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Association between metabolic visceral fat score and left ventricular hypertrophy in individuals with type 2 diabetes

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Abstract

Background Left ventricular hypertrophy (LVH), a hallmark of early-stage heart failure (HF), is a common complication in individuals with type 2 diabetes mellitus (T2DM). Metabolic Visceral Fat Score (METS-VF), a novel metric for estimating visceral adiposity, may provide valuable insights into LVH risk. This study explores the association between METS-VF and LVH in T2DM and compare its predictive performance to traditional abdominal obesity indices.

Methods This cross-sectional study included 4,988 adults with T2DM. Participants were stratified into quartiles based on METS-VF. Logistic regression models assessed the association between METS-VF and LVH. Restricted cubic spline analyses evaluated nonlinear relationships, while stratified analyses explored subgroups effects. Receiver operating characteristic (ROC) curves compared the predictive performance of METS-VF with other indices.

Results LVH prevalence increased across METS-VF quartiles (Quartile 1: 7.9%; Quartile 2: 13.0%; Quartile 3: 20.0%; Quartile 4: 31.0%; $P < 0.001$). Higher METS-VF was independently associated with LVH (OR: 9.79; 95% CI: 6.16–15.76; $P < 0.001$). A nonlinear relationship was observed between METS-VF and LVH, with a steeper risk increase above specific thresholds. Stratified analyses showed that the positive association between METS-VF and LVH was consistent. METS-VF outperformed traditional indices in predicting LVH (AUC: 0.68; 95% CI: 0.66–0.70).

Conclusions METS-VF is strongly associated with LVH in T2DM, demonstrating superior predictive performance compared to traditional indices. METS-VF is a practical, cost-effective tool for early cardiac risk stratification, facilitating timely interventions to mitigate HF risk in T2DM populations.

Keywords Metabolic visceral fat score, Left ventricular hypertrophy, Type 2 diabetes mellitus, Heart failure

Introduction

Left ventricular hypertrophy (LVH) is a common structural cardiac complication in type 2 diabetes mellitus (T2DM) and represents a key marker of subclinical heart failure (HF) [1]. LVH is associated with an increased risk of heart failure with preserved ejection fraction (HFpEF), ischemic heart disease, and sudden cardiac death, making its early detection critical for cardiovascular risk stratification [2]. Importantly, studies have demonstrated that early identification and reversal of LVH can significantly reduce cardiovascular morbidity and mortality in patients with T2DM, emphasizing the need for effective screening tools [3].

Among the pathophysiological mechanisms linking T2DM to LVH, visceral adipose tissue (VAT) accumulation has been recognized as a major contributor to

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adverse cardiac remodeling. Unlike subcutaneous adipose tissue (SAT), VAT is metabolically active, promoting a pro-inflammatory, lipotoxic, and insulin-resistant environment that accelerates myocardial fibrosis, hypertrophy, and diastolic dysfunction [4–6]. Despite its clinical significance, VAT is challenging to measure accurately in routine practice. Advanced imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI) provide direct VAT assessment but are costly, time-consuming, and impractical for large-scale screening. In contrast, traditional anthropometric measures like body mass index (BMI), waist circumference (WC), and waist-to-hip ratio (WHR) do not distinguish VAT from SAT and fail to capture the true cardiometabolic burden of visceral fat [7].

To overcome these limitations, novel VAT indices such as the abdominal volume index (AVI), lipid accumulation product (LAP), and Chinese visceral adiposity index (CVAI) have been proposed as more accurate surrogates of VAT and better predictors of cardiovascular risk [8]. However, these indices still have limitations, as they do not fully integrate metabolic parameters. Recently, the Metabolism Score for Visceral Fat (METS-VF) has emerged as a promising composite index that incorporates demographic factors (age, sex), anthropometric measures (BMI, WC) and metabolic markers [fasting plasma glucose (FPG), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C)] to provide a more dynamic assessment of VAT and its metabolic impact. METS-VF has been validated as a strong predictor of T2DM onset, hypertension, and metabolic dysfunction, and recent evidence suggests its potential role in assessing cardiovascular risk [9–11]. However, its relationship with LVH in T2DM patients remains largely unexplored, despite the well-established role of VAT in cardiac remodeling and HF progression.

This cross-sectional study aims to evaluate the association between METS-VF and LVH in patients with T2DM and to determine whether METS-VF outperforms conventional VAT indices in predicting LVH. We hypothesize that higher METS-VF is associated with increased LVH prevalence, reflecting the role of visceral adiposity-driven metabolic dysfunction in subclinical myocardial remodeling. By addressing this knowledge gap, our findings could inform early cardiac risk stratification strategies and help guide preventive interventions in T2DM patients at risk for HF.

Methods

Study population

This cross-sectional study was conducted at Capital Medical University Affiliated Beijing Luhe Hospital, a National Metabolic Management Centers (MMC) in China. Details of the MMC project have been described previously [12]. Between October 2017 and July 2023, a total of 7,255 participants aged 18 years or older and diagnosed with diabetes mellitus (DM) were recruited from the Department of Endocrinology. Diagnosis of T2DM was based on the 1999 Health Organization criteria. For this analysis, we excluded participants with non-T2DM types (n = 400), those with a history of atherosclerotic cardiovascular disease, heart failure (HF), or Malignant carcinoma (n = 1447), and those with missing echocardiographic data (n = 303) or incomplete METS-VF calculations (n = 117). A flow diagram detailing patient enrollment is provided in Fig. 1.

The study protocol was approved by the Ethics Committee of Capital Medical University Affiliated Beijing Luhe Hospital. All procedures confirmed to the ethical guidelines of the 1975 Declaration of Helsinki, and all participants provided consent prior to participation.

Data collection

Participants completed a standardized questionnaire and underwent clinical and laboratory examinations during both registration and follow-up visits. All data were recorded in an MMC-specialized electronic medical record system. Trained staff collected the following baseline demographic and clinical data:

- Demographics: Age, sex, education level (categorized as middle school or below and high school or above).
- Lifestyle factors: Smoking and drinking status (categorized as never, current, or former), and weight loss in the previous year (categorized as no loss, < 5 kg, and > 5 kg).
- Anthropometric and physical measurements:

  Height and weight were recorded twice (barefoot and in lightweight clothing) to the nearest 0.1 cm and 0.1 kg, respectively.
  Waist circumference (WC) was measured at the midpoint between the iliac crest and the lower rib margin, and hip circumference (HC) was measured at the widest part of the buttocks.
  BMI was calculated as weight divided by height squared (kg/m^2), and waist-to-hip ratio (WHR) was calculated as WC divided by HC.
  Blood pressure (BP) and heart rate (HR) were measured twice using a calibrated automated electronic device (HEM-752 FUZZY, Omron, China) after a 5-min rest; the mean of the two readings was used for analysis.
  Visceral and subcutaneous fat (VAT and SAT) were assessed using Dual-energy X-ray absorptiometry (DXA) (GE/Lunar Radiation Corp, Madison, WI).

