

Original articles

Non-obese adults with prediabetes in Central Vietnam: Metabolic risk factors and associations with the triglyceride-glucose index

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Abstract

Background: Insulin resistance can occur even in non-obese individuals, particularly among Asian populations, where visceral adiposity contributes to metabolic risk. The triglyceride-glucose (TyG) index has emerged as a simple and reliable surrogate marker of insulin resistance. In Vietnam, evidence linking the TyG index to metabolic abnormalities in this group is still scarce.

Objective: To describe the metabolic characteristics of non-obese adults with prediabetes in Central Vietnam and to examine the associations between the TyG index and selected anthropometric and lipid-related risk factors.

Methods: A cross-sectional study of 104 non-obese adults (BMI < 25 kg/m²), aged 20 - 65 years, with newly diagnosed prediabetes (ADA 2022 criteria) consecutively enrolled from community health centers in Quang Binh, Hue, and Da Nang City (October 2022 to October 2025). Anthropometric, glycaemic, and lipid parameters were assessed. An elevated TyG index (≥ 8.5) was used as a surrogate marker of insulin resistance. Pearson correlation and multiple linear regression were performed; triglycerides were excluded from the multivariable model as a direct mathematical component of the TyG formula.

Results: Mean age 50.56 ± 9.68 years; mean BMI of 23.39 ± 1.88 kg/m². Central adiposity (WHtR ≥ 0.5) was present in 67.3%, and 76.0% exhibited elevated TyG index. Dyslipidemia was prevalent: TC ≥ 5.2 mmol/L in 55.8%, hypertriglyceridaemia in 42.3%, elevated non-HDL-C in 73.1%. TyG correlated significantly with TC/HDL-C ($r = 0.532$), non-HDL-C ($r = 0.479$), TC ($r = 0.395$), and WC ($r = 0.243$). In multivariable regression, independent predictors of TyG were: TC ($\beta = 0.463$, $p < 0.001$), HDL-C ($\beta = -0.303$, $p = 0.001$), female sex ($\beta = -0.287$, $p = 0.002$), and fasting plasma glucose ($\beta = 0.255$, $p = 0.002$); adjusted $R^2 = 0.371$, $F(8,95) = 8.589$, $p < 0.001$.

Conclusions: In non-obese Vietnamese adults with prediabetes, the TyG index was significantly associated with central adiposity and atherogenic dyslipidaemia, with total cholesterol emerging as its strongest independent predictor. TyG may serve as a practical surrogate marker for cardiometabolic risk assessment in resource-limited primary care settings. Further prospective studies are needed to confirm these findings and establish clinical utility.

Keywords: prediabetes; non-obese; TyG index; visceral adiposity; insulin resistance; Central Vietnam.

1. INTRODUCTION

The metabolic phenotype of dysglycemia in Asians differs from obesity-related models observed in Western populations. Asians tend to accumulate more visceral fat despite having lower body mass index (BMI), leading to insulin resistance and dyslipidemia even at normal BMI levels. This metabolically unhealthy normal-weight (MONW) phenotype highlights the limitations of BMI in assessing metabolic risk [1-3].

The triglyceride-glucose (TyG) index, calculated as $\ln [\text{TG (mg/dL)} \times \text{FPG (mg/dL)} / 2]$, is a simple and reliable surrogate marker of insulin resistance and a strong predictor of type 2 diabetes mellitus, even in non-obese individuals [4, 5].

In Vietnam, national surveys show rising trends in abdominal obesity and dyslipidemia despite a relatively low average BMI, suggesting that visceral fat accumulation may contribute to the growing burden of prediabetes and diabetes [6, 7]. However, data on insulin resistance and TyG index among non-obese Vietnamese adults remain limited. No previous study has examined TyG index as a surrogate marker for insulin resistance in the non-obese, prediabetic subgroup in Central Vietnam. Furthermore, the rationale for using the TyG index beyond conventional glycemic parameters (FPG, HbA1c) lies in its ability to simultaneously capture both glucose and lipid metabolism, thereby reflecting insulin resistance more comprehensively. Importantly, non-obese

Asian individuals remain at high metabolic risk due to visceral adiposity, which is not captured by BMI alone. Therefore, this study aimed to describe the metabolic characteristics of non-obese adults with prediabetes in Central Vietnam and to examine the associations between the TyG index and selected anthropometric and lipid-related risk factors.

