Clinical and Applied Thrombosis/Hemostasis
Volume 28: 1-7

DOI: 10.1177/10760296221094030
journals.sagepub.com/home/cat
SAGE

The Association of Triglyceride Glucose index for Coronary Artery Disease in Postmenopausal Women

Jingyi Liu, MM[1,2], Haiwei Bu, MM[2], Zhenjiang Ding, MM[2], Ying Zhang, MM[2], Yongjie Chen, PhD[3], and Yuxia Gao, MD[1]

Abstract

This study aimed to explore the association between the triglyceride glucose (TyG) index and coronary artery disease (CAD) in postmenopausal women. This study enrolled 869 postmenopausal women and classified them into two groups: CAD group (n = 538) and control group (n = 331). The TyG index was significantly higher in patients with CAD than in controls ($P < 0.05$).Receiver operator characteristic curves showed that the TyG index was more discriminative for CAD than for control group, and after adjusting for the traditional clinical prognostic factors, including age (>60 years), diabetes, ischemic stroke, systolic blood pressure (≥140), and ejection fraction (<50%), we found that the TyG index could be an independent risk factor for CAD ($P < 0.05$). The risk of increased TyG index was greater in the <50 years subgroup than in the >50 years subgroup ($P < 0.05$). The TyG index may be a valuable clinical predictor of CAD risk in postmenopausal women.

Keywords

triglyceride glucose index, coronary artery disease, Gensini score, postmenopausal women

Date received: 28 November 2021; revised: 12 March 2022; accepted: 28 March 2022.

Introduction

Globally, coronary artery disease (CAD) is a major cause of morbidity and mortality,[1] especially in postmenopausal women.[2] The postmenopausal phase has been considered an important factor for developing metabolic syndrome. Insulin resistance (IR) is regarded as a decline in insulin in promoting glucose uptake and utilization, which is a marker of metabolic imbalance.[3] Previous research has shown that IR is strongly related to the development and progression of coronary atherosclerosis, plaque characteristics, and cardiovascular (CV) outcomes.[4] In addition, IR is involved in inflammation,[5] endothelial dysfunction, and cardiac autonomic function,[6] which is reported to play a vital role in the pathogenesis of CAD.[7] Increasing evidence in postmenopausal women suggests a protective effect of endogenous estrogen against CAD, whereas its deficiency exacerbates the process of CAD after menopause.[8]

The triglyceride glucose (TyG) index is a novel, simple, and inexpensive alternative index of IR[9] and has been proven to be a good marker for predicting subclinical CAD in the absence of traditional risk factors.[10]The formula is ln [TG(mg/dL) × fasting glucose(mg/dL)/2) to calculate TyG index.[4]

However, the association between the TyG index and CAD in postmenopausal women remains unclear. Therefore, this study aimed to examine whether the TyG index is a valuable biomarker for predicting the presence and severity of CAD in postmenopausal women.

Methods

Participants

This is a single center, retrospective, observational study.A total of 869 inpatients with suspected CAD were consecutively enrolled in this study, between December 2015 and January

[1] Department of Cardiology, General Hospital of Tianjin Medical University, Tianjin, China
[2] Department of Cardiology, The Affiliated Hospital of Chengde Medical University, Chengde, HeBei, China
[3] Department of Epidemiology and Statistics, School of Public Health, Tianjin Medical University, Tianjin, China

Corresponding Author:
Yuxia Gao,M.D., Address Department of Cardiology, Tianjin Medical University General Hospital, Tianjin Medical University, No. 154, Anshan Road, Heping District, Tianjin 300052, China.
Email: tjgaoyuxia@126.com

2019, at the Affiliated Hospital of Chengde Medical College. They were divided into two groups based on coronary angiography (CAG): the CAD group (n = 538) and the control group (n = 331). The inclusion criteria for the CAD group included postmenopausal women and suspected CAD with any stenosis of coronary arteries or ≥50% stenosis of the left main, left anterior descending, left circumflex, right coronary, or their main branches.The exclusion criteria were as follows: (1) infections, malignancies, hematopoietic or immune system disease of any form, or coronary embolism (2) hypertrophic cardiomyopathy and (3) connective tissue disease combined with coronary artery vasculitis. The Gensini score is a comprehensive score that assesses the extent of CAD burden on angiography. This score is calculated as the sum of the severity scores assigned depending on the degree of angiographic luminal stenosis in each segment of the coronary artery, exponentially increasing by the severity.of lesions (25%, 50%, 75%, 90%, 99%, and 100% coronary stenosis), with a cumulative effect according to multiple lesions and lesion location. The study complies with the Declaration of Helsinki.