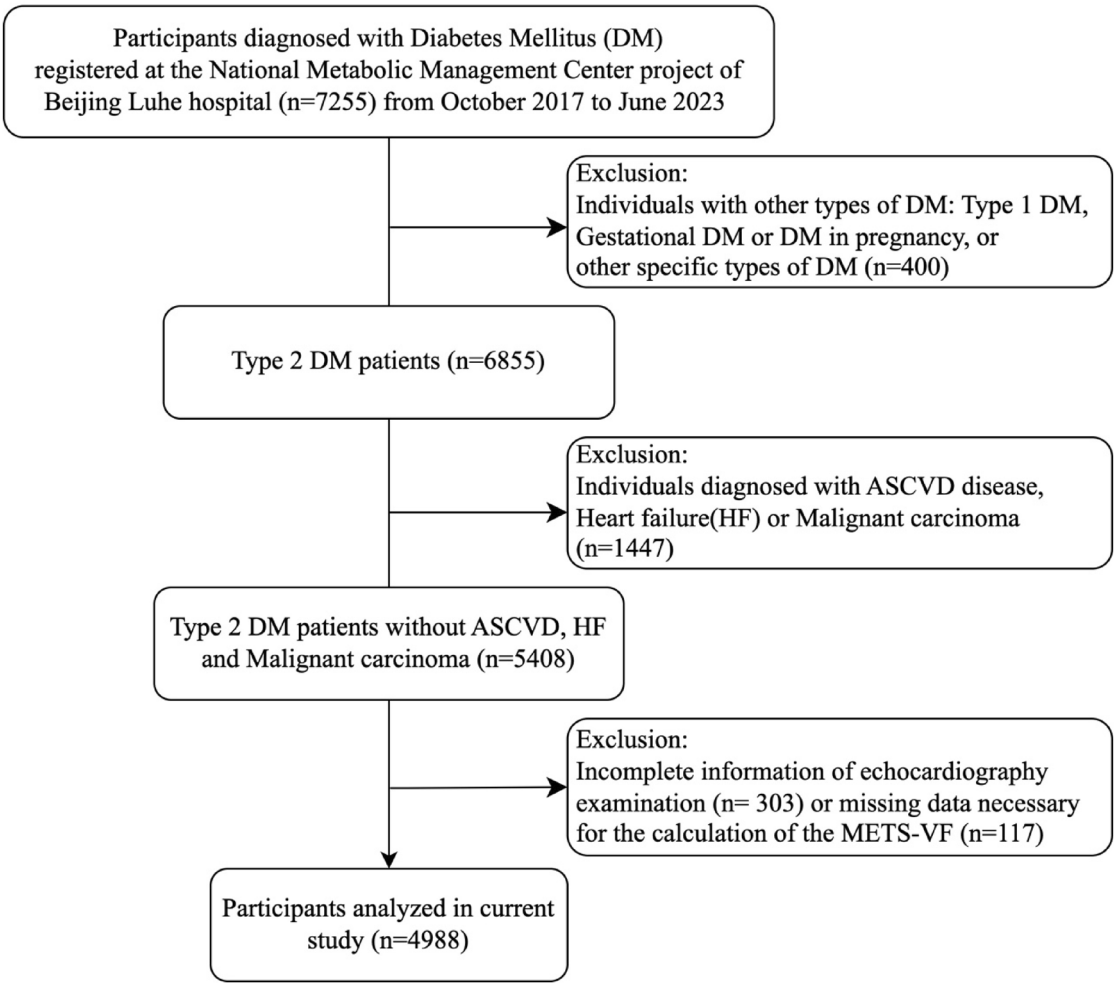

Fig. 1 Flowchart of the selection process of study subjects. ASCVD atherosclerotic cardiovascular disease, METS-VF Metabolic Visceral Fat Score

- Medication history: The use of insulin, metformin, sodium-glucose cotransporter-2 inhibitors (SGLT2i), glucosidase inhibitors, glinides, sulfonylureas, dipeptidyl peptidase 4 inhibitors (DPP4i), glucagon-like peptide-1 receptor agonists (GLP-1 RA), beta-blockers, renin-angiotensin system inhibitors (RASi) including angiotensin receptor-neprilysin inhibitors (ARNI), angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), calcium channel blockers (CCBs), diuretics, statins, and anti-platelet drugs, was also recorded.
- Laboratory Measurements: After an overnight fast, blood samples were collected, and the following biochemical parameters were measured:

  Lipid profiles: Triglyceride (TG), total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C).

  Liver function tests: Alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALP), γ-glutamyl transferase (GGT), albumin,

  Kidney function: creatinine (Cr), blood urea nitrogen (BUN), uric acid (UA), urinary albumin/creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation for individuals of Asian descent.

  Fasting blood glucose(FBG), 2-h post-glucose challenge blood glucose, insulin, and C-peptide levels.

  Hemoglobin A1c (HbA1c): Measured using high-performance liquid chromatography.

  Other laboratory indices: Complete blood counts [red blood cell count (RBC), white blood cell count (WBC), platelet count (PLT), hemoglobin (Hb), hematocrit (Hct), and mean corpuscular hemoglobin (MCH)] were also measured.

- Insulin resistance (IR) indices were calculated:
- Homeostasis model assessment of insulin resistance (HOMA-IR),

$$\text{HOMA - IR} = \frac{\text{Fasting insulin } (\mu\text{IU/mL}) \times \text{FBG (mmol/L)}}{22.5}$$

- Metabolic score for insulin resistance (METS-IR) [13]

$$\text{METS - IR} = \frac{\text{Ln}\left[2 \times \text{FBG}\left(\text{mg/dL}\right) + \text{TG}\left(\text{mg/dL}\right)\right] \times \text{BMI}}{\text{Ln}\left(\text{HDL - C}\left(\text{mg/dL}\right)\right)}$$

- Triglyceride glucose (TyG) [14]

$$\text{TyG index} = \text{Ln}\left[\frac{\text{FBG}\left(\text{mg/dL}\right) \times \text{TG}\left(\text{mg/dL}\right)}{2}\right]$$

Abdominal obesity indices

The following formulas were used to calculate METS-VF [15] and other abdominal obesity indices:

$$\begin{aligned}\text{METS - VF} &= 4.466 + 0.011 \times (\text{Ln (METS - IR)})^3 \\ &+ 3.239 \times (\text{Ln (WHtR)})^3 + 0.319 \\ &\times \text{Sex} + 0.594 \times \left(\text{Ln}\left(\text{Age}\right)\right).\end{aligned}$$

where Sex is coded as male = 1 and female = 0.

Abdominal Volume Index (AVI)[16]

$$\text{AVI} = \frac{\left(2 \times \text{WC}^2 + 0.7 \times \text{WC - HC}^2\right)}{1000}$$

where WC is waist circumference in cm and HC is hip circumference in cm.

Lipid Accumulation Product (LAP)[17]:

$$\text{LAP in male} = (\text{WC- } 65) \times \text{TG}$$

$$\text{LAP in female} = (\text{WC- } 58) \times \text{TG}$$

where WC is waist circumference in cm and TG is triglycerides in mmol/L.

Cardiometabolic Index (CMI) [18]:

$$\text{CMI} = \frac{\text{TG}}{\text{HDL - C}} \times \frac{\text{WC}}{\text{Height}}$$

where TG is in mg/dL, HDL-C is in mg/dL, WC is in cm, and Height is in cm.

Chinese Visceral Adiposity Index (CVAI) [19]:

Males:

$$\begin{aligned}CVAI &= -267.93 + 0.68 \times \text{Age} + 0.03 \times \text{BMI} + 4.00 \\ &\times \text{WC} + 22.00 \times \text{Lg (TG)} - 16.32 \times \text{HDL - C}\end{aligned}$$

Females:

$$\begin{aligned}CVAI &= -187.32 + 1.71 \times \text{Age} + 4.32 \times \text{BMI} + 1.12 \\ &\times \text{WC} + 39.76 \times \text{Lg (TG)} - 11.66 \times \text{HDL - C}\end{aligned}$$

where Age is in years, BMI is body mass index in kg/m^2, WC is waist circumference in cm, TG is triglycerides in mmol/L, and HDL-C is in mmol/L.

Echocardiography assessment

Echocardiographic assessments were performed using the General Electric Healthcare Vivid E9 ultrasound system (GE, Illinois, USA). Experienced echocardiographers, blinded to clinical details, evaluated LV dimensions and wall thickness according to the American Society of Echocardiography recommendations. The following indices were used to evaluate LV structure and function:

LV mass (LVM): Derived from the Devereux formula according to the ASE recommendations

$$\text{LVM} = 0.8 \times [1.04 \times (\text{LV internal diameter} + \text{interventricular septal diameter} + \text{posterior wall diameter})^3 - (\text{LV internal diameter})^3] + 0.6\ \text{g}$$

LV mass index (LVMI): LVM indexed to height to the power of 2.7 (m$^{2.7}$). LVH was defined by LVMI > 46.7 g/m$^{2.7}$ in women and > 49.2 g/ m$^{2.7}$ in men [21].

Relative wall thickness (RWT): Calculated as septal wall thickness plus posterior wall thickness divided by LV end-diastolic diameter.

LV end-diastolic dimension (LVEDD), LV end-systolic dimension (LVESD) and LV ejection fraction (LVEF).

Left ventricular geometric patterns were defined as follows: normal geometry (no LVH, RWT ≤ 0.42), concentric remodelling (no LVH, RWT > 0.42), concentric hypertrophy (LVH present, RWT > 0.42), and eccentric hypertrophy (LVH present, RWT ≤ 0.42).

Statistical analysis

Continuous variables were expressed as mean ± SD or median (IQR) for normal or skewed distributions, respectively. Categorical variables were reported as frequencies and percentages (%). To compare baseline characteristics across quartiles of METS-VF and between groups with or without LVH, one-way analysis of variance (ANOVA) was used for continuous variables, and the Chi-squared test was applied for categorical variables.

Logistic regression models were used to assess the association between METS-VF and LVH risk and LV geometry types, with odds ratios (ORs) and 95% confidence intervals (CIs) calculated for each model and adjusted for confounders in three models: Model 1: Adjusted for age, sex, DM duration, level of education, smoking status, drinking status, and weight loss in last 1 year.

Model 2: further adjusted for SBP, HR, VAT, SAT and HT.

Model 3: further adjusted for FBG, fasting insulin and C-peptide, 2-h insulin and C-peptide, HOMA-IR, TG, TC, LDL-c, ALT, ALB, eGFR, ACR category, BUN, Hb, RBC, WBC, HCT, MCH, and medication use.