2. MATERIALS AND METHODS

2.1. Study Design and Participants:

A cross-sectional study was conducted from October 2022 to October 2025 in community health centers in Quang Binh, Thua Thien Hue, and Da Nang, Central Vietnam. Individuals aged 20 - 65 years attending routine health check-ups were screened

for eligibility. Those meeting all inclusion criteria were consecutively invited to participate; no eligible individual refused to enroll. A total of 104 non-obese adults (BMI < 25 kg/m²) with newly diagnosed prediabetes according to ADA 2022 criteria (fasting plasma glucose 5.6 - 6.9 mmol/L and/or HbA1c 5.7 - 6.4%) were enrolled and included in the final analysis (Figure 1). Exclusion criteria included: known diabetes or current use of hypoglycemic medications; use of lipid-lowering agents (statins or fibrates); use of systemic corticosteroids within the preceding 3 months; use of hormonal therapy (including oral contraceptives); chronic liver or kidney disease; thyroid dysfunction; acute or chronic inflammatory diseases; pregnancy; and alcohol use disorder.

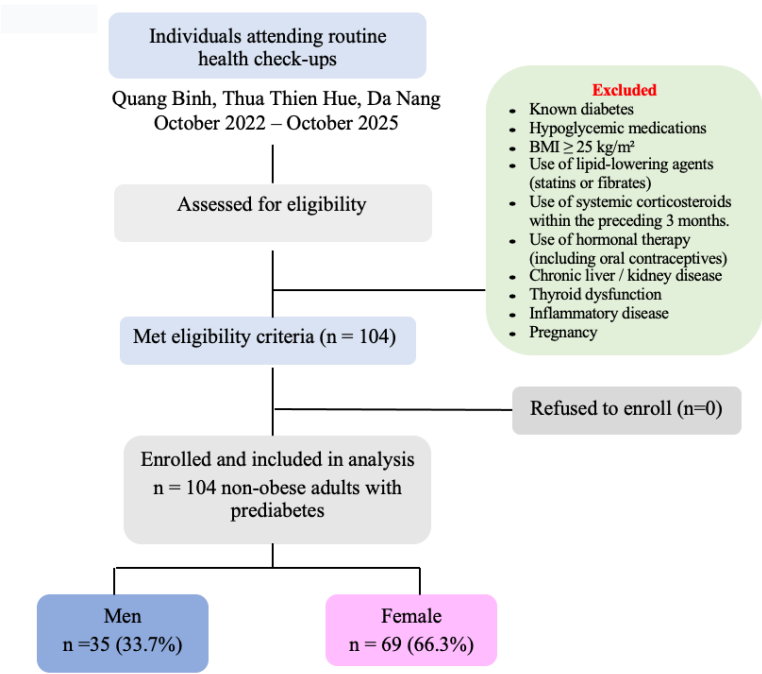


Figure 1. Participant Flow Diagram

2.2. Definitions

Non-obesity: BMI < 25 kg/m² according to WHO criteria for Asian populations [8].

Prediabetes: FPG 5.6 - 6.9 mmol/L and/or HbA1c 5.7 - 6.4% (ADA 2022) [9].

Elevated TyG index (surrogate marker suggestive of insulin resistance): TyG ≥ 8.5, a threshold derived from validation studies in Asian populations showing optimal sensitivity (~80%) and specificity (~84%) for predicting insulin resistance in non-diabetic adults [5].

Central obesity: WC ≥ 90 cm (men) or ≥ 80 cm (women) (IDF criteria for Asians); WHtR ≥ 0.5 [10, 11].