Demographic and Clinical Data

Baseline demographic and clinical data were recorded. Body mass index was calculated by direct measurements as weight divided by the square of height. Blood pressure was measured using an oscillometric method (Omron, Hoofddorp, Netherlands). Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TGs), and low-density lipoprotein cholesterol (LDL-C) levels were measured using standard enzymatic methods. Left ventricular end-diastolic dimension, left ventricular end-systolic diameter, left ventricular ejection fraction, calcification of the aorta, and abnormal ventricular wall motion were also recorded. EThe traditional risk factors for CAD, such as diabetes, hypertension, dyslipidemia, ischemic stroke, current smoking, and family history of CAD were assessed. The atherosclerosis index (AI) was calculated as (TC − HDL-C)/HDL-C.[11] Atherogenic index of plasma (AIP) was calculated using log10(TG/HDL-C).[12] The lipoprotein combine index (LCI) was calculated using the formula TC × TG × LDL-C/HDL-C.[13] TyG was calculated as ln [TG(mg/dL) × fasting glucose(mg/dL)/2).[4] Natural menopause was defined as the absence of menstruation over a period of 12 months.[14] In our research,we study the correlation and diagnostic value of TyG index and AI,AIP and LCI.

Statistical Analyses

Statistical analyses were performed using the SPSS (version 26.0; SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to analyze continuous data with normal or skewed distributions. Mean ± standard deviation and quartile median were used to express normal and skewed continuous data, respectively. To investigate differences between the groups, the t-test was applied for normally distributed continuous variables, the Mann-Whitney U-test for continuous variables with abnormal distribution, and the Chi-squared test for categorical variables. Bivariate correlations between the TyG index and risk factors were calculated using Spearman's rank correlation, and Pearson correlation was used to prove the association of TyG index and other continuous data, including demographics, risk factors, and biomarkers. Gensini score according to the maximum Yorden index through ROC curve to divided into 2 groups.Logistic regression analysis was performed to investigate unadjusted and multivariable-adjusted associations of the TyG index with CAD. A two-tailed P value of <0.05 was considered statistically significant.

Results

Baseline Characteristics of the CAD and Control Groups

The prevalence of hyperlipidemia, hypertension, diabetes mellitus, ischemic stroke, current smoker, and family history were higher in CAD group than control group. Furthermore, age, heart rate, systolic blood pressure (SBP), AI, AIP, LCI, and TyG index were significantly higher in the CAD group as compared to the control group (all $P < 0.05$). Similarly, the left ventricle end-systolic diameters and Gensini score were higher in the CAD group than in the control group; however, the ejection fraction was lower in the CAD group (all $P < 0.001$). The proportion of calcification of the aorta and abnormal ventricular wall motion were common in the CAD group. Moreover, levels of TC, TG, LDL-C, and blood glucose were higher in the CAD group than in the control group (all $P < 0.05$) (Table 1).

TyG index According to Different Coronary Artery Severity

AI, LCI, and TyG index were significantly higher in the Gensini≥38.75 group than the Gensini <38.75 group(all $P < 0.05$).Although AIP was no significant difference between the two groups, we also found AIP in the Gensini≥38.75 group was greater than the Gensini <38.75 group. (Table 2).

Association of the TyG index and Traditional CAD Risks Factors

Table 3 demonstrated that the TyG index had a significant positive correlation with hyperlipidemia, hypertension, diabetes mellitus, TC, TG, LDL-C, AI, AIP, LCI, blood glucose, and Gensini score (Rs = 0.352, 0.117, 0.360, 0.230, 0.666, 0.080, 0.336, 0.623, 0.518, 0.107, 0.476, and 0.199, respectively; all $P < 0.05$). Moreover, the TyG index was negatively correlated with HDL-C (Rs = -0.191; $P < 0.05$).

Receiver-Operating Characteristic Curve Analyses of the CAD and Control Group Subjects

The area under the curve (AUC) of the AI, AIP, LCI and TyG index were 0.578,0.570,0.583 and 0.615,respectively. The

Table 1. Baseline Characteristics of Both the Coronary Artery Disease and Control Groups.