To explore linear or non-linear associations between METS-VF and LVH risk, restricted cubic spline (RCS) regression models were used to fit dose–response curves. ORs and 95% CIs were derived from the logistic regression model, with the 10th percentile set as the reference value (OR = 1.00) and knots placed at the 10th, 50th and 90th percentiles of the ln-transformed concentrations. Stratified analyses were performed across subgroups based on age (< 50 or > = 50 years), sex (male or female), DM duration (< 36 months or > = 36 months), education level (< High school or > = High school), BMI (< 24 kg/m 2 or > = 24 kg/m 2), HbA1c (< 7% or > = 7%), HOMA-IR (< 2 OR > = 2), HT (yes or no), and ACR category (< 30 mg/g, 30–300 mg/g or > = 300 mg/g). Interaction effects were evaluated using the likelihood ratio test, comparing models with and without interaction terms. Receiver operating characteristic (ROC) analysis was conducted to evaluate the diagnostic performance of METS-VF and other abdominal indices (WC, HC, VAT, BMI, WHR, AVI, LAP, CMI, CVAI) in predicting LVH risk. Areas under the curve (AUC) of receiver operating characteristics (ROCs) were calculated to assess each marker's ability to predict LVH risk, with optimal cutoff values identified by Youden's index. All analyses were performed using RStudio (version 2023.09.1 + 494 for Mac OS), employing packages such as gtsummary, rcssci, tidyverse, broom, ggplot2, splines, forestploter, and lcmm. All tests were two-tailed, and a P value of less than 0.05 was considered statistically significant.

Results

A total of 7255 patients with DM were recruited for the MMC project from October 2017 to June 2023. Among these, 4988 patients with T2DM were included in this analysis (Fig. 1). The mean age of participants was 49.8 ± 12.4 years, with 41.8% of participants being female. The median METS-VF was 6.95 (IQR 6.64–7.21), with quartiles defined as follows: Quartile 1: 4.53–6.64, Quartile 2: 6.64–6.95, Quartile 3: 6.95–7.20, and Quartile 4: 7.20–8.09.

Clinical characteristics and echocardiographic parameters

Table 1 presents the clinical characteristics of participants stratified by METS-VF quartiles. Compared to those in lower quartiles, participants in higher METS-VF quartiles were generally: older, more likely to be male, and had lower education levels (all P < 0.01), more likely to be current smokers or drinkers (P < 0.01) and exhibited greater stability in weight (P < 0.01). Additionally, higher METS-VF quartiles were associated with an increased prevalence of hypertension (HT), dyslipidemia, arrhythmia, and fatty liver. Patients in higher quartiles also had significantly higher levels of SBP, DBP, height, weight, WC, HC, VAT, and SAT (all P < 0.01). Biochemical parameters such as HbA1c, fasting blood glucose (FBG), fasting insulin, C-peptide levels, and insulin resistance indices (HOMA-IR, METS-IR, TyG) were also higher in the higher quartiles of METS-VF, while HDL-C, albumin (ALB), eGFR, and platelets (PLT) were lower (all P < 0.01). Use of medications, including insulin, metformin, SGLT2i, GLP-1 RA, β-blockers, renin-angiotensin blockers, and statins, was more common in the higher METS-VF quartiles (all P < 0.01). Baseline characteristics comparing LVH and non-LVH T2DM patients are detailed in Supplementary Table 1.

Echocardiographic parameters, shown in Fig. 2, revealed that participants in higher METS-VF quartiles had significantly: greater interventricular septal thickness and LV posterior wall thickness, larger LV end-diastolic dimension, LV end-systolic dimension and LAD, higher LVMI, and RWT (all P < 0.01). Higher METS-VF quartiles were also associated with a higher prevalence of abnormal LV ejection fraction (LVEF) (P < 0.01).

Association between METS-VF and left ventricular structure and function

Table 2 presents the association between METS-VF and LVH prevalence. The overall prevalence of LVH was 17.9%. Higher METS-VF quartiles were associated with a significantly higher prevalence of LVH: Q1: 7.9%, Q2: 13.0%; Q3: 20.0% Q4: 31.0%, P < 0.001). Similarly, higher METS-VF quartiles were associated with an increased risk of concentric hypertrophy (Q1: 5.85%; Q2: 7.62%; Q3: 13.0%; Q4: 18.8%, P < 0.001) and eccentric hypertrophy (Q1: 2.00%; Q2: 5.37%; Q3: 6.98%; Q4: 12.1%, P < 0.001). When analyzed as a continuous variable, each 1-unit increase in METS-VF was significantly associated with a higher risk of LVH: Crude Model: OR (95% CI): 5.32 (4.32—6.57), P < 0.001; Model 1: OR (95% CI): 6.91 (5.41–8.88), P < 0.001; Model 2: OR (95% CI): 7.88 (5.30–11.82), P < 0.001; Model 3: OR (95% CI): 9.79 (6.15–15.76), P < 0.001. The highest METS-VF Q(Q4) showed the strongest association with LVH across all models, with ORs ranging from 5.26 (4.16–6.70) in the crude model to 5.97 (3.86–9.32) in Model 3.

For eccentric hypertrophy, METS-VF quartiles were associated with a higher risk in the following order: Q2:

Table 1 Baseline characteristics of T2D patients according to METS-VF quartiles

Characteristic	Overall, N = 4,988	Quartile 1 (4.53–6.64)	Quartile 2 (6.64–6.95)	Quartile 3 (6.95–7.20)	Quartile 4 (7.20–8.09)	p-value
Age, (years)[a]	49.75 ± 12.38	46.06 ± 11.99	48.24 ± 12.10	50.81 ± 12.10	53.90 ± 11.95	**< 0.001**
Female Sex, n(%)	2087 (41.8%)	713 (57.2%)	575 (46.1%)	492 (39.5%)	307 (24.6%)	**< 0.001**
DM duration, (months)[b]	72.66 ± 80.90	66.71 ± 76.27	72.15 ± 80.70	73.60 ± 79.77	78.18 ± 86.24	0.090
Education Level, n(%)						**< 0.001**
Middle school or below	1896 (38.3%)	370 (29.7%)	453 (36.5%)	468 (37.8%)	605 (49.1%)	
High school or above	3060 (61.7%)	874 (70.3%)	787 (63.5%)	771 (62.2%)	628 (50.9%)	
Smoking Status, n(%)						**< 0.001**
Former/ Current	1986 (39.8%)	367 (29.4%)	454 (36.4%)	530 (42.5%)	635 (50.9%)	
Drinking Status, n(%)						**< 0.001**
Former/ Current	2315 (46.4%)	447 (35.8%)	552 (44.3%)	608 (48.8%)	708 (56.8%)	
Weight loss In Last 1 Year, n(%)						**< 0.001**
No loss	3325 (66.7%)	764 (61.3%)	795 (63.8%)	868 (69.6%)	898 (72.0%)	
< 5kg	991 (19.9%)	285 (22.9%)	268 (21.5%)	238 (19.1%)	200 (16.0%)	
≥ 5kg	672 (13.5%)	198 (15.9%)	184 (14.8%)	141 (11.3%)	149 (11.9%)	
Comorbidity, n (%)						
Hypertension	2869 (57.5%)	478 (38.3%)	663 (53.2%)	807 (64.7%)	921 (73.9%)	**< 0.001**
Dyslipidemia	3756 (75.3%)	816 (65.4%)	948 (76.0%)	995 (79.8%)	997 (80.0%)	**< 0.001**
Arrythmia	588 (11.8%)	120 (9.6%)	147 (11.8%)	138 (11.1%)	183 (14.7%)	**0.001**
Fatty Liver	1,093 (21.9%)	158 (12.7%)	277 (22.2%)	312 (25.0%)	346 (27.7%)	**< 0.001**
Physical Measurements						
Height, (cm)[a]	166.67 ± 8.77	166.22 ± 8.72	166.26 ± 8.99	166.57 ± 8.97	167.61 ± 8.32	**< 0.001**
Weight, (kg)[a]	75.50 ± 14.82	65.42 ± 10.47	72.28 ± 11.54	77.57 ± 12.42	86.72 ± 15.48	**< 0.001**
Waist Circumstance, (cm)[a]	94.86 ± 10.55	83.58 ± 6.03	91.98 ± 5.23	97.68 ± 5.69	106.20 ± 8.77	**< 0.001**
Hip Circumstance, (cm)[a]	101.16 ± 8.58	94.77 ± 5.86	99.21 ± 6.01	102.67 ± 6.80	108.00 ± 9.16	**< 0.001**
SBP, (mmHg)[a]	132.93 ± 17.35	127.77 ± 16.33	131.86 ± 16.99	135.37 ± 17.84	136.73 ± 16.81	**< 0.001**
DBP, (mmHg)[a]	81.50 ± 11.66	78.98 ± 10.86	81.20 ± 11.17	82.77 ± 12.17	83.04 ± 11.96	**< 0.001**
HR, (bpm)[a]	84.83 ± 12.61	85.28 ± 12.31	84.95 ± 12.39	84.41 ± 13.05	84.68 ± 12.67	0.321
Visceral adipose tissue, (%)[a]	108.60 ± 42.06	73.83 ± 27.75	96.91 ± 26.98	116.04 ± 31.61	147.58 ± 40.75	**< 0.001**
Subcutaneous adipose tissue, (%)[a]	206.21 ± 71.86	151.69 ± 48.78	189.93 ± 52.90	220.24 ± 57.49	262.91 ± 74.36	**< 0.001**
Laboratory test						
HbA1C, (%)[a]	8.89 ± 2.21	8.77 ± 2.44	8.81 ± 2.18	8.86 ± 2.08	9.10 ± 2.10	**< 0.001**
FBG (mmol/L)[a]	9.30 ± 3.84	8.75 ± 3.67	9.22 ± 3.86	9.39 ± 3.72	9.83 ± 4.03	**< 0.001**
2h-BG, (mmol/L)[a]	14.31 ± 4.74	13.97 ± 4.80	14.40 ± 4.77	14.44 ± 4.62	14.44 ± 4.76	0.053
Fasting Insulin, (mU/l)[b]	10.30 (6.17, 16.05)	7.75 (4.62, 12.01)	9.52 (6.07, 15.41)	11.24 (7.01, 16.91)	12.84 (7.88, 20.45)	**< 0.001**
2h-Insulin, (mU/l)[b]	39.78 (21.37, 64.93)	33.09 (17.55, 52.77)	38.90 (20.15, 65.83)	41.69 (23.93, 68.09)	47.83 (25.78, 75.71)	**< 0.001**
Fasting C-peptide, (ng/mL)[b]	2.18 (1.49, 2.97)	1.80 (1.23, 2.38)	2.13 (1.48, 2.85)	2.37 (1.66, 3.08)	2.56 (1.75, 3.57)	**< 0.001**
2h- C-peptide, (ng/mL)[b]	5.34 (3.37, 7.58)	4.68 (2.93, 6.76)	5.27 (3.33, 7.57)	5.52 (3.61, 7.63)	5.87 (3.68, 8.37)	**< 0.001**
HOMA-IR[b]	3.92 (2.18, 6.77)	2.77 (1.57, 4.75)	3.65 (2.14, 6.14)	4.40 (2.55, 7.23)	5.25 (2.88, 9.34)	**< 0.001**
METS-IR[a]	44.47 ± 9.03	37.34 ± 6.27	42.46 ± 6.58	46.07 ± 6.86	52.01 ± 9.08	**< 0.001**
TyG[a]	9.40 ± 0.87	9.12 ± 0.83	9.39 ± 0.89	9.50 ± 0.81	9.59 ± 0.85	**< 0.001**
TG, (mmol/L)[b]	1.60 (1.09, 2.46)	1.27 (0.89, 1.93)	1.59 (1.08, 2.44)	1.73 (1.22, 2.66)	1.80 (1.27, 2.71)	**< 0.001**
TC, (mmol/L)[a]	5.05 ± 1.34	5.04 ± 1.26	5.15 ± 1.52	5.07 ± 1.26	4.94 ± 1.29	**< 0.001**
HDL-c, (mmol/L)[a]	1.19 ± 0.29	1.28 ± 0.31	1.21 ± 0.29	1.17 ± 0.28	1.11 ± 0.26	**< 0.001**
LDL-c, (mmol/L)[a]	3.23 ± 0.91	3.19 ± 0.91	3.28 ± 0.95	3.27 ± 0.91	3.17 ± 0.87	**< 0.001**
ALT, (U/L)[b]	22.00 (15.00, 36.00)	19.00 (13.00, 29.00)	21.00 (15.00, 35.00)	24.00 (17.00, 40.00)	27.00 (18.00, 41.50)	**< 0.001**
AST, (U/L)[b]	19.00 (15.00, 25.00)	17.00 (14.00, 22.00)	18.00 (15.00, 24.00)	19.00 (15.00, 27.00)	20.00 (16.00, 29.00)	**< 0.001**
ALP, (U/L)[b]	76.00 (64.00, 92.00)	72.00 (61.00, 88.75)	77.00 (65.00, 92.00)	77.00 (65.00, 94.00)	78.00 (64.00, 94.00)	**< 0.001**
GGT, (U/L)[b]	28.00 (19.00, 44.00)	22.00 (16.00, 33.00)	26.00 (19.00, 40.00)	32.00 (21.50, 49.00)	32.00 (23.00, 52.00)	**< 0.001**
ALB, (g/L)[a]	44.90 ± 4.32	45.11 ± 4.30	45.06 ± 4.51	45.03 ± 4.27	44.39 ± 4.16	**< 0.001**
Creatinine, (μmol/L)[a]	68.07 ± 18.61	63.43 ± 15.77	66.89 ± 18.34	68.40 ± 16.17	73.56 ± 22.07	**< 0.001**
eGFR, (mL/min/1.73m^2)[a]	101.45 ± 17.56	106.07 ± 15.98	102.98 ± 17.24	100.43 ± 16.80	96.32 ± 18.68	**< 0.001**
eGFR < 60 mL/min/1.73m^2, n(%)	115 (2.3%)	10 (0.8%)	26 (2.1%)	22 (1.8%)	57 (4.6%)	**< 0.001**

Table 1 (continued)

