Evaluation of Cardiac Functions By Speckle Tracking Echocardiography in Type 2 Diabetes Mellitus With Hyperlipidemia

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Abstract

Background

To investigate the value of two-dimensional speckle tracking echocardiography (2D-STE) in evaluating cardiac functions in type 2 diabetes mellitus (T2DM) with hyperlipidemia.

Methods

Sixty T2DM patients with normal left ventricular ejection fraction (LVEF) and poorly-controlled blood glucose were selected. Among these, thirty had hyperlipidemia. Thirty age- and gender-matched healthy individuals were recruited as the normal control group. Longitudinal strain of left ventricular segments, left ventricular global longitudinal strain (LV GLS), left atrial global longitudinal strain (LA GLS), right ventricular global longitudinal strain (RV GLS) and right atrial global longitudinal strain (RA GLS) were measured by 2D-STE.

Results

(1) Compared with the normal control group, LV GLS in T2DM group and T2DM with hyperlipidemia group decreased ($P < 0.05$), but there was no significant difference of LV GLS between T2DM group and T2DM with hyperlipidemia group ($P > 0.05$). Compared with the normal control group and T2DM group, longitudinal strain of middle segment of LV in T2DM with hyperlipidemia group decreased ($P < 0.05$). (2) There was a significant difference in LA GLS among the three groups. LA GLS of T2DM with hyperlipidemia group was lower compared with the normal control and T2DM group ($P < 0.05$). (3) Compared with the normal control group, RV GLS in T2DM group and T2DM with hyperlipidemia group was lower ($P < 0.05$), but there was no significant difference of RV GLS between T2DM group and T2DM with hyperlipidemia group ($P > 0.05$). RA GLS in T2DM with hyperlipidemia group decreased ($P < 0.05$) compared to the normal control group and T2DM group.

Conclusion

Speckle tracking echocardiography can effectively evaluate cardiac dysfunction in patients with T2DM. LA GLS and RA GLS can be used as potential markers of cardiac dysfunction in T2DM with hyperlipidemia, and provide the basis for early clinical diagnosis and treatment.

1. Introduction

Type 2 diabetes mellitus (T2DM) is one of the global epidemic diseases and metabolic syndrome, which can lead to extensive myocardial injury, reduce myocardial compliance,[1] and cause cardiac systolic and

diastolic dysfunction.[2] T2DM, which is closely related to atherosclerosis, can give rise to multiple cardiovascular complications and increase the mortality of cardiovascular diseases.[3]

Dyslipidemia is a common metabolic disorder in T2DM. Its main manifestations are the increase of triglyceride (TG), total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C). It is related to metabolic factors such as insulin resistance, and it promotes vascular atherosclerosis, causes cardiac lipid accumulation, and increases myocardial ischemia / reperfusion injury.[4] Studies have shown that T2DM and hyperlipidemia are independent risk factors for cardiovascular diseases,[5] which can increase the risk of macrovascular and microvascular complications.

Two-dimensional speckle tracking echocardiography (2D-STE) can be used to evaluate myocardial motion by tracking myocardial natural acoustic spots. It is a reliable technique for quantifying multi-plane myocardial deformation due to no angle dependence.[6] The decrease of myocardial strain can reduce the myocardial work efficiency. To some extent, the myocardial strain can reflect the cardiac function.[7] Therefore, this study intends to analyze the changes of cardiac structure and functions in T2DM with or without hyperlipidemia by using myocardial deformation parameters, and to seek early diagnostic indicators of myocardial dysfunction in T2DM.