Factors	CAD Group (n = 538)	Control Group (n = 331)	χx2/Z	*p*-Value
Age	62.00 (57.00, 67.00)	60.00 (56.00, 65.00)	−6.064	<0.001
BMI (kg/m^2)	25.00 (22.90, 27.30)	25.90 (23.90, 29.00)	−2.970	0.003
Hyperlipidemia (n, %)	150.00 (27.90)	97.00 (29.30)	0.204	0.651
Hypertension (n, %)	389.00 (72.30)	163.00 (49.20)	47.028	<0.001
T2DM (n, %)	169.00 (31.40)	56.00(16.90)	22.436	<0.001
Ischemic stroke (n, %)	99.00 (18.40)	37.00 (11.20)	8.099	0.004
Current smoker (n, %)	37.00 (6.90)	10.00 (3.00)	5.956	0.015
Family history of CAD (n, %)	54.00 (10.00)	27.00 (8.20)	0.857	0.355
HR (bpm)	74.00 (66.00, 83.00)	73.00 (64.00, 82.00)	−2.341	0.019
SBP (mm Hg)	140.00 (125.00, 155.00)	135.00 (120.00, 149.00)	−5.496	<0.001
DBP (mm Hg)	80.00 (72.00, 90.00)	80.00 (72.00, 90.00)	−0.592	0.554
LVEDD (mm)	48.00 (46.00, 50.00)	48.00 (46.00, 50.00)	0.112	0.911
LVESD (mm)	32.00 (30.00, 35.00)	31.00 (29.00, 34.00)	−2.821	0.005
EF (%)	61.00 (56.00, 66.00)	62.00 (58.50, 67.00)	−4.539	<0.001
Calcification of the aorta (n, %)	69.00 (19.90)	27.00 (9.30)	14.043	<0.001
Abnormal ventricular wall motion (n, %)	122.00 (33.90)	40.00 (13.30)	37.600	<0.001
TC (mmol/L)	4.60 ± 1.70	4.30 ± 1.20	3.002	0.003
TG (mmol/L)	1.60 (1.10, 2.40)	1.40 (1.10, 2.10)	−3.375	0.001
LDL-C (mmol/L)	2.40 ± 0.90	2.2 ± 0.8	2.995	0.003
HDL-C (mmol/L)	1.10 (0.98, 1.30)	1.20 (0.98, 1.30)	−1.732	0.083
LCI	15.64 (8.02, 29.64)	13.60(6.99, 26.85)	−4.335	<0.001
AIP	0.18 (−0.02, 0.38)	0.15(−0.05, 0.35)	−3.474	0.001
AI	2.90 (2.16, 3.91)	2.78 (2.07, 3.65)	−3.937	<0.001
TyG	9.10 (8.6, 9.6)	8.90 (8.50, 9.40)	−5.641	<0.001
BUN (mmol/L)	5.40 (4.50, 6.50)	5.20 (4.50, 6.40)	−1.682	0.093
Blood glucose (mmol/L)	6.30 (5.40, 8.60)	5.80 (5.10,7.20)	−6.599	<0.001
Genisi Score	43.00 (34.50, 72.50)	13.00 (9.25, 15.00)	−23.170	<0.001

AI: atherosclerosis index, AIP: Atherogenic index of plasma, BMI: body mass index, BUN: blood urea nitrogen, CAD: coronary artery disease, DBP: diastolic blood pressure, EF: Ejection fraction, HDL-C: high-density lipoprotein cholesterol, LCI: lipoprotein combine index, LDL-C: low-density lipoprotein cholesterol, LVEDD: left ventricular end-diastolic dimension, LVESD: left ventricular end-systolic dimension, SBP: systolic blood pressure, T2DM: type-2 diabetes mellitus, TC: total cholesterol, TG: triglyceride, TyG: triglyceride glucose.

Table 2. TyG Index According to Different Coronary Artery Severity.

Variables	Gensini Score ≥ 38.75	Gensini Score < 38.75	*Z*	*P*
TyG	9.243 (8.743,9.733)	8.905 (8.584,9.431)	−2.560	0.010
AI	3.009 (2.190,3.930)	2.453 (1.703,3.495)	−2.478	0.013
AIP	0.182 (−0.022,0.387)	0.143 (0.018,0.280)	0.759	0.448
LCI	16.026 (8.444,31.088)	12.525 (4.700,22.111)	−2.401	0.016

AI: atherosclerosis index, AIP: Atherogenic index of plasma, LCI: lipoprotein combine index,TyG: triglyceride glucose.

optimal diagnostic cut-off point for AI,AIP,LCI and TyG index were found to be 2.793, 0.029, 13.687 and 9.432, respectively. Surprisingly, the AUC of TyG was higher than that of AI, AIP, and LCI (Table 4).