Characteristic	Overall, N = 4,988	Quartile 1 (4.53–6.64)	Quartile 2 (6.64–6.95)	Quartile 3 (6.95–7.20)	Quartile 4 (7.20–8.09)	p-value
UACR Category(mg/g), n(%)						**< 0.001**
< 30	3,683 (73.8%)	989 (79.3%)	960 (77.0%)	906 (72.7%)	828 (66.4%)	
30–300	1,061 (21.3%)	215 (17.2%)	234 (18.8%)	276 (22.1%)	336 (26.9%)	
≥ 300	244 (4.9%)	43 (3.4%)	53 (4.3%)	65 (5.2%)	83 (6.7%)	
BUN, (mmol/L)[a]	5.01 ± 1.60	4.82 ± 1.48	4.97 ± 1.59	5.03 ± 1.52	5.23 ± 1.77	**< 0.001**
UA, (μmol/L)[a]	333.05 ± 92.80	310.04 ± 90.10	329.59 ± 88.95	341.62 ± 93.38	350.87 ± 93.72	**< 0.001**
Hb, (g/L)[a]	148.14 ± 17.25	144.79 ± 17.60	148.01 ± 17.13	148.99 ± 17.04	150.78 ± 16.67	**< 0.001**
RBC, ($10^{\wedge 12}$/L)[a]	4.89 ± 0.72	4.81 ± 0.53	4.89 ± 0.53	4.92 ± 1.10	4.95 ± 0.55	**< 0.001**
WBC, ($10^{\wedge 9}$/L)[a]	6.77 ± 1.92	6.53 ± 1.90	6.72 ± 1.98	6.84 ± 1.79	6.99 ± 1.99	**< 0.001**
PLT, ($10^{\wedge 9}$/L)[a]	238.11 ± 61.86	247.11 ± 66.39	244.72 ± 62.80	235.25 ± 60.48	225.40 ± 54.89	**< 0.001**
Hematocrit, (L/L)[a]	0.44 ± 0.06	0.44 ± 0.05	0.45 ± 0.06	0.45 ± 0.06	0.45 ± 0.05	**< 0.001**
MCH, (fL)[a]	30.51 ± 6.38	30.41 ± 8.57	30.29 ± 2.02	30.80 ± 9.04	30.55 ± 1.79	**< 0.001**
hsCRP, (mmol/L)[b]	1.65 (0.74, 3.77)	1.03 (0.48, 2.39)	1.58 (0.72, 3.55)	1.87 (0.89, 3.88)	2.36 (1.16, 5.01)	**< 0.001**
Echocardiographic examination						
Left atrial diameter, (mm)[a]	32.25 ± 3.95	30.31 ± 3.46	31.66 ± 3.55	32.86 ± 3.75	34.17 ± 3.95	**< 0.001**
Interventricular septum, (mm)[a]	9.73 ± 1.48	9.27 ± 1.27	9.53 ± 1.39	9.85 ± 1.52	10.28 ± 1.51	**< 0.001**
LV posterior wall, (mm)[a]	9.64 ± 1.37	9.21 ± 1.30	9.42 ± 1.27	9.76 ± 1.35	10.16 ± 1.36	**< 0.001**
LV end-diastolic dimension, (mm)[a]	46.39 ± 4.42	44.96 ± 3.98	46.05 ± 4.36	46.61 ± 4.31	47.93 ± 4.47	**< 0.001**
LV end-systolic dimension, (mm)	28.25 ± 3.97	27.15 ± 3.57	28.11 ± 3.75	28.48 ± 4.29	29.26 ± 3.95	**< 0.001**
LV ejection faction, (%)	69.49 ± 5.27	70.04 ± 5.25	69.57 ± 5.09	69.55 ± 5.29	68.79 ± 5.37	**< 0.001**
LVM	156.97 ± 41.67	139.37 ± 32.89	150.27 ± 38.25	160.52 ± 40.29	177.71 ± 44.55	**< 0.001**
LVMI, (g/m^2)	80.83 ± 19.48	76.83 ± 16.96	79.17 ± 18.78	81.80 ± 19.97	85.51 ± 20.91	**< 0.001**
Relative wall thickness	0.42 ± 0.06	0.41 ± 0.06	0.42 ± 0.06	0.42 ± 0.07	0.43 ± 0.07	**< 0.001**
LVH	1,521 (30.5%)	217 (17.4%)	324 (26.0%)	413 (33.1%)	567 (45.5%)	**< 0.001**
Geometry type						**< 0.001**
0 Normal	2,095 (42.0%)	641 (51.4%)	568 (45.5%)	505 (40.5%)	381 (30.6%)	
1	1,372 (27.5%)	389 (31.2%)	355 (28.5%)	329 (26.4%)	299 (24.0%)	
2	888 (17.8%)	139 (11.1%)	168 (13.5%)	247 (19.8%)	334 (26.8%)	
3	633 (12.7%)	78 (6.3%)	156 (12.5%)	166 (13.3%)	233 (18.7%)	
Baseline Medications, n (%)						
Insulin usage	1,418 (28.4%)	382 (30.6%)	339 (27.2%)	326 (26.1%)	371 (29.8%)	0.042
Metformin	3,311 (66.4%)	760 (60.9%)	841 (67.4%)	839 (67.3%)	871 (69.8%)	**< 0.001**
SGLT2i	764 (15.3%)	150 (12.0%)	183 (14.7%)	193 (15.5%)	238 (19.1%)	**< 0.001**
Glucosidase Inhibitors	1,207 (24.2%)	334 (26.8%)	293 (23.5%)	275 (22.1%)	305 (24.5%)	**0.045**
Thiazolidinediones	77 (1.5%)	14 (1.1%)	23 (1.8%)	18 (1.4%)	22 (1.8%)	0.444
Glinides	56 (1.1%)	13 (1.0%)	14 (1.1%)	16 (1.3%)	13 (1.0%)	0.933
Sulfonylureas	1,284 (25.7%)	279 (22.4%)	349 (28.0%)	352 (28.2%)	304 (24.4%)	**0.001**
DPP4i	1,000 (20.0%)	279 (22.4%)	241 (19.3%)	243 (19.5%)	237 (19.0%)	0.127
GLP-1 RA	465 (9.3%)	53 (4.3%)	93 (7.5%)	122 (9.8%)	197 (15.8%)	**< 0.001**
β-blockers	238 (4.8%)	32 (2.6%)	40 (3.2%)	66 (5.3%)	100 (8.0%)	**< 0.001**
RASi	867 (17.4%)	113 (9.1%)	154 (12.3%)	247 (19.8%)	353 (28.3%)	**< 0.001**
CCBs	738 (14.8%)	95 (7.6%)	164 (13.2%)	207 (16.6%)	272 (21.8%)	**< 0.001**
Diuretics	121 (2.4%)	14 (1.1%)	23 (1.8%)	30 (2.4%)	54 (4.3%)	**< 0.001**
Statins	1,178 (23.6%)	231 (18.5%)	289 (23.2%)	308 (24.7%)	350 (28.1%)	**< 0.001**
Anti-platelet drugs	444 (8.90)	65 (5.21)	95 (7.61)	120 (9.62)	164 (13.15)	**< 0.001**

SBP Systolic blood pressure, DBP Diastolic blood pressure, HR Heart rate, HbA1c glycosylated hemoglobin A1c, FBG fasting blood glucose, 2h-BG 2-h blood glucose after a 75g oral glucose tolerance test, HOMA-IR homeostasis model assessment of insulin resistance, TG triglyceride, TC total cholesterol, HDL-c high-density lipoprotein-cholesterol, LDL-c low-density lipoprotein-cholesterol, ALT alanine transaminase, AST aspartate transaminase, ALP alkaline phosphatase, GGT γ-glutamyl transferase, ALB albumin, eGFR estimated glomerular filtration rate, ACR albumin-Creatinine Ratio, BUN blood urea nitrogen, UA uric acid, Hb hemoglobin, RBC red blood cell, WBC white blood cell, PLT platelet count, MCH mean corpuscular hemoglobin, hsCRP high-sensitivity C-reactive protein, LV left ventricular, LVMI left ventricular mass index, SGLT2i Sodium-glucose transport protein 2 inhibitors, DPP4i dipeptidyl peptidase 4 inhibitors, GLP-1 RA Glucagon- like peptide-1 receptor agonists, RASi including angiotensin receptor-neprilysin inhibitors (ARNI), angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs), CCBs calcium channel blockers

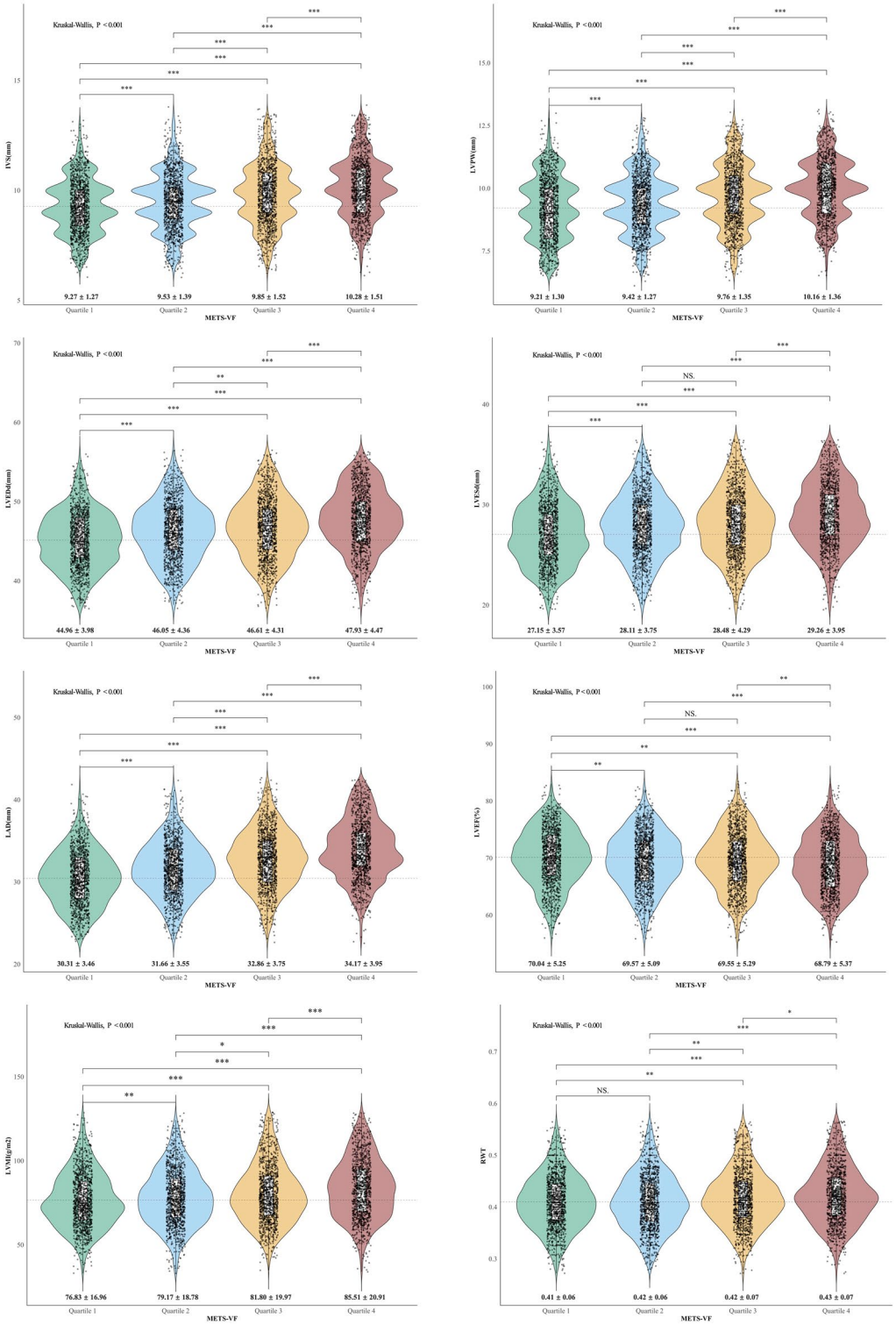

Fig. 2 The violin plots demonstrating the distribution of echocardiographic parameters among patients in different METS-VF groups. METS-VF Metabolic score for visceral fat. IVS interventricular septal thickness, LVPW LV posterior wall thickness, LVEDd LV end-diastolic dimension, LVESd LV end-systolic dimension, LAD left atrial diameter, LVEF LV ejection fraction, LVMI LV mass index, RWT relative wall thickness. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$, NS. P value of o significance

