Scientific Research Publishing

World Journal of Cardiovascular Diseases, 2025, 15(12), 613-630
https://www.scirp.org/journal/wjcd
ISSN Online: 2164-5337
ISSN Print: 2164-5329

Triglyceride-Glucose Index and Arterial Stiffness: A Cross-Sectional Study in a Chinese Population

Xue Wang[1*], Jinlin Lv[1*], Yuluan Xiang[1], Xiaobo Wang[2#], Lihua Li[1#]

[1]Department of Gerontology, The First Affiliated Hospital of Dali University, Dali, China
[2]School of Basic Medicine, Dali University, Dali, China
Email: 18855483027@qq.com, arlene9943@163.com, 467868950@qq.com, #wxb4320062@163.com, #lilihuayncn@163.com

How to cite this paper: Wang, X., Lv, J.L., Xiang, Y.L., Wang, X.B. and Li, L.H. (2025) Triglyceride-Glucose Index and Arterial Stiffness: A Cross-Sectional Study in a Chinese Population. *World Journal of Cardiovascular Diseases*, **15**, 613-630.
https://doi.org/10.4236/wjcd.2025.1512054

Received: October 10, 2025
Accepted: December 14, 2025
Published: December 17, 2025

Abstract

Background: Arterial stiffness (AS) is an important indicator of cardiovascular health. The triglyceride-glucose (TyG) index is a practical substitute for insulin resistance and has been linked to AS. We investigated the cross-sectional relationships between the TyG index (compared to HOMA-IR) and various measures of AS in adults from Dali, China. **Methods:** In 715 participants, we measured carotid-femoral pulse wave velocity (cfPWV), brachial-ankle PWV (baPWV), central augmentation index (cAIx), pulse pressure amplification (PPA), and ankle-brachial index (ABI), along with TyG and HOMA-IR. Linear regression and multivariable models estimated their associations. We also assessed how well TyG and HOMA-IR could detect existing elevated AS, defined as cfPWV > 10 m/s. **Results:** In unadjusted analyses, the TyG index was positively associated with baPWV ($r = 0.1999$, $P < 0.001$) and cfPWV ($r = 0.2959$, $P < 0.001$), but not with cAIx, PPA, or ABI. After adjusting for traditional cardiovascular risk factors, the relationship between TyG and cfPWV remained significant ($\beta = 0.1800$ m/s, 95% CI: 0.01 - 0.35; $P = 0.0436$), while relationships with baPWV, cAIx, PPA, and ABI were not significant. Participants in the highest TyG index tertile had higher odds of elevated AS than those in the lowest tertile (OR = 2.45; 95% CI: 1.43 - 4.20; $P = 0.0011$). ROC analysis showed TyG had better discrimination for existing elevated AS (AUC = 0.884) than HOMA-IR (AUC = 0.697; $P < 0.0001$). **Conclusions:** The TyG index is cross-sectionally associated with central arterial stiffness (cfPWV) and shows a stronger ability to identify existing elevated AS than HOMA-IR in this population.

*These authors contributed equally to this work.
#Corresponding author.

Keywords

Triglyceride-Glucose Index, Arterial Stiffness, Pulse Wave Velocity, Central Hemodynamics, Insulin Resistance

1. Introduction

Arterial stiffness (AS) is an important marker of cardiovascular health and predicts hypertension, stroke, and heart failure [1] [2]. Traditional indicators of AS, such as brachial-ankle pulse wave velocity (baPWV), carotid-femoral pulse wave velocity (cfPWV), pulse pressure amplification (PPA), ankle-brachial index (ABI), and central augmentation index (cAIx), are commonly used to evaluate vascular health. Epidemiological studies have shown that metabolic dysregulation, including hyperglycemia and dyslipidemia, significantly contributes to cardiovascular diseases, which remain the leading cause of mortality worldwide. Our research team has previously investigated the association between thyroid hormone levels and AS, focusing on how hormonal changes impact vascular health [3]. More recently, we found a link between the Triglyceride-Glucose (TyG) index and blood pressure levels [4].

The TyG index has become a simple and reliable marker for assessing insulin resistance. In fact, recent studies have shown a strong link between high TyG index levels and increased AS across different populations, including those with type 2 diabetes and hypertension [5]-[7]. Notably, the TyG index has been shown to surpass traditional insulin resistance markers, such as the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), in predicting AS [7]-[9]. However, most previous research has been limited to either specific populations or isolated measures of AS [5] [10]-[12], which limits the understanding of the complex relationship between the TyG index and AS. The exact mechanisms by which the TyG index affect AS remain unclear and require further research using multiple AS indicators.

The relationship between the TyG index and AS may differ significantly among different demographic groups, especially within Asian populations, where genetic predispositions, lifestyle choices, and dietary habits substantially influence metabolic health outcomes [13]-[15]. Investigating the association of the TyG index with established AS markers—baPWV, cfPWV, PPA, ABI, and cAIx—can help improve cardiovascular risk assessment. Although large studies have associated the TyG index with AS, most have focused on a single metric within a cohort, rarely compared the TyG index against HOMA-IR, and seldom considered differences between central elastic and peripheral muscular arteries. To fill these gaps, we use a comparative, integrative approach that evaluates the TyG index and HOMA-IR side-by-side across various AS phenotypes, with clear attention to differences between central elastic and peripheral muscular arteries, thus improving clinical relevance in a representative Chinese cohort.

2. Research Methods

2.1. Study Population

This cross-sectional study utilized data from an ongoing population-based study investigating multiple cardiovascular risk factors in Dali, Yunnan Province, China [3] [4]. While the cohort overlaps with our previous research, the focus of this study is entirely different, exploring new data points and relationships that have not been analyzed before. Specifically, it examines the predictive value of the TyG index relative to HOMA-IR for AS, which was the focus of our earlier work. Participants were recruited from two communities within Dali. From October to December 2018, invitations were sent to all residents aged 18 years or older, and 764 individuals (70%) agreed to participate.

The study protocol was approved by the Ethics Committee of Dali University and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent. A total of 49 participants were then excluded based on the following criteria: lack of blood samples (n = 6), missing arterial measurements (n = 38), or incomplete anthropometric data (n = 5). Consequently, the final analytical cohort consisted of 715 participants.

