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ORIGINAL RESEARCH

Association of the Triglyceride Glucose Index and Epicardial Adipose Tissue with Type 2 Diabetes Mellitus Complicated by Coronary Atherosclerotic Heart Disease

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Background: To examine the role of the triglyceride glucose(TyG) index and epicardial adipose tissue(EAT) in the progression of type 2 diabetes mellitus (T2DM) with concurrent coronary atherosclerotic heart disease(CHD), thereby establishing a theoretical foundation for early clinical intervention in CHD among T2DM patients.

Methods: This retrospective study analysed 96 patients with T2DM who underwent Coronary CT angiography at the Department of Endocrinology, Qinhuangdao First Hospital, between January 2020 and June 2023. Participants were categorised into two groups: those with T2DM and CHD (n=48) and those with T2DM alone (n=48). Clinical indicators were compared between the groups. Logistic regression analysis assessed the association between T2DM with CHD and both the TyG index and EAT. Receiver operating characteristic (ROC) curve analysis evaluated the predictive value of these parameters.

Results: Significant differences ($P < 0.05$) were found between the two groups in weight, uric acid(UA), triglycerides(TG), high-density lipoprotein cholesterol (HDL-C), epicardial adipose tissue volume (EATV) and the TyG index. Multivariate analysis identified both EATV and the TyG index as independent risk factors for T2DM complicated by CHD ($P < 0.05$). ROC curve analysis demonstrated that both EATV and TyG index showed statistically significant predictive power for CHD development in T2DM patients ($P<0.05$). The EATV exhibited an AUC of 0.739 with a predictive cut-off value of 128.45 mL (sensitivity 66.7%, specificity 72.9%). The TyG index demonstrated an AUC of 0.724 with a predictive cut-off value of 9.47 (sensitivity 58.3%, specificity 81.2%).

Conclusion: The TyG index and EAT are significant risk factors for CHD in T2DM patients. These findings provide a theoretical basis for early screening, monitoring, and clinical intervention in this population.

Keywords: triglyceride glucose index, epicardial adipose tissue, type 2 diabetes mellitus, coronary atherosclerotic heart disease

Introduction

In recent years, the prevalence of type 2 diabetes mellitus (T2DM) has risen steadily, with projections suggesting that more than 1.31 billion people worldwide will be affected by diabetes by 2050, representing a major global public health challenge.[1] Chronic hyperglycemia, along with pancreatic β-cell dysfunction, insulin resistance, and immune-inflammatory responses, worsens the pathological progression of T2DM and contributes to the development of both microvascular and macrovascular complications, including diabetic retinopathy, nephropathy, neuropathy, atherosclerosis, and cardiovascular diseases.[2] Coronary atherosclerotic heart disease (CHD), a chronic ischaemic condition resulting from coronary artery stenosis, presents most severely as acute coronary syndrome (ACS). The incidence and mortality rates of CHD continue to increase due to population ageing and the widespread adoption of unhealthy lifestyles.[3] Compared with non-diabetic individuals, diabetic patients exhibit greater infiltration of inflammatory macrophages and

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T-cells in coronary arteries, leading to more severe vascular damage.[4] Epidemiological evidence indicates that each 1% increase in glycated haemoglobin (HbA1c) is associated with an estimated 11–16% higher risk of cardiovascular events.[5] Patients with coronary artery disease (CAD) complicated by glucose metabolism disorders often present with multi-vessel lesions and face an increased likelihood of subsequent adverse cardiovascular outcomes. Consequently, the early identification of high-risk individuals among T2DM and CAD patients may lead to improved prognoses.