Table 2 Association between baseline METS-VF and LVH, LV Geometry type in people with T2DM

METS-VF	Per 1 unit	Quartile 1 (4.53–6.64)	Quartile 2 (6.64–6.95)	Quartile 3 (6.95–7.20)	Quartile 4 (7.20–8.09)
LVH					
Prevalence, n (%)	895 (17.9%)	98 (7.9%)	162 (13.0%)	249 (20.0%)	386 (31.0%)
Crude model	5.32 (4.32–6.57), P < 0.001	1.00 (reference)	1.75 (1.35–2.29), P < 0.001	2.93 (2.29–3.77), P < 0.001	5.26 (4.16–6.70), P < 0.001
Model 1	6.91 (5.41–8.88), P < 0.001	1.00 (reference)	1.80 (1.37–2.37), P < 0.001	3.13 (2.41–4.09), P < 0.001	6.25 (4.79–8.22), P < 0.001
Model 2	7.88 (5.30–11.82), P < 0.001	1.00 (reference)	1.61 (1.20–2.19), P < 0.001	2.54 (1.86–3.48), P < 0.001	4.67 (3.22–6.83), P < 0.001
Model 3	9.79 (6.16–15.76), P < 0.001	1.00 (reference)	1.61 (1.20–2.19), P < 0.001	2.96 (2.07–4.26), P < 0.001	5.97 (3.86–9.32), P < 0.001
LV geometry type					
Normal geometry					
Prevalence, n(%)	2,398 (48.1%)	694 (55.7%)	657 (52.7%)	584 (46.8%)	463 (37.1%)
Concentric remodeling, Odds ratio (95% confidence interval) of concentric remodelling vs. normal geometry					
Prevalence, n(%)	1,695 (34.0%)	455 (36.5%)	428 (34.3%)	414 (33.2%)	398 (31.9%)
Crude model	1.21 (1.06–1.39), P = 0.006	1.00 (reference)	0.99 (0.84–1.18), P = 0.941	1.08 (0.91–1.29), P = 0.375	1.31 (1.10–1.57), P = 0.003
Model 1	1.14 (0.98–1.01), P = 0.097	1.00 (reference)	0.95 (0.80–1.23), P = 0.743	1.03 (0.86–1.23), P = 0.743	1.23 (1.01–1.50), P = 0.037
Model 2	0.85 (0.70–1.03), P = 0.093	1.00 (reference)	0.87 (0.72–1.06), P = 0.163	0.88 (0.71–1.10), P = 0.272	0.98 (0.73–1.31), P = 0.904
Model 3	0.80 (0.62–1.07), P = 0.140	1.00 (reference)	0.91 (0.73–1.13), P = 0.372	0.92 (0.71–1.18), P = 0.372	1.06 (0.76–1.48), P = 0.739
Concentric hypertrophy, Odds ratio (95% confidence interval) of concentric hypertrophy vs. normal geometry					
Prevalence, n(%)	565 (11.3%)	73 (5.85%)	95 (7.62%)	162 (13.0%)	235 (18.8%)
Crude model	5.15 (3.99–6.65), P < 0.001	1.00 (reference)	1.38 (0.995–1.90), P = 0.054	2.64 (1.96–3.55), P < 0.001	4.83 (3.62–6.44), P < 0.001
Model 1	5.54 (4.13–7.44), P < 0.001	1.00 (reference)	1.31 (0.94–1.82), P = 0.113	2.48 (1.82–3.39), P < 0.001	4.74 (3.44–6.52), P < 0.001
Model 2	5.27 (3.83–7.25), P < 0.001	1.00 (reference)	1.08 (0.75–1.56), P = 0.670	1.84 (1.27–2.66), P = 0.001	3.13 (2.00–4.90), P < 0.001
Model 3	5.79 (3.40–9.88), P < 0.001	1.00 (reference)	0.98 (0.64–1.49), P = 0.908	1.96 (1.29–3.00), P = 0.002	3.41 (2.01–5.78), P < 0.001
Eccentric hypertrophy, Odds ratio (95% confidence interval) of eccentric hypertrophy vs. normal geometry					
Prevalence, n(%)	330 (6.62%)	25 (2.00%)	67 (5.37%)	87 (6.98%)	151 (12.1%)
Crude model	7.06 (5.05–9.87),P < 0.001	1.00 (reference)	2.83 (1.77–4.54), P < 0.001	4.14 (2.62–6.54), P < 0.001	9.05 (2.62–14.05), P < 0.001
Model 1	12.32 (8.25–18.39), P < 0.001	1.00 (reference)	3.30 (2.01–5.40), P < 0.001	5.42 (3.34–8.82), P < 0.001	14.69 (9.01–23.95), P < 0.001
Model 2	13.56 (9.04–20.33), P < 0.001	1.00 (reference)	3.03 (1.79–5.14), P < 0.001	4.29 (2.46–7.49), P < 0.001	10.41 (5.48–19.80), P < 0.001
Model 3	21.64 (12.86–36.4), P < 0.001	1.00 (reference)	4.63 (2.38–8.99), P < 0.001	8.47 (4.20–17.11), P < 0.001	26.06 (11.60–58.51), P < 0.001

METS-VF Metabolic score for visceral fat. LVH left ventricular hypertrophy. OR odds ratio, CI confidence interval

Crude Model: unadjusted model

Model 1: adjusted for age, sex, diabetes duration, level of education, smoking status, drinking status, weight loss in last 1 year

Model 2: Model 1 + further adjusted for SBP, HR, VAT, SAT, HT

Model 3: Model 2 + further adjusted for FBG, fasting insulin and C-peptide, 2-h insulin and C-peptide, HOMA-IR, TG, TC, LDL-c, ALT, ALB, eGFR, ACR category, BUN, Hb, RBC, WBC, HCT, MCH, antidiabetic agents (SGLT2i, GLP-1 RA, sulfonylureas, glucosidase inhibitor), antihypertensive agents (β-blockers, renin angiotensin blockers, CCBs, diuretic), statins, anti-platelet drugs

SBP systolic blood pressure, HR heart rate, VAT visceral adipose tissue, SAT subcutaneous adipose tissue, HT hypertension, FBG fasting blood glucose, HOMA-IR homeostasis model assessment of insulin resistance, TG triglycerides, TC total cholesterol, LDL-c low-density lipoprotein-cholesterol, ALT Alanine transaminase, ALB Albumin, eGFR estimated glomerular filtration rate, ACR Albumin-Creatinine Ratio, BUN blood urea nitrogen, Hb Hemoglobin, RBC red blood cell, WBC white blood cell, HCT Hematocrit, MCH mean corpuscular hemoglobin, SGLT2i Sodium-glucose transport protein 2 inhibitors, GLP1-RA Glucagon- like peptide-1 receptor agonists, CCBs calcium channel blockers

OR (95% CI): 2.83 (1.77–4.54), P < 0.001; Q3: OR (95% CI): 4.14 (2.62–8.82), P < 0.001; Q4: OR (95% CI): 9.05 (2.62–14.05), P < 0.001]. These associations remained significant after adjusting for potential covariates in Model 3: [Q2: OR (95% CI): 4.63 (2.38–8.99), P < 0.001; Q3: OR (95% CI): 8.47 (4.20–17.11), P < 0.001; Q4: OR (95% CI): 26.06 (11.60–58.51), P < 0.001]. However, no significant association was found for concentric remodeling. Results were similar when METS-VF was treated as a continuous variable. After adjusting for covariates, each 1-unit increase in METS-VF was linked to a 57.9% higher risk of concentric LVH [OR (95% CI): 5.79 (3.83–9.88), P < 0.001] and a 216.4% higher risk of eccentric hypertrophy [OR (95% CI): 21.64 (12.86–36.4), P < 0.001]. Associations

between other abdominal obesity indices and LVH are shown in Supplementary Table 2.

