Early Detection of Regional and Global Left Ventricular Myocardial Function Using Strain and Strain-rate Imaging in Patients with Metabolic Syndrome

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Abstract

Background: Strain and strain-rate imaging (SRI) have been found clinically useful in the assessment of cardiac systolic and diastolic function as well as providing new insights in deciphering cardiac physiology and mechanics in cardiomyopathies, and identifying early subclinical changes in various pathologies. The aim of this study was to evaluate the regional and global left ventricular (LV) myocardial function in metabolic syndrome (MS) with SRI so that we can provide more myocardial small lesions in patients with MS, which is robust and reliable basis for early detection of LV function.

Methods: Thirty-nine adults with MS were enrolled in the study. There was a control group of 39 healthy adults. In addition to classic echocardiographic assessment of LV global functional changes, SRI was used to evaluate regional and global LV function. Including: Peak systolic strain (S), peak systolic strain-rate (SR-s), peak diastolic strain-rate (SR-e).

Results: There were no statistically significant differences between MS and controls in all traditional parameters of LV systolic function. On the other hand, significant differences were observed between MS and the control group in most of the parameters of S, SR-s, SR-e in regional LV function. Multiple stepwise regression analyses revealed that S and SR significantly were negatively correlated with blood pressure, waist circumference, fasting plasma glucose, uric acid, suggesting that risk factories were relevant to regional systolic dysfunction.

Conclusion: In MS with normal LV ejection fraction, there was regional myocardial dysfunction, risk factors contributed to the impairment of systolic and diastolic function of the regional myocardium. Assessment of myocardial function using SRI could be more accurate in MS patient evaluation than conventional echocardiography alone.

Key words: Metabolic Syndrome; Regional Left Ventricular Function; Strain and Strain-rate Imaging

INTRODUCTION

Metabolic syndrome (MS) is a condition characterized by the accumulation of multiple risk factors (insulin resistance, hyperglycemia, dyslipidemia, hypertension, visceral obesity) for cardiovascular disease in an individual with a background of obesity and/or lack of exercise.[1] Even more with the change of the modern lifestyle and diet structure, incidence of MS increased year by year, it is greatly endangering people's health. However, it is not known whether MS is also associated with abnormal cardiac function. If MS indicates persons who have already developed abnormal left ventricular (LV) function, early recognition of MS would be important. Several studies have shown that sub-clinical diastolic dysfunction is evident in most patients with obesity and MS with either increased or no interval change in LV systolic function at rest. A study by Sasso *et al.*[2] demonstrated a reduced increase in ejection fraction (EF) in obese subjects during dynamic exercise using radionuclide ventriculography; however,[3] EF is a load-dependent measurement of systolic function that may not accurately reflect the subtle changes of early myocardial dysfunction.

Strain and strain-rate imaging (SRI) are a new noninvasive method for assessment of myocardial function. SRI has been found clinically useful in the assessment of cardiac systolic and diastolic function as well as providing new insights in deciphering cardiac physiology and mechanics in cardiomyopathies, and identifying early sub-clinical changes in various pathologies. Due to its ability to differentiate

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between active and passive movement of myocardial segments, to quantify components of myocardial function, such as longitudinal myocardial shortening, that are not visually assess-able, it allows comprehensive assessment of myocardial function and the spectrum of potential clinical applications is very wide. Strain (S) and strain-rate (SR) data are helpful for therapeutic decisions and also useful for follow-up evaluations of therapeutic results in cardiology. S and SR data also provide valuable prognostic information, especially prediction of future reverse remodeling in myocardial small lesions.[4,5] Moreover, it seems that the regional dysfunction can be detected earlier than global dysfunction.[6]

The review explains the fundamental concepts of deformation imaging, describes in a comparative manner the two major deformation imaging methods (tissue Doppler imaging [TDI]-derived and two-dimensional [2D] speckle tracking, 2D-strain derived). Stefani et al.[7] reported that the strain can be computed in athletes by both TDI-derived and 2D-strain derived methods. The goal of our study was to assess LV regional function in patients with MS by TDI-derived imaging methods so that we can provide more myocardial small lesions in patients with MS, which is robust and reliable basis for early detection of LV function.

