18	<.01
Maximum Left atrial area, cm^2	15.46±2.64	25.74±7.11	<.01
Minimum Left atrial area, cm^2	7.52±2.11	17.2±7.04	<.01
Maximum left atrial volume, mL	41.9±11.25	94.1±37.39	<.01
Minimum left atrial volume, mL	12.95±5.54	49.2±33.88	<.01

HOCM=hypertrophic obstructive cardiomyopathy, HR=heart rate, LV=left ventricular, LVEF=left ventricular ejection fraction.

Layer-specific strain analysis by speckle tracking echocardiography from apical 4-chamber, 2-chamber and long-axis views of normal and HOCM subjects.

	Normal (n=21)	HOCM (n=44)	P value
Total left atrial strain, %	−20.43±3.79	−6.83±4.19	<.01
4-chamber GS	−20.03±2.87	−13.45±4.83	<.01
4-chamber endocardial layer, %/sec	−22.04±2.88	−15.31±5.55	<.01
4-chamber mid-myocardial layer, %/sec	−19.77±2.81	−13.46±4.83	<.01
4-chamber epicardial layer, %/sec	−17.81±2.81	−11.78±4.28	<.01
GS endo BaseSept, %	−17.48±3.28	−6.88±5.91	<.01
GS endo midSept, %	−21.67±2.59	−11.49±6.98	<.01
GS endo apSept, %	−28.67±5.25	−24.8±11.54	.151
GS endo apLat, %	−25±6.35	−20.54±10.34	.076
GS endo midLat, %	−19.24±4.33	−12.27±5.94	<.01
GS endo BaseLat, %	−18.29±5.09	−11.73±6.09	<.01
2-chamber GS, %	−19.71±3.21	−13.86±4.99	<.01
2-chamber endocardial layer, %/sec	−22.04±3.72	−15.6±5.28	<.01
2-chamber mid-myocardial layer, %/sec	−19.71±3.21	−13.73±4.86	<.01
2-chamber epicardial layer, %/sec	−17.73±2.82	−12.16±4.41	<.01
GS endo BaseInf, %	−21.29±3.27	−14.58±7.3	<.01
GS endo midInf, %	−23.14±3.57	−13.61±5.09	<.01
GS endo apInf, %	−29.29±6.5	−22.06±10.46	<.01
GS endo apAnt, %	−22.43±8.19	−21.94±11.81	.869
GS endo midAnt, %	−18.1±5.34	−12.25±6.95	<.05
GS endo BaseAnt, %	−19.67±4.82	−8.53±5.31	<.01
3-chamber GS	−19.46±3.4	−14.34±4.59	<.01
3-chamber endocardial layer, %/sec	−21.72±3.83	−16.56±5.39	<.01
3-chamber mid-myocardial layer, %/sec	−19.64±2.88	−14.36±4.61	<.01
3-chamber epicardial layer, %/sec	−17.81±2.39	−12.48±4.04	<.01
GS endo BasePost, %	−20.9±6.83	−17.13±7.15	.054
GS endo midPost, %	−21.81±4.69	−13.29±5.9	<.01
GS endo apPost, %	−27.19±8.66	−20.53±9.47	<.05
GS endo apAntSept, %	−26.1±8.31	−25.39±10.91	.799
GS endo midAntSept, %	−20.52±4.02	−14.61±9.2	<.01
GS endo BaseAntSept, %	−17.62±4.96	−7.84±6.66	<.01

Ant=anterior wall, ap=apical segment, Base=basal segment, GS endo=global strain of endocardium, GS=global strain, HOCM=hypertrophic obstructive cardiomyopathy, Inf=inferior wall, Lat=lateral wall, mid=middle segment, Post=posterior wall, Sept=septum.

except for in apical segment. The absolute values of layer peak systolic strains from endocardium to mid-myocardium and epicardium in the basal septum of the HOCM group were significantly lower than those of the normal group [(−6.88± 5.91) vs (−17.48±3.28)%, $P<.01$; (−7.56±6.1) vs (−17.52± 2.91)%, (−7.9±5.24) vs (−17.81±2.84)%, respectively, $P<.01$]. The layer strain of endocardium in the basal septal segment was lower than those in other segments. Figure 1 shows the layer-specific strain analysis by speckle tracking echocardiography.