2.2. Fieldwork

Two experienced physicians measured brachial blood pressure (BP) in each participant using a mercury sphygmomanometer. Before measurements, participants sat and rested for at least 5 minutes. Five consecutive readings were taken, and their average was used for analysis. Mean arterial pressure (MAP) was calculated as follows: MAP = (systolic BP + 2 × diastolic BP)/3. The brachial pulse pressure (PP) was calculated as the difference between systolic and diastolic BP. The same physicians administered a standardized questionnaire to collect data on medical history, smoking status, alcohol consumption, and medication use. Hypertension was defined as follows: brachial systolic BP ≥ 140 mm Hg, diastolic BP ≥ 90 mm Hg; or current use of antihypertensive medications [16]. A trained physician carefully conducted anthropometric measurements. Body mass index (BMI) was calculated by dividing body weight (kg) by the square of height (m^2).

Venous blood samples were collected from the participants following an overnight fast to measure various biochemical markers, including plasma glucose, hemoglobin A1c, insulin, serum creatinine, and a comprehensive lipid profile. Plasma glucose, insulin, and serum levels of total cholesterol, triglycerides, high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), and creatinine levels were assessed using standardized laboratory methods at the First Affiliated Hospital of Dali University. Diabetes mellitus was diagnosed if any of the following criteria were met: fasting plasma glucose ≥ 7.0 mmol/L; hemoglobin A1c ≥ 6.5%; or current use of antidiabetic medications [17]. The TyG index was calculated as follows: TyG = ln [fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL)/2] [18]. The HOMA-IR was calculated as follows: HOMA-IR = (fasting plasma insulin [μU/mL] × fasting plasma glucose [mmol/L])/22.5 [19].

2.3. Measurement of AS Indexes

To ensure the stability and accuracy of measurements, a qualified physician conducted all arterial assessments using applanation tonometry after the participants had rested in a supine position for 15 minutes. The participants were instructed to abstain from smoking, engaging in intense physical activity, and consuming alcohol or caffeine-containing beverages for at least 2 hours before the examination. Arterial waveforms were recorded utilizing a high-fidelity SPC-301 micromanometer (Millar Instruments, Houston, Texas, USA) connected to a laptop computer running SphygmoCor version 7.1 software (Actor Medical, Sydney, Australia).

Before beginning the SphygmoCor recordings, pulse wave calibration was performed using the average of two consecutive brachial BP readings taken in the supine position. Calibration was carried out with a validated Omron HEM-7051 oscillometric BP monitor (Omron Healthcare, Kyoto, Japan). The SphygmoCor software employs a validated generalized transfer function to calculate the aortic pulse wave from the radial arterial signal [20]. Subsequently, aortic systolic and diastolic BP values were derived from this calculated aortic pulse wave. The central augmentation index (cAIx) was determined as the ratio of augmented pressure to central PP, expressed as a percentage: cAIx = (augmented pressure/central PP) × 100%. PPA was calculated as the ratio of peripheral PP to central PP: PPA = (peripheral PP/central PP) × 100%.

cfPWV was measured using a standardized protocol. The physician sequentially recorded right carotid and femoral waveforms, each lasting 12 seconds. The transit time was determined from the delay between the foot points of the waveforms, using simultaneous electrocardiogram recordings. The travel distance was calculated as the difference between the sternal notch-to-femoral and sternal notch-to-carotid distances. cfPWV was then calculated as the ratio of the travel distance to transit time. AS was defined as a cfPWV > 10 m/s [21].

Pulse wave measurements were performed using the Vascular Profiler-1000 device (Omron Healthcare, Kyoto, Japan), which utilizes the oscillometric cuff method. Trained physicians applied pressure cuffs to the participants' arms and ankles while they rested in a supine position for 10 minutes. The device simultaneously recorded pulse waves in both ankles and measured blood pressure in all four limbs. The ankle-brachial index (ABI) was calculated for each leg as the ratio of the ankle systolic blood pressure to the higher of the two brachial systolic blood pressures. The lower of the two ABI values (left and right) was used for analysis. The brachial-ankle pulse wave velocity (baPWV) was calculated with the formula: baPWV = L/PTT, where L is the path length, determined as (La − Lb), with La representing the distance from the suprasternal notch to the ankle, and Lb the distance from the suprasternal notch to the brachium. The pulse transit time (PTT) is the duration between the brachial and ankle waveforms. The device automatically estimated these distances based on the subject's height. The higher

value of the left and right baPWV was used for analysis.

2.4. Statistical Analysis

Database management and statistical analyses were performed using SAS version 9.4 and EmpowerStats version 4.1. To compare means and proportions, analysis of variance (ANOVA) and chi-squared or Fisher's exact test were employed, respectively. The TyG index and HOMA-IR were analyzed as continuous and categorical (tertile) variables, respectively, to examine their relationships with AS. Single (Model 1) and multiple (Model 2) linear and logistic regression analyses were conducted to examine the associations of the TyG index and HOMA-IR with AS measures and the prevalence of elevated AS (cfPWV > 10 m/s). Model 2 was adjusted for sex, age, body height, BMI, pulse rate, serum creatinine, brachial MAP, current smoking status, current alcohol intake, and current antihypertensive treatment. We assessed and compared the predictive abilities of the TyG index and HOMA-IR for AS using receiver operating characteristic (ROC) analysis. For each index, the area under the ROC curve (AUC) was calculated. A two-tailed P value below 0.05 was considered statistically significant.

3. Results

3.1. Characteristics of the Study Population

Among the 715 participants, 467 (65.31%) were women, and 226 (31.61%) had hypertension. Table 1 presents the characteristics of the study population across the TyG index tertiles. Notably, there were significant differences in several indicators of AS. The cfPWV, a gold standard measure of AS, showed a progressive increase across the TyG index tertiles (T1: 9.68 ± 2.00 m/s; T2: 10.42 ± 1.93 m/s; T3: 11.09 ± 1.87 m/s; P < 0.001). Similarly, baPWV demonstrated a significant upward trend (T1: 1460.60 ± 399.35 cm/s; T2: 1536.40 ± 347.68 cm/s; T3: 1640.33 ± 378.72 cm/s; P < 0.001). Although it did not reach statistical significance, cAIx also showed an increasing trend across the TyG index tertiles (T1: 27.23% ± 12.48%; T2: 28.81% ± 10.23%; T3: 29.54% ± 12.04%; P = 0.087). There was no significant difference in PPA and ABI across the TyG index tertiles (P = 0.173). These findings suggest a potential association between higher TyG index values and AS.

3.2. Correlation Analysis of the TyG Index and HOMA-IR with AS Indicators

Table 2 shows the correlations between AS indicators and both the TyG index and HOMA-IR. The TyG index exhibited significant positive correlations with baPWV (r = 0.1999, 95% confidence interval [CI]: 0.1285 - 0.2693, P < 0.001) and cfPWV (r = 0.2959, 95% CI: 0.2276 - 0.3614, P < 0.001). Similarly, HOMA-IR was positively correlated with baPWV (r = 0.1151, 95% CI: 0.0420 - 0.1868, P = 0.0021) and cfPWV (r = 0.1679, 95% CI: 0.0957 - 0.2384, P < 0.001). Neither the TyG index nor HOMA-IR showed significant correlations with cAIx, PPA, or

ABI (all P > 0.05).