METHODS

Subjects and methodology

Thirty-nine adults with MS were enrolled in the study. All were recruited from the out-patient cardiology, diabetes and general medicine clinics of General Hospital of Ningxia Medical University from December 2010 to August 2012. Thirty-nine healthy adults with no MS and matched for age and gender comprised the control group. A diagnosis of MS was assigned according to the International Diabetes Federation in 2005 which is fit for Asians MS diagnostic criteria: Waist circumference (W. circum.) 90/80 cm (men/women) plus any two of the following four factors: Increased triglyceride level l150 mg/dL or a specific treatment for this lipid abnormality; reduced high-density lipoprotein cholesterol <40/50 mg/dL (men/women) or a treatment specific for this lipid abnormality; raised blood pressure (BP): Systolic BP (SBP) ≥130 or diastolic BP (DBP) ≥85 mmHg or treatment of previously diagnosed hypertension; raised fasting plasma glucose (FPG) ≥100 mg/dL or treatment of previously diagnosed type two diabetes.[8,9]

Exclusion criteria were the following: Chronic kidney disease; a history or findings of cardiovascular disease including heart failure symptoms or systolic dysfunction; coronary artery disease (through exercise testing); significant valvular heart disease (i.e., greater than mild valvular insufficiency, or stenosis) and/or hypertrophic cardiomyopathy; pregnancy or lactation; and/or major systemic illness.

All groups had a full medical history and clinical examination including BP measurement, anthropometric measures, systemic examination, biochemical tests including lipid profile, FPG and echocardiographic studies. W. circum., a validated estimate of visceral adiposity, was measured to the nearest 0.5 cm.[10] Systemic hypertension was defined according to the Joint National Committee VII criteria, as a BP > 140/90 mmHg and/or under current antihypertensive therapy.[11]

Echocardiographic evaluation M-mode and 2D echocardiographic studies were performed with a GE phased array ultrasonoscope (VIVID-7 dimension, USA) using a 3.5 MHz transducer.

Parameters of left ventricular structure

Left ventricular dimensions (systolic diameter and diastolic diameter) were measured at end diastole (R wave of electrocardiogram) and end systole (maximum posterior motion of septum) and were indicated by d and s, respectively. All were detected in the parasternal long-axis view during M-mode tracing according to the recommendation of the American Society of echocardiography.[12]

Parameters of systolic function

Left ventricular end-diastolic and end-systolic volumes (EDV and ESV) were calculated according to Abraham et al.; stroke volume was calculated as the difference between EDV and ESV; EF was calculated as EF% =100 × (EDV − ESV)/EDV.
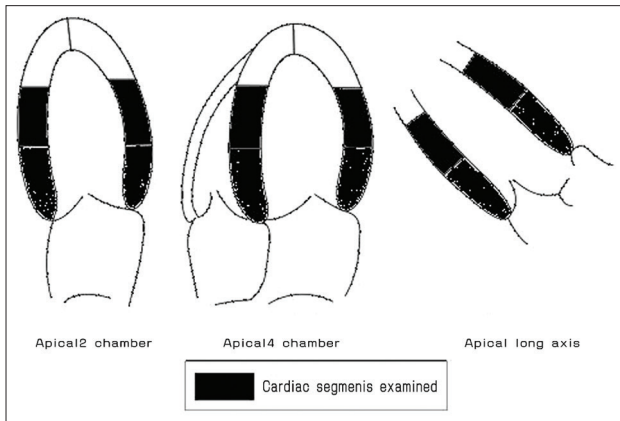
Parameters of diastolic function

Assessment of diastolic function was obtained by pulsed-wave Doppler of transmitral flow patterns recorded in the apical four-chamber view where the sample volume was best positioned in the LV at the tips of the valve leaflets. Mitral flow early-phase filling velocity (E), peak atrial phase filling velocity (A), and E/A ratio were measured.

