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The adverse effect of metabolic syndrome on left ventricular global strains and myocardial energetic efficiency in non-ischemic dilated cardiomyopathy patients: a cardiac magnetic resonance study

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Abstract

Background Metabolic syndrome (MetS) is a known risk factor for cardiovascular dysfunction; however, its impact on left ventricular (LV) global strains and myocardial energetic efficiency in non-ischemic dilated cardiomyopathy (NIDCM) remains inadequately understood. This study aimed to investigate the effect of MetS on LV dysfunction in NIDCM patients using cardiovascular magnetic resonance (CMR) imaging.

Methods A total of 557 NIDCM patients (378 without MetS and 179 with MetS) who underwent CMR examination were included. CMR-derived LV strains, remodeling index (LVRI), global function index (LVGFI), and indexed myocardial energetic efficiency (MEEI) were assessed and compared between the groups. The independent determinants of LV global longitudinal peak strain (GLPS), LVRI, LVGFI, and MEEI were evaluated using multivariable linear regression analyses.

Results Compared to NIDCM patients without MetS, those with MetS had significantly lower LVSVI, LVEF, and LVGFI, along with higher LVMI and LVRI (all $p < 0.05$). However, no significant differences were found in LVEDVI and LVESVI (both $p > 0.05$). In terms of LV strain, the NIDCM(MetS+) group exhibited worse global peak strain and peak diastolic strain rate in all three directions, as well as decreased radial and longitudinal peak systolic strain rate (PSSR) compared to the NIDCM (MetS−) group (all $p < 0.05$), while circumferential PSSR did not differ significantly ($p > 0.05$). The MEEI was significantly lower in the NIDCM(MetS+) group compared to the NIDCM(MetS−) group (0.30 [0.20, 0.45] ml/s/g vs. 0.39 [0.25, 0.58] ml/s/g, $p < 0.001$). Multivariable analysis identified the presence of MetS as an independent determinant of LV GLPS ($\beta = 0.211$, $p < 0.001$), LVRI ($\beta = 0.147$, $p = 0.003$), and MEEI ($\beta = -0.160$, $p < 0.001$).

Conclusion The presence of MetS worsens LV function, remodeling, and myocardial energetic efficiency in patients with NIDCM, as evidenced by declines in LV strain, global function parameters, and indexed myocardial energetic

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efficiency. These findings suggest that addressing metabolic abnormalities may be crucial for improving LV function and outcomes for patients with NIDCM.

Keywords Metabolic syndrome, Non-ischemic dilated cardiomyopathy, Magnetic resonance imaging, Myocardial strain, Myocardial energetic efficiency

Introduction

Metabolic syndrome (MetS) is a well-established risk factor for cardiovascular disease, encompassing a cluster of metabolic abnormalities, including abdominal obesity, hypertension, dyslipidemia, and impaired glucose metabolism [1, 2]. These factors have been associated with adverse changes in the cardiovascular structure and function of various cardiac pathologies, including left ventricular (LV) dysfunction and remodeling [3, 4]. Non-ischemic dilated cardiomyopathy (NIDCM), a condition characterized by LV dilation and systolic dysfunction in the absence of significant coronary artery disease, is particularly vulnerable to the detrimental effects of metabolic disturbances [5]. Furthermore, several studies have highlighted the potential role of the triglyceride–glucose (TyG) index, a simple surrogate marker for insulin resistance, in predicting outcomes of cardiovascular dysfunction [6, 7]. Therefore, it is crucial to elucidate the specific effects of MetS on LV changes and the relationship between the TyG index and LV function in NIDCM patients.

Recent advancements in cardiac imaging, particularly cardiac magnetic resonance (CMR), have enabled more accurate and comprehensive assessments of LV function [8]. The CMR-derived strain parameters are crucial for understanding the pathophysiology of NIDCM, as they allow for early detection of subtle changes in myocardial function before significant structural damage occurs [9]. CMR is also capable of measuring LV mass, volumes, LV remodeling index (LVRI), and LV global function index (LVGFI), providing a detailed evaluation of myocardial deformation and function [10]. Myocardial energetic efficiency (MEE), which reflects the heart's ability to convert chemical energy from oxidative metabolism into mechanical work, is an important indicator of cardiac function in patients with MetS [11, 12]. Impaired MEE has been identified as an independent predictor of adverse cardiovascular outcomes [12]. Previous studies have established a simple, non-invasive method to estimate myocardial MEE by assessing stroke work and myocardial oxygen consumption [3, 13]. However, the additive effects of MetS and the TyG index on LV function in NIDCM patients have not been thoroughly investigated.

To better understand how metabolic abnormalities impact the LV function and to identify potential biomarkers for early detection and risk assessment in NIDCM patients, we conducted this study to investigate the effects of MetS on LV global strains and MEEI, as well as to assess the relationship between the TyG index and LV dysfunction.

Methods and materials

Study population

The study was approved by the Biomedical Research Ethics Committee of our hospital, and informed consent was waived due to its retrospective nature. We included consecutive patients diagnosed with NIDCM who underwent CMR examinations between January 2014 and December 2024. The diagnosis of NIDCM was based on criteria established by the World Health Organization/International Society and Federation of Cardiology [14]. The exclusion criteria were as follows: (1) significant coronary artery stenosis, defined as ≥ 50% stenosis of a major coronary artery confirmed by invasive coronary angiography or computed tomography coronary angiography, a history of myocardial infarction, or prior coronary revascularization; (2) other cardiac conditions, such as primary cardiomyopathies, congenital heart diseases, infiltrative cardiomyopathies, or severe valvular heart disease; (3) severe renal failure, defined as an estimated glomerular filtration rate (eGFR) < 30 mL/min/1.73 m^2; (4) inadequate images because of arrhythmia or poor image quality; (5) incomplete clinical records.