2. Methods

2.1 Study population

Sixty T2DM patients with normal left ventricular ejection fraction (LVEF) and poorly-controlled blood glucose were recruited from January, 2019 to May, 2020. Among them, thirty T2DM patients were complicated with hyperlipidemia. Inclusion criteria for T2DM patients were as follows: fasting plasma glucose (FPG) ≧ 7.0mmol/L, hemoglobin A1c (HbA1c) ≧ 6.5%.[8] The inclusion criteria for patients with hyperlipidemia: TC ≧ 6.2mmol/L or TG ≧ 2.3mmol/L.[2] Exclusion criteria: type 1 diabetes mellitus and other special types of diabetes mellitus, coronary heart diseases, arrhythmia, congenital heart diseases, moderate to severe valvular heart diseases, primary cardiomyopathy, thyroid diseases, liver and kidney dysfunction. Thirty age- and gender-matched healthy volunteers served as the normal control group.

All individual participants included in the study provided informed consent. We conducted our studies in compliance with the Declaration of Helsinki. The study protocol (No. HEC- KYJJ2020-025-01) was approved by the Ethics Committee of the First College of Clinical Medical Sciences, China Three Gorges University.

2.2 Clinical and laboratory examinations

The height, weight, heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), the duration of T2DM and drug treatments were collected for all participants. Laboratory tests included blood routine, FPG, HbA1c, TC, TG, LDL-C, high-density lipoprotein cholesterol (HDL-C), blood urine nitrogen

(BUN), serum creatinine (SCr) and serum uric acid (UA). We evaluated carotid atherosclerosis and the echogenicity of the largest plaque using duplex ultrasound.

2.3 Echocardiography

We performed standard echocardiography using a commercially available ultrasound system with Philips EPIQ 7C (Philips Healthcare, Andover, MA, USA). Conventional 2D and Doppler techniques were used with a X5-1 transducer (1.0 ~ 5.0 MHz). All subjects were placed in the left decubitus position with calm breathing and connected with three lead electrocardiogram. According to the ASE recommendations, left atrial end systolic diameter (LAD), interventricular septum (IVS) thickness, left ventricular posterior wall (LVPW) thickness, left ventricular end diastolic diameter (LVDd), left ventricular end systolic diameter (LVDs), left ventricular ejection fraction (LVEF), right ventricular diastolic diameter (RVDd) and tricuspid annular plane systolic excursion (TAPSE) were measured by transthoracic echocardiography. High frequency dynamic images of the apical four-, two- and three-chamber long axis views were collected for 3–5 consecutive cardiac cycles. After examinations, all images were imported into Philips QLAB10.7 workstation. In these images, the endocardial planes of the LV, LA, RV and right atrial (RA) were manually delineated, and the data of ventricular longitudinal segments were generated. Values of LV global longitudinal strain (LV GLS), LA global longitudinal strain (LA GLS), RV global longitudinal strain (RV GLS) and RA global longitudinal strain (RA GLS) were automatically calculated by the workstation.

2.4 Reproducibility

Thirty patients were randomly selected for evaluating the repeatability of the overall longitudinal strain of LV, LA, RV and RA. After repeated analysis by the same examiner (ZD) and another experienced examiner (RL), the consistency between the two examiners was evaluated.

2.5 Statistical Analysis

SPSS 25.0 software was used for statistical analysis. All continuous data were summarized as mean ± standard deviation (SD). One-way ANOVA was used to evaluate the statistical differences among the groups. The categorical variables were compared by chi-square test. The ratio of frequency was used for counting data. $P < 0.05$ was considered as statistically significant.

3. Results

3.1 Clinical Characteristics

There was no significant difference in age, gender, body mass index (BMI), blood test, SCr and BUN among the three groups. Compared with the normal control group, FPG, HbA1c, TC, TG, LDL-C and UA increased in T2DM and T2DM with hyperlipidemia groups (all $P<0.05$). Compared with the T2DM group, TC, TG, LDL-C and UA were higher in T2DM with hyperlipidemia group (all $P<0.05$). All these results were in Table 1.

3.2 Routine echocardiographic parameters

There was no significant difference in LAD, IVS, LVPW, LVDd, LVDs, RVDd, LVEF, TAPSE, E/A, E/e' among the three groups (P>0.05), as illustrated in Table 2.