Parameters of strain-rate imaging

Myocardial S and SR was derived from TDI. S and SR were derived from S and SR curves[13] obtained by placing a sample bar (10 mm) in the basal and middle segments of the septal, lateral, inferior, anterior and posterior walls from the apical four-chamber [Figure 1], two-chamber and long axis views. S and SR curves were extracted from color TDI by standard software (GE Vingmed). SR data were recorded from the basal and middle segments, using standard apical views at a high frame rate (>100 frames/s). The region of interest [Figure 2] inter-ventricular septum was constant at 5 mm^2 during the whole trial and was tracked automatically throughout the systole. The basal and mid-myocardial layer were sampled in each segment and maintained at the same position during the cardiac cycle by manually tracking wall motion [Figure 2], but we excluded data if we were unable to obtain a smooth strain curve or the angle between the scan line and wall was >20°. Peak systolic strain was defined as the greatest value on the strain curve, and peak systolic strain-rate (SR-s) was measured from the strain curve as previously validated. At the same time SR-s, peak early and late diastolic strain-rate (SR-e and SR-a), and peak systolic strain from the same sample volume within the same cardiac cycle. SR data were averaged from 4 to 6 cycles.[14]

Peak systolic strain-rate represents the maximal rate of deformation in systole, systolic strain represents the

Figure 1: Cardiac segments examined with strain-rate imaging.

Figure 2: Strain and strain-rate imaging from the mid-inferoseptal myocardial segment from a study subject. Peak systolic strain is the peak percentage long-axis shortening; peak systolic strain-rate is the peak rate of shortening; peak diastolic strain-rate is the peak rate of length.

magnitude of deformation between end diastole used as a reference point and end systole.

Ethics

Informed consent was obtained from all adults. The aim and the value of the work were explained in a simplified manner for them. There was no harm inflicted on them. The study was approved by the Ethics Committee of Faculty of General Hospital of Ningxia Medical University.
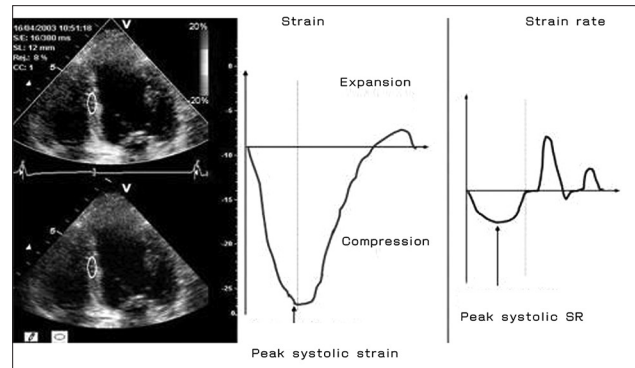
Statistical analysis

The analysis was carried out by a computer program (SPSS Version 17, SPSS Inc., IL, USA). Descriptive data are reported as mean ± standard deviation. According to the type of data, the Student's unpaired t-test was used for statistical comparisons between two groups. The P value was set at <0.05 for statistically significant results. Multiple regression analysis was utilized to assess the relationships between risk factories and S or SR. To determine the reproducibility of the S and SR derived from TDI, S and SR analyses of ten subjects were repeated by an additional investigator and by the same primary reader 1 day later. During these repeated analyses, the investigators were blinded to the results of both prior measurements.

RESULTS

No significant difference was observed among the groups regarding age, sex, heart rate in Table 1. There were significant differences in W. circum., BP, FPG, cholesterol, low density lipoprotein, uric acid (UA) and triglycerides between MS and the control groups.

Echocardiographic Doppler data are shown in Table 2. There were no statistically significant differences between MS and controls in all parameters of LV structural and systolic function exclude diastolic function.

Strain and SR date were possible in 100% of the 900 attempted segments from the 78 echocardiographic studies with technically adequate images. S and SR data shown in Table 3. Compared with controls, patients with MS had significantly impaired resting S ($P < 0.000$) and SR-s ($P < 0.000$), SR-e ($P < 0.000$), SR-a ($P < 0.05$).