The definition of MetS was based on the joint interim statement from the International Diabetes Federation Task Force on Epidemiology and Prevention (2009) [15]. According to this definition, the diagnosis of MetS is made when at least three of the following five risk factors are present: (1) elevated waist circumference (with definitions varying by population and country); (2) elevated triglycerides (TG) [≥ 150 mg/dL (1.7 mmol/L)] or specific treatment for this lipid abnormality; (3) reduced high-density lipoprotein cholesterol (HDL-c) [< 40 mg/dL (1.0 mmol/L) in males; <50 mg/dL (1.3 mmol/L) in females] or specific treatment for this lipid abnormality; (4) elevated blood pressure (systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 85 mmHg) or treatment for previously diagnosed hypertension (HTN); and (5) elevated fasting blood glucose (FBG) [> 100 mg/dL (5.6 mmol/L)] or a history of type 2 diabetes mellitus (T2DM). For patients without waist circumference measurements, body mass index (BMI) was used as a substitute, with a BMI > 25 kg/m^2 considered equivalent to exceeding the waist circumference threshold for MetS [3, 16, 17]. Patients with fewer than two risk factors

were classified as non-MetS. Finally, 557 inpatients were assigned to the NIDCM(MetS−) ($n = 378$, age 50 (36, 61), 251 males) and NIDCM(Mets+) ($n = 179$, age 50 (40, 61) years, 131 males) groups. Additionally, TyG index was calculated as Ln[fasting triglycerides (mg/ dl) × fasting glucose (mg/dl)/2] [18]. To assess the influence of the TyG index on LV function, NIDCM patients were divided into two groups according to the median TyG index (Calculated as Ln[fasting triglycerides (mg/ dl) × fasting glucose (mg/dl)/2]) [19]: TyG index < 8.79 ($n = 279$) and TyG index ≥ 8.79 groups ($n = 278$).

Data regarding the sex, age, history of smoking and drinking, presence of HTN or T2DM, FBG, glycated hemoglobin (HbA1c), BMI, SBP, DBP, heart rate (HR), serum cholesterol levels, creatinine, estimated glomerular filtration rate (eGFR), N-terminal pro-brain natriuretic peptide (NT-proBNP), cardiac troponin T (cTnT) and medical history were collected from the digital medical records of the patients. HTN was defined as a SBP ≥ 140 mmHg and/or a DBP ≥ 90 mmHg at rest, measured on more than two occasions, or current antihypertensive treatment [20]. T2DM was diagnosed according to American Diabetes Association guidelines [21]. BMI was calculated as weight (kg) divided by height squared (m^2), with obesity defined as a BMI ≥ 25 kg/m^2.

CMR scanning protocol

CMR exams for all subjects were conducted in the supine position using a 3.0 T whole-body magnetic resonance scanner (MAGNETOM Skyra or Tim Trio, Siemens Medical Solutions, Erlangen, Germany). A standard ECG-triggering device and end-inspiratory breath holding were used during the procedure. To obtain cine images, a retrospectively gated balanced steady-state free precession (b-SSFP) sequence was employed with the following parameters: repetition time (TR) 2.81/3.4 ms, echo time (TE) 1.22/1.3 ms, flip angle 38°/50°, slice thickness 8 mm, field of view (FOV) 250 × 300 mm^2 or 340 × 285 mm^2, and matrix size 208 × 139 or 256 × 166. The cine images included short-axis slices from the mitral valve annulus to the LV apex, as well as four-chamber and two-chamber long-axis views.

CMR data analysis

Data were analyzed using commercial software (cvi42; Circle Cardiovascular Imaging, Inc., Calgary, AB, Canada). The epicardial and endocardial LV contours were manually traced by two experienced observers (each with more than 6 years of experience with CMR) at both the end-diastolic and end-systolic phases, excluding the papillary muscles. LV indices, including end-diastolic volume (LVEDV), end-systolic volume (LVESV), stroke volume (LVSV), and LV mass, were obtained and indexed to body surface area. LV ejection fraction (LVEF) was also calculated. The LVRI was calculated as LVM/LVEDV. The LVGFI was calculated using the following formula [22]: LVGFI=(LVSV/[(LVEDV + LVESV)/2 + (LVM/1.05)]) × 100.

LV global strain parameters, including global peak strain (PS), peak systolic strain rate (PSSR), and peak diastolic strain rate (PDSR) in the radial, circumferential, and longitudinal directions, were assessed on short-axis and long-axis cine images (2-chamber and 4-chamber views) using a feature-tracking module (Fig. 1).

MEE was estimated as: SBP×SV/ SBP×HR = SV/HR [23], where HR is expressed in seconds (HR/60). Since MEE is closely related to LV mass, it was normalized to LV mass in order to obtain an estimate of energetic expenditure per unit of myocardial mass, represented as indexed MEE (MEEI, ml/s/g).

Variability analysis

To assess intra-observer variability, a researcher measured LV global strain parameters twice in 40 randomly selected subjects, with a 1-month interval between measurements. Subsequently, a second researcher, blinded to the first researcher's results, reanalyzed the measurements. The variability between observers was then evaluated based on the results from both researchers, who were independent and double-blinded.