Insulin resistance (IR) represents the primary pathophysiological mechanism underlying type 2 diabetes mellitus (T2DM), being prevalent in most diabetic patients and demonstrating a close association with adverse cardiovascular events.[6] Research indicates that IR promotes inflammatory states, vascular endothelial dysfunction and dyslipidaemia, which may serve as key mechanisms driving coronary heart disease (CHD) progression.[7] Triglycerides (TG) and related metabolic markers, particularly the triglyceride-glucose (TyG) index, are widely recognised as surrogate indicators of IR and have been consistently associated with various cardiovascular diseases including coronary atherosclerosis, CHD and acute coronary syndrome (ACS).[8,9] Epicardial adipose tissue (EAT), as a visceral adipose tissue (VAT) component, envelops the cardiac surface, distributes along major coronary artery branches and maintains direct myocardial contact. This metabolically active tissue exerts both local and systemic effects through paracrine and endocrine secretion of bioactive substances.[10] While EAT normally secretes both pro-inflammatory and anti-inflammatory cytokines to maintain vascular homeostasis, increased EAT thickness has been associated with elevated cardiovascular risk and may represent a significant risk factor for atherosclerotic disorders.[11,12] Due to its unique metabolic and anatomical characteristics, EAT has emerged as an important clinical biomarker for cardiovascular diseases. Despite known links between insulin resistance and CHD, the predictive value of TyG index and EAT remains unclear. This study primarily investigates the correlation between the TyG index, epicardial adipose tissue volume (EATV) and T2DM complicated by CHD, aiming to provide further insights into these interrelated metabolic and cardiovascular parameters.

Materials and Methods

General Data

A total of 96 patients with type 2 diabetes mellitus (T2DM) who underwent Coronary CT angiography at the Department of Endocrinology, Qinhuangdao First Hospital between January 2020 and June 2023 were retrospectively enrolled. The patients were divided into two groups: The T2DM with coronary heart disease (CHD) group (n=48) and the T2DM-only group (n=48). This study was approved by the Ethics Committee of Qinhuangdao First Hospital.

Diagnostic Criteria

Diabetes Mellitus

The diagnostic criteria for diabetes mellitus were based on the Chinese Guidelines for the Prevention and Treatment of Diabetes (2020 Edition).[13] Diabetes diagnosis required either the presence of typical symptoms (including polydipsia, polyuria, polyphagia or unexplained weight loss) alongside any of the following biochemical criteria: fasting venous plasma glucose (FPG) ≥7.0 mmol/L, 2-hour oral glucose tolerance test (OGTT) venous plasma glucose ≥11.1 mmol/L, glycated haemoglobin (HbA1c) ≥6.5%, or random venous plasma glucose ≥11.1 mmol/L. For asymptomatic patients, diabetes was confirmed when two separate glucose measurements (either at the same or different time points, excluding random glucose measurements) met or exceeded these diagnostic thresholds, ensuring diagnostic accuracy through rigorous biochemical confirmation in accordance with established clinical guidelines.

Coronary Atherosclerotic Heart Disease

CHD was diagnosed when coronary computed tomography angiography (CTA) demonstrated ≥50% stenosis in at least one of the three major coronary arteries.[14]

Exclusion Criteria

(1) Type 1 diabetes, specific types of diabetes, or gestational diabetes; (2) Severe pericardial disease, cardiomyopathy, valvular heart disease or acute coronary artery disease; (3) Severe hepatic or renal dysfunction; (4) History of glucocorticoid use; (5) Recent acute infection; (6) Incomplete or insufficient medical records.

Method

Determination of Basic Human Body Parameters

The patient's age, gender, height, weight, heart rate, history of hypertension, and other general conditions were recorded. The body mass index (BMI) was calculated using the formula BMI = weight (kg) / height^2 (m^2).

The Following Parameters Were Measured

Glycated haemoglobin (HbA1c), fasting plasma glucose (FPG), alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), uric acid (UA), creatinine (CREA), serum urea nitrogen (BUN), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). The TyG index was calculated using SPSS 25.0 software. TyG index = ln[TG (mg/dL) × FPG (mg/dL)/2].