Univariate Logistic Regression Analysis of CAD Risk Factors

Univariate associations with CAD were examined for the baseline demographic and clinical characteristics, and increasing age, SBP, TC, TG, LDL-C, AI, AIP, LCI, and TyG were found to have a significant univariate association. The following variables were shown to be statistically significant in the univariate associations: hyperlipidemia, hypertension, diabetes mellitus, ischemic stroke, calcification of the aorta, and ejection fraction <50% (Table 5).

Multiple Logistic Regression Analysis of CAD Risk Factors

The multivariate logistic regression model showed that age of >60 years, diabetes mellitus, ischemic stroke, SBP of ≥140, ejection fraction of <50%, and TyG of ≥9.432 were independent risk factors for CAD, and the odds ratios of these factors were 2.031 (1.465, 2.815), 1.625 (1.080, 2.445), 1.782 (1.104, 2.877), 1.933 (1.409, 2.651), 3.736 (2.068, 6.748), and 1.876 (1.299, 2.710), respectively (all $P < 0.05$). (Table 6).

Comparison of the TyG index with CAD Subgroups of Different Ages

To evaluate the association between age-specific CAD and the TyG index, we established logistic regression analysis models 1

and 2 with different adjusted CAD risk factors. The association was consistent after further adjustment for hypertension, diabetes mellitus, and ischemic stroke. When adjusting for ejection fraction of <50%, SBP of ≥140, as well as for hypertension, diabetes mellitus, and ischemic stroke, a necessary association between the TyG index and CAD was predominant (all $P < 0.05$, Table 7).

Discussion

To the best of our knowledge, at present, there is limited information about the TyG index and its association with CAD in postmenopausal women. In the present study, we found that the TyG index was prominently associated with the prevalence of CAD in postmenopausal women. In addition, we also found that the TyG index could predict CAD in women at different menopausal ages.

CAD is the leading cause of morbidity and mortality worldwide.[15] In the view of CAD progression, numerous conditions such as postmenopausal status,[16] dyslipidaemia, diabetes mellitus have been regarded as high-risk factors.[17] For this reason, it is necessary to discover new indicators for predicting CAD in postmenopausal women. In previous studies, the AI was a classical index to measure the degree of atherosclerosis. It is the ratio of a series of risk factors to protective factors involved in the process of atherosclerosis and possesses more clinical value than any other single index.[18] Furthermore, Si *et al* reported that the LCI was higher in acute coronary artery disease patients than in non-CAD patients.[19] However, there was no association between AI, LCI, and CAD in postmenopausal women who underwent CAG. AIP is correlated with the atherosclerosis burden.[20] In previous study, AIP was positively related to the severity of CAD in elderly individuals and was superior to other lipid profiles.[21] So we compared the differences between AIP and TyG for coronary artery disease in postmenopausal women.In addition, Wang *et al* demonstrated that the AIP, as a biomarker, assists in the prevention of CAD in the Chinese population.[12] Guo *et al* and Wu *et al* observed that AIP could be a powerful independent predictor of CAD risk in postmenopausal women.[13,22]

A recent study showed that the TyG index was an inexpensive clinical surrogate marker for IR,which expresses a noticeable underlying disorder contributing to CAD.[23] IR causes arteriosclerosis due to chronic hyperinsulinemia. Chronic hyperinsulinemia increases the synthesis of very low-density lipoprotein cholesterol, proliferation of vascular smooth muscle cells, transport of LDL cholesterol into arterial smooth muscle cells, and activation of inflammation genes.[24] In addition, IR stimulates the progression of CAD by disrupting glucose metabolism, weakening systemic lipid metabolism, and causing endothelial dysfunction.[25] There are connections between the TyG index and a fair amount of CAD risk, including arterial stiffness and coronary artery calcification.[26–28] A series of reports have shown that the TyG index predicts CAD severity[29] and CV outcomes in patients with non-ST-segment elevation ACS[30] and myocardial infarction.[31,32] Moreover, the TyG index could be a valuable predictor of adverse CV outcomes after PCI in patients with type 2 diabetes mellitus and ACS.[9]

Epidemiological studies have mentioned that the rates of morbidity and mortality due to CAD are markedly higher in postmenopausal women than in premenopausal women.[33] The onset of heart disease in women reportedly occurs much later than that in men, and young women suffer less from heart disease due to the vascular protective action of estrogen, which prevents atherosclerosis.[34,35] Mazzuca found that estrogen binding to estrogen receptors (ERs) could promote vasodilation[36] and reduce the response of blood vessels to the progression of atherosclerosis.[37] Loss of estrogen early in life might destroy vascular

Table 3. Correlation Between the Triglyceride Glucose Index and Other Variables.