Statistical analysis

Continuous data are presented as either the mean ± SD or median (25th percentile, 75th percentile), depending on the results of normality testing. To assess differences between the NIDCM (MetS−) and NIDCM (MetS+) groups, Student's t-test or the Mann–Whitney U test was used. Categorical variables are presented as counts (percentages) and were compared using the chi-square test or Fisher's exact test, as appropriate. Spearman correlation analysis was used to assess relationships between variables. The association between MetS, TyG index and LV parameters in the NIDCM cohort was evaluated using multivariable linear regression, including variables with no collinearity and a P value < 0.1 in univariable analysis. Inter-observer and intra-observer agreement for LV strain measurements were quantified using intraclass correlation coefficients (ICCs). Statistical analysis was performed using SPSS (version 23.0, IBM SPSS Inc., Armonk, NY, USA) or GraphPad Prism (version 9.0, GraphPad Software Inc., San Diego, CA, USA). A two-tailed P value of < 0.05 was considered statistically significant.

Results

Baseline characteristics

The baseline demographic and clinical characteristics of the study cohort are summarized and compared in

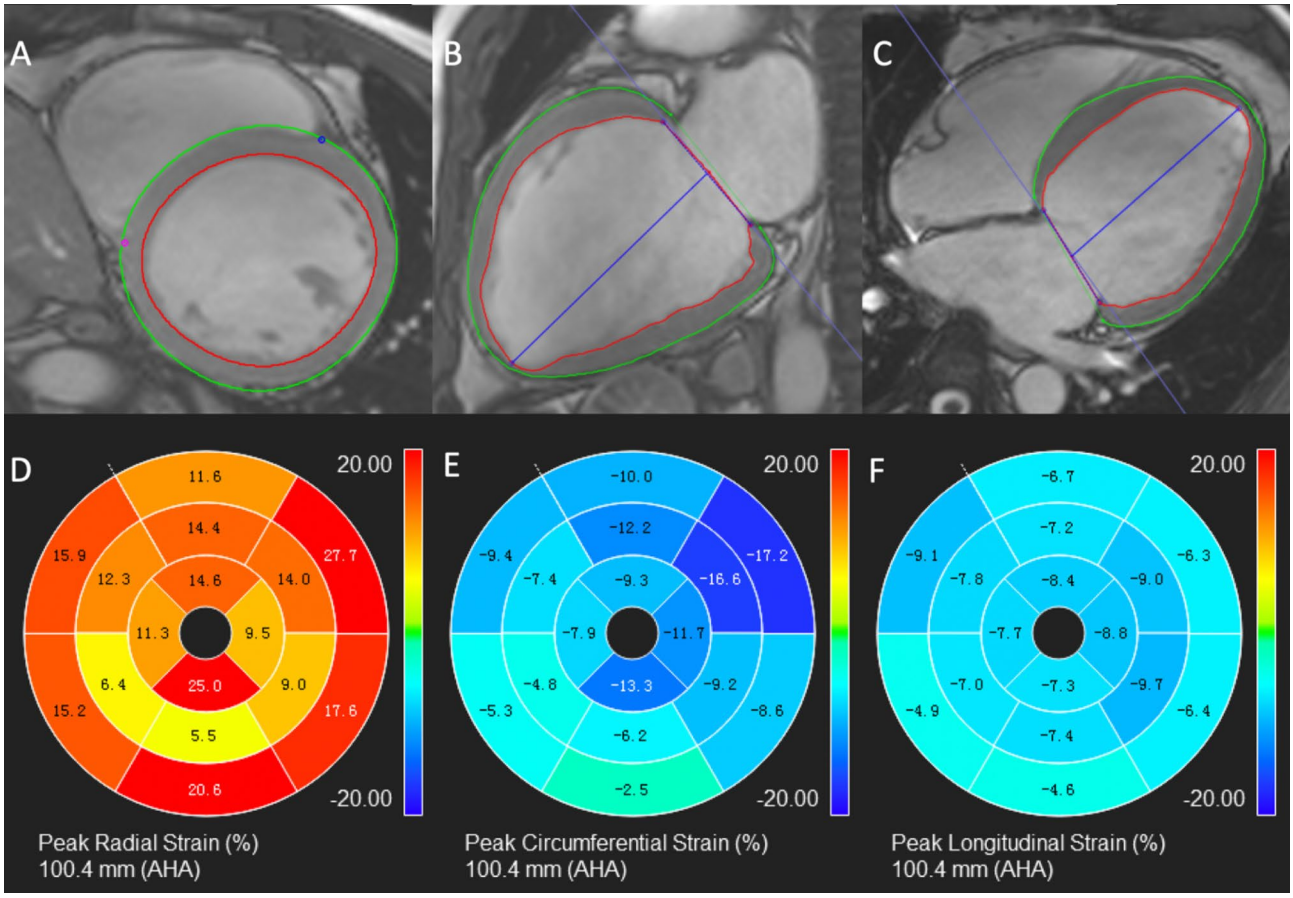

Fig. 1 Measurement of LV global peak strain. MR tissue tracking in short-axis (**A**), and long-axis 2-chamber (**B**), 4-chamber (**C**) cine images at end-diastole. The 3-dimensional pseudo-color maps of LV global peak strain in radial (**D**), circumferential (**E**), and longitudinal (**F**) directions

Table 1. Compared with the NIDCM(MetS−) group, BMI, HR, SBP, DBP, HbA1c, FBG, TG, Total cholesterol (TC), HDL-c, and low-density lipoprotein cholesterol (LDL-c) were higher in the NIDCM(MetS+) group ($p < 0.05$), while age, sex, and the history of smoking and drinking showed no statistically significant differences. The incidences of T2DM, HTN, and hyperlipidemia were significantly higher in the NIDCM(MetS+) group compared to the NIDCM(MetS−) group (all $p < 0.001$). The TyG index was significantly higher in the NIDCM(MetS+) group than in the NIDCM(MetS−) group (9.28 [8.97, 9.70] vs. 8.58 [8.26, 8.90], $p < 0.001$). Additionally, the NIDCM(MetS+) group had higher creatinine and cTnT levels than the NIDCM(MetS−) group ($p < 0.05$, respectively). There was no statistically significant difference in NT-proBNP between the NIDCM(MetS−) and the NIDCM(MetS+) groups ($p > 0.05$).