Table 1. Characteristics of the study population according to the TyG index.

Variables	T1 (<8.54)	T2 (8.54 - 9.00)	T3 (>9.0)	P
N	238	238	239	
Age, years	48.99 ± 14.28	52.49 ± 12.20	54.07 ± 10.49	<0.001
Body mass index, kg/m^2	22.30 ± 3.05	24.32 ± 3.14	25.09 ± 3.14	<0.001
Body height, cm	159.63 ± 6.94	161.33 ± 8.61	162.04 ± 7.92	0.003
Total cholesterol, mmol/L	4.44 ± 0.74	4.82 ± 0.85	5.15 ± 1.04	<0.001
Triglyceride, mmol/L	0.93 (0.78 - 1.09)	1.52 (1.32 - 1.71)	2.45 (2.07 - 3.19)	<0.001
Fasting blood glucose, mmol/L	5.04 ± 0.43	5.44 ± 0.78	6.42 ± 2.29	<0.001
LDL-C, mmol/L	2.27 ± 0.54	2.68 ± 0.63	2.74 ± 0.78	<0.001
HDL-C, mmol/L	1.44 ± 0.25	1.35 ± 0.22	1.38 ± 0.22	<0.001
Serum creatinine, μmol/L	69.80 ± 16.13	72.48 ± 19.12	73.48 ± 18.93	0.072
Brachial MAP, mm Hg	87.14 ± 13.19	91.40 ± 11.68	94.94 ± 12.18	<0.001
Brachial PP, mm Hg	41.06 ± 11.93	40.75 ± 10.41	43.61 ± 11.50	0.010
Aortic PP, mm Hg	36.34 ± 9.80	36.87 ± 8.29	37.89 ± 9.38	0.176
Pulse rate, beats/minute	70.60 ± 8.44	71.87 ± 8.81	73.56 ± 9.11	0.001
baPWV, cm/s	1460.60 ± 399.35	1536.40 ± 347.68	1640.33 ± 378.72	<0.001
cfPWV, m/s	9.68 ± 2.00	10.42 ± 1.93	11.09 ± 1.87	<0.001
cAIx, %	27.23 ± 12.48	28.81 ± 10.23	29.54 ± 12.04	0.087
PPA, %	115.07 ± 27.41	112.73 ± 27.94	117.43 ± 26.77	0.173
ABI	1.07 ± 0.10	1.08 ± 0.08	1.08 ± 0.08	0.427
TyG index	8.19 ± 0.26	8.77 ± 0.14	9.53 ± 0.54	<0.001
HOMA-IR	1.77 ± 0.91	2.77 ± 2.02	4.13 ± 3.03	<0.001
Insulin, μIU/mL	7.91 ± 4.02	11.37 ± 7.27	14.42 ± 8.59	<0.001
Women, n (%)	180 (75.63%)	155 (65.13%)	132 (55.23%)	<0.001
Current smoking, n (%)	32 (13.45%)	49 (20.59%)	64 (26.78%)	0.001
Current drinking, n (%)	18 (7.56%)	21 (8.82%)	42 (17.57%)	<0.001
Diabetes mellitus, n (%)	1 (0.42%)	19 (7.98%)	56 (23.43%)	<0.001
Hypertension, n (%)	45 (18.91%)	74 (31.09%)	107 (44.77%)	<0.001
Current antihypertensive treatment, n (%)	28 (11.76%)	47 (19.75%)	64 (26.78%)	<0.001

T1, T2, and T3 refer to the first tertile, second tertile, and third tertile of the TyG index, respectively. The values in the table are presented as mean ± standard deviation, median (interquartile range), or n (%). TyG index, triglyceride-glucose index; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; MAP, mean blood pressure; PP, pulse pressure; baPWV, brachial-ankle pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cAIx, central augmentation index; PPA, pulse pressure amplification; ABI, ankle-brachial index; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.

Table 2. Correlation analysis of the TyG index and HOMA-IR with AS indicators.

Variable	TyG index r (95% CI)	P	HOMA-IR R (95% CI)	P
baPWV, cm/s	0.1999 (0.1285, 0.2693)	<0.001	0.1151 (0.0420, 0.1868)	0.0021
cfPWV, m/s	0.2959 (0.2276, 0.3614)	<0.001	0.1679 (0.0957, 0.2384)	<0.001
cAIX, %	0.0689 (−0.0045, 0.1415)	0.0656	0.0727 (−0.0006, 0.1453)	0.0521
PPA, %	0.0463 (−0.0271, 0.1192)	0.2162	−0.0119 (−0.0852, 0.0616)	0.7516
ABI	0.0530 (−0.0204, 0.1259)	0.1566	−0.0363 (−0.1094, 0.0372)	0.3330

TyG index, triglyceride-glucose index; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; AS, arterial stiffness; baPWV, brachial-ankle pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cAIX, central augmentation index; PPA, pulse pressure amplification; ABI, ankle-brachial index.

3.3. Linear Regression Analysis of the TyG Index and HOMA-IR with AS Indicators

Univariate regression analysis showed significant links between the TyG index and AS indexes, especially with baPWV and cfPWV in the unadjusted model (Model 1). After adjusting for confounders (Model 2), the TyG index still had a significant connection with cfPWV (β = 0.18 m/s, 95% CI: 0.01 - 0.35, P = 0.0436). The highest TyG tertile (T3) continued to be significantly associated with cfPWV compared to the lowest tertile (T1) in the adjusted model (β = 0.31 m/s, 95% CI: 0.04 - 0.58, P = 0.0240). Conversely, HOMA-IR showed significant relationships with baPWV, cfPWV, and cAIx in the unadjusted model, but these relationships weakened and became non-significant after adjusting for confounders. Neither the TyG index nor HOMA-IR showed significant associations with PPA or ABI in either model. These results suggest that the TyG index may be a reliable marker of AS, especially cfPWV, compared with HOMA-IR, even after accounting for traditional cardiovascular risk factors (Table 3 and Table 4).

Table 3. Multivariate regression analysis of the TyG index with AS indicators.