3.3 The longitudinal strain of LV each segment, LV GLS and LA GLS by 2D-STE

Compared with the normal control group, LV GLS in T2DM and T2DM with hyperlipidemia groups was lower (P<0.05); there was no significant difference of LV GLS between T2DM and T2DM with hyperlipidemia groups (P>0.05). Compared with the normal control group, the longitudinal strain of middle segment in LV anterior wall, inferior wall, posterior wall, anterior septum, posterior septum and lateral wall in T2DM and T2DM with hyperlipidemia groups were lower (P<0.05), while the longitudinal strain of other segments had no significant difference (P>0.05). In T2DM with hyperlipidemia group, the longitudinal strain of middle segments of LV was lower (P<0.05) compared with T2DM group, as shown in Table 3 and Figure 1.

Compared with the normal control group, the level of LA GLS in T2DM group and T2DM with hyperlipidemia group was lower (P<0.05). Compared with T2DM group, LA GLS in T2DM with hyperlipidemia group decreased (P<0.05).

3.4 RV GLS and RA GLS by 2D-STE

Compared with the normal control group, RV GLS in T2DM group and T2DM with hyperlipidemia group decreased (P<0.05). There was no significant difference in RV GLS between T2DM and T2DM with hyperlipidemia group (P>0.05), as seen in Table 3 and Figure 2. Compared with the normal control group, RA GLS in T2DM and T2DM with hyperlipidemia groups was lower (P<0.05). RA GLS in T2DM with hyperlipidemia group decreased (P<0.05) compared with T2DM group.

4. Discussion

T2DM is a common risk factor for cardiovascular disease. Hyperglycemia, insulin resistance and impaired signal transductions of cardiac insulin metabolism can reduce myocardial glucose uptake, increase the production of myocardial reactive oxygen species, induce mitochondrial dysfunction, and reduce the content of ATP and $Ca2^{+}$ in cardiomyocytes, which can lead to myocardial autophagy and myocardial necrosis, and impair myocardial metabolism and function.[9] In the early stage, DM can cause myocardial fibrosis and increased stiffness, leading to reduced LV diastolic filling, enlarged atrial size, and increased blood flow filling and LV end diastolic pressure; in the second stage, LV hypertrophy, cardiac remodeling, diastolic dysfunction and heart failure with normal LVEF occurred.[10] With the development of diabetic cardiomyopathy, diastolic dysfunction and cardiac compliance falling may coexist with systolic dysfunctions, leading to reduced LVEF.[11] Hyperlipidemia is a common clinical metabolic disease, characterized by an abnormal increase of lipid and lipoprotein levels,[1] which can cause myocardial hypertrophy, fibrosis, myocardial cell apoptosis, LV systolic and diastolic subclinical

function damage,[12] accelerate atherosclerosis, aggravate cardiac dysfunction and cardiovascular events in T2DM patients.[13]

There was a significant difference of serum UA levels among the three studied groups. Elevated serum UA is a metabolic syndrome, which can interfere with glucose uptake, aggravate insulin resistance, inhibit the bioavailability of nitric oxide, and induce endothelial dysfunction. It is associated with the progression of T2DM,[14] and is an independent predictor of cardiovascular disease.[15] Studies have found that serum UA level increases gradiently with cardiovascular risk factors, and the possibility of atherosclerosis significantly elevated as well.[16] The levels of TC and LDL-C can affect the formation of atherosclerotic plaques, promote inflammatory reaction and oxidative stress, damage vascular wall elastin, induce the production of superoxide, and lead to atherosclerosis; besides, serum UA and cholesterol levels have a synergistic effect on the aggravation of atherosclerosis due to their overlapping mechanism.[17]