Comparison of CMR results among NIDCM with and without MetS

The LV functional and strain parameters are presented in Table 2. Compared with the NIDCM patients without MetS, those with MetS had decreased LVSVI, LVEF, and LVGFI, and increased LVMI, LVRI (all $p < 0.05$). There was no statistically significant difference in LVEDVI and LVESVI between the two groups ($p > 0.05$). In terms of LV strain parameters, NIDCM patients with MetS had worse LV global PS and PDSR in radial, circumferential, and longitudinal directions (all $p < 0.05$), as well as worse radial and longitudinal PSSR compared to those without MetS, while no significant difference was found in circumferential PSSR between these two groups ($p > 0.05$) (Fig. 2). In addition, the MEEI was significantly lower in the NIDCM(MetS+) group than in the NIDCM(MetS−) group (0.30 [0.20, 0.45] ml/s/g vs. 0.39 [0.25, 0.58] ml/s/g, $p < 0.001$).

Comparison of CMR-derived parameters between NIDCM with higher and lower TyG index

The CMR results between the TyG index < 8.79 group and the TyG index ≥ 8.79 group in patients with NIDCM are summarized in Table 3. There were no significant differences in LV CMR-derived parameters between higher and lower TyG index groups, including LVEDVI, LVESVI, LVSVI, LVEF, LVMI, LVGFI, LV PS, PDSR, and PSSR in all three directions, as well as MEEI (all $p > 0.05$).

Table 1 The baseline demographic and clinical characteristics of the study cohort

	NIDCM(MetS−) *N* = 378	NIDCM(MetS+) *N* = 179	*P* value
Age, years	50 (36, 61)	50 (40, 61)	0.478
Male, n	251 (66)	131 (73)	0.118
BMI, kg/m^2	22.65 (20.30, 22.62)	26.7 (24.84, 29.03)	< 0.001
SBP, mmHg	108 (97, 120)	116 (103, 130)	< 0.001
DBP, mmHg	72 (62, 80)	80 (68, 90)	< 0.001
Heart rate, b.p.m.	77.1 (66.5, 91.6)	81.9 (73.3, 93.9)	0.002
Smoking history, n	156 (41)	77 (43)	0.713
Drinking history, n	122 (32)	65 (36)	0.337
T2DM, n	46 (12)	62 (34)	< 0.001
HTN, n	36 (10)	84 (47)	< 0.001
Hyperlipidemia, n	152 (40)	145(81)	< 0.001
HbA1c, %	5.9 (5.6, 6.4)	6.4 (6.0, 7.3)	< 0.001
FBG, mmol/L	5.4 (4.8, 6.3)	6.7 (5.8, 8.1)	< 0.001
TG, mmol/L	1.14 (0.89, 1.59)	1.92 (1.46, 2.71)	< 0.001
TC, mmol/L	4.01 (3.28, 4.68)	4.21 (3.60, 5.01)	0.005
HDL-c, mmol/L	1.15 (0.89, 1.42)	0.91 (0.75, 1.06)	< 0.001
LDL-c, mmol/L	2.39 (1.84, 2.96)	2.60 (2.08, 3.10)	0.009
TyG index	8.58 (8.26, 8.90)	9.28 (8.97, 9.70)	< 0.001
Creatinine, μmol/L	81 (68, 98)	89 (75, 111)	< 0.001
eGFR, mL/ min/1.73 m^2	88.03 (71.33, 101.92)	85.49 (68.95, 96.21)	0.049
NT-proBNP, pg/mL	1608.50(788.75, 3791.50)	2206.00(658.50, 4465.50)	0.160
cTnT, ng/L	17.75 (11.08, 31.63)	20.75 (13.38, 39.98)	0.027

All values are presented as mean ± SD or median (Q1–Q3) or N (%)

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; T2DM, type 2 diabetes mellitus; HTN, hypertension; HbA1c, glycated hemoglobin; TyG index, triglyceride glucose index; FBG, Fasting blood glucose; TG, Triglycerides; TC, Total cholesterol; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; NT- proBNP, N-terminal pro-brain natriuretic peptide; cTnT, cardiac troponin T

Table 2 CMR parameters among NIDCM patients with and without MetS

	NIDCM(MetS−) *N* = 378	NIDCM(MetS+) *N* = 179	*P* value
LV Functional parameters			
LVEDVI (mL/m^2)	169.41 (138.44, 210.51)	170.53 (139.85, 204.39)	0.985
LVESVI (mL/m^2)	133.92 (97.98, 173.77)	140.23 (103.16, 168.85)	0.454
LVSVI (mL/m^2)	36.04 (26.83, 47.46)	32.01 (24.16, 42.90)	0.013
LVEF (%)	21.13 (14.82, 29.10)	19.56 (13.62, 25.94)	0.034
LVMI (g/m^2)	70.31 (59.41, 83.69)	77.57 (64.22, 90.50)	< 0.001
LVRI (g/mL)	0.41 (0.35, 0.48)	0.45 (0.38, 0.52)	< 0.001
LVGFI	16.51 (10.99, 22.94)	14.16 (9.84, 18.96)	0.004
LV Strain parameters			
GRPS, %	8.18 (5.33, 12.22)	6.07 (4.41, 8.39)	< 0.001
GCPS, %	− 6.43 (− 8.80, − 4.49)	− 5.32 (− 7.29, − 4.21)	< 0.001
GLPS, %	− 5.27 (− 7.22, − 3.83)	− 4.03 (− 5.49, − 2.71)	< 0.001
Radial PSSR, 1/sec	0.57 (0.40, 0.78)	0.49 (0.35, 0.61)	< 0.001
Circumferential PSSR, 1/sec	− 0.40 (− 0.54, − 0.30)	− 0.37 (− 0.50, − 0.28)	0.053
Longitudinal PSSR, 1/sec	− 0.37 (− 0.48, − 0.29)	− 0.31 (− 0.44, − 0.25)	0.004
Radial PDSR, 1/sec	− 0.59 (− 0.91, − 0.42)	− 0.52 (− 0.70, − 0.37)	0.001
Circumferential PDSR, 1/sec	0.48 (0.34, 0.68)	0.41 (0.33, 0.56)	< 0.001
Longitudinal PDSR, 1/sec	0.40 (0.30, 0.52)	0.35 (0.27, 0.46)	0.001
MEEI, ml/s/g	0.39 (0.25, 0.58)	0.30 (0.20, 0.45)	< 0.001