Variables	Model 1	P	Model 2	P
baPWV, cm/s				
TyG	116.82 (74.80, 158.85)	<0.0001	9.69 (−21.13, 40.51)	0.5380
TyG tertile				
T1	Reference		Reference	
T2	75.80 (8.27, 143.33)	0.0281	−10.65 (−56.25, 34.95)	0.6473
T3	179.74 (112.28, 247.20)	<0.0001	20.65 (−27.59, 68.89)	0.4018
cfPWV, m/s				
TyG	0.91 (0.69, 1.13)	<0.0001	0.18 (0.01, 0.35)	0.0436
TyG tertile				
T1	Reference		Reference	

Continued

T2	0.74 (0.40, 1.09)	<0.0001	0.11 (−0.14, 0.37)	0.3737
T3	1.41 (1.07, 1.76)	<0.0001	0.31 (0.04, 0.58)	0.0240
cAIx, %				
TyG	1.23 (−0.08, 2.53)	0.0656	0.05 (−1.25, 1.34)	0.9441
TyG tertile				
T1	Reference		Reference	
T2	1.58 (−0.51, 3.66)	0.1397	0.09 (−1.83, 2.01)	0.9258
T3	2.31 (0.22, 4.39)	0.0304	0.63 (−1.40, 2.66)	0.5408
PPA, %				
TyG	1.94 (−1.13, 5.01)	0.2162	−1.69 (−4.97, 1.58)	0.3116
TyG tertile				
T1	Reference		Reference	
T2	−2.33 (−7.25, 2.58)	0.3527	−4.49 (−9.34, 0.35)	0.0696
T3	2.37 (−2.54, 7.28)	0.3451	−2.84 (−7.97, 2.28)	0.2769
ABI				
TyG	0.01 (−0.00, 0.02)	0.1566	0.01 (−0.00, 0.02)	0.2799
TyG tertile				
T1	Reference		Reference	
T2	0.01 (−0.01, 0.02)	0.3732	0.01 (−0.01, 0.02)	0.4980
T3	0.01 (−0.01, 0.03)	0.2040	0.01 (−0.01, 0.02)	0.3666

Data are presented as β (95% CI) and P value. T1, T2, and T3 refer to the first tertile, second tertile, and third tertile, respectively. Model 1: unadjusted; Model 2: adjusted for sex, age, body height, body mass index, pulse rate, serum creatinine, brachial mean blood pressure, current smoking, current drinking, and current antihypertensive treatment. TyG index, triglyceride-glucose index; AS, arterial stiffness; baPWV, brachial-ankle pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cAIX, central augmentation index; PPA, pulse pressure amplification; ABI, ankle-brachial index.

Table 4. Multivariate regression analysis of the HOMA-IR with AS indicators.

Variables	Model 1	P	Model 2	P
baPWV, cm/s				
HOMA-IR	18.56 (6.79, 30.32)	0.0021	6.81 (−1.72, 15.34)	0.1180
HOMA-IR tertile				
T1	Reference		Reference	
T2	10.59 (−58.03, 79.22)	0.7623	9.49 (−36.16, 55.13)	0.6839
T3	71.48 (2.85, 140.10)	0.0416	33.89 (−20.20, 87.98)	0.2199
cfPWV, m/s				
HOMA-IR	0.14 (0.08, 0.20)	<0.0001	0.01 (−0.04, 0.06)	0.7170
HOMA-IR tertile				
T1	Reference		Reference	

Continued

T2	0.21 (−0.14, 0.57)	0.2419	0.02 (−0.23, 0.27)	0.8750
T3	0.78 (0.42, 1.14)	<0.0001	0.11 (−0.19, 0.42)	0.4577
cAIx, %				
HOMA-IR	0.36 (−0.00, 0.72)	0.0521	0.08 (−0.27, 0.44)	0.6446
HOMA-IR tertile				
T1	Reference		Reference	
T2	0.89 (−1.20, 2.97)	0.4044	−0.25 (−2.17, 1.67)	0.7994
T3	3.13 (1.05, 5.21)	0.0033	1.41 (−0.86, 3.68)	0.2243
PPA, %				
HOMA-IR	−0.14 (−0.99, 0.71)	0.7516	−0.39 (−1.30, 0.52)	0.3998
HOMA-IR tertile				
T1	Reference		Reference	
T2	−2.83 (−7.76, 2.09)	0.2600	−2.03 (−6.89, 2.82)	0.4126
T3	−1.96 (−6.89, 2.97)	0.4357	−1.55 (−7.31, 4.20)	0.5973
ABI				
HOMA-IR	−0.00 (−0.00, 0.00)	0.3330	−0.00 (−0.00, 0.00)	0.2748
HOMA-IR tertile				
T1	Reference		Reference	
T2	0.00 (−0.01, 0.02)	0.5906	0.00 (−0.01, 0.02)	0.6179
T3	−0.00 (−0.02, 0.01)	0.6231	−0.00 (−0.02, 0.02)	0.7352

Data are presented as β (95% CI) and P value. T1, T2, and T3 refer to the first tertile, second tertile, and third tertile, respectively. Model 1: unadjusted; Model 2: adjusted for sex, age, body height, body mass index, pulse rate, serum creatinine, brachial mean blood pressure, current smoking, current drinking, and current antihypertensive treatment. AS, arterial stiffness; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; baPWV, brachial-ankle pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cAIX, central augmentation index; PPA, pulse pressure amplification; ABI, ankle-brachial index.

3.4. Logistic Regression Analysis of the TyG Index and HOMA-IR with the Prevalent Elevated AS

Table 5 shows the multivariate regression analysis of the TyG index and HOMA-IR with AS. In the unadjusted model (Model 1), both the TyG index and HOMA-IR were significantly associated with AS. The TyG index showed a stronger relationship (odds ratio [OR] = 3.38, 95% CI: 2.52 to 4.55, P < 0.0001) than HOMA-IR (OR = 1.21, 95% CI: 1.11 to 1.31, P < 0.0001). After adjusting for confounders (Model 2), the TyG index still had a significant link to AS (OR = 1.88, 95% CI: 1.30 to 2.73, P = 0.0008), but the link between HOMA-IR and AS was no longer significant (OR = 1.06, 95% CI: 0.95 to 1.19, P = 0.3023). Notably, the highest tertile of the TyG index (T3) remained significantly associated with AS in the adjusted model (OR = 2.45, 95% CI: 1.43 to 4.20, P = 0.0011). In contrast, the highest

tertile of HOMA-IR lost its significant link after adjustment. These results suggest that the TyG index may be a stronger and more independent marker of AS than HOMA-IR, even after accounting for various cardiovascular risk factors (Table 5, Figure 1).

Table 5. Multivariate regression analysis of the TyG index and HOMA-IR with AS.