Dyslipidemia: Defined based on NCEP ATP III guidelines [12].

2.3. Statistical Analysis

Statistical analyses were performed using SPSS version 18.0. The normality of continuous variables was assessed using the Shapiro-Wilk test. All continuous variables were found to be approximately normally distributed. Results were expressed as mean ± standard deviation (SD) or percentages. Pearson’s correlation coefficient was used to assess relationships between variables. Multiple linear regression identified independent predictors of TyG index using a pre-specified model; variables were selected based on clinical relevance and significant univariate correlations (p < 0.05). A two-tailed p-value < 0.05 was considered statistically significant.

2.4. Ethical Considerations

This study was approved by the Biomedical Research Ethics Committee of Hue University of Med-

icine and Pharmacy (H2022/037, dated May 26th, 2022). All participants provided written informed consent.

3. RESULTS

3.1. Baseline Characteristics of the Study Population

The study included 104 non-obese adults with prediabetes (35 men, 69 women) with a mean age of 50.56 ± 9.68 years. Mean BMI was 23.39 ± 1.88 kg/m², with 66.3% in the 23.0-24.9 kg/m² range (overweight by Asian criteria but below the WHO obesity cut-off). Mean WC was 83.86 ± 6.43 cm in men and 78.71 ± 7.14 cm in women. Central adiposity (WHtR ≥ 0.5) was observed in 67.3% (Table 1).

Table 1. Clinical and anthropometric characteristics

Characteristic	Total (n = 104)	Men (n = 35)	Women (n = 69)
Age (years), mean $\pm$ SD	50.56 ± 9.68	49.31 ± 9.75	51.19 ± 9.65
Anthropometrics			
BMI (kg/m ²), mean $\pm$ SD	23.39 ± 1.88	-	-
BMI 23 - 24.9 kg/m ² , n(%)	69 (66.3%)	-	-
Waist circumference (cm), mean $\pm$ SD	-	83.86 ± 6.43	78.71 ± 7.14
WC ≥ 90 cm (men) or ≥ 80 cm (women), n(%)	52/104 (50%)	9/35 (25.7%)	43/69 (62.3%)
WHtR, mean $\pm$ SD	0.51 ± 0.05	-	-
WHtR ≥ 0.5 , n(%)	70 (67.3%)	-	-

3.2. Glycemia and TyG index

Mean FPG was 5.60 ± 0.59 mmol/L and mean HbA1c was $5.89 \pm 0.32\%$. Mean TyG index was 8.88 ± 0.48 , higher in men (9.16 ± 0.58) than women (8.74 ± 0.36). Elevated TyG index (≥ 8.5) was found in 76.0% of all participants (Table 2).

Table 2. Glycemia and TyG index

Characteristic	Total (n = 104)	Male (n = 35)	Female (n = 69)
Glycemic Parameters			
FPG (mmol/L), mean $\pm$ SD	5.60 ± 0.59	5.61 ± 0.56	5.59 ± 0.61
HbA1c (%), mean $\pm$ SD	5.89 ± 0.32	5.91 ± 0.23	5.88 ± 0.36
TyG index			
TyG index, mean $\pm$ SD	8.88 ± 0.48	9.16 ± 0.58	8.74 ± 0.36
TyG index ≥ 8.5 , n(%)	79/104 (76.0%)	30/35 (85.7%)	49/69 (71.0%)

3.3. Lipid Profile and Atherogenic Indices

More than half of participants had TC ≥ 5.2 mmol/L (55.8%), and 73.1% showed elevated non-HDL-C (≥ 3.4 mmol/L). Hypertriglyceridaemia was present in 42.3%. Low HDL-C was present in 41.3% (men 34.3%, women 44.9%). The TC/HDL-C ratio was ≥ 4 in 54.8% (Table 3).