LV, left ventricular; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index; LVSVI, left ventricular stroke volume index; LVEF, left ventricular ejection fraction; LVRI, left ventricular remodeling index; LVGFI, left ventricular global function index; GRPS, global radial peak strain; GCPS, global circumferential peak strain; PSSR, peak systolic strain rate; PDSR, peak diastolic strain rate; MEEI, indexed myocardial energetic efficiency

Univariable and multivariable linear regression analyses of LV GLPS, LVRI, LVGFI, and MEEI

Table 4 shows the univariate and multivariate correlations of LV GLPS, LVRI, LVGFI, and MEEI. For all NIDCM patients in this cohort, the presence of MetS, TyG index, male sex, BMI, history of smoking and drinking, HR, creatinine, and NT-proBNP were positively associated with GLPS and LVRI, while eGFR was negatively associated with both GLPS and LVRI. cTnT was negatively associated with GLPS. In addition, the presence of MetS, male sex, history of smoking and drinking, HR, creatinine, NT-proBNP, and cTnT were negatively associated with LVGFI and MEEI.

After multivariable adjustment, the presence of MetS, male sex, HR, and NT-proBNP were independent determinants of decreased LV GLPS (β = 0.211, 0.184, 0.330, and 0.212, all $p < 0.05$). The presence of MetS, male sex, and BMI were independent determinants of LVRI (β = 0.147, 0.127, and 0.104, all $p < 0.05$). Male sex, HR, and NT-proBNP were independent determinants of LVGFI (β = − 0.135, − 0.354, and − 0.166, all $p < 0.05$). The presence of MetS, male sex, and NT-proBNP were independent determinants of MEEI (β = − 0.160, − 0.129, and − 0.192, all $p < 0.05$).

Interobserver and intraobserver variability

The intra- and inter-observer variability of CMR parameters are summarized in Table 5. Interobserver and intraobserver ICC were within the ranges of 0.906–0.969 and 0.897–0.987, respectively.