Factors	Rs	*P*-value
Hyperlipidemia	0.352	<0.001
Hypertension	0.117	<0.001
T2DM	0.360	<0.001
TC	0.230	<0.001
TG	0.666	<0.001
LDL	0.080	0.019
HDL	−0.191	<0.001
AI	0.336	<0.001
AIP	0.623	<0.001
LCI	0.518	<0.001
Blood glucose	0.476	<0.001
Gesini score	0.199	<0.001

AI, atherosclerosis index, AIP, Atherogenic index of plasma, HDL-C, high-density lipoprotein cholesterol, LCI, lipoprotein combine index, LDL-C, low-density lipoprotein cholesterol, Rs,Ratio scale, T2DM, type-2 diabetes mellitus, TC, total cholesterol, TG, triglyceride, TyG, triglyceride glucose.

Table 4. The Receiver Operating Characteristic Curves of the Triglyceride Glucose Index, Atherosclerosis Index, Atherogenic Index of Plasma, and Lipoprotein Combine Index.

Variables	AUC	SE	*P*	95% CI	Se (%)	Sp (%)	Cut off
TyG	0.615	0.020	<0.001	0.577–0.654	40.5	78.1	9.432
AI	0.578	0.020	<0.001	0.539–0.617	56.0	60.5	2.793
AIP	0.570	0.020	0.001	0.530–0.609	71.6	40.8	0.029
LCI	0.583	0.020	<0.001	0.544–0.622	55.6	58.3	13.687

AI: atherosclerosis index, AIP: Atherogenic index of plasma, AUC: area under the curve, CI: confidence interval, LCI: lipoprotein combine index, SE:Standard Error, Se: sensitivity, Sp:specificity, TyG: triglyceride glucose

function and promote the expression of inflammatory factors at a younger age, which could further damage vascular function.[38] Androgen and sex hormone-binding globulin are also associated with the risk of CV disease.[39] Androgen and sex hormone-binding globulin are associated with both postmenopausal major adverse cardiovascular events[40] and serious CAD risk factors. Menopause is a complex physiological process resulting from the declined production and secretion of ovarian hormones, with increasing age, estradiol levels decrease until menopause, when they are of the same level as seen in men (5-20 pg/ml).[41] Endothelial function provides the mechanisms leading to the development and progression of atherosclerosis with menopause, and coronary endothelial function is impaired by both traditional and non-traditional CAD risk factors that predict future CV events.[42,43] Lambrinoudaki *et al* demonstrated that the TyG index was relevant to arterial stiffness, mainly in lean postmenopausal women.[44] In a recent study, Nakagomi *et al*[45] studied sex differences in the association between the TyG index and arterial stiffness, and in contrast to previous studies, our study showed that the TyG index was of more predictive value for CAD in postmenopausal women than AI, AIP, and LCI. In this retrospective cohort study, we also found that the TyG index was a strong and independent risk factor for CAD in postmenopausal women, providing incremental diagnostic value compared to the well-known, traditional CV risk factors. Additionally, the TyG index is prominently associated with well-known CV risk factors.

Menopause age might be regard as a crucial factor in the risk stratification of CV disease in women.[38] According to the SWAN study's methodology, using 50 to differentiate premenopause and postmenopause.[17] In our study, we observed a subgroup of CAD patients and found that the TyG index was a crucial predictor of CAD, independent of the traditional CV risk factors, which might support the hypothesis that the TyG index may predict CAD in postmenopausal women.

Table 5. Univariate Logistic Regression Analysis of the Association Between Coronary Artery Disease and Other Various Factors.

Variables	OR	95% CI	*P*
Age > 60(years)	2.800	1.720–4.559	<0.001
Hyperlipidemia	1.482	1.112–1.976	0.007
Hypertension	2.210	1.365–3.581	0.001
T2DM	2.340	1.311–4.175	0.004
Ischemic stroke	2.596	1.225–5.504	0.013
SBP≥140mm Hg	1.719	1.063–2.780	0.027
EF < 50%	2.778	1.154–6.688	0.023
Calcification of the aorta	2.436	1.513–3.920	<0.001
TC > 5.2 (mmol/L)	1.733	1.234–2.434	0.001
TG > 1.7 (mmol/L)	1.392	1.005–1.837	0.019
LDL-C > 3.4 (mmol/L)	1.846	1.179–2.890	0.007
AI≥ 2.793	1.939	1.468–2.561	<0.001
AIP≥0.029	1.527	1.157–2.015	0.003
LCI≥13.687	1.830	1.387–2.415	<0.001
TyG≥9.432	2.788	1.607–4.835	<0.001

AI: atherosclerosis index, AIP: Atherogenic index of plasma, CI: confidence interval, EF: Ejection fraction, LCI: lipoprotein combine index, LDL-C: low-density lipoprotein cholesterol, OR: odds ratio, SBP: systolic blood pressure, T2DM: type-2 diabetes mellitus, TC: total cholesterol, TG: triglyceride, TyG: triglyceride glucose.