EAT Measurement method

In collaboration with the Department of Radiology, the original cardiac CT images in Digital Imaging and Communications in Medicine (DICOM) format from eligible patients meeting the inclusion and exclusion criteria were collected from our hospital's Picture Archiving and Communication System (PACS). The acquired DICOM images were transferred to a Philips workstation where two physicians with over two years of experience in coronary artery disease diagnosis, who were blinded to the image grouping, independently performed manual delineation of the epicardial adipose tissue (EAT) area. During delineation, the CT value range was first set to −190 to −30 Hounsfield units (HU). From the lower margin of the right pulmonary artery trunk to the diaphragmatic level, the cardiac region was delineated layer by layer along the pericardium. Upon completion of delineation, the software automatically displayed the EAT area and generated the epicardial adipose tissue volume (EATV) value (see Figure 1).

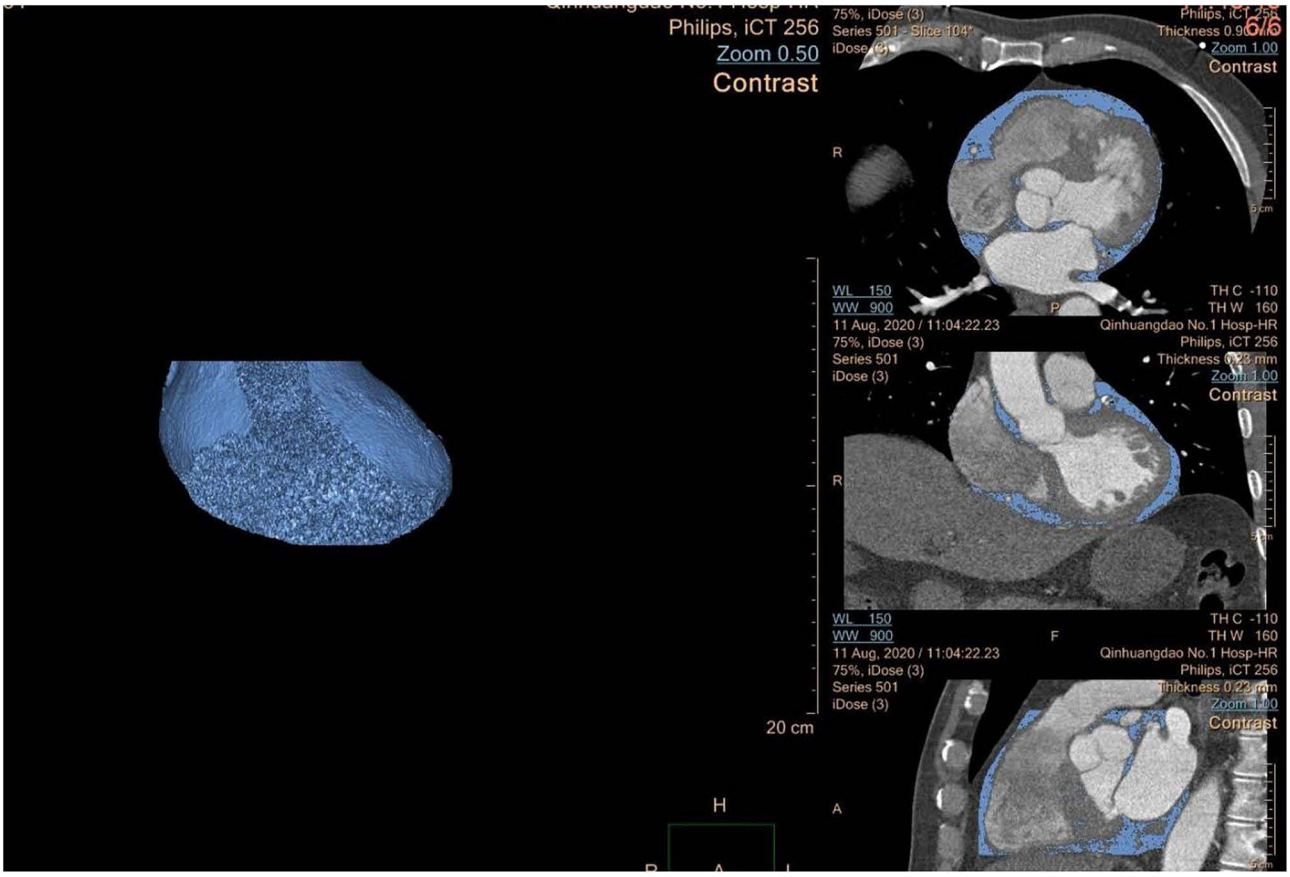

Figure 1 Epicardial adipose tissue volume (EATV). The pericardial adipose tissue range is displayed (blue area).