3.3. Baseline characteristics and conventional echocardiographic measurements of HOCM patients receiving ASA

The baseline characteristics of 15 of 44 HOCM patients receiving ASA are listed in Table 3. The mean age was (39±8) years old, and 10 patients (66.7%) were males. All HOCM patients were optimally treated with β blockers, calcium channel blockers, or a combination of these drugs, and subjected to continued medical treatment during follow-up. None of the patients had received surgical myectomy or PM implantation before.

The interventricular septum thickness decreased after ASA, without a significant difference though. In contrast, the interventricular septum/posterior wall ratio plummeted. One year after ASA, systolic velocity S', early diastolic velocity E' and late diastolic velocity A' by tissue Doppler imaging showed a decreasing tendency in septal LV segments, inferring the improvement of LV diastolic function (Table 4).

3.4. Layer-specific strain analysis by speckle tracking echocardiography at baseline and 1 year after ASA

The baseline segmental and average LV strains from apical 4-chamber, 2-chamber and long-axis views are summarized in Table 5. After ASA, the LV longitudinal strain was elevated from basal to apical LV segments. Meanwhile, the layer strain of LV endocardium in the basal septum significantly increased during 1-year follow-up [(−8.29±4.81) vs (−4.85±4.18)%, $P<.05$].

4. Discussion

In this study, we found that the layer strains from inner to mid-myocardial and outer layer in basal and middle myocardium segments were lower in the HOCM group than in the normal group. For the HOCM group, the absolute value of layer strain in basal endocardium segment was significantly lower than those of other segments. Besides, the layer peak systolic strain of LV endocardium in the basal septum increased during 1-year follow-up.

The longitudinal systolic myocardial strain can reflect the myocardial function of any patients. Previous studies have demonstrated that such strain significantly reduced in symptomatic or non-symptomatic HOCM patients retaining LV function.[15–17]