Table 3. Lipid profile and atherogenic indices (n = 104)

Parameter	Cut-off	n (%)
Total Cholesterol (TC)	≥ 5.2 mmol/L	58 (55.8)
LDL-C	≥ 3.4 mmol/L	47 (45.2)
Triglycerides (TG)	≥ 1.7 mmol/L	44 (42.3)
Low HDL-C (sex-specific criteria)*	See note	43 (41.3)
Non-HDL-C	≥ 3.4 mmol/L	76 (73.1)
TC/HDL-C ratio	≥ 4	57 (54.8)

*Low HDL-C defined as < 1.0 mmol/L in men and < 1.3 mmol/L in women. Sex-specific rates: men 12/35 (34.3%); women 31/69 (44.9%).

3.4. Univariate Correlation Analysis of Factors Associated with the TyG Index

TyG correlated strongly with TG ($r = 0.820$, $p < 0.001$), partly inherent to the formula and significantly with TC/HDL-C ($r = 0.532$), non-HDL-C ($r = 0.479$), TC ($r = 0.395$), and WC ($r = 0.243$). Correlations with BMI and WHtR were not significant (Table 4).

Table 4. Correlations of TyG index with metabolic risk factors

Variable	Correlation Coefficient (r)	p-value
WC	0.243	< 0.05
BMI	0.152	> 0.05
WHtR	0.057	> 0.05
TC	0.395	< 0.05
TG	0.820	< 0.05
HDL-C	0.220	< 0.05
Non-HDL-C	0.479	< 0.05
TC/HDL-C	0.532	< 0.05

** The strong correlation between TyG and TG ($r = 0.820$) is partly inherent, as TG are a direct component of the TyG index calculation. This finding should not be interpreted as an independent association.*

3.5. Univariate Correlations of HbA1c

HbA1c showed weak but significant positive correlations with non-HDL-C ($r = 0.196$, $p < 0.05$) and TC/HDL-C ratio ($r = 0.217$, $p < 0.05$). No significant correlations were observed with anthropometric indices or other lipid parameters (Table 5). These findings suggest that HbA1c may have limited utility in capturing the broader spectrum of metabolic dysregulation beyond glycaemic control in this non-obese prediabetic population.

Table 5. Univariate correlations of HbA1c with metabolic risk factors

Variable	Correlation Coefficient (r)	p-value
WC	0.097	> 0.05
BMI	-0.042	> 0.05
WHtR	0.047	> 0.05
Total Cholesterol (TC)	0.150	> 0.05
Triglycerides (TG)	0.105	> 0.05
HDL-C	-0.125	> 0.05
Non-HDL-C	0.196	< 0.05
TC/HDL-C	0.217	< 0.05

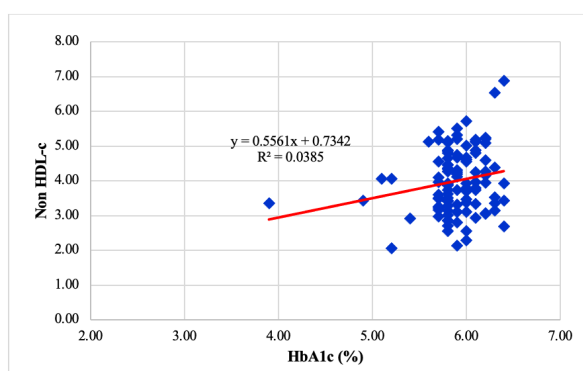


Figure 2. Correlations of HbA1c with Non HDL-C

3.6. Multivariable Linear Regression: Independent Predictors of TyG index

In the multivariable linear regression model, four variables were identified as independent predictors of the TyG index (Table 6): total cholesterol ($\beta = 0.463$, $p < 0.001$), HDL-C ($\beta = -0.303$, $p = 0.001$),

female sex ($\beta = -0.287$, $p = 0.002$), and fasting plasma glucose ($\beta = 0.255$, $p = 0.002$). The model explained 42.0% of TyG variance (adjusted $R^2 = 0.371$, $F(8, 95) = 8.589$, $p < 0.001$). Age, waist circumference, systolic blood pressure, and HbA1c were not independently associated with TyG after adjustment.