Variables	Model 1	P	Model 2	P
TyG	3.38 (2.52, 4.55)	<0.0001	1.88 (1.30, 2.73)	0.0008
TyG tertile				
T1	Reference		Reference	
T2	2.09 (1.45, 3.03)	<0.0001	1.22 (0.73, 2.03)	0.4417
T3	5.14 (3.47, 7.61)	<0.0001	2.45 (1.43, 4.20)	0.0011
HOMA-IR	1.21 (1.11, 1.31)	<0.0001	1.06 (0.95, 1.19)	0.3023
HOMA-IR tertile				
T1	Reference		Reference	
T2	1.27 (0.88, 1.81)	0.1994	1.05 (0.63, 1.74)	0.8600
T3	2.47 (1.70, 3.58)	<0.0001	1.54 (0.85, 2.78)	0.1549

Data are presented as OR (95% CI) P value. Model 1: unadjusted; Model 2: adjusted for sex, age, body height, body mass index, pulse rate, serum creatinine, brachial mean blood pressure, current smoking, current drinking, and current antihypertensive treatment. TyG index, triglyceride-glucose index; AS, arterial stiffness; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.

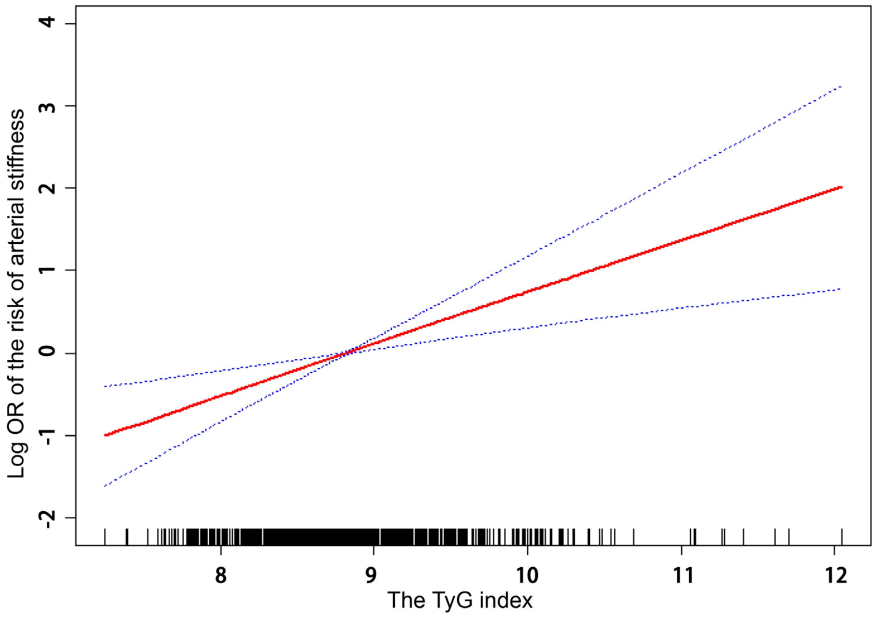

Figure 1. Smoothed curve fitting of the relationship between the TyG index and AS (cfPWV > 10 m/s). Values were adjusted for sex, age, body height, body mass index, pulse rate, serum creatinine, brachial mean blood pressure, current smoking, current drinking, and current antihypertensive treatment. AS, arterial stiffness.

3.5. Comparisons of the Predictive Capabilities of the TyG Index and HOMA-IR for AS

The diagnostic performance of the TyG index and HOMA-IR for AS was evaluated using ROC analysis. Model 1 (TyG index) demonstrated an excellent AUC (0.884; 95% CI: 0.860 - 0.908) compared with Model 2 (HOMA-IR), which had an AUC of 0.697 (95% CI: 0.659 - 0.735), with a statistically significant difference ($P < 0.0001$). Model 1 exhibited higher sensitivity (84.58%) and specificity (75.93%) than Model 2, which had 70.18% sensitivity and 61.42% specificity. Additionally, Model 1 showed greater accuracy (80.65%) than Model 2 (66.20%) (Figure 2). The net reclassification index (NRI) indicated a significant decrease in both events and non-events in Model 1 compared with Model 2 (NRI = −0.2890, $P < 0.0001$). The integrated discrimination index (IDI) also reflected a significant decrease (IDI = −0.2890, $P < 0.0001$). Taken together, these findings indicate that the TyG index demonstrates an excellent predictive capability for AS compared with HOMA-IR. The TyG index not only demonstrates enhanced diagnostic accuracy but also superior reclassification performance.

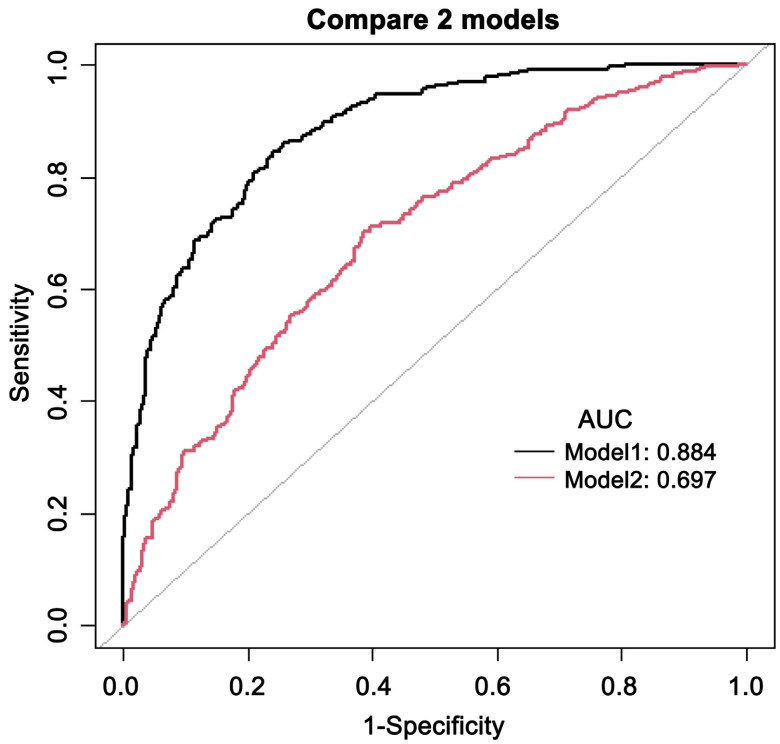

Figure 2. Comparison of ROC curves for the TyG Index and HOMA-IR in predicting AS (cfPWV > 10 m/s). Values were adjusted for sex, age, body height, body mass index, pulse rate, serum creatinine, brachial mean blood pressure, current smoking, current drinking, and current antihypertensive treatment. AS, arterial stiffness. Model 1(TyG Index), Model 2 (HOMA-IR).