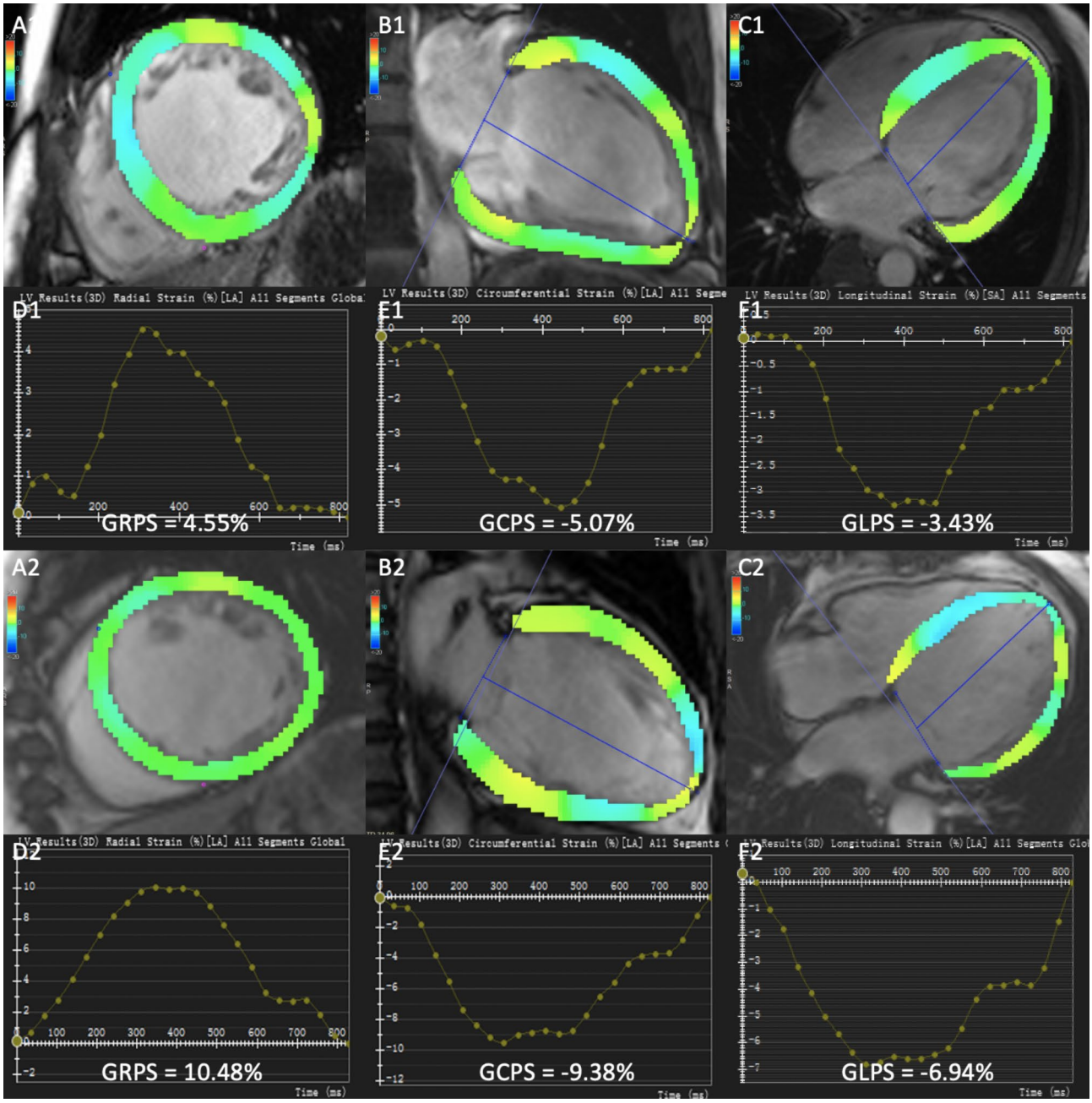

Fig. 2 Two groups of the representative CMR imaging. LV pseudo-color images of short-axis(**A1** and **A2**), long-axis four-(**B1** and **B2**) and two-chamber(**C1** and **C2**) cine images at the end-diastole and CMR imaging derived the global radial(**D1** and **D2**), circumferential(**E1** and **E2**), and longitudinal(**F1** and **F2**) peak strain curves. **A1**–**F1** a NIDCM(MetS+) subject, **A2**–**F2** a NIDCM(MetS–) patient

Discussion

The main findings of our study were as follows: (1) the presence of MetS significantly worsens LV function in NIDCM patients, as evidenced by a decrease in LVEF, LV strain and strain rate, as well as increase LVRI, and decrease LVGFI; (2) MEEI was significantly decreased in the NIDCM patients with MetS compared to those without MetS; (3) the higher TyG index was mildly correlated with decreased LV strains, while no significant differences in LV structural and functional parameters between the higher and lower TyG index subgroups; (4) multivariable linear regression analysis showed that the presence of MetS was an independent determinant of impaired LV GLPS, increased LVRI, and decreased MEEI. These findings suggest that the presence of MetS worsens LV function and myocardial energetic efficiency and contributes to more pronounced LV remodeling in NIDCM

Table 3 CMR parameters among NIDCM patients with higher and lower TyG index

	NIDCM (TyG index < 8.79) *N* = 279	NIDCM (TyG index ≥ 8.79) *N* = 278	*P* value
LV Functional parameters			
LVEDVI (mL/m^2)	167.84 (141.33, 215.63)	170.60 (135.89, 204.33)	0.311
LVESVI (mL/m^2)	134.25 (100.76, 179.55)	135.92 (98.87, 166.96)	0.477
LVSVI (mL/m^2)	35.70 (25.88, 46.48)	33.84 (25.66, 46.38)	0.417
LVEF (%)	20.52 (14.36, 28.06)	20.99 (14.44, 27.68)	0.778
LVMI (g/m^2)	70.95 (60.35, 84.05)	73.30 (61.54, 87.39)	0.376
LVRI (g/mL)	0.42 (0.35, 0.49)	0.43 (0.37, 0.50)	0.092
LVGFI	15.68 (10.53, 21.69)	16.05 (10.77, 22.07)	0.898
LV Strain parameters			
GRPS, %	7.41 (5.18, 11.23)	6.89 (4.61, 10.84)	0.212
GCPS, %	− 6.23 (− 8.46, − 4.38)	− 5.65 (− 8.02, − 4.33)	0.213
GLPS, %	− 4.98 (− 6.98, − 3.72)	−4.78 (− 6.69, − 3.06)	0.063
Radial PSSR, 1/sec	0.54 (0.40, 0.74)	0.52 (0.36, 0.72)	0.235
Circumferential PSSR, 1/sec	− 0.39 (− 0.51, − 0.29)	− 0.38 (− 0.53, − 0.30)	0.804
Longitudinal PSSR, 1/sec	− 0.36 (− 0.47, − 0.28)	− 0.36 (− 0.48, − 0.27)	0.944
Radial PDSR, 1/sec	− 0.57 (− 0.87, − 0.41)	− 0.56 (− 0.82, − 0.39)	0.372
Circumferential PDSR, 1/sec	0.48 (0.33, 0.66)	0.44 (0.34, 0.62)	0.180
Longitudinal PDSR, 1/sec	0.40 (0.29, 0.51)	0.38 (0.29, 0.49)	0.702
MEEI, ml/s/g	0.36 (0.23, 0.55)	0.34 (0.22, 0.52)	0.283

LV, left ventricular; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index; LVSVI, left ventricular stroke volume index; LVEF, left ventricular ejection fraction; LVRI, left ventricular remodeling index; LVGFI, left ventricular global function index; GRPS, global radial peak strain; GCPS, global circumferential peak strain; PSSR, peak systolic strain rate; PDSR, peak diastolic strain rate; MEEI, indexed myocardial energetic efficiency

patients, highlighting the adverse impact of metabolic abnormalities on cardiac structure and function.