Table 6. Multiple linear regression: independent predictors of TyG index (n = 104)

Variables	Unstandardized Coefficients		Standardized Coefficients (Beta)	95% CI for B		p
	B	SE		Lower B	Upper B	
Model: R ² = 0.420, Adjusted R ² = 0.371, F(8, 95) = 8.589, p < 0.001, SE = 0.3873						
Constant	8.818	0.975	—	6.881	10.754	< 0.001
Age (years)	-0.002	0.005	-0.031	-0.011	0.008	0.742
Sex (female vs. male)	-0.295	0.091	-0.287	-0.476	-0.114	0.002
WC (cm)	-0.001	0.006	-0.018	-0.013	0.011	0.846
Systolic BP (mmHg)	-0.001	0.003	-0.024	-0.006	0.005	0.791
FPG (mmol/L)	0.208	0.066	0.255	0.078	0.339	0.002
HbA1c (%)	-0.170	0.121	-0.113	-0.410	0.070	0.162
TC (mmol/L)	0.240	0.046	0.463	0.148	0.331	< 0.001
HDL-C (mmol/L)	-0.474	0.141	-0.303	-0.754	-0.194	0.001

* B = unstandardized coefficient; SE = standard error; β = standardized coefficient; CI = confidence interval. TG excluded as direct mathematical component of TyG formula. Dependent variable: TyG index.

4. DISCUSSION

4.1. Pathophysiological Basis of the Non-obese Prediabetes Phenotype

In this cross-sectional study of non-obese adults with prediabetes in Central Vietnam, a high prevalence of elevated TyG index values was observed in more than three-quarters of participants. Central adiposity and atherogenic dyslipidemia were also common despite normal BMI ranges. These findings highlight the metabolic heterogeneity of prediabetes in Asian populations and suggest that insulin resistance can develop independently of general obesity.

The pathophysiological basis of the non-obese prediabetes phenotype lies in the concept of ectopic fat deposition. Among Asians, a genetic predisposition toward lower adiponectin levels and a higher proportion of visceral adipose tissue relative to subcutaneous fat has been well documented [1, 13]. Visceral adiposity promotes hepatic insulin resistance, increasing VLDL secretion and driving hypertriglyceridaemia, while intramyocellular lipid accumulation reduces peripheral glucose uptake, exacerbating hyperglycaemia even at normal BMI.

4.2. Comparison with National and Regional Data

Our findings align with recent Vietnamese data. Pham et al. reported that abdominal obesity

in Vietnam tripled over six years despite stable mean BMI, demonstrating dissociation between general and central adiposity [6]. Dang et al. further documented that dyslipidemia, particularly isolated hypertriglyceridemia, is the most common metabolic abnormality in Vietnamese adults, affecting over 30% of those with normal BMI [7]. The present study extends these observations by demonstrating that among non-obese prediabetic Vietnamese adults in Central Vietnam, the prevalence of hypertriglyceridemia (42.3%) and elevated TyG (76.0%) is substantially higher than in the general population, underscoring the added metabolic burden conferred by early glucose dysregulation. These data are derived from three provinces in Central Vietnam and may not be fully representative of the entire Vietnamese population. Therefore, the application of the MONW concept to all Asian populations should be made cautiously without further validation across different regions.

4.3. TyG Index, Central Adiposity, and Lipid Profile

The predominance of elevated TyG index in this non-obese cohort, together with the high prevalence of central adiposity and atherogenic dyslipidemia, supports the concept that metabolic dysfunction in

Asian individuals often operates independently of overall adiposity [4]. The pattern observed - visceral fat accumulation driving hepatic insulin resistance despite normal BMI - is consistent with the MONW phenotype described in prior Asian studies [3, 11].