4. Discussion

Our study revealed a significant association between the TyG index and AS indi-

cators, especially cfPWV. This link remained even after adjusting for traditional cardiovascular risk factors. The TyG index had a better predictive ability for AS than HOMA-IR, as shown by a larger AUC. Importantly, the TyG index stayed independently associated with AS after controlling for confounding factors, while HOMA-IR did not. These findings highlight the potential of the TyG index as a biomarker for detecting elevated AS prevalence. The absence of significant links between the TyG index and other vascular parameters, such as cAIx, PPA, and ABI, emphasizes the complexity of the relationship between metabolic indices and AS.

Our study provided intriguing insights into the relationship between the TyG index and various indicators of AS, revealing both consistent and inconsistent findings with previous research. Notably, we observed significant associations between the TyG index and cfPWV, as well as between cfPWV and AS diagnosis. This finding aligns with and expands upon recent studies that have reported similar correlations [22] [23]. In a cohort of 2830 elderly participants from the Northern Shanghai Study, Zhao *et al.* demonstrated that an elevated TyG index was significantly associated with prevalent elevated AS, baPWV ≥ 1800 cm/s, ABI ≤ 0.9, microalbuminuria, and chronic kidney disease. [22] Similarly, in a Beijing community-based study involving 6015 participants, Ji *et al.* reported that a higher TyG index was associated with increased cfPWV, baPWV, and urinary albumin-to-creatinine ratio (UACR), as well as with increased risks of cfPWV ≥ 10 m/s, baPWV ≥ 1800 cm/s, and UACR ≥ 30 mg/g [23]. Importantly, the TyG index predicted a cfPWV > 10 m/s—a guideline-defined threshold indicating significant subclinical target-organ damage. Because exceeding this cut-off signals advanced arterial stiffening and heightened cardiovascular risk, TyG-based stratification can help prioritize intensified risk management and closer surveillance, offering practical value where cfPWV testing is not routinely available.

However, our results differ from earlier reports in several ways. After adjusting for standard cardiovascular risk factors, the TyG index was not independently linked to baPWV, cAIx, ABI, or PPA, although its connection with cfPWV remained strong. These differences might be due to variations in cohort makeup, measurement procedures, regional environmental and lifestyle factors, and the distinct physiology of central versus peripheral arteries. Notably, cfPWV directly measures central aortic stiffness, while baPWV combines central and muscular segments and is more affected by current blood pressure and wave reflections. The differing associations—seen for cfPWV but not for baPWV and other measures—suggest that insulin resistance may mainly impact elastic central arteries, with smaller or less clear effects on peripheral or wave-reflection indices. Overall, these findings highlight the diversity of arterial stiffness types and emphasize the importance of comprehensive, phenotype-specific cardiovascular evaluation.

Our findings carry significant clinical and research implications. While they verify the potential usefulness of the TyG index in predicting central AS, they also warn against relying too much on a single marker for cardiovascular risk assess-

ment. The absence of association with other vascular indicators suggests that the link between insulin resistance and vascular function is complex and may not impact all aspects of arterial health equally.

Our findings indicate several promising avenues for future research. Longitudinal studies are necessary to assess the predictive ability of the TyG index for changes over time in various vascular parameters. Additionally, understanding the mechanisms underlying differences in associations across vascular beds could provide valuable insights into the pathophysiology of AS in relation to insulin resistance.

The superiority of the TyG index over HOMA-IR in predicting AS, as demonstrated in our study, aligns with findings from an Italian cohort [24], where the TyG index was a better predictor of carotid atherosclerosis. Similarly, Wang *et al.* [7] demonstrated that the TyG index showed a stronger and more independent association with AS than HOMA-IR in patients with type 2 diabetes, particularly in older individuals with longer disease duration and poor glycemic control. Beyond predictive performance, the TyG index is more practical for clinical use because it does not require an insulin assay. Insulin measurements are less routinely available in many clinical laboratories and are typically more costly and variable across assay platforms. In contrast, fasting triglyceride and glucose tests used to calculate the TyG index are widely accessible, standardized, and included in routine metabolic panels. These consistent results across different populations suggest that the TyG index could be a more reliable and practical indicator of insulin resistance than HOMA-IR. Future research should determine optimal TyG index cutoff points to improve its clinical utility and aid early detection interventions [19].

The association between the TyG index and AS likely involves multiple complex pathophysiological mechanisms [25]. Insulin resistance can cause endothelial dysfunction, an early sign of AS. The hyperglycemic and hypertriglyceridemic state reflected by a high TyG index increases oxidative stress, which directly damages vascular endothelial cells [26] [27]. Chronic low-grade inflammation, metabolic disturbances, and activation of the renin-angiotensin-aldosterone system may also play a role there [25] [28]. Recent research suggests that changes in autonomic nervous system function, heightened platelet activation, and adipose tissue dysfunction may contribute to how insulin resistance affects the body AS [25] [27] [29] [30]. Lastly, our previous findings indicate that the TyG index is independently linked to both brachial and aortic diastolic blood pressure and serves as a predictor of hypertension. This finding introduces an additional pathway through which a high TyG index may contribute to AS. Specifically, insulin resistance, as reflected by a high TyG index, may lead to elevated blood pressure, a known risk factor for AS. This cascade effect could further amplify insulin resistance's impact on vascular health.

Our study has several notable strengths, including the evaluation of multiple AS indicators and the use of multivariate analysis to control confounding factors.

We also compared the predictive abilities of the TyG index and HOMA-IR and demonstrated a dose-response relationship with tertile analysis, which strengthens our findings. However, there are some limitations. First, its cross-sectional design prevents us from making causal conclusions, and we cannot establish the temporal link between insulin resistance and AS. Second, despite using multivariable adjustment, residual confounding might still be present; specifically, important lifestyle and behavioral factors—like dietary patterns (e.g., sodium intake, macronutrient quality) and physical activity (intensity, duration)—were not measured in enough detail and therefore not included, which could affect both insulin resistance and AS. Future cohorts should prioritize repeated 24-hour urinary sodium, detailed dietary recalls with nutrient profiling and pattern scores (e.g., DASH adherence), and objective accelerometry-derived moderate-to-vigorous physical activity, sedentary time, and fitness measures to reduce residual confounding. Third, because the study population was from a single region in China, this may limit the generalizability of the results to other settings with different genetics, environments, and healthcare systems. Future studies should therefore validate these findings in other ethnic groups and in more diverse Chinese populations (across regions, urban-rural contexts, and healthcare tiers) to confirm the broader utility of the TyG index as a cardiovascular risk marker. Lastly, measurement variability across different arterial stiffness endpoints, along with their sensitivity to blood pressure and heart rate, may have weakened the associations observed for non-central measures.