Table 1: Clinical and biochemical characteristics of MS and control groups

Characteristics	n = 39		t	P
	MS	Controls		
Age (year)	50.90 ± 4.37	50.35 ± 5.32	0.421	0.676
Sex (male/female)	22/17	19/17	0.541	0.751
Heart rate (bmp)	74.08 ± 5.76	72.11 ± 4.88	0.403	0.534
SBP (mmHg)	157.9 ± 18.78	116.27 ± 10.07	8.908	0.000
DBP (mmHg)	99.17 ± 9.61	73.23 ± 6.97	9.352	0.000
W. circum. (cm)	91.93 ± 3.81	77.00 ± 4.01	15.170	0.000
Cholesterol (mmol/L)	4.61 ± 1.35	3.60 ± 0.53	3.610	0.001
HDL (mmol/L)	0.96 ± 0.23	0.99 ± 0.25	−0.491	0.628
LDL (mmol/L)	2.99 ± 0.93	2.25 ± 0.42	3.897	0.000
Triglyceride (mmol/L)	3.66 ± 2.05	1.60 ± 0.53	4.110	0.000
FPG (mmol/L)	9.78 ± 3.81	4.84 ± 0.50	5.353	0.000
UA (μmol/L)	341.7 ± 82.47	257.46 ± 66.53	4.093	0.000

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; W. circum.: Waist circumference; HDL: High-density lipoprotein cholesterol; LDL: Low-density lipoprotein cholesterol; FPG: Fasting plasma glucose; UA: Uric acid; MS: Metabolic syndrome. P: Comparison between MS and controls.

Table 2: Conventional echocardiographic data in MS and control groups

Characteristics	n = 39		t	P
	MS	Controls		
End DD (mm)	49.00 ± 3.20	46.69 ± 2.94	2.78	0.089
End SD (mm)	30.34 ± 2.50	28.85 ± 2.68	1.089	0.285
EF (%)	67.65 ± 3.62	67.96 ± 4.03	0.244	0.809
FS (%)	37.93 ± 2.91	38.00 ± 3.50	0.378	0.709
E (m/s)	0.69 ± 0.15	0.88 ± 0.12	−3.92	0.001
A (m/s)	0.89 ± 0.17	0.61 ± 0.10	5.82	0.000
E/A ratio	0.81 ± 0.21	1.47 ± 0.23	−7.51	0.000

End SD: End systolic diameter; End DD: End diastolic diameter; EF: Ejection fraction; E: E velocity; A: A velocity; MS: Metabolic syndrome. P: Comparison between MS and controls.

In multiple regression analysis, regarding the separate contribution to mid-aivs longitudinal peak strain in the four-chamber view of each parameter among all risk factors

Table 3: Strain and strain-rate data in MS and control groups

Segments	MS				Controls			
	S	SR-s	SR-e	SR-a	S	SR-s	SR-e	SR-a
Bas-aivs	−22.00 ± 3.88	−1.37 ± 0.39	1.73 ± 0.68	1.88 ± 0.95	−27.42 ± 6.11#	−2.26 ± 1.16*	3.04 ± 1.11※	1.80 ± 0.80&
Bas-lat	−20.05 ± 5.15	−1.46 ± 0.44	1.78 ± 0.81	1.51 ± 0.79	−26.86 ± 9.10#	−1.98 ± 1.02*	2.72 ± 1.09※	1.84 ± 0.88&
Mid-aivs	−21.41 ± 4.56	−1.56 ± 0.37	1.72 ± 0.45	1.74 ± 0.43	−28.53 ± 3.35#	−1.65 ± 0.78*	2.69 ± 0.54※	2.09 ± 0.31&
Mid-lat	−14.64 ± 3.82	−0.99 ± 0.29	1.34 ± 0.51	1.12 ± 0.48	−24.40 ± 7.40#	−2.12 ± 1.28*	2.65 ± 1.09※	1.56 ± 0.74&
Bas-ant	−17.91 ± 8.37	−1.18 ± 0.61	1.27 ± 0.54	1.45 ± 0.80	−27.02 ± 6.18#	−2.36 ± 0.88*	2.69 ± 0.94※	1.68 ± 0.73&
Bas-inf	−19.83 ± 5.25	−1.31 ± 0.35	1.55 ± 0.67	1.53 ± 0.56	−26.36 ± 8.02#	−2.22 ± 0.88*	2.66 ± 0.84※	1.75 ± 0.65&
Mid-ant	−14.03 ± 4.18	−0.89 ± 0.30	1.15 ± 0.49	0.90 ± 0.41	−23.41 ± 8.10#	−2.06 ± 0.90*	2.45 ± 1.02※	1.50 ± 0.57&
Mid-inf	−19.06 ± 5.69	−1.23 ± 0.48	1.42 ± 0.55	1.46 ± 0.56	−27.18 ± 7.28#	−2.06 ± 0.69*	2.62 ± 0.93※	1.59 ± 0.63&
Bas-bivs	−18.54 ± 6.59	−1.26 ± 0.55	1.48 ± 0.93	1.37 ± 0.48	−26.81 ± 7.65#	−2.37 ± 0.95*	2.65 ± 1.10※	1.69 ± 0.77&
Bas-post	−19.25 ± 7.64	−1.61 ± 0.79	1.75 ± 0.81	1.48 ± 0.79	−25.97 ± 5.97#	−2.11 ± 0.87*	2.48 ± 1.04※	1.65 ± 0.73&
Mid-bivs	−16.96 ± 4.29	−1.10 ± 0.49	1.26 ± 0.45	1.33 ± 0.56	−25.40 ± 6.30#	−2.18 ± 0.91*	2.21 ± 0.75※	1.55 ± 0.54&
Mid-post	−18.16 ± 6.54	−1.37 ± 0.95	1.38 ± 0.71	1.05 ± 0.47	−26.86 ± 9.21#	−2.29 ± 1.10*	2.49 ± 0.93※	1.61 ± 0.82&