There was no significant difference in LAD, IVS, LVPW, LVDd, LVDs, LVEF, RVDd, TAPSE, E/A, E/e' and other conventional echocardiography parameters among the three groups, indicating that conventional echocardiography has limitations. Compared with the normal control and T2DM group, the longitudinal strain of LV middle segment in T2DM with hyperlipidemia group decreased, which may be related to the compensatory response of subepicardial muscle fiber torsion.[18] LV GLS in T2DM group and T2DM with hyperlipidemia group decreased compared to the normal control group, suggesting that hyperglycemia and insulin resistance can lead to cardiomyocyte hypertrophy, myocardial microvascular endothelial cell dysfunction, intercellular collagen deposition, expansion of extracellular space, and increase of myocardial stiffness and myocardial degeneration.[19] As an atherosclerotic factor, lipids can trigger the release of pro-inflammatory cytokines, promote the thickening of arterial intima and reduce myocardial blood flow.[20] Moreover, excessive lipid metabolites can easily result in myocardial fibrosis and reduced ventricular diastolic compliance and diastolic energy,[21] and thus impaired myocardial function.

RV GLS in T2DM group and T2DM with hyperlipidemia group decreased, indicating that RV function was impaired in two groups. Kang et al[22] found that DM was associated with RV systolic and diastolic dysfunction. LV diastolic and systolic dysfunction caused by T2DM may have an important influence on RV due to the ventricular interdependence. The two ventricles are combined anatomically through their muscle fiber anatomical structure and IVS, exhibiting an interdependent physiological feature,[23] which is related to the interwoven subendocardial fibers of IVS between RV and LV, indicating that diabetes inducing myocardial diffuse fibrosis can affect eccentric hypertrophy, diastolic and systolic dysfunction of the two ventricles. Due to the non-antagonistic vasoconstriction by T2DM,[22] systemic macrovascular and microvascular functions were impaired and the arterial tension was increased by causing myocardial endothelial cell dysfunction, inflammatory response, calcium homeostasis change and substrate metabolism.[24] In this study, the middle strain of LV was consistent with RV in two T2DM groups. There was no significant difference of RV GLS between T2DM group and T2DM with hyperlipidemia group, which might be due to the fact that DM and lipid treatment had been carried out for a period of time, possibly affecting the improvement of RV function and mechanics.[25]

Compared with the normal control group, LA GLS in T2DM group and T2DM with hyperlipidemia group decreased ($P < 0.05$), indicating that both T2DM and T2DM with hyperlipidemia groups had LA dysfunction. Hyperglycemic toxicity can cause abnormal myocardial composition, mainly due to excessive collagen accumulation,[26] resulting in increased extracellular volume of cardiomyocytes and LA fibrosis in patients with T2DM, which may be the main factor for the decrease of LA GLS. However, there was no significant difference of LV and RV GLS between T2DM and T2DM with hyperlipidemia groups, but LA GLS significantly decreased ($P < 0.05$), suggesting that LA GLS in T2DM with hyperlipidemia group was more sensitive than LV and RV GLS, which may have an association with dyslipidemia and BMI. Dyslipidemia aggravates myocardial injury and dysfunction by causing myocardial interstitial fibrosis, impaired calcium homeostasis, mitochondrial dysfunction and microvascular lesions.[27] The higher BMI, to a certain extent, can increase blood capacity, promote cardiac enlargement and lead to the decrease of atrial GLS.[28]