We herein used speckle tracking echocardiography to analyze the deformation of endocardial, mid-myocardial and epicardial layers.[18] The strains of the 3 layers demonstrated a significant pressure gradient, with the greatest deformation in the endocardial layer. The gradient reflects differences between the functions of endocardial and epicardial layers. However, this study showed that the absolute layer strain of endocardium in basal segment was significantly lower than those of other segments in both groups, especially in the HOCM group. Veselka
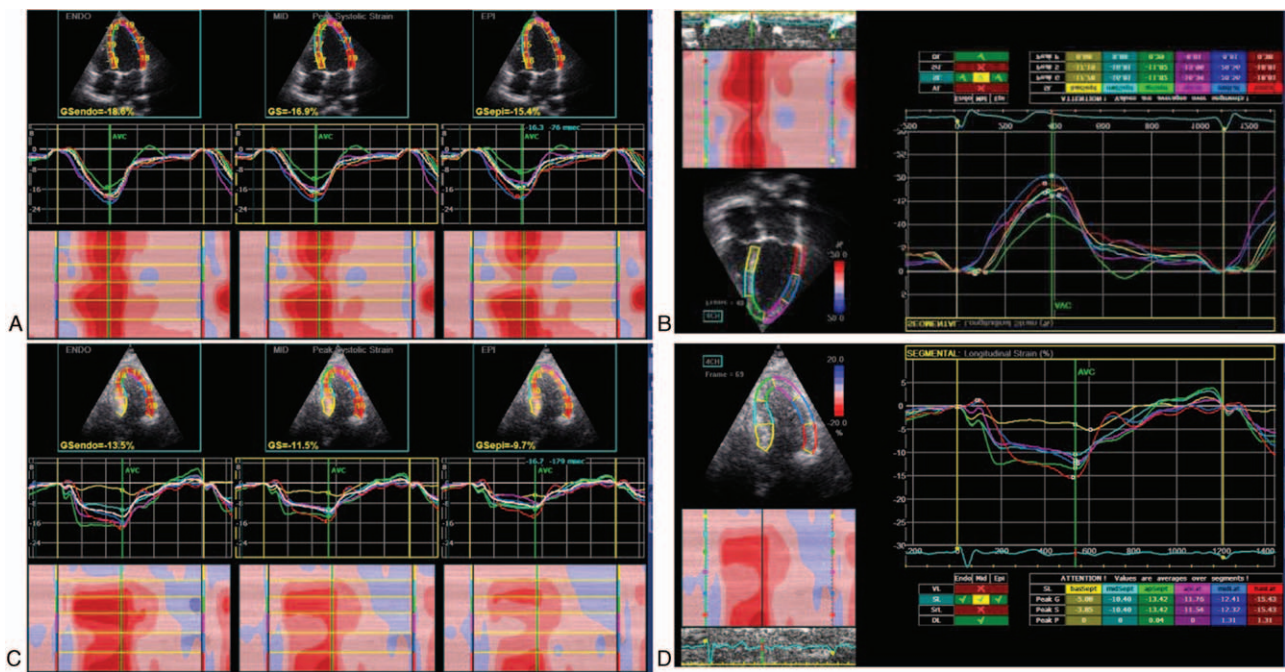
Figure 1. Layer-specific strain analysis of a HOCM patient by speckle tracking echocardiography from apical 4-chamber view compared to that of a normal individual. A: Layer-specific strain analysis of a normal individual; B: longitudinal strain in a normal individual; C: layer-specific strain analysis of a HOCM patient; D: longitudinal strain in a HOCM patient. HOCM = Hypertrophic obstructive cardiomyopathy.

et al reported that substantial reduction of the LV outflow tract pressure gradient after ASA was not accompanied by significant improvement of the average LV longitudinal systolic deformation.[6] They further proved that the longitudinal systolic strain increased regionally in the basal segments of the myocardium remote from the target area of alcohol ablation during a 3-year follow-up. Regardless, the differences between the strains of each layer in HOCM patients before and after ASA remain largely unknown.

Baseline characteristics of HOCM subjects (n = 15).

Variable	Value
Age, year	39 ± 8
Male gender, n (%)	10 (66.7)
NYHA class I, n (%)	3 (20)
NYHA class II, n (%)	6 (40)
NYHA class III or IV, n (%)	6 (40)
Syncope, n (%)	2 (13.3)
Chest Pain, n (%)	7 (46.7)
Dyspnea, n (%)	5 (33.3)
Family history of HCM, n (%)	1 (6.7)
Systolic blood pressure, mmHg	128 ± 9
Diastolic blood pressure, mmHg	74 ± 9
Sustained VT/VF, n (%)	4 (26.7)
Use of beta-blockers, n (%)	13 (86.7)
Use of calcium channel blockers, n (%)	4 (26.7)
Use of anti-arrhythmia drugs, n (%)	4 (26.7)
Use of ICD, n (%)	0

HOCM = hypertrophic obstructive cardiomyopathy, ICD = implantable cardioverter defibrillator, NYHA = New York Heart Association, VF = ventricular fibrillation, VT = ventricular tachycardia.

Non-transmural infarction can be accurately differentiated from non-infarction by using deformation analysis of the endocardial layer.[18,19] ASA can mitigate LV obstruction by injecting a small amount of 95% ethanol solution into an appropriate septal branch of the left anterior descending artery, followed by basal septum necrosis and shrinkage. Until now, the risk/benefit of ASA in comparison with surgical myectomy is still elusive, and the endocardial or transmural myocardial function has seldom been tested. In this study, ASA indeed exerted therapeutic effects on local transmural myocardial contraction. Thus, septum layer strain may be 1 of the most important echocardiographic determinants of global LV longitudinal systolic function in HOCM patients, and 1 of the most crucial predictors of LV functional improvement after ASA.