S ($^{#}P<0.000$) and SR-s (*$P<0.000$), SR-e (※$P<0.000$), SR-a (&$P<0.05$), P: Comparison between MS and controls. Bas-aivs: Basal of after left ventricular interval; Bas-lat: Basal of lateral wall; Mid-aivs: middle of after left ventricular interval; Mid-lat: Middle of lateral wall; Bas-ant: Basal of anterior wall; Bas-Inf: Basal of inferior wall; Mid-ant: Middle of anterior wall; Mid-inf: Middle of inferior wall; Bas-bivs: Basal of before left ventricular interval; Bas-post: Basal of posterior wall; Mid-bivs: Middle of before left ventricular interval; Mid-post: Middle of posterior wall; S: Peak systolic strain; SR-s: Peak systolic strain-rate; SR-e: Peak early diastolic strain-rate; SR-a: Peak late diastolic strain-rate; MS: Metabolic syndrome.

selected, SBP, DBP, W. circum., FPG, UA, were found to be significantly negatively related with S and SR [Table 4].

Scheme showing the interrelations between MS and cardiovascular disease via complex reactions and endothelial dysfunction. These induce vascular stiffness and increased myocardial oxygen consumption in spite of down-regulated per-fusion. This unfavorable constellation results in cardiomyocyte stress (S and SR declined) and myocardial dysfunction as the first stage of diabetic cardiomyopathy.

DISCUSSION

## Metabolic syndrome and left ventricular function

In the strong heart study, MS was found to be associated with reduced LV systolic and diastolic function and LV dimensions, while LV mass and relative wall thickness were found to be higher in the MS group.[15] Ahn *et al*.[16] reported that W. circum. was found to have the most significant effect on LV functional and geometric change. The latter is corroborated by the improvement in diastolic function with improvement in metabolic control of diabetes by specific medical therapy or lifestyle modification. Accordingly, diastolic dysfunction reflects the structural and metabolic milieu in the myocardium, and may allow targeted therapeutic interventions to modulate cardiac metabolism to prevent heart failure in insulin resistance and diabetes.[17]

In our study, we found functional modifications of the LV in patients with MS including diastolic and systolic function. In the current study, even in the isolated metabolic group who were nondiabetic and nonhypertensive and had a body mass index (BMI) significantly lower than the MS group, the altered global ventricular performance may be mediated by other potential mechanisms. This may contribute to insulin resistance [Figure 3], hypertriglyceridemia with

Table 4: Multiple regression analysis of association with between strain and strain-rate and risk factors