Compared with the normal control group, RA GLS in T2DM group and T2DM with hyperlipidemia group decreased; compared with T2DM group, RA GLS in T2DM with hyperlipidemia group decreased ($P < 0.05$), indicating that RA function was impaired in T2DM and T2DM with hyperlipidemia groups. It was found that HbA1c was independently correlated with RA GLS.[25] Bening et al have proved that diabetes had a significant impact on the RA fiber contractility, and RA contractility of diabetic patients was significantly lower than that of non-diabetic patients.[23] The sub-endocardial fibrosis of T2DM atrium leads to the decrease of the elasticity of the atrial wall, which may cause atrial enlargement and dysfunction. Blood glucose inhibits RA myofilament function by changing calcium homeostasis. Hyperlipidemia can aggravate the accumulation of myocardial lipid, result in myocardial stiffness and systolic and diastolic dysfunction. Ventricular diastolic dysfunction is an independent predictor of atrial phase function, and changes in atrial function can indicate ventricular diastolic dysfunction.[29] In this study, there was no significant difference of LV GLS and RV GLS between T2DM and T2DM with hyperlipidemia groups, while LA GLS and RA GLS significantly decreased, indicating that LA and RA function changes earlier than LV and RV, suggesting that T2DM group and T2DM with hyperlipidemia group may have ventricular diastolic dysfunction at an early stage.

5. Limitation

There is an increasing evidence that gender differences are important in epidemiology, pathophysiology, treatment and outcomes in cardiovascular diseases. Among the three groups, we included a similar proportion of men and women, but the impact of gender was not specifically mentioned. Due to the small number of included samples, only 2D myocardial longitudinal strain study was carried out in these patients. The single dyslipidemia group was not included, so the effect of dyslipidemia on myocardial strain was not analyzed. Because of the 3D nature of the heart geometry, 2D measurements may have substantial differences to the real dimensions. At present, three-dimensional ultrasound is being widely used, so we will continue to use new ultrasonic technologies for the further study in the future.

6. Conclusion

T2DM can cause changes in cardiac structure and function. Longitudinal strain of myocardium can effectively evaluate cardiac dysfunctions in T2DM patients. LA GLS and RA GLS are more sensitive than LV GLS and RV GLS, and can be used as potential markers to predict the changes of cardiac function in T2DM patients with hyperlipidemia, which provides the basis for early clinical diagnosis and treatment.

Declarations

Compliance with Ethical Standards

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Conflict of Interest: All authors declared they have no conflict of interest.

Ethical approval: This article is approved by the Ethics Committee of the First College of Clinical Medical Sciences, China Three Gorges University (No. HEC-KYJJ2020-025-01).

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Tables

Table 1 Baseline characteristics of the study population ($\bar{x}\pm s$).

Indicators	Control group (n=30)	T2DM group (n=30)	T2DM with hyperlipidemia group (n=30)
Age (y)	54.2±8.9	54.4±9.3	54.9±9.8
Gender (male)	19 (63.3%)	19 (63.3%)	20 (66.7%)
Smoking (n, %)	8 (26.7%)	9 (30%)	10 (33.3%)
SBP (mmHg)	118.6±7.4	121.7±8.3	123±7
DBP (mmHg)	75.5±4.5	74.5±5.6	75±4
Duration of T2DM (y)	-	6.5±5.1	7.9±7.6
BSA (m2)	1.5934±0.2323	1.6934±0.3239	1.7214±0.3942
BMI (kg/m2)	23.7±3.71	23.92±2.93	24.25±1.96
HR (bpm)	75±12	74±15	76±12
RBC (x10^12/L)	4.42±0.65	4.41±0.61	4.54±0.43
WBC (x10^9/L)	6.02±1.78	6.32±1.86	6.36±1.58
PLT (x10^9/L)	195±60	191±48	189±59
Neutrophil (x10^9/L)	3.45±1.20	3.39±1.18	3.51±1.01
Lymphocyte (x10^9/L)	1.78±0.43	1.74±0.50	1.82±0.35
FPG (mmol/L)	4.39±0.80	9.56±2.54*	11.05±2.45*
HbA1c (%)	4.85±0.65	9.45±1.77*	9.98±1.95*
TC (mmol/L)	4.07±0.75	4.05±0.72	5.32±1.31*#
TG (mmol/L)	1.41±0.45	1.26±0.51	5.24±0.72*#
LDL-C (mmol/L)	2.32±0.65	2.39±0.67	3.66±0.91*#
HDL-C (mmol/L)	1.19±0.29	1.15±0.26	1.12±0.33
SCr (umol/L)	62.50±15.61	65.61±16.61	71.55±17.35
BUN (mmol/L)	5.41±2.36	5.55±1.73	5.42±1.60
UA (umol/L)	289.64±54.22	322.34±57.66*	371.41±58.81*#
Carotid US			
IMT(≥1mm)	8(26.7%)	14(46.7%)	19(63.3%)
Echolucent plaque	4(13.3%)	7(23.3%)	10(33.3%)