5. Conclusion

Our study underscores the clinical potential of the TyG index as a predictor of AS, with a notably stronger and more consistent association with central stiffness measured by cfPWV—particularly at the guideline-relevant threshold of cfPWV > 10 m/s—than with peripheral/mixed measures such as baPWV, for which associations were absent or attenuated. While the TyG index showed statistically significant links, these correlations were modest, reflecting the multifactorial nature of insulin resistance and vascular health. The differential pattern across stiffness endpoints suggests that the TyG index may preferentially capture pathways more relevant to central (aortic) stiffening rather than peripheral arterial properties, warranting cautious interpretation when using non-central measures. Future large-scale, prospective studies should validate these findings across diverse populations, delineate population-specific modifiers, and clarify the clinical contexts in which the TyG index is most predictive. Such work will help establish the TyG index as a cost-effective and implementable tool for cardiovascular risk assessment and management.

Acknowledgements

We thank all the staff and participants of the Dali cohort for their invaluable contributions. We thank LetPub (https://www.letpub.com.cn/) for its linguistic assis-

tance during the preparation of this manuscript.

Author Contributions

L.L.H. conceptualized and designed the study. W.X., L.J.L., X.Y.L., and W.X.B. contributed to data acquisition. L.L.H. conducted the statistical analyses and composed the initial draft of the manuscript. All authors have thoroughly reviewed and approved the final version of the manuscript.

Funding

Our work was supported by the National Natural Science Foundation of China (grant numbers 82260076 and 81860084), the Xingdian Famous Physician Special Project of Yunnan Province, the Youth Top-notch Talents Program of the Ten Thousand Talents Plan of Yunnan Province, Yunnan Key Specialties, and the Key Discipline of the First Affiliated Hospital of Dali University.

Availability of Data and Materials

The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The Ethics Committee of Dali University approved the study. Participants provided informed consent before inclusion.

Conflicts of Interest

The authors declare no conflicts of interest in this work.

References

[1] Zhong, H., Shao, Y., Guo, G., Zhan, Y., Liu, B., Shao, M., *et al.* (2023) Association between the Triglyceride-Glucose Index and Arterial Stiffness: A Meta-Analysis. *Medicine*, **102**, e33194. https://doi.org/10.1097/md.0000000000033194

[2] Muhammad, I.F., Bao, X., Nilsson, P.M. and Zaigham, S. (2023) Triglyceride-Glucose (tyg) Index Is a Predictor of Arterial Stiffness, Incidence of Diabetes, Cardiovascular Disease, and All-Cause and Cardiovascular Mortality: A Longitudinal Two-Cohort Analysis. *Frontiers in Cardiovascular Medicine*, **9**, Article ID: 1035105. https://doi.org/10.3389/fcvm.2022.1035105

[3] Jamal, M.T., Li, Q., Li, Q., Liang, W., Wang, L., Wei, J., *et al.* (2020) Association of Thyroid Hormones with Blood Pressure and Arterial Stiffness in the General Population: The Dali Study. *The Journal of Clinical Hypertension*, **23**, 363-372. https://doi.org/10.1111/jch.14154

[4] Sun, Y., Hu, N., Huang, X., Liu, Z., Li, Q., Li, Q., *et al.* (2024) Central and Peripheral Blood Pressures in Relation to the Triglyceride-Glucose Index in a Chinese Population. *Cardiovascular Diabetology*, **23**, Article No. 3. https://doi.org/10.1186/s12933-023-02068-z

[5] Wu, S., Xu, L., Wu, M., Chen, S., Wang, Y. and Tian, Y. (2021) Association between

Triglyceride-Glucose Index and Risk of Arterial Stiffness: A Cohort Study. *Cardiovascular Diabetology*, **20**, Article No. 146. https://doi.org/10.1186/s12933-021-01342-2

[6] Li, M., Zhan, A., Huang, X., Hu, L., Zhou, W., Wang, T., *et al.* (2020) Positive Association between Triglyceride Glucose Index and Arterial Stiffness in Hypertensive Patients: The China H-Type Hypertension Registry Study. *Cardiovascular Diabetology*, **19**, Article No. 139. https://doi.org/10.1186/s12933-020-01124-2

[7] Wang, S., Shi, J., Peng, Y., Fang, Q., Mu, Q., Gu, W., *et al.* (2021) Stronger Association of Triglyceride Glucose Index than the HOMA-IR with Arterial Stiffness in Patients with Type 2 Diabetes: A Real-World Single-Centre Study. *Cardiovascular Diabetology*, **20**, Article No. 82. https://doi.org/10.1186/s12933-021-01274-x

[8] Lee, S.B., Ahn, C.W., Lee, B.K., Kang, S., Nam, J.S., You, J.H., *et al.* (2018) Association between Triglyceride Glucose Index and Arterial Stiffness in Korean Adults. *Cardiovascular Diabetology*, **17**, Article No. 41. https://doi.org/10.1186/s12933-018-0692-1

[9] Tan, L., Liu, Y., Liu, J., Zhang, G., Liu, Z. and Shi, R. (2023) Association between Insulin Resistance and Uncontrolled Hypertension and Arterial Stiffness among US Adults: A Population-Based Study. *Cardiovascular Diabetology*, **22**, Article No. 311. https://doi.org/10.1186/s12933-023-02038-5

[10] Wang, Y., Wang, S., Pan, X., Meng, P., Yang, J., Zhang, Y., *et al.* (2023) Association between Triglyceride-Glucose Index and Arterial Stiffness Reflected by Carotid Pulse-wave Velocity in Stage 1 Hypertension and Individuals with Normal/elevated Blood Pressure. *The Journal of Clinical Hypertension*, **25**, 199-212. https://doi.org/10.1111/jch.14639

[11] Yan, Y., Wang, D., Sun, Y., Ma, Q., Wang, K., Liao, Y., *et al.* (2022) Triglyceride-Glucose Index Trajectory and Arterial Stiffness: Results from Hanzhong Adolescent Hypertension Cohort Study. *Cardiovascular Diabetology*, **21**, Article No. 33. https://doi.org/10.1186/s12933-022-01453-4