Table 6. Multivariate Logistic Regression Analysis of the Risk of Coronary Artery Disease and the Triglyceride Glucose Index.

Factor	OR	95% CI	*P*
Age > 60(years)	2.031	1.465–2.815	<0.001
T2DM	1.625	1.080–2.445	0.020
Ischemic stroke	1.782	1.104–2.877	0.018
SBP≥140(mm Hg)	1.933	1.409–2.651	<0.001
EF < 50%	3.736	2.068–6.748	<0.001
TyG≥9.432	1.876	1.299–2.710	0.001

CI: confidence interval, EF: Ejection fraction, OR: odds ratio, SBP: systolic blood pressure, T2DM: type 2 diabetes mellitus, TyG: triglyceride glucose.

Limitations

Our study has several potential limitations. First, the sample size of the patients in this study was relatively small. We need a lager sample size to compare such parameters between pre- and postmenopausal CAD subjects and subanalyse the CAD group based on disease severity.Second, this was a single-center study, and patient selection may have been subject to bias.

Table 7. Post-menopausal Age-specific Associations Between the Triglyceride Glucose Index and Other Selected Coronary Artery Disease-related Factors.

	Age≥50 (N = 583)			Age < 50 (N = 286)		
	CAD Group (n = 369)	Non-CAD Group (n = 214)		CAD Group (n = 169)	Non-CAD Group (n = 117)	
	OR	95% CI	*P*	OR	95% CI	*P*
Unadjusted	2.062	1.455–2.923	<0.001	3.230	1.823–5.724	<0.001
Model1-adjusted	1.779	1.220–2.595	0.003	2.602	1.415–4.787	0.002
Model2-adjusted	1.615	1.074–2.431	0.021	2.882	1.484–5.597	0.002

Model 1: Hypertension, T2DM, Ischemic stroke.
Model 2: Model1 + EF < 50%, SBP≥140mm Hg.
CAD: coronary artery disease, CI: confidence interval, EF: Ejection fraction, OR: odds ratio,SBP: systolic blood pressure, T2DM: type-2 diabetes mellitus

Conclusion

We found that the TyG index was a strong, independent CV risk factor, providing important, novel insights into the diagnosis of CAD in postmenopausal women. In particular, menopausal age of <50 years was a much stronger predictor than menopausal age >50 years for the diagnosis of CAD.

Acknowledgements

We are grateful for the assistance of doctors and technicians in the Division of Cardiology, The Affiliated Hospital of Chengde Medical University. We thank Dr Yongjie Chen for helping with the statistical analysis.

Author Contributions

JY.L. contributed to the conception and design of the study, the acquisition, analysis, and interpretation of the data, and drafting of the manuscript. YX.G.contributed to the methodology and validation.HW.B., ZJ.D., Y.Z., and YJ.C. contributed to the data acquisition. YX.G. contributed to the critical revision of the manuscript for important intellectual content. All authors approved the final version of the manuscript for publication.

Authors' Note

Ethical approval to report this case was obtained from the Institutional Review Board of The Affiliated Hospital of Chengde Medical University. Written informed consent was obtained from the patients for their anonymized information to be published in this article.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical Approval

The protocol was approved by the Institutional Ethics Committee of the Affiliated Hospital of Chengde Medical University (Number: CYFYLL2022150).

Informed Consent

Participants provided informed consent.

ORCID iDs

Jingyi Liu https://orcid.org/0000-0001-7123-0119
Zhenjiang Ding https://orcid.org/0000-0002-1243-8419
Ying Zhang https://orcid.org/0000-0002-4822-4295
Yuxia Gao https://orcid.org/0000-0002-9246-320X