The high prevalence of elevated TyG and its significant correlations with atherogenic lipid indices TC/HDL-C ($r = 0.532$), non-HDL-C ($r = 0.479$), TC ($r = 0.395$) confirm that TyG reflects both insulin resistance and atherogenic dyslipidaemia. However, the very strong correlation with TG ($r = 0.820$) is partly mathematical, as triglycerides are a direct component of the TyG formula. Despite this inherent limitation, the TyG index remains a useful surrogate marker because it integrates both glucose and triglyceride pathways. The lack of significant correlation between TyG and BMI further supports that TyG is more closely related to metabolic dysfunction than to overall adiposity. These findings are consistent with previous reports demonstrating that elevated TyG levels are associated with higher TG concentrations, reduced HDL-C, and increased cardiovascular risk. This relationship aligns with the physiological link between insulin resistance and reduced triglyceride clearance due to impaired lipoprotein lipase activity [4].

The high prevalence of increased TyG index among non-obese prediabetic individuals highlights the presence of underlying insulin resistance even in the absence of obesity. These findings are consistent with those of Lim et al. (2014), who emphasized that ectopic fat deposition in the liver and muscle often occurring without elevated BMI is a key driver of insulin resistance in Asians [13]. This aligns with evidence from Asian populations suggesting that lean individuals can still exhibit metabolic disturbances related to visceral adiposity and ectopic fat deposition. Although the mean BMI in this study was within the normal range, more than two-thirds of participants presented with central adiposity ($WHtR \geq 0.5$), supporting the concept of the MONW phenotype. Our data from Central Vietnam contribute regional evidence highlighting the potential limitations of BMI-based metabolic screening in this population.

4.4. Multivariable Analysis: Independent Predictors of the TyG Index

In the multivariable model, 4 variables were independent predictors of TyG: TC, HDL-C, female sex, and fasting plasma glucose (adjusted $R^2 = 0.371$, $F(8,95) = 8.589$, $p < 0.001$).

TC was the strongest predictor ($\beta = 0.463$, $p < 0.001$), reflecting the shared pathophysiological

basis of elevated TC and TyG: hepatic insulin resistance drives increased VLDL synthesis, simultaneously elevating TG and TC as parallel biomarkers of the same upstream metabolic dysfunction [4, 5]. TC therefore does not cause TyG elevation, but increases concurrently as a marker of the same atherogenic state. HDL-C showed a strong negative independent association ($\beta = -0.303$, $p = 0.001$). Hepatic insulin resistance increases VLDL secretion, accelerating HDL-C catabolism through CETP-mediated lipid exchange, simultaneously elevating TG and lowering HDL-C. This confirms that TyG and HDL-C are reciprocal markers of the same atherogenic metabolic state consistent with the inverse association reported in Korean and Chinese community-based cohorts [5].

FPG remained independently associated with the TyG index ($\beta = 0.255$, $p = 0.002$) after adjustment for HbA1c, suggesting that variations in fasting glucose within the prediabetic range contribute substantially to TyG variability through impaired regulation of hepatic glucose output. This finding complements the lipid-axis contributions of TC and HDL-C. Female sex was independently associated with lower TyG ($\beta = -0.287$, $p = 0.002$), consistent with oestrogen-mediated upregulation of lipoprotein lipase and suppression of hepatic VLDL secretion. However, the mean age of women was 51.19 years, suggesting a substantial proportion of peri-menopausal participants. Therefore, this protective hormonal effect may have been attenuated and should be interpreted cautiously. Notably, WC lost statistical significance in the multivariable model despite significant univariate correlation ($r = 0.243$), suggesting that visceral adiposity influences TyG indirectly through downstream lipid and glucose dysregulation captured by TC, HDL-C, and FPG rather than as a direct independent effect. Similarly, HbA1c showed no independent association with TyG, reflecting its distinct metabolic domains: HbA1c captures chronic glycaemic exposure, whereas TyG integrates glucose and lipid dysfunction simultaneously. In this cohort with narrow HbA1c variation ($5.89 \pm 0.32\%$), HbA1c lacks discriminatory power for TyG variability, supporting the complementary clinical utility of TyG beyond conventional glycaemic indices.