Calcified plaque	-	3(10%)	6(20%)
Treatment			
Metformin (n, %)	-	20 (66.7%)	27 (%)
Sulfonylureas (n, %)	-	13 (43.3%)	15 (50.0%)
Insulin (n, %)	-	23 (76.7%)	26 (86.7%)
α-GSDI (n, %)	-	17 (56.7%)	16 (53.3%)
TZDs (n, %)	-	5 (16.7%)	4 (13.3%)
Statins (n, %)	-	-	11 (36.7%)
Fibrates (n, %)	-	-	13 (43.3%)

T2DM, type 2 diabetes mellitus; SBP, systolic blood pressure; DBP, diastolic blood pressure; BSA, body surface area; BMI, body mass index; HR, heart rate; RBC, red blood cell; WBC, white blood cell; PLT, platelet; FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; TC, total cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SCr, serum creatinine; BUN, blood urine nitrogen; UA, uric acid; US, ultrasound; IMT, intima-media thickness; α-GSDI, α-glucosidase inhibitor; TZDs, Thiazolidinediones.

*P<0.05 vs control group.

#P<0.05 vs T2DM group.

Table 2 Comparison of conventional echocardiography parameters among three groups ($\bar{x}$ ±s).

Indicators	Control group (n=30)	T2DM group (n=30)	T2DM with hyperlipidemia group (n=30)
LAD (mm)	30.5±2.8	31.7±3.7	32.5±3.3
LA volume (ml)	26.0±3.8	26.2±3.3	27.5±3.6
IVS (mm)	8.4±0.9	8.6±0.9	9.1±0.9
LVPW (mm)	8.3±0.9	8.2±1.0	8.6±1.0
LVDd (mm)	46.4±3.5	46.5±4.2	47.2±4.9
LVDs (mm)	30.5±3.5	30.8±4.4	30.5±4.2
LVEF (%)	65.5±4.9	64.4±4.2	64.2±4.5
RVDd (mm)	28±4	30±3	31±3
TAPSE (mm)	22±2	21±2	21±2
E/A	0.96±0.17	0.93±0.13	0.92±0.14
E/e'	0.82±0.16	0.85±0.15	0.78±0.14

T2DM, type 2 diabetes mellitus; LAD, left atrial diameter; LA, left atrial; IVS, inter-ventricular septum; LVPW, left ventricular posterior wall; LVDd, left ventricular end diastolic diameter; LVDs, left ventricular end systolic diameter; LVEF, left ventricular ejection fraction; RVDd, right ventricular diastolic diameter; TAPSE, tricuspid annular plane systolic excursion.

Table 3 Comparison of myocardial strain parameters among three groups ($\bar{x}\pm s$).

T2DM, type 2 diabetes mellitus; LV, left ventricular; GLS, global longitudinal strain; RV, right ventricular; LA, left atrial; RA, right atrial.

*$P<0.05$ vs control group.

†$P<0.01$ vs control group.

#$P<0.05$ vs T2DM group.