References

1. Malakar AK, Choudhury D, Halder B, et al. A review on coronary artery disease, its risk factors, and therapeutics. *J Cell Physiol*. 2019;234(10):16812-16823.
2. Lee SN, Cho JY, Eun YM, et al. Associations between osteoporosis and coronary artery disease in postmenopausal women. *Climacteric*. 2016;19(5):458-462.
3. Hill MA, Yang Y, Zhang L, et al. Insulin resistance, cardiovascular stiffening and cardiovascular disease. *Metabolism*. 2021;119:154766.
4. Cho YR, Ann SH, Won KB, et al. Association between insulin resistance, hyperglycemia, and coronary artery disease according to the presence of diabetes. *Sci Rep*. 2019;9(1):6129.
5. Beverly JK, Budoff MJ. Atherosclerosis: pathophysiology of insulin resistance, hyperglycemia, hyperlipidemia, and inflammation. *J Diabetes*. 2020;12(2):102-104.
6. Poon AK, Whitsel EA, Heiss G, et al. Insulin resistance and reduced cardiac autonomic function in older adults: the atherosclerosis risk in communities study. *BMC Cardiovasc Disord*. 2020;20(1):217.
7. Ding X, Wang X, Wu J, et al. Triglyceride-glucose index and the incidence of atherosclerotic cardiovascular diseases: a meta-analysis of cohort studies. *Cardiovasc Diabetol*. 2021;20(1):76.
8. Liu N, Chen J, Zhang K, et al. A community-based study of the relationship between coronary artery disease and osteoporosis in Chinese postmenopausal women. *Coron Artery Dis*. 2016;27(1):59-64.
9. Alizargar J, Bai CH, Hsieh NC, et al. Use of the triglyceride-glucose index (TyG) in cardiovascular disease patients. *Cardiovasc Diabetol*. 2020;19(1):8.
10. Park GM, Cho YR, Won KB, et al. Triglyceride glucose index is a useful marker for predicting subclinical coronary artery disease in the absence of traditional risk factors. *Lipids Health Dis*. 2020;19(1):7.
11. Maleki MH, Mousavi M, Kazemi T, et al. Comparison of atherogenic index and lipid profiles in candidates for coronary artery bypass graft surgery versus normal people. *Pak J Pharm Sci*. 2018;31(5):1899-1902.
12. Wang L, Chen F, Xiaoqi C, et al. Atherogenic index of plasma is an independent risk factor for coronary artery disease and a higher SYNTAX score. *Angiology*. 2021;72(2):181-186.
13. Wu TT, Gao Y, Zheng YY, et al. Atherogenic index of plasma (AIP): a novel predictive indicator for the coronary artery disease in postmenopausal women. *Lipids Health Dis*. 2018;17(1):197.
14. Nelson HD. Menopause. *Lancet*. 2008;371(9614):760-770.
15. West HW, Antoniades C. Imaging and targeting coronary artery inflammation. *Antioxid Redox Signal*. 2021;34(15):1217-1243.
16. Colpani V, Baena CP, Jaspers L, et al. Lifestyle factors, cardiovascular disease and all-cause mortality in middle-aged and elderly women: a systematic review and meta-analysis. *Eur J Epidemiol*. 2018;33(9):831-845.
17. Reeh J, Therming CB, Heitmann M, et al. Prediction of obstructive coronary artery disease and prognosis in patients with suspected stable angina. *Eur Heart J*. 2019;40(18):1426-1435.
18. Frontini MG, Srinivasan SR, Xu JH, et al. Utility of non-high-density lipoprotein cholesterol versus other lipoprotein measures in detecting subclinical atherosclerosis in young adults (The Bogalusa Heart Study). *Am J Cardiol*. 2007;100(1):64-68.