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Statistical Analysis Was Performed Using SPSS 25.0 Software

The study analysed participants in two groups: those with T2DM combined with CHD and those with T2DM alone. For normally distributed data, mean values ± standard deviation (x±s) were used, with *t*-tests employed for intergroup comparisons. Non-normally distributed data were represented by median (P25, P75), with non-parametric tests applied. Count data were presented as [n (%)] and compared using χ^2-tests. Multivariate risk analysis was conducted using logistic regression. Receiver operating characteristic (ROC) curves were utilised to calculate the area under the curve (AUC) for evaluating the predictive capabilities of TyG and EATV, with optimal cutoff points determined by the maximum Jordon index. Significant differences were defined as P<0.05.

Result

Comparison of Clinical Data Between the Two Groups

No statistically significant differences were observed in age, gender, height, heart rate, BMI, or history of hypertension between the T2DM group and the T2DM with CHD group (P≥0.05). However, body weight was significantly higher in the T2DM with CHD group compared to the T2DM group alone (P<0.05) (see Table 1).

Comparison of Laboratory Data Between the Two Groups

No statistically significant differences were observed in FPG, HbA1c, ALT, AST, GGT, BUN, CREA, TC or LDL-C between the T2DM group and the T2DM with CHD group (P≥0.05). However, UA, TG, HDL-C, EATV and TyG levels were significantly higher in the T2DM with CHD group compared to the T2DM alone group (P<0.05) (see Table 2).

Table 1 Comparison of General Data Between the Two Groups

Group	T2DM Alone Group	T2DM with CHD Group	t/z/x^2	p
Number of cases	48	48	–	–
Age	58.77±8.89	59.42±8.59	−0.362	0.718
Sex (man)	24 (50%)	32 (66.7%)	2.743	0.098
Height	166.21±8.87	169.08±8.58	−1.615	0.110
Weight	70.01±8.01	75.76±12.63	−2.662	0.009
BMI	25.35±2.42	26.28±2.99	−1.857	0.066
Heart rate	82.67±10.59	84.56±11.13	−0.855	0.395
History of hypertension (yes)	29 (60.4%)	35 (72.9)	1.688	0.194

Table 2 Comparison of Laboratory Data Between the Two Groups

Group	T2DM Alone Group	T2DM with CHD Group	t/z	p
Number of cases	48	48		
FPG	7.18 (5.89, 8.19)	7.05 (6.40, 8.75)	−0.780	0.435
HbA1c	7.30 (6.53, 8.70)	7.85 (6.83, 8.70)	−1.052	0.293
AST	18.15 (15.23, 21.68)	18.50 (16.50, 24.13)	−0.707	0.479
ALT	17.65 (14.83, 28.98)	20.00 (14.45, 29.98)	−0.608	0.543
GGT	24.95 (18.55, 41.18)	28.30 (21.20, 52.28)	−1.385	0.166

(*Continued*)

Table 2 (Continued).

Group	T2DM Alone Group	T2DM with CHD Group	t/z	p
UA	334.82±73.32	375.50±85.90	−2.496	0.014
BUN	6.08±1.71	6.30±1.64	−0.666	0.507
CREA	61.58±14.38	67.40±15.43	−1.913	0.059
TG	1.38 (1.07, 1.97)	2.30 (1.44, 3.60)	−4.100	<0.001
TC	4.89±1.16	5.04±1.61	−0.538	0.592
HDL-C	1.09±0.17	0.95±0.26	3.137	0.002
LDL-C	2.69±0.87	2.71±1.07	−0.237	0.926
EATV	117.33±27.17	148.72±40.58	−4.452	<0.001
TyG	8.99±0.62	9.56±0.76	−4.018	<0.001

Multivariate Logistic Regression Analysis of Risk Factors for T2DM with CHD

The significant variables identified in Tables 1 and 2 were incorporated into a multivariate logistic regression model. The analysis revealed that both EATV and TyG were independent risk factors for T2DM combined with CHD (P<0.05) (see Table 3).