This study has some limitations. First, a small number of HOCM patients, particularly those after ASA, were enrolled, because we excluded the patients without high-quality 2D echocardiographic images for speckle tracking analysis and 1-year follow-up. However, the myocardial global strain and layer strain of the HOCM group had already shown a significant increasing tendency, which can be considered as the predictors of LV function improvement for the patients receiving ASA. We hypothesized that the layer strains of endocardium in the basal septum of HOCM and normal groups decreased by $(17.48 \pm 3.28)\%$ and $(6.88 \pm 5.91)\%$ respectively. Accordingly, a total of 10 patients were needed to detect a power of 0.8 (Type II error = 0.2, $\alpha = 0.05$, 2-tailed). Because of the considerable uncertainty of patients lost during follow-up, the enrollment was enlarged to 11 patients (10% increment). Therefore, the 65 patients enrolled in this study were enough to estimate the layer strain of endocardium in the basal septum of HOCM patients. Second, this study was a nonrandomized and single-center retrospective trial which was mainly related to

Table 4

Conventional echocardiographic findings at baseline and 1 year after ASA of HOCM subjects (n=15).

Characteristics	Baseline	1-Year follow-up	P value
HR, bpm	68.79±7.9	72.14±6.59	.233
LV end-diastolic diameter, mm	46.14±5.83	47.64±5.57	.492
LV end-systolic diameter, mm	26.07±3.43	29.43±5.56	.065
LVEF, %	74.07±4.71	68.21±9.32	<.05
Interventricular septum thickness, mm	18.29±7.97	14.29±4.75	.119
Posterior wall thickness, mm	11.79±2.42	12.57±3.69	.512
Interventricular septum/posterior wall ratio	1.54±0.58	1.09±0.46	<.05
Maximal wall thickness, mm	26.85±8.06	21.86±8.2	.124
Left atrial diameter, mm	44±4.16	44.71±5.15	.697
Maximum Left atrial area, cm^2	26.25±4.74	28.61±16.2	.619
Minimum Left atrial area, cm^2	19.68±7.2	16.42±6.87	.24
Maximum left atrial volume, mL	95.46±26.2	76.69±33.93	.122
Minimum left atrial volume, mL	53.48±35.92	43.93±30.65	.463
Right ventricular diameter, mm	29.31±5.48	28.57±3.34	.675
LVOT max, m/s	3.19±1.24	2.48±1.16	.14
LVOT pressure gradient, mmHg	46.35±38.75	29.75±28.92	.217
AVI, cm	24.43±11	17.04±3.83	.131
MR (grading, 1–4)	1.63±0.52	1.5±0.67	.663
Mitral E velocity, m/s	0.6±0.16	0.67±0.24	.344
Mitral A velocity, m/s	0.78±0.23	0.75±0.22	.73
E/A ratio	0.78±0.41	0.91±0.72	.565
E deceleration time, ms	248.38±75.3	265.08±93.19	.62
Lateral E′, cm/s	16.1±2.5	11.1±2.1	<.05
Lateral A′, cm/s	16.9±2.8	8.0±4.0	.058
Lateral E/E′	9.21±10.94	11.94±7.37	.445
Septal S′, cm/s	13.7±2.19	6.0±2.0	<.05
Septal E′, cm/s	10.5±1.75	4.0±2.0	<.05
Septal A′, cm/s	14.9±2.27	13.3±2.3	.051
Septal E/E′	10.41±11.41	12.42±9.9	.622
IVCT, ms	93.54±23.21	111±30.46	.119
IVRT, ms	114.62±25.64	129±24.31	.164
ET, ms	259.31±39.31	259.92±37.68	.969

A′ = late diastolic velocity by tissue Doppler imaging, ASA = Alcohol septal ablation, AVI = aortic velocity integral, E′ = early diastolic velocity by tissue Doppler imaging, ET = ejection time, HOCM = hypertrophic obstructive cardiomyopathy, HR = heart rate, IVCT = isovolumetric contraction time, IVRT = isovolumetric relaxation time, LV = left ventricular, LVEF = left ventricular ejection fraction, LVOT = left ventricular outflow tract, MR = mitral regurgitation, S′ = systolic velocity by tissue Doppler imaging.