19. Si Y, Liu J, Han C, et al. The correlation of retinol-binding protein-4 and lipoprotein combine index with the prevalence and diagnosis of acute coronary syndrome. *Heart Vessels*. 2020;35(11):1494-1501.
20. Wu J, Zhou Q, Wei Z, Wei J, Cui M. Atherogenic Index of plasma and coronary artery disease in the adult population: a meta-analysis. *Front Cardiovasc Med*. 2021;8:817441.
21. Huang H, Yu X, Li L, et al. Atherogenic index of plasma is related to coronary atherosclerotic disease in elderly individuals: a cross-sectional study. *Lipids Health Dis*. 2021;20(1):68.
22. Guo Q, Zhou S, Feng X, et al. The sensibility of the new blood lipid indicator–atherogenic index of plasma (AIP) in menopausal women with coronary artery disease. *Lipids Health Dis*. 2020;19(1):27.
23. Hong S, Han K, Park CY. The triglyceride glucose index is a simple and low-cost marker associated with atherosclerotic cardiovascular disease: a population-based study. *BMC Med*. 2020;18(1):361.
24. Di Pino A, DeFronzo RA. Insulin resistance and atherosclerosis: implications for insulin-sensitizing agents. *Endocr Rev*. 2019;40(6):1447-1467.
25. Ormazabal V, Nair S, Elfeky O, et al. Association between insulin resistance and the development of cardiovascular disease. *Cardiovasc Diabetol*. 2018;17(1):122.
26. Chiu TH, Tsai HJ, Chiou HC, et al. A high triglyceride-glucose index is associated with left ventricular dysfunction and atherosclerosis. *Int J Med Sci*. 2021;18(4):1051-1057.
27. Park K, Ahn CW, Lee SB, et al. Elevated TyG index predicts progression of coronary artery calcification. *Diabetes Care*. 2019;42(8):1569-1573.
28. Won KB, Park EJ, Han D, et al. Triglyceride glucose index is an independent predictor for the progression of coronary artery calcification in the absence of heavy coronary artery calcification at baseline. *Cardiovasc Diabetol*. 2020;19(1):34.
29. Wang L, Cong HL, Zhang JX, et al. Triglyceride-glucose index predicts adverse cardiovascular events in patients with diabetes and acute coronary syndrome. *Cardiovasc Diabetol*. 2020;19(1):80.
30. Mao Q, Zhou D, Li Y, Wang Y, Xu SC, Zhao XH. The Triglyceride-Glucose index predicts coronary artery disease severity and cardiovascular outcomes in patients with non-ST-segment elevation acute coronary syndrome. *Dis Markers*. 2019;2019:6891537.
31. Tian X, Zuo Y, Chen S, et al. Triglyceride-glucose index is associated with the risk of myocardial infarction: an 11-year prospective study in the Kailuan cohort. *Cardiovasc Diabetol*. 2021;20(1):19.
32. Zhang Y, Ding X, Hua B, et al. High triglyceride-glucose index is associated with adverse cardiovascular outcomes in patients with acute myocardial infarction. *Nutr Metab Cardiovasc Dis*. 2020;30(12):2351-2362.
33. Xu R, Cheng XC, Zhang Y, et al. Association of severity of coronary lesions with bone mineral density in postmenopausal women. *Arq Bras Cardiol*. 2018;110(3):211-216.
34. Sousa NFDS, Lima MG, Cesar CLG, et al. Active aging: prevalence and gender and age differences in a population-based study. *Cad Saude Publica*. 2018;34(11):e00173317.
35. Dugani SB, Moorthy MV, Li C, et al. Association of lipid, inflammatory, and metabolic biomarkers with age at onset for incident coronary heart disease in women. *JAMA Cardiol*. 2021;6-(4):437-447.
36. Mazzuca MQ, Mata KM, Li W, et al. Estrogen receptor subtypes mediate distinct microvascular dilation and reduction in [Ca2 +]I in mesenteric microvessels of female rat. *J Pharmacol Exp Ther*. 2015;352(2):291-304.
37. Zhu D, Chung HF, Dobson AJ, et al. Age at natural menopause and risk of incident cardiovascular disease: a pooled analysis of individual patient data. *Lancet Public Health*. 2019;4(11):e553-e564.
38. Knowlton AA, Lee AR. Estrogen and the cardiovascular system. *Pharmacol Ther*. 2012;135(1):54-70.
39. Sutton-Tyrrell K, Wildman RP, Matthews KA, et al. Sex-hormone-binding globulin and the free androgen index are related to cardiovascular risk factors in multiethnic premenopausal and perimenopausal women enrolled in the Study of Women Across the Nation (SWAN). *Circulation*. 2005;111(10):1242-1249.
40. Rexrode KM, Manson JE, Lee IM, et al. Sex hormone levels and risk of cardiovascular events in postmenopausal women. *Circulation*. 2003;108(14):1688-1693.
41. Chiu MH, Heydari B, Batulan Z, et al. Coronary artery disease in post-menopausal women: are there appropriate means of assessment? *Clin Sci (Lond)*. 2018;132(17):1937-1952.
42. Reriani M, Sara JD, Flammer AJ, et al. Coronary endothelial function testing provides superior discrimination compared with standard clinical risk scoring in prediction of cardiovascular events. *Coron Artery Dis*. 2016;27(3):213-220.
43. Mathews L, Iantorno M, Schär M, et al. Coronary endothelial function is better in healthy premenopausal women than in healthy older postmenopausal women and men. *PLoS One*. 2017;12(10):e0186448.
44. Lambrinoudaki I, Kazani MV, Armeni E, et al. The TyG Index as a marker of subclinical atherosclerosis and arterial stiffness in lean and overweight postmenopausal women. *Heart Lung Circ*. 2018;27(6):716-724.
45. Nakagomi A, Sunami Y, Kawasaki Y, et al. Sex difference in the association between surrogate markers of insulin resistance and arterial stiffness. *J Diabetes Complications*. 2020;34(6):107442.