RCS analysis

RCS analysis explored the potential non-linear relationship between METS-VF and the likelihood of LVH (Fig. 3). METS-VF values in the analysis ranged from 5.799 to 8.043, excluding outliers. In the crude model, a roughly linear relationship METS-VF and LVH was observed (P < 0.001, P nonlinear = 0.223) (Fig. 3A-1). After full adjustment for relevant confounding factors, the relationship between METS-VF and LVH became non-linear. Specifically, below a METS-VF threshold of 6.368, the likelihood of LVH increased gradually. However, above this threshold, the risk of LVH increased more rapidly (P < 0.001, P nonlinear = 0.008) (Fig. 3A-2). Among males, a similar linear relationship was observed in both unadjusted model (P < 0.001, P nonlinear = 0.121) (Fig. 3B-1) and adjusted models (P < 0.001, P nonlinear = 0.081) (Fig. 3B-2), with a rapid increase in risk above a METS-VF of 6.479. For females, a non-linear relationship was also present, with an inflection point at 6.264 (unadjusted: P < 0.001, P nonlinear = 0.032, adjusted: P < 0.001, P nonlinear = 0.039) (Fig. 3C-1, C-2). The potential non-linear relationship between other abdominal indices and the LVH risk were explored in Supplementary Fig. 1.

Stratified analyses

A stratified analysis was conducted to evaluate the relationship between METS-VF and LVH across different patient subgroups, including factors such as age, sex, diabetes duration, education level, smoking/drinking status, BMI, HbA1c, HOMA-IR, hypertension (HT), and UACR (Fig. 4). No significant interactions were observed across these subgroups. However, when stratifying by UACR, a differential effect was noted. Specifically, METS-VF was strongly associated with LVH in patients with UACR < 300 mg/g. In this group, higher METS-VF levels were linked to an increased risk of LVH (OR (95% CI): 5.47 (3.79-7.95), P < 0.001). In contrast, no significant association was observed in patients with UACR > = 300 mg/g [OR (95% CI) 1.33 (0.18-11.45), P = 0.784]. Overall, the positive association between METS-VF and LVH remained consistent across other population subgroups, including those based on age, sex, and clinical variables.

ROC analyses of the METS-VF and other abdominal obesity indices with LVH risk

Receiver Operating Characteristic (ROC) curve analyses were performed to compare the predictive value of METS-VF with other abdominal obesity indices for LVH risk. As shown in Fig. 5 and Supplementary Table 3, METS-VF demonstrated the highest Area Under the Curve (AUC) of 0.68 (95% CI 0.66–0.70), indicating its moderate ability to predict LVH risk relative to the other indices. In particular, the ROC curve analysis identified a METS-VF cutoff value of 7.11 for optimal prediction of LVH. This cutoff provided a sensitivity of 56.09% and a specificity of 70.34%, balancing the ability to correctly identify LVH cases while minimizing false positives.

Discussion

This cross-sectional study of 4988 adults with T2DM demonstrates a significant, nonlinear association between the METS-VF index and the likelihood of LVH. Higher METS-VF quartiles were linked to increased LV wall thickness, LV dimensions, and LVMI, even after adjusting for key confounders such as age, sex, BMI, smoking, alcohol consumption, glycemic control, and hypertension status. Notably, the association was stronger for eccentric hypertrophy, and METS-VF outperformed traditional abdominal obesity indices (e.g., waist circumference, hip circumference, and visceral adipose tissue) in predicting LVH risk, underscoring its potential as an accessible tool for early cardiac risk stratification in T2DM patients.

The relationship between metabolic risk factors and heart failure (HF) has been well established, with T2DM representing a major risk factor for both HF onset and progression [22]. Screening for subclinical heart failure, such as LVH, is crucial, as early identification of LV remodeling could allow for timely interventions that delay the onset of overt HF, ultimately improving patient outcomes [3, 23, 24]. Our findings are consistent with previous research suggesting that the interplay between metabolic dysfunction and structural cardiac changes in T2DM patients is multifactorial, involving not only glycemic control but also factors like VAT accumulation, insulin resistance, and inflammation [25, 26]. VAT, in particular, has been increasingly recognized as a key driver of LV remodeling, given its ability to release pro-inflammatory cytokines and free fatty acids that contribute to myocardial stress and fibrosis [4, 27–29]. As an easily quantifiable metric of VAT, METS-VF offers a practical tool to assess this risk, particularly in clinical

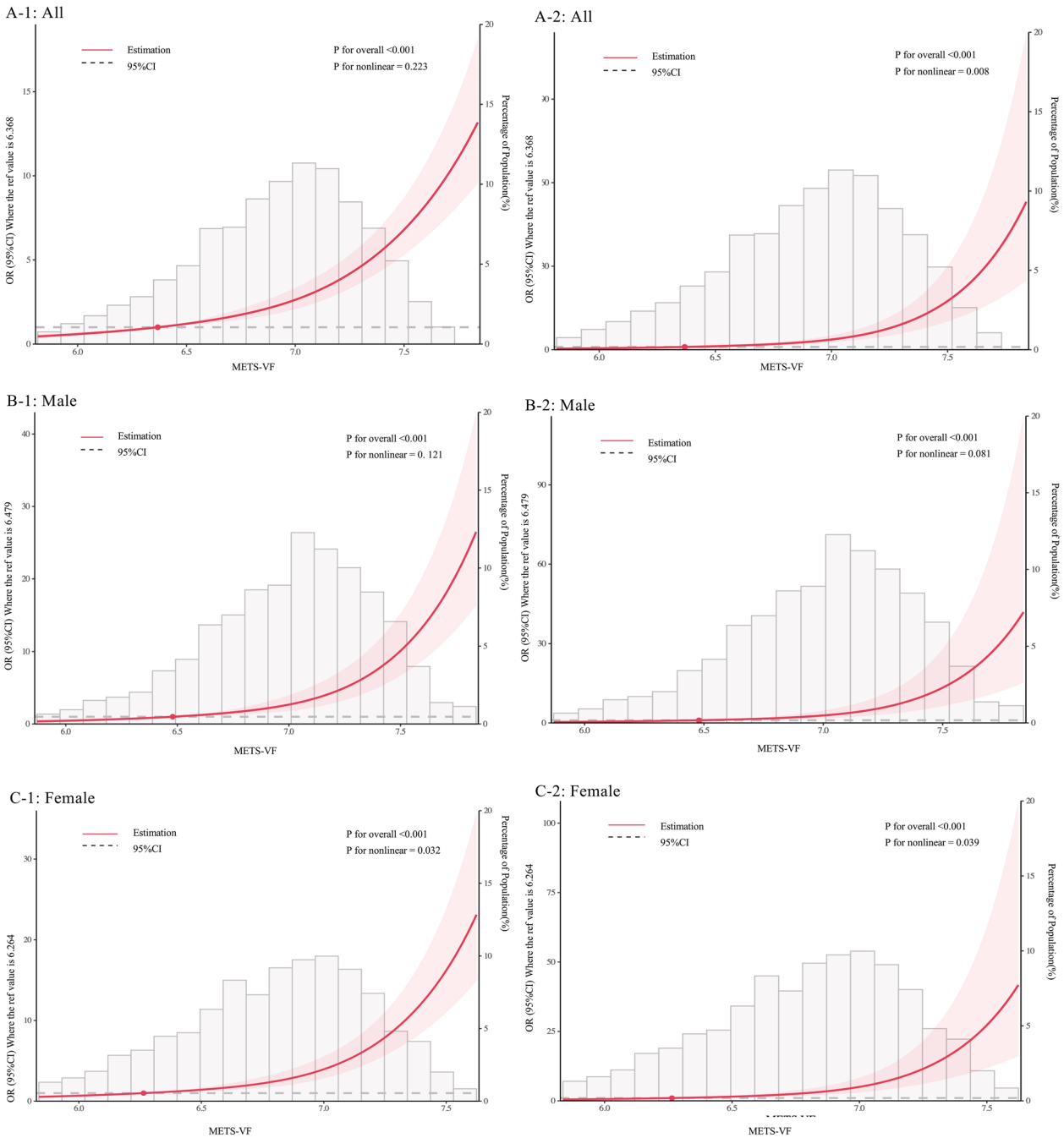

Fig. 3 The dose–response relationship between METS-VF and the risk of LVH. (A-1) Crude model with all participants; (A-2) Adjusted model with all participants; (B-1) Crude model with male participants; (B-2) Adjusted model with male participants; (C-1) Crude model with female participants; (C-2) Adjusted model with female participants. The red line represents the odds ratio based on the restricted cubic spline models, and the shad area represents the 95% confidence interval (knots on the 10th, 50th,90th percentiles). The dash line represents the OR equal to 1. The 10th percentiles were set as the reference values. The model was adjusted for age, sex, diabetes duration, level of education, smoking status, drinking status, weight loss in last 1 year, SBP, HR, VAT, AT, HT, FBG, fasting insulin and C-peptide, 2-h insulin and C-peptide, HOMA-IR, TG, TC, LDL-c, ALT, ALB, eGFR, ACR category, BUN, Hb, RBC, WBC, HCT, MCH, SGLT2i, GLP-1 RA, sulfonylureas, glucosidase Inhibitor, β-blockers, renin angiotensin blockers, CCBs, diuretic, statins, anti-platelet drugs

settings where advanced imaging techniques such as MRI or CT are unavailable. This index integrates metabolic and anthropometric data, making it a more accessible and cost-effective measure compared to traditional methods [11].