Variables	βn ± SE		P	
	S	SR	S	SR
SBP (mmHg)	−0.402	−0.515	0.002	0.000
DBP (mmHg)	−0.464	−0.494	0.000	0.000
W. circum. (cm)	−0.501	−0.556	0.000	0.000
Serum cholesterol (mmol/L)	−0.192	−0.220	0.160	0.107
LDL (mmol/L)	−0.327	−0.227	0.055	0.096
Triglyceride (mmol/L)	−0.078	−0.304	0.573	0.054
FPG (mmol/L)	−0.375	−0.345	0.005	0.010
UA (umol/L)	−0.375	−0.345	0.005	0.001

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; W. circum.: Waist circumference; LDL: Low density lipoprotein; FPG: Fasting plasma glucose; UA: Uric acid.
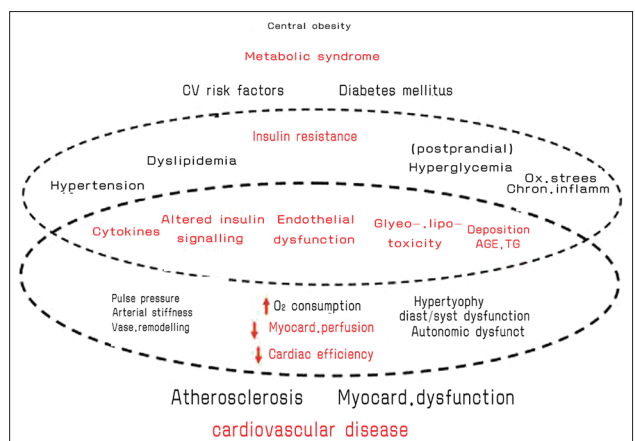
Figure 3: The interrelations between metabolic syndrome and cardiovascular disease.

subsequent impaired endothelial dysfunction, abnormalities in myocardial per fusion and/or metabolic substrate

utilization, inflammation and oxidative stress, interstitial fibrosis, impaired ventricular – vascular interaction, etc. In conclusion, the current study shows that MS groups have an associated abnormal LV global performance.

Strain and strain-rate data compared to traditional echocardiographic parameters

Strain and SR are less affected by passive myocardial motion and tend to be uniform throughout the LV in normal subjects. These noninvasive techniques are rapidly evolving and expanding. Strain values enable the differentiation of normal from abnormal contraction in the ischemic myocardium.[18] The high sensitivity of longitudinal strain to detect functional impairment due to subtle ischemia could be explained by the predominant longitudinal orientation of subendocardial fibers, which are more susceptible to ischemia.[19,20]

Tissue Doppler imaging associated with SRI[21] has shown its value in early detection of MS, and a measurable SR depression has been regarded as the earliest sign of LV regional systolic dysfunction in patients with MS long before a clinical manifestation of heart failure. Our study also showed that a subtle systolic decline of the SR appeared in MS, but the LV ejection fraction remained unchanged in the MS groups. Longitudinal fibers, as a consequence of their prominent subendocardial location, are more vulnerable to fibrosis and hemodynamic overload. Thus, subendocardial long-axis function may be impaired long before circular fiber dysfunction develops in the mid-wall or radial fiber dysfunction in the subepicardial layers. Consequently, subendocardial long-axis function is viewed as a potential marker of subclinical LV dysfunction in several disease conditions. Similarly, longitudinal fibers are more susceptible to ischemia than radial fibers. Thus, there appears to be an entire spectrum of systolic function abnormalities, ranging from normal systolic heart function to systolic heart failure, with heart failure with normal EF being located in between.