Figures

Indicators	Control group (n=30)	T2DM group (n=30)	T2DM with hyperlipidemia group (n=30)
LV anterior wall			
Basal	21.8±5.5	21.7±4.5	21.4±5.2
Mid	20.4±3.4	18.4±3.7*	16.2±4.7*#
Apical	22.3±3.5	22.1±3.9	22.0±3.4
LV inferior wall			
Basal	19.4±4.1	18.5±2.9	18.2±2.7
Mid	19.0±3.5	17.7±4.2 †	15.3±2.8 †#
Apical	27.4±4.9	27.2±5.1	26.8±5.6
LV posterior wall			
Basal	18.2±5.5	18.0±5.4	17.9±4.6
Mid	23.4±3.0	21.2±4.4*	19.8±4.7*#
Apical	21.6±4.2	22.0±4.8	21.4±4.5
LV anterior septum			
Basal	16.5±3.5	16.4±4.9	15.8±4.3
Mid	22.5±3.5	19.5±4.3*	17.7±5.0*#
Apical	28.2±4.3	27.8±5.9	27.2±5.5
LV posterior septum			
Basal	20.8±4.3	19.8±4.5	19.7±4.8
Mid	21.9±4.3	18.1±5.1*	16.1±4.5*#
Apical	28.2±5.1	27.9±5.5	27.7±5.7
LV lateral wall			
Basal	21.8±4.6	21.5±4.9	22.0±5.5
Mid	21.6±4.6	19.1±4.6*	17.1±4.5*#
Apical	23.0±4.6	22.0±5.5	21.0±4.3
LV GLS	23.2±4.5	20.3±3.1*	20.1±3.5*
RV lateral wall			
Basal	19.8±4.6	18.7±4.9	18.1±4.4

Mid	23.8±4.6	20.5±4.6*	17.8±3.1 †#
Apical	20.9±4.9	18.5±6.2	18.4±6.8
RV septum			
Basal	16.4±3.7	14.8±6.1	14.7±5.0
Mid	25.0±4.0	20.3±4.9*	16.5±6.4 †#
Apical	25.7±8.1	24.5±6.5	24.2±5.7
RV GLS	22.1±5.5	18.8±3.8*	18.2±3.4*
LA GLS	35.3±3.2	29.3±2.8*	23.2±2.4 †#
RA GLS	36.2±2.0	27.4±5.1 †	23.7±3.6 †#