Comparison of Predictive Cut-off Values for T2DM with CHD

ROC curve analysis demonstrated that both EATV and TyG index showed statistically significant predictive power for CHD development in T2DM patients (P<0.05). The EATV exhibited an AUC of 0.739 with a predictive cut-off value of 128.45 mL (sensitivity 66.7%, specificity 72.9%). The TyG index demonstrated an AUC of 0.724 with a predictive cut-off value of 9.47 (sensitivity 58.3%, specificity 81.2%) (see Table 4 and Figure 2).

Table 3 Multivariate Logistic Regression Analysis of Risk Indicators for T2DM with CHD

Index	β	*P*	OR	95% CI
EATV	0.033	<0.001	1.033	1.015–1.051
TyG	1.377	0.001	3.964	1.821–8.629
Constant	−17.066	<0.001	0.000	–

Table 4 Comparison of Predictive Cut-off Points and Values of Different Indicators for T2DM with CHD

	Predictive Cutoff Value	AUC	95% CI	Sensitivity (%)	Specificity (%)
EATV	128.45	0.739	0.641–0.837	66.7	72.9
TyG	9.47	0.724	0.622–0.825	58.3	81.2

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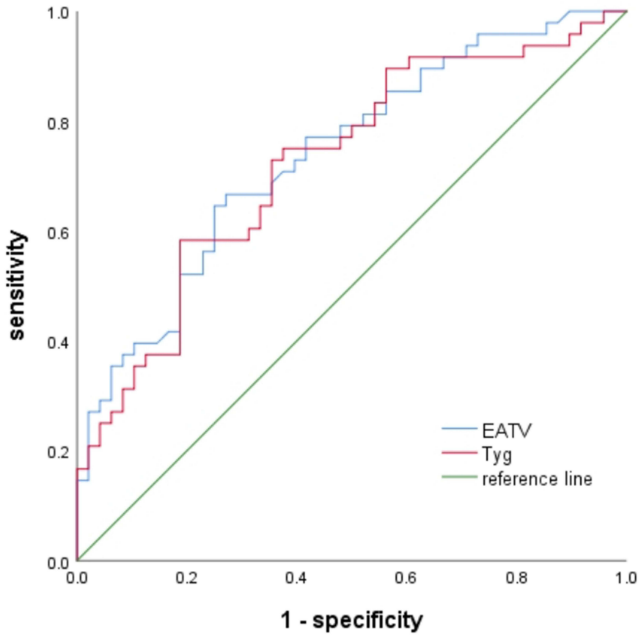

Figure 2 ROC curves of different indicators predicting the risk of CHD in T2DM patients.

Discussion

Previous studies have demonstrated that insulin resistance (IR) correlates strongly with cardiovascular disease risk across diverse populations, particularly in individuals diagnosed with T2DM and insulin-resistant coronary artery disease.[15] Atherosclerosis is a disease characterized by lipid accumulation of endothelial cells and activation of inflammatory cells. The formation and development of atherosclerotic plaque is the most important pathophysiological process in CHD. Recent research has reported that insulin resistance is involved in the formation and remodeling of coronary artery plaques, independent of traditional risk factors such as age, smoking, and hypertension.[16] Inflammatory stimulation, oxidative stress, abnormal glucose metabolism and endothelial cell damage create favorable conditions for abnormal lipid deposition. These factors usually promote each other, forming a vicious circle, and eventually leading to disease progression. Endothelial damage is considered as the core link, and insulin resistance is the key factor mediating endothelial damage, thus promoting the occurrence of atherosclerosis.[17] IR frequently triggers systemic inflammation, vascular endothelial dysfunction, oxidative stress, glucose metabolism disorders, and lipid metabolism disorders, ultimately accelerating atherosclerosis progression.[18] This may explain the potential mechanism underlying CHD development in T2DM patients.