Left ventricular EF was the main parameter to describe global heart function. However, new features of echocardiography S and SR provide precise information especially of regional myocardial function.[22] The EF is the traditional measure of systolic LV function and has proved to be a strong predictor of clinical outcome in dilated cardiomyopathy and a variety of cardiac diseases.[23] The EF bears a curvilinear relationship with EDV and an inverse correlation with after load.[23] However, normalization by EDV makes it insensitive to sub clinical myocardial dysfunction in the normal-sized heart or the small hypertrophied ventricle. The normal-sized heart of patients with MS and correspondingly overweight body habits frequently results in poor delineation of the endocardial borders, which are prerequisites for the quantitative assessment of EF. Similarly, the traditional measurements of diastolic function, the Doppler-derived mitral valve inflow velocity pattern and its derivatives, have proved difficult to interpret because of the pseudo normal pattern, that defines grade two dysfunction, but may be mistaken for the normal pattern.[24] While other traditional Doppler echocardiographic parameters enable only semi quantitative assessment of diastolic function and cannot reliably distinguish perturbations in loading conditions from altered diastolic functions, SRI enables detailed quantification of global and regional diastolic function.

Strain and strain-rate in metabolic syndrome

Increased myocardial stiffness and reduce myocardial deformation ability, S and SR will reduce. In our study, S and SR were all declined in MS [Table 3], while the papillary muscle level of the S and SR were higher than those of the mitral level [Figure 4]. So there were regional myocardial dysfunctions in patients with MS. The impact of sub-clinical cardiac involvement in MS remains to be assessed. MS may have decreases of myocardial systolic and early diastolic velocities on TDI, even if they appear to have normal systolic and diastolic function on conventional echocardiography.[25]

Our multiple regression analysis, regarding the separate contribution to S and SR of each parameter among all risk factors selected, BP, W. circum., FPG, UA were found to significantly negatively related with S and SR [Table 4]. Increased relative risk of LV hypertrophy due to the presence of MS, once excluded age and BP, seems mediated mainly by BMI (as an index of increased adiposity), which works as both a hemodynamic and nonhemodynamic factor.[26,27] The amount of lipid stored in ectopic depots in MS is directly correlated and that pericardium lipid in particular appeared correlated to cardiac function in a site-specific manner that appeared independent of insulin resistance and BMI.[28] One possibility is that pericardia lipid itself exerts a restrictive pressure on
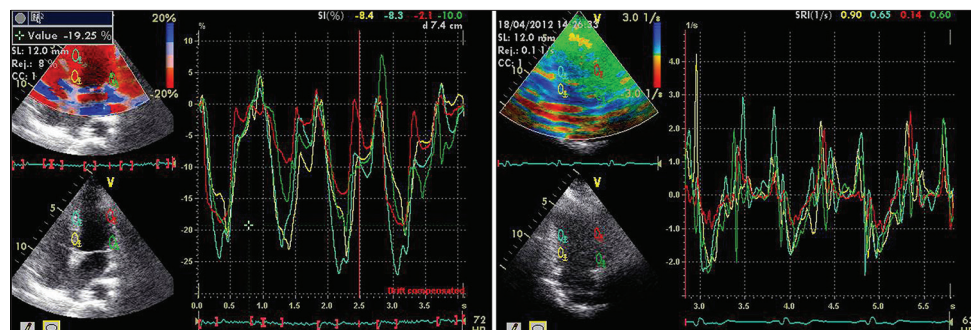

Figure 4: Strain and strain-rate imaging from the basal and mid myocardial segments from a study subject.

myocardial expansion in diastole, and thereby slightly reduces cardiac diastolic filling, and thus affects cardiac output. Another possibility is that pericardia lipid elaborates signaling molecules that affect cardiac function via direct venous drainage, and in this way affects diastolic function.[29] All these small changes cannot be found by traditional echocardiographic parameters, while SRI can detect easily. Irregular distribution of systolic S and SR induces in the LV segments may indicate that MS leads to heterogeneous myocardial involvement also in the early period.[30]

Limitations

As with flow Doppler, TDI-derived imaging methods are angle dependent. With the currently available technology, SR has a relatively poor signal to noise ratio and is load sensitive. S measurements are made along a single ultrasound scan line.

Conclusions

Strain and SR derived from SRI reflect an improved assessment of myocardial contraction and quantification of regional myocardial deformation in humans.[31] Particularly in the clinical setting, cardiovascular risk factors contributing to altered SRI measures occur before the development of global LV dysfunction.[32] In addition, the technology's allure lies primarily in its portability, relative affordability, widespread availability, noninvasive nature, and rapid real-time imaging capabilities.[33] SRI should be used routinely and become a standard research tool for MS.

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