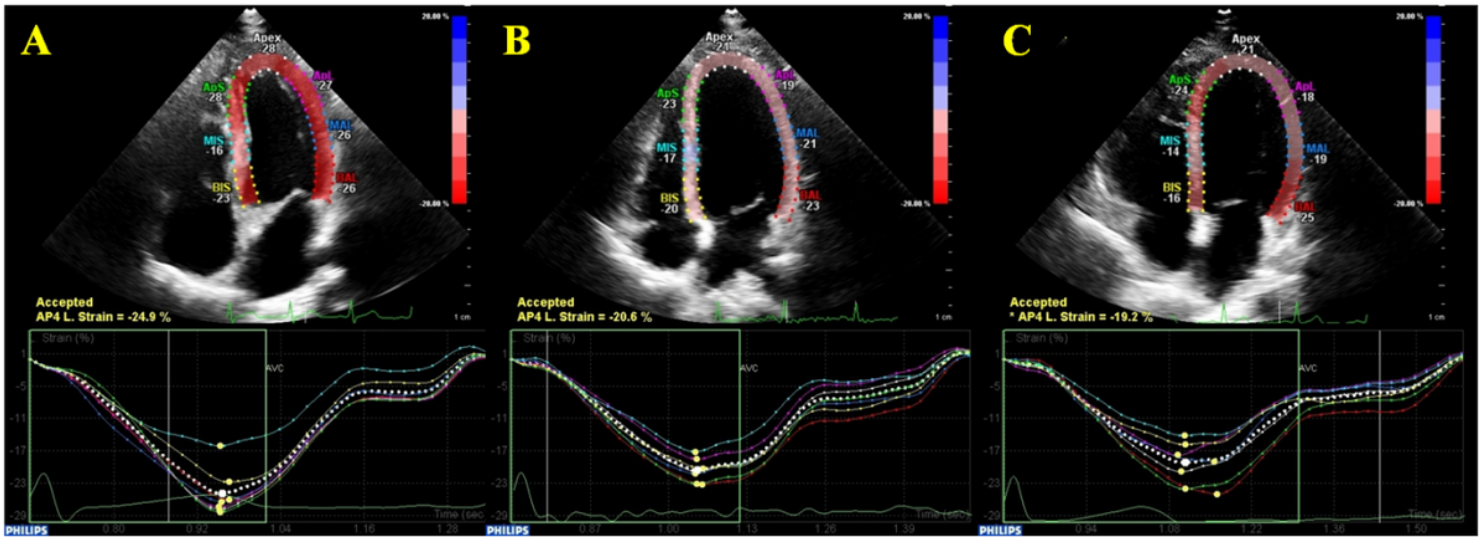

Figure 1

In T2DM with hyperlipidemia group, the longitudinal strain of middle segments of LV was lower (P<0.05) compared with T2DM group, as shown in Table 3 and Figure 1.

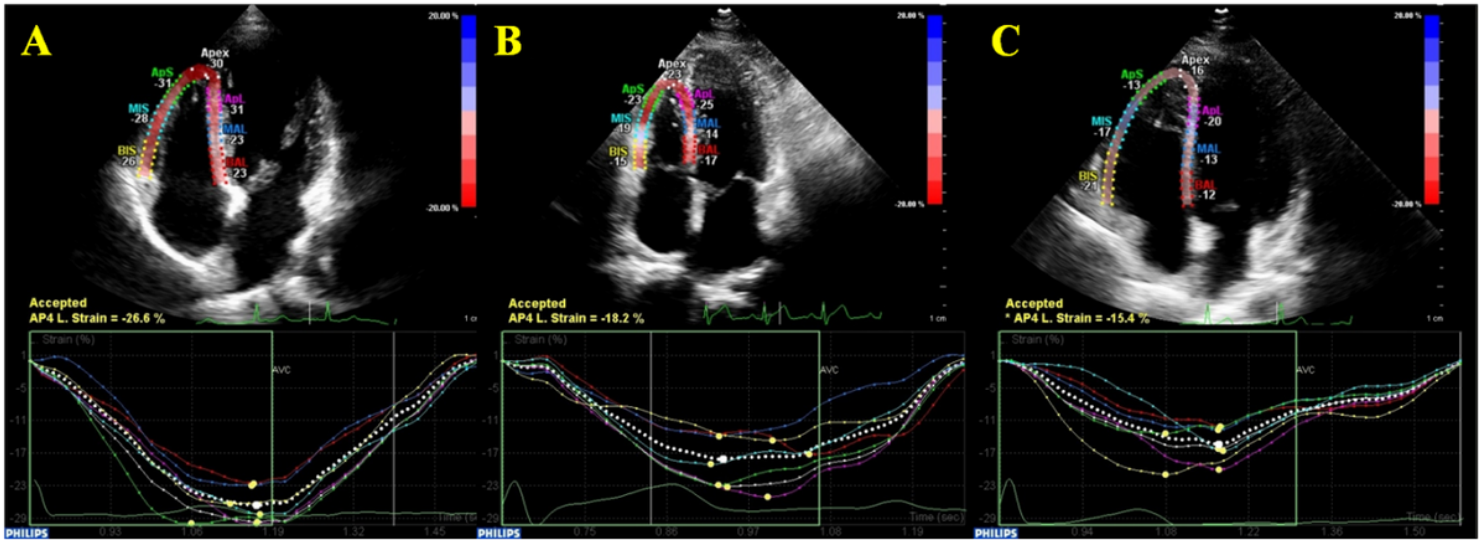

Figure 2

There was no significant difference in RV GLS between T2DM and T2DM with hyperlipidemia group (P>0.05), as seen in Table 3 and Figure 2.