The hyperinsulinaemic-euglycaemic clamp test is considered the gold standard for diagnosing IR. However, its clinical application remains limited due to high costs, complex procedures, and time-consuming requirements.[19] The triglyceride-glucose (TyG) index incorporates both triglycerides (TG) and fasting blood glucose. Elevated TG levels increase free fatty acids (FFAs), thereby reducing insulin sensitivity.[20] Sustained FFAs resulting from elevated TG may suppress AMP-activated protein kinase activity and promote TG accumulation, ultimately altering insulin signalling in pancreatic α-cells and inducing excessive glucagon secretion.[21] Recent evidence increasingly suggests that the TyG index serves as a simple and reliable surrogate marker for estimating IR, demonstrating comparable efficacy to the homeostasis model assessment of insulin resistance (HOMA-IR).[8,9] Compared to the hyperinsulinaemic-euglycaemic clamp test, the TyG index exhibits high sensitivity (96.5%) and specificity (85.0%) for detecting IR.[22] Recent studies have reported that serum triglyceride glucose product index not only reflects insulin resistance, but also is related to inflammation, endothelial dysfunction, glucose lipid metabolism disorder, thrombosis and other atherosclerosis factors.[20]

Li et al[23] enrolled a total of 518 patients with T2DM who underwent coronary angiography (CAG) as study participants. These patients were subsequently stratified into either the T2DM group or the T2DM with CHD group

based on diagnostic findings. Multivariate analysis adjusted for age, sex, smoking status, hypertension history, and use of hypoglycaemic/lipid-lowering medications demonstrated that the TyG index was an independent risk factor for CHD development in T2DM patients, with an AUC of 0.717 for CHD prediction. Furthermore, higher TyG index values showed significant correlation with both increased numbers of coronary artery stenoses and greater severity of CHD within the cohort. A retrospective cohort study[24] using data from the National Health and Nutrition Examination Survey (NHANES) 2007–2016 demonstrated that among elderly diabetic patients in the United States, the prevalence of coronary artery disease (CAD) showed a positive correlation with increasing TyG index. The highest TyG quartile was associated with a 48.0% increased cardiovascular disease (CVD) risk, and there was a U-shaped association between TyG index and CVD risk. Liu et al[25] analysed data from 2,440 young diabetic patients in NHANES (2001–2018), identifying a U-shaped relationship between TyG index and both all-cause mortality and CVD mortality in this population, with threshold values determined to be 9.18 and 9.16 for CVD and all-cause mortality respectively. The TyG index represents an independent risk factor for CAD and is also independently associated with severe CAD stenosis.[26] A study by Su et al[27] demonstrated that elevated TyG index levels can identify high-risk patients with multivessel CAD. Furthermore, increased TyG index correlates with severe dyslipidaemia, progressively impaired glucose metabolism status, and progression of coronary artery stenosis. A significant association between TyG index and CAD severity was observed in patients with diabetes mellitus (DM), whereas no such association was found in non-DM patients. However, Zhao et al[28] demonstrated that the TyG index showed a positive correlation with CAD severity across all glucose metabolism statuses, except in patients undergoing insulin therapy. In contrast, Wu et al[29] reported that while the TyG index was significantly associated with CAD, no correlation was observed between the TyG index and CAD severity regardless of glucose metabolism status. Park et al[30] concluded that the TyG index serves as an effective biomarker for the early detection of subclinical coronary atherosclerosis, even in the absence of traditional cardiovascular risk factors.