MetS, a cluster of metabolic dysregulations, is identified as a significant risk factor for the development of heart failure (HF). The association between MetS and HF, as well as HF prognosis, is further supported by clinical studies [24, 25]. Dilated cardiomyopathy accounts for up to one-third of HF cases and is one of the leading causes of malignant ventricular arrhythmias and sudden cardiac death [26]. This study indicates that NIDCM patients who coexist with MetS have worse LV function and remodeling, including lower LVSVI, LVEF, and LVGFI, along with increased LVMI and LVRI. Regarding myocardial strain, NIDCM patients with MetS showed significantly worse LV strains and strain rates. These findings are consistent with previous studies that have demonstrated that MetS accelerates adverse LV remodeling, contributing to worsened cardiac function [27, 28], particularly pronounced in patients with additional cardiovascular conditions, such as obstructive coronary artery disease and myocardial infarction [4, 29]. The pathophysiological mechanisms underlying the additive effect of MetS on LV dysfunction in NIDCM patients are complex and multifactorial, involving increased abdominal obesity, elevated proinflammatory cytokines, and oxidative stress, all of which lead to reduced coronary blood flow and subendocardial hypoperfusion [28, 30, 31]. These factors contribute to cell apoptosis, tissue fibrosis, and impaired calcium handling, resulting in excitation-contraction coupling abnormalities [32]. Additionally, systolic dyssynchrony worsens due to MetS-related LV remodeling [33, 34, 35]. Interestingly, our study did not observe significant changes in LV volumes, such as LVEDVI and LVESVI, between NIDCM patients with and without MetS. This is in line with prior research, which suggests that metabolic abnormalities associated with MetS primarily influence myocardial function and mass without leading to significant alterations in LV dilation and volume [3].

MEEI, which is relatively simple to calculate and can be obtained through non-invasive exams like echocardiography or CMR, has recently become an important indicator for assessing myocardial structure, function, and oxygen consumption [13, 23]. NIDCM is characterized by an imbalance between LV performance and myocardial energy consumption. Therefore, MEEI in NIDCM patients is crucial, as it reflects the relationship between oxygen consumption and LV performance and outcomes [36, 37]. In NIDCM patients, despite markedly impaired LV work, the oxygen cost of contraction remains relatively unchanged, resulting in a decrease in the mechanical efficiency of contraction [36]. Previous studies have shown that low MEEI is associated with an increased risk of HF events. Additionally, prior studies have confirmed that MetS exacerbates MEEI, and impaired MEE has been linked to various cardiovascular diseases and metabolic factors [11, 38]. Similarly, our study found that MEEI was lower in NIDCM patients with MetS compared to those without MetS, and the presence of MetS was an independent determinant of MEEI. The underlying mechanisms of this injury are related to metabolic and hemodynamic changes, including insulin resistance, concentric LV geometry, LV diastolic dysfunction, and discrete systolic dysfunction.

Furthermore, the TyG index, a marker of insulin resistance, was higher in the NIDCM patients with MetS than those without, but no significant differences in LV structural and functional parameters were found between NIDCM patients with higher and lower TyG index. A higher TyG index showed mild correlations with decreased LV GLPS and increased LVRI. This weak association possibly suggests that while the TyG index primarily reflects one aspect of metabolic

Table 4 Determinants of impaired LV function in NIDCM patients

	GLPS				LVRI				LVGFI				MEEI			
	Univariable		Multivariable		Univariable		Multivariable		Univariable		Multivariable		Univariable		Multivariable	
	r	*P* value	β	*P* value	r	*P* value	β	*P* value	r	*P* value	β	*P* value	r	*P* value	β	*P* value
MetS	0.256	< 0.001	0.211	< 0.001	0.156	< 0.001	0.147	0.003	− 0.123	0.004			− 0.169	< 0.001	− 0.160	< 0.001
TyG index	0.089	0.035			0.109	0.010			− 0.020	0.644			− 0.065	0.124		
Age, y	0.068	0.107			0.075	0.075			0.018	0.667			0.022	0.604		
Male, n	0.181	< 0.001	0.184	< 0.001	0.181	< 0.001	0.127	0.005	− 0.158	< 0.001	− 0.135	0.001	− 0.162	< 0.001	− 0.129	0.004
BMI, kg/m^2	0.095	0.025			0.211	< 0.001	0.104	0.038	− 0.004	0.927			− 0.080	0.060		
Smoking, n	0.119	0.005			− 0.124	0.004			− 0.124	0.003			− 0.107	0.012		
Drinking, n	0.085	0.044			0.125	0.003			− 0.115	0.007			− 0.116	0.006		
HR, b.p.m.	0.410	< 0.001	0.330	< 0.001	0.021	0.621			− 0.273	< 0.001	− 0.354	< 0.001	− 0.622	< 0.001		
Creatinine, μmol/L	0.220	< 0.001			0.108	0.010			− 0.132	0.002			− 0.131	0.002		
eGFR, mL/ min/1.73 m^2	−0.091	0.040			− 0.052	0.245			0.045	0.318			0.046	0.303		
NT-proBNP, pg/mL	0.403	< 0.001	0.212	< 0.001	− 0.165	< 0.001			− 0.386	< 0.001	− 0.166	< 0.001	− 0.330	< 0.001	− 0.192	< 0.001
cTnT, ng/L	0.238	< 0.001			0.061	0.158			− 0.246	< 0.001			− 0.215	< 0.001		

MetS, metabolic syndrome; TyG index, triglyceride glucose index; BMI, body mass index; eGFR, estimated glomerular filtration rate; HR, heart rate; NT- proBNP, N-terminal pro-brain natriuretic peptide; cTnT, cardiac troponin T; GLPS, global longitudinal peak strain; LVRI, left ventricular remodeling index; LVGFI, left ventricular global function index; MEEI, indexed myocardial energetic efficiency

The multivariable linear regression of GLPS combined MetS, TyG index, age, male sex, BMI, history of smoking and drinking, HR, creatinine, eGFR, NT-proBNP, and cTnT

The multivariable linear regression of LVRI combined MetS, TyG index, age, male sex, BMI, history of smoking and drinking, creatinine, and NT-proBNP

The multivariable linear regression of LVGFI combined MetS, age, male sex, history of smoking and drinking, HR, creatinine, NT-proBNP, and cTnT