[12] Guo, W., Zhu, W., Wu, J., Li, X., Lu, J., Qin, P., *et al.* (2021) Triglyceride Glucose Index Is Associated with Arterial Stiffness and 10-Year Cardiovascular Disease Risk in a Chinese Population. *Frontiers in Cardiovascular Medicine*, **8**, Article ID: 585776. https://doi.org/10.3389/fcvm.2021.585776

[13] Lip, G.Y.H. and Boos, C.J. (2005) Ethnic Differences in Arterial Responses, Inflammation, and Metabolic Profiles: Possible Insights into Ethnic Differences in Cardiovascular Disease and Stroke. *Arteriosclerosis, Thrombosis, and Vascular Biology*, **25**, 2240-2242. https://doi.org/10.1161/01.atv.0000188507.89134.ef

[14] Ma, R.C.W. and Chan, J.C.N. (2013) Type 2 Diabetes in East Asians: Similarities and Differences with Populations in Europe and the United States. *Annals of the New York Academy of Sciences*, **1281**, 64-91. https://doi.org/10.1111/nyas.12098

[15] Williams, R. and Periasamy, M. (2020) Genetic and Environmental Factors Contributing to Visceral Adiposity in Asian Populations. *Endocrinology and Metabolism*, **35**, 681-695. https://doi.org/10.3803/enm.2020.772

[16] Liu, J. (2020) Highlights of the 2018 Chinese Hypertension Guidelines. *Clinical Hypertension*, **26**, Article No. 8. https://doi.org/10.1186/s40885-020-00141-3

[17] Buysschaert, M., Medina, J., Buysschaert, B. and Bergman, M. (2016) Definitions (and Current Controversies) of Diabetes and Prediabetes. *Current Diabetes Reviews*, **12**, 8-13. https://doi.org/10.2174/1573399811666150122150233

[18] Sánchez-Íñigo, L., Navarro-González, D., Fernández-Montero, A., Pastrana-Delgado, J. and Martínez, J.A. (2016) The Tyg Index May Predict the Development of Cardiovascular Events. *European Journal of Clinical Investigation*, **46**, 189-197.

https://doi.org/10.1111/eci.12583

[19] Tahapary, D.L., Pratisthita, L.B., Fitri, N.A., Marcella, C., Wafa, S., Kurniawan, F., *et al.* (2022) Challenges in the Diagnosis of Insulin Resistance: Focusing on the Role of HOMA-IR and Tryglyceride/Glucose Index. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, **16**, Article 102581. https://doi.org/10.1016/j.dsx.2022.102581

[20] Gallagher, D., Adji, A. and Orourke, M. (2004) Validation of the Transfer Function Technique for Generating Central from Peripheral Upper Limb Pressure Waveform. *American Journal of Hypertension*, **17**, 1059-1067. https://doi.org/10.1016/j.amjhyper.2004.05.027

[21] Williams, B., Mancia, G., Spiering, W., *et al.* (2018) 2018 ESC/ESH Guidelines for the Management of Arterial Hypertension *European Heart Journal*, **39**, 3021-3104.

[22] Zhao, S., Yu, S., Chi, C., Fan, X., Tang, J., Ji, H., *et al.* (2019) Association between Macro- and Microvascular Damage and the Triglyceride Glucose Index in Community-Dwelling Elderly Individuals: The Northern Shanghai Study. *Cardiovascular Diabetology*, **18**, Article No. 95. https://doi.org/10.1186/s12933-019-0898-x

[23] Ji, W., Gao, L., Sun, P., Jia, J., Li, J., Wang, X., *et al.* (2022) Association of the Triglyceride-Glucose Index and Vascular Target Organ Damage in a Beijing Community-Based Population. *Frontiers in Cardiovascular Medicine*, **9**, Article ID: 948402. https://doi.org/10.3389/fcvm.2022.948402

[24] Irace, C., Carallo, C., Scavelli, F.B., De Franceschi, M.S., Esposito, T., Tripolino, C., *et al.* (2013) Markers of Insulin Resistance and Carotid Atherosclerosis. a Comparison of the Homeostasis Model Assessment and Triglyceride Glucose Index. *International Journal of Clinical Practice*, **67**, 665-672. https://doi.org/10.1111/ijcp.12124

[25] Mechanick, J.I., Farkouh, M.E., Newman, J.D. and Garvey, W.T. (2020) Cardiometabolic-Based Chronic Disease, Adiposity and Dysglycemia Drivers. *Journal of the American College of Cardiology*, **75**, 525-538. https://doi.org/10.1016/j.jacc.2019.11.044

[26] Guglielmi, V., Dalle Grave, R., Leonetti, F. and Solini, A. (2024) Female Obesity: Clinical and Psychological Assessment toward the Best Treatment. *Frontiers in Endocrinology*, **15**, Article ID: 1349794. https://doi.org/10.3389/fendo.2024.1349794

[27] Das, D., Shruthi, N.R., Banerjee, A., Jothimani, G., Duttaroy, A.K. and Pathak, S. (2023) Endothelial Dysfunction, Platelet Hyperactivity, Hypertension, and the Metabolic Syndrome: Molecular Insights and Combating Strategies. *Frontiers in Nutrition*, **10**, Article ID: 1221438. https://doi.org/10.3389/fnut.2023.1221438

[28] Cooper, S.A., Whaley-Connell, A., Habibi, J., Wei, Y., Lastra, G., Manrique, C., *et al.* (2007) Renin-Angiotensin-Aldosterone System and Oxidative Stress in Cardiovascular Insulin Resistance. *American Journal of Physiology-Heart and Circulatory Physiology*, **293**, H2009-H2023. https://doi.org/10.1152/ajpheart.00522.2007

[29] Valensi, P. (2021) Autonomic Nervous System Activity Changes in Patients with Hypertension and Overweight: Role and Therapeutic Implications. *Cardiovascular Diabetology*, **20**, Article No. 170. https://doi.org/10.1186/s12933-021-01356-w

[30] Chandrasekaran, P. and Weiskirchen, R. (2024) Cellular and Molecular Mechanisms of Insulin Resistance. *Current Tissue Microenvironment Reports*, **5**, 79-90. https://doi.org/10.1007/s43152-024-00056-3

List of Abbreviations

TyG Index: Triglyceride-Glucose Index;
baPWV: Brachial-Ankle Pulse Wave Velocity;
cfPWV: Carotid-Femoral Pulse Wave Velocity;
PPA: Pulse Pressure Amplification;
ABI: Ankle-Brachial Index;
cAIx: Central Augmentation Index;
HOMA-IR: Homeostasis Model Assessment of Insulin Resistance;
BP: Blood Pressure;
BMI: Body Mass Index;
MAP: Mean Arterial Pressure;
UACR: Urinary Albumin-to-Creatinine Ratio.