As the gold standard for diagnosing CHD, CAG is not only invasive but also costly. The distribution of adipose tissue represents a critical risk factor for cardiovascular diseases, with increasing attention being paid to the regional distribution and metabolism of adipose tissue. EAT accumulates on the cardiac surface, covering approximately 80% of the adult heart, predominantly over the right ventricle.[31] Although morphologically similar to pericardial adipose tissue, EAT demonstrates distinct embryonic origins and vascularisation patterns. EAT functions as a major source of both anti-inflammatory and pro-inflammatory adipokines, exerting significant effects on cardiac function and morphology.[32] Increased epicardial fat thickness appears to elevate the risk of metabolic syndrome, thereby contributing to the development of CAD. Consequently, EAT may represent a potential biomarker for cardiovascular risk.[11,12]

In a study of 503 patients undergoing coronary angiography, Shambu et al[33] measured EAT thickness, revealing significantly greater mean EAT thickness in the CAD group compared with controls. Furthermore, increased EAT thickness showed significant correlation with both CAD incidence and severity in diabetic patients. The prevalence of single-vessel, double-vessel, and triple-vessel disease demonstrated progressive elevation with increasing EAT thickness, suggesting that EAT may not only indicate the presence of CAD but also reflect the extent of coronary involvement.[34] A study[35] demonstrated that compared with the non-ACS group, the ACS group showed significantly higher EATV and epicardial adipose tissue density (EATD). Increased EATV was significantly associated with larger volumes of fibrotic plaques and lipid-rich plaques in the coronary arteries, particularly in the right coronary artery and left anterior descending artery.[36] Consistent with these findings, Gao et al[37] reported that both EATV and EATD correlated with high-risk plaque (HRP). For every standard deviation increase in EATD, the risk of HRP was 3.120 times higher than that of non-HRP; similarly, for every standard deviation increase in EATV, the HRP risk was 1.499 times greater than that of non-HRP. In the study by Bertaso et al,[38] an independent association was observed between EAT and cardiovascular risk factors, coronary artery calcification, and carotid artery stenosis. Moreover, EATV was identified as an independent risk factor for left ventricular diastolic dysfunction in patients with CAD, with an AUC of 0.79 for diagnosing left ventricular diastolic dysfunction in CAD patients.[39] Additionally, research has demonstrated[40] that EATV is significantly associated with coronary atherosclerosis in T2DM patients without a history of CAD. In a study conducted by Uygur et al,[41] 127 patients with DM were prospectively followed. The study population was stratified into two groups based on the occurrence of major adverse cardiovascular events (MACE). Results demonstrated that EATV was an independent

predictor of long-term MACE in T2DM patients without prior coronary events, with an AUC of 0.820 and an optimal cut-off value of 123.2 mL.

The findings indicate that EATV and TyG index are independent risk factors for CHD in patients with T2DM. These indicators serve as cost-effective early risk assessment tools to identify high-risk groups for CHD in T2DM patients, providing a basis for targeted interventions against cardiovascular disease in diabetic patients.

This study has several limitations. As a retrospective study with a limited sample size, it included only patients from Qinhuangdao city, which may limit the generalisability of the findings to other regions. To improve the accuracy and reduce potential bias in future research, a multi-centre, multi-regional study design with larger sample sizes should be adopted, encompassing broader age ranges, ethnic groups, disease conditions and other relevant variables.

Data Sharing Statement

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

Ethical Approval

The present trial followed the Declaration of Helsinki (2000) and had approval from the Ethics committee of First Hospital of Qinhuangdao [2024W015]. The requirement for informed consent was waived by the Institutional Review Board of Ethics committee of First Hospital of Qinhuangdao because of the retrospective nature of the study.

Author Contributions

Binbin Liu: Investigation, Formal analysis, Data curation, Writing – original draft. Juan Du: Investigation, Formal analysis, Data curation, Writing – original draft. Ziru Niu: Investigation, Formal analysis, Data curation, Conceptualization, Writing – original draft. Qiang Lu: Data curation, Formal analysis, Investigation, Funding acquisition, Writing – review and editing. All authors took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no commercial or financial relationships that could be construed as a potential conflict of interest. The authors declare that they have no conflicts of interest.

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