The multivariable linear regression of MEEI combined MetS, age, male sex, BMI, history of smoking and drinking, creatinine, NT-proBNP, and cTnT

Table 5 Interobserver and intraobserver variabilities of CMR parameters

	Interobserver		Intraobserver	
	ICC	95%CI	ICC	95%CI
LVEDVI, mL/m^2	0.959	0.923–0.978	0.961	0.927–0.979
LVESVI, mL/m^2	0.969	0.942–0.983	0.968	0.940–0.983
LVMI, g/m^2	0.946	0.901–0.971	0.945	0.898–0.970
PS, %	0.923	0.859–0.958	0.944	0.898–0.970
Radial	0.951	0.909–0.974	0.987	0.975–0.993
Circumferential	0.915	0.845–0.954	0.961	0.927–0.979
Longitudinal	0.934	0.879–0.965	0.931	0.873–0.963
PSSR, 1/sec	0.906	0.830–0.949	0.923	0.860–0.959
Radial	0.918	0.851–0.956	0.924	0.861–0.959
Circumferential	0.939	0.888–0.967	0.937	0.884–0.966
Longitudinal	0.909	0.835–0.951	0.915	0.845–0.954
PDSR, 1/sec	0.915	0.846–0.954	0.897	0.814–0.944
Radial				
Circumferential				
Longitudinal				

LV, left ventricular; LVEDVI, left ventricular end-diastolic volume index; LVESVI, left ventricular end-systolic volume index; LVMI, left ventricular mass index; ICC, intraclass correlation coefficient; CI, confidence interval; PS, peak strain; PSSR, peak systolic strain rate; PDSR, peak diastolic strain rate

status—triglycerides and blood glucose levels—its impact on LV function is likely more indirect. LV function can be influenced by multiple factors, such as cardiac structure, hemodynamics, and neuroendocrine activation. Therefore, the TyG index may not fully capture these complex interactions, resulting in a weaker or nonsignificant impact on LV strains, remodeling, and MEEI in patients with NIDCM.

This study found that male sex was an independent determinant of LV GLPS, LVRI, LVGFI, and MEEI. Men with NIDCM may be more prone to myocardial fibrosis, resulting in increased myocardial stiffness and decreased longitudinal systolic function [39]. Estrogen is known to have protective effects on the heart in women, and the absence of this protection in men may contribute to a greater vulnerability to myocardial dysfunction [40]. Moreover, men are more susceptible to the components of MetS (e.g., high blood pressure, diabetes), which can exacerbate LV dysfunction and impair myocardial energetic efficiency, and contribute to more pronounced LV remodeling. These findings are consistent with previous studies suggesting that sex differences may play a significant role in the progression of heart disease, particularly in conditions like NIDCM [39]. Our study also highlighted that patients with NIDCM and MetS exhibited higher cTnT levels compared to those without MetS. Elevated cTnT levels are indicative of myocardial injury and can serve as a marker of cardiac stress. Additionally, NT-proBNP was identified as an independent determinant of LV GLPS and MEEI, with elevated levels of NT-proBNP reflecting increased cardiac load, myocardial fibrosis, and neuroendocrine activation [41]. These factors contribute to the decline in myocardial contractile function, further supporting the link between metabolic abnormalities, inflammation, and myocardial dysfunction.

The limitations of the current study were as follows: (1) it was a retrospective study, and patients were recruited from a single and tertiary referral center; therefore, potential bias may exist; (2) myocardial fibrosis is a key factor in the prognosis of NIDCM patients, and the relationship between myocardial fibrosis and LV dysfunction requires further investigation in future studies; (3) heart rate can be an unstable indicator. Although we excluded patients with arrhythmias that could affect the measurements and collected heart rate data in a quiet state, some potential limitations may remain; (4) long-term follow-up data on clinical outcomes, such as cardiovascular events or mortality, were not available. Future studies with larger sample sizes are needed to provide a more comprehensive analysis of the associations between impaired imaging parameters and poor outcomes.

Conclusion

This study highlights the significant association between MetS and LV dysfunction in NIDCM patients. The presence of MetS worsens LV deformation, remodeling, and MEEI, contributing to pronounced LV remodeling in patients with NIDCM. It was independently associated with impaired LV GLPS, LVRI, and MEEI. These findings suggest that addressing metabolic abnormalities may provide valuable insights into the progression of NIDCM and guide therapeutic strategies aimed at mitigating further cardiac deterioration.

Abbreviations

MetS	Metabolic syndrome
NIDCM	Non-ischemic dilated cardiomyopathy
LV	Left ventricular
CMR	Cardiac magnetic resonance
LVRI	Left ventricular remodeling index
LVGFI	Left ventricular global function index
MEE	Myocardial energetic efficiency
MEEI	Indexed myocardial energetic efficiency
TyG	Triglyceride–glucose index
T2DM	Type 2 diabetes mellitus
HTN	Hypertension
GRPS	Global radial peak strain
GCPS	Global circumferential peak strain
GLPS	Global longitudinal peak strain
LGE	Late gadolinium enhancement
HR	Heart rate

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Author contributions

MTS, YL and ZGY designed the study. MTS interpreted the data and wrote the manuscript. MTS and YL analyzed the data and gave advice on data presentation. MTS, KS, and WFYwere responsible for collecting and sorting statistical data. JW, LJ, SQY participated in editing and review of the manuscript. YG, YJ, and XML supervised the overall study and reviewed the manuscript. YL and ZGY are the guarantor of this work and had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study protocol was approved by the Biomedical Research Ethics Committee of our hospital. Informed consent was waived due to the retrospective nature of the research. The patient-sensitive data were protected with full confidentiality.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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