Our results support earlier findings indicating that METS-VF is an effective measure of metabolic

Subgroups	LVH (n/N)	Odd Ratio(95%CI)	P value	METS-VF	P for interaction
Age					0.339
Age<50	244/2406	14.01(6.31-32.27)	<0.001		
Age≥50	651/2582	8.44(4.97-14.57)	<0.001		
Sex					0.118
Male	373/2901	13.04(6.28-27.82)	<0.001		
Female	522/2087	9.31(5.07-17.55)	<0.001		
DM duration					0.89
<36	301/2245	13.97(6.07-33.48)	<0.001		
≥36	577/2669	8.86(4.98-16.06)	<0.001		
Education level					0.475
Middle school or below	486/1896	7.37(2.70-21.40)	<0.001		
High school or above	400/3060	10.64(6.25-18.42)	<0.001		
Smoking status					0.409
0	698/3547	9.35(5.37-16.60)	<0.001		
1	197/1441	12.39(5.18-30.81)	<0.001		
Drinking status					0.108
0	796/4367	9.70(5.41-17.78)	<0.001		
1	99/621	11.68(5.37-22.16)	<0.001		
BMI					0.707
<24	119/1089	2.91(1.16-7.60)	0.026		
≥24	776/3899	15.53(8.63-28.39)	<0.001		
HBA1c					0.189
<7	206/1174	7.37(2.70-21.40)	<0.001		
≥7	683/3789	10.64(6.25-18.42)	<0.001		
HOMA-IR					0.639
<2	184/1071	3.24(1.46-7.48)	0.005		
≥2	682/3775	17.25(9.73-31.17)	<0.001		
HT					0.3
No	217/2119	6.25(2.79-14.61)	<0.001		
Yes	678/2869	11.75(6.65-21.16)	<0.001		
ACR category					0.425
<30	584/3683	11.61(6.57-20.95)	<0.001		
30-300	240/1061	12.01(4.59-33.18)	<0.001		
≥300	71/244	1.33(0.18-11.45)	0.784		

Fig. 4 Stratified analyses of the association between METS-VF and LVH risk in T2DM patients. The model was adjusted for age, sex, diabetes duration, education level, smoking status, drinking status, weight loss in last 1 year, SBP, HR, VAT, SAT, HT, FBG, fasting insulin and C-peptide, 2-h insulin and C-peptide, HOMA-IR, TG, TC, LDL-c, ALT, ALB, eGFR, ACR category, BUN, Hb, RBC, WBC, HCT, MCH, SGLT2i, GLP-1 RA, sulfonylureas, glucosidase inhibitor, β-blockers, renin angiotensin blockers, CCBs, diuretic, statins, anti-platelet drugs (excluding the variable for subgroup stratification)

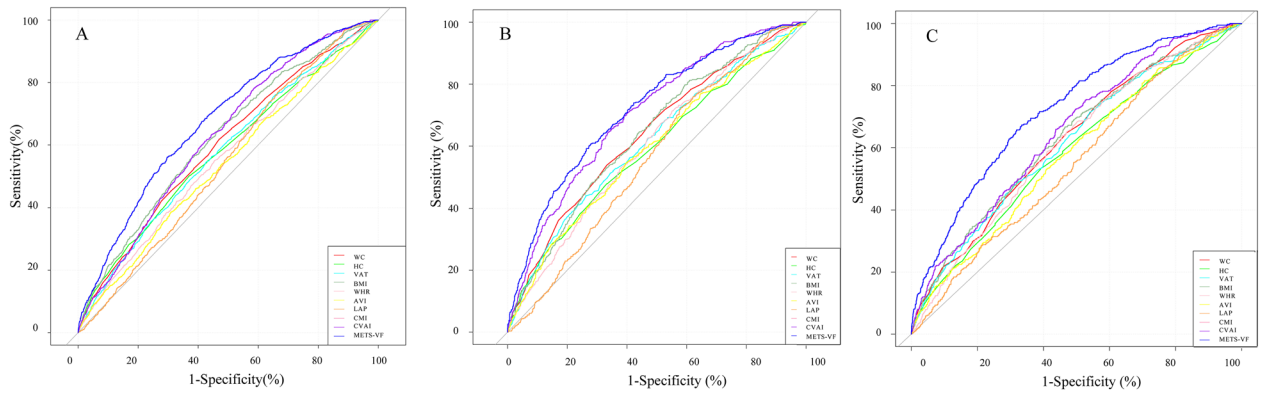

Fig. 5 Comparison of predictive performances for LVH between METS-VF and other o abdominal obesity indices. **A** all participants **B** female **C** male. ROC Receiver operating characteristic. LVH left ventricular hypertrophy, WC waist circumference, HC hip circumstance, VAT visceral adipose tissue, BMI body mass index, WHR waist-to-hip ratio, AVI abdominal volume index, LAP lipid accumulation product, CMI cardiometabolic Index, CVAI Chinese visceral adiposity index, METS-VF metabolic score for visceral fat

dysfunction, with associations not only with cardiovascular risk [15, 30–32], but also with kidney disease [10], non-alcoholic fatty liver disease[33], cardiovascular mortality [34] and overall mortality [35]. However, few studies have explored the specific relationship between METS-VF and LV remodeling in T2DM patients. This

study fills an important gap in the literature by highlighting the utility of METS-VF in predicting LVH, particularly eccentric hypertrophy. Interestingly, our stratified analyses revealed distinct patterns between sexes, with males exhibiting a linear relationship between METS-VF and LVH, while females showed a nonlinear association, suggesting the influence of sex hormones, hypertension, and hypoalbuminemia on LV remodeling [36, 37]. Furthermore, the results of this study challenge some previous findings, such as those by Hamid et al., who did not find a significant association between T2DM without hypertension and alterations in LV structure [38]. In contrast, our data suggest that METS-VF is an independent predictor of LVH in T2DM patients, regardless of hypertension status. This emphasizes the crucial role of VAT in LVH progression, which may not be fully captured by traditional indices like waist circumference or BMI.

Our findings have important implications for clinical practice. METS-VF, which combines easily obtainable clinical data (BMI, waist-to-height ratio, blood glucose, triglycerides, HDL cholesterol, and demographic factors), provides a cost-effective and practical method for VAT estimation. It could serve as a valuable tool for identifying T2DM patients at elevated risk for LVH, enabling earlier interventions to prevent or slow the progression of heart failure. Importantly, our sensitivity analyses demonstrated that METS-VF is a strong predictor of LVH even in individuals with normal BMI, further highlighting its superior ability to assess visceral adiposity compared to traditional measures of obesity. Despite these strengths, there are several limitations to this study. The cross-sectional design limits our ability to infer causality between METS-VF and LVH. As a single-center study, our results may not be generalizable to other populations, and there may be unmeasured confounders that could have influenced the observed associations. Additionally, our analysis focused on baseline METS-VF values without considering longitudinal changes in METS-VF or LVH progression. This is an important avenue for future research, as prospective studies would be better positioned to assess the temporal relationship between METS-VF and LVH and to explore whether interventions targeting METS-VF could mitigate LV remodeling and reduce heart failure risk in T2DM populations.

Conclusion

In conclusion, our study suggests that METS-VF is a promising index for identifying T2DM patients at high risk for LVH, particularly eccentric hypertrophy. Its strong predictive capability, independent of traditional abdominal obesity indices and hypertension status, underscores its potential as a valuable clinical tool for early cardiac risk stratification and targeted intervention in this high-risk population.

Supplementary Information

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Supplementary material 1.

Author contributions

Lu Wang wrote the main manuscript text and prepared all tables and figures. Lu Wang, Jing Ke and Bin Cao conceptualized and designed the study. Simo Liu, Qianqian Zhao, Haolin Gong, Yuan Fang, Zhaohui Zheng, Caiguo Yu, Nannan Wu and Yan Ma were responsible for data collection. Di Wang, Ke Yu, Longyan Yang and Dong Zhao contributed to data analysis and interpretation. All authors reviewed the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics and consent to participate

Not applicable.

Consent to publish

Not applicable.

Competing interests

The authors declare no competing interests.

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