Table 5

Layer-specific strain analysis by speckle tracking echocardiography from apical 4-chamber, 2-chamber and long-axis views at baseline and 1 year after ASA of HOCM subjects (n=15).

Characteristics	Baseline	1-year follow-up	P value
Total left atrial strain, %	−5.85±3.19	−7.58±3.37	.254
4-chamber GS	−11.45±4.95	−13.96±6.22	.258
4-chamber endocardial layer, %/sec	−13.32±5.7	−16.37±7.07	.23
4-chamber mid-myocardial layer, %/sec	−11.45±4.95	−13.98±6.22	.255
4-chamber epicardial layer, %/sec	−9.82±4.22	−12.05±5.53	.253
GSendo BaseSept, %	−4.85±4.18	−8.29±4.81	<.05
GSendo midSept, %	−8.23±5.78	−12.43±7.42	.115
GSendo apSept, %	−22.62±12.98	−27.57±12.36	.319
GSendo apLat, %	−17.85±11.04	−23.07±9.16	.191
GSendo midLat, %	−10.92±6.34	−12.14±5.82	.607
GSendo BaseLat, %	−10.31±6.52	−11.93±8.33	.581
2-chamber GS, %	−12.48±6.04	−12.05±5.44	.853
2-chamber endocardial layer, %/sec	−13.72±5.91	−13.95±5.99	.922
2-chamber mid-myocardial layer, %/sec	−12.1±5.58	−12.05±5.46	.98
2-chamber epicardial layer, %/sec	−10.76±4.89	−10.5±4.81	.892
GS endo BasInf, %	−13.54±8.3	−13.15±5.24	.889
GS endo midInf, %	−12.38±5.85	−12.31±4.35	.97
GS endo apInf, %	−18.62±11.37	−19.38±10.15	.857
GS endo apAnt, %	−17.23±12.13	−20.23±12.17	.535
GS endo midAnt, %	−9.46±7.07	−10.77±7.14	.643
GS endo BaseAnt, %	−8.38±7.54	−6.46±3.82	.42
3-chamber GS	−11.65±4.3	−12.99±5.4	.488
3-chamber endocardial layer, %/sec	−13.52±5.36	−15.31±6.47	.451
3-chamber mid-myocardial layer, %/sec	−11.65±4.3	−12.99±5.4	.488
3-chamber epicardial layer, %/sec	−10.05±3.63	−11.06±4.46	.534
GS endo BasPost, %	−14.85±7.95	−13.38±7.6	.636
GS endo midPost, %	−9.92±4.59	−10.38±5.16	.812
GS endo apPost, %	−16.08±8.48	−19.62±9.29	.32
GS endo apAntSept, %	−20.23±11.77	−25.23±9.88	.252
GS endo midAntSept, %	−10.69±9.52	−13.69±9.08	.419
GS endo BaseAntSept, %	−5.85±7	−7.15±8	.661

Ant = anterior wall, ap = apical segment, ASA = Alcohol septal ablation, Base = basal segment, GS endo = global strain of endocardium, GS = global strain, HOCM = hypertrophic obstructive cardiomyopathy, Inf = inferior wall, Lat = lateral wall, mid = middle segment, Post = posterior wall, Sept = septum.

echocardiographic indices. Future prospective studies will require a systematic protocol to assess the long-term relationship between the changes of myocardial strain and other clinical outcomes such as B-type natriuretic peptide and obstruction pressure gradient.

In summary, the layer-specific strains of HOCM patients measured by tissue Doppler echocardiography decreased significantly compared to those of normal individuals. The increased specific layer strain of LV endocardium in the basal septum may be a valid marker of echocardiographic improvement in HOCM patients receiving ASA.

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