The remaining unexplained TyG variance (adjusted $R^2 = 0.371$) suggests that unmeasured factors, fasting insulin, adipokines, dietary composition, and physical activity may contribute substantially. Future studies incorporating direct insulin measurement are warranted.

4.5. TyG Index versus HbA1c: Comparative Clinical Utility

HbA1c showed only modest correlations with lipid indices, suggesting it reflects glycemic control but not the broader spectrum of insulin resistance related metabolic disturbances. Thus, TyG index was significantly associated with several metabolic risk markers and may serve as a potential surrogate marker for early detection of metabolic abnormalities in non-obese individuals with prediabetes. Similar observations have been reported by Park et al. (2021), who found the TyG index to outperform HOMA-IR in predicting metabolic syndrome [5]. These results support the complementary use of TyG alongside HbA1c in metabolic risk assessment of non-obese prediabetic adults.

4.6. Clinical Implications

The high prevalence of elevated TyG index despite normal BMI suggests that insulin resistance may precede the onset of overt obesity in this population. Incorporating WHtR and TyG into clinical screening, rather than relying solely on BMI, could improve early detection of cardiometabolic risk. TyG may serve as a cost-effective tool in primary healthcare settings where insulin assays are unavailable. Identifying MONW phenotype enables timely lifestyle interventions before progression to diabetes. Ethnic-specific thresholds for metabolic risk assessment may be warranted in Asian populations, though further validation studies are needed before formal recommendations.

4.7. Strengths and Limitations

Strengths: This study provides novel evidence on the metabolic characteristics of non-obese Vietnamese adults with prediabetes in Central Vietnam, emphasizing the potential clinical utility of the TyG index and WHtR.

Limitations: (1) The cross-sectional design limits causal inference. (2) The absence of a normoglycemic control group restricts direct comparison. (3) Insulin resistance was estimated indirectly using the TyG index (a surrogate marker) rather than measured by the reference standard (euglycemic-hyperinsulinemic clamp) or HOMA-IR. (4) Small sample ($n = 104$); consecutive sampling may limit community generalisability. (5) Most participants had BMI 23.0 - 24.9 kg/m² (overweight by Asian criteria), so the MONW concept should be applied cautiously. (6) Data from three Central Vietnam provinces only; results may not be representative of all Vietnamese populations.

5. CONCLUSION

In non-obese Vietnamese adults with prediabetes from Central Vietnam, insulin resistance is highly prevalent and closely associated with central adiposity and atherogenic dyslipidaemia. In multivariable analysis, total cholesterol ($\beta = 0.463$), HDL-C ($\beta = -0.303$), female sex ($\beta = -0.287$), and fasting plasma glucose ($\beta = 0.255$) were independent predictors of the TyG index, together explaining 42.0% of its variance (adjusted $R^2 = 0.371$). Total cholesterol was the strongest predictor, reflecting shared hepatic insulin resistance driving VLDL overproduction. The TyG index may serve as a simple and practical surrogate marker for metabolic risk assessment in this population. However, given the cross-sectional design and small sample size, longitudinal studies with direct measures of insulin resistance are needed to confirm these findings and establish clinical utility.

REFERENCES

1. Rhee EJ. Diabetes in Asians. *Endocrinol Metab.* 2015;30(3):263-269.
2. Stefan N, Schick F, Häring HU. Causes, characteristics, and consequences of metabolically unhealthy normal weight in humans. *Cell Metab.* 2017 Aug 1;26(2):292-300.
3. Pluta W, Dudzińska W, Lubkowska A. Metabolic Obesity in People with Normal Body Weight (MONW)-Review of Diagnostic Criteria. *Int J Environ Res Public Health.* 2022 Jan 6;19(2):624. doi: 10.3390/ijerph19020624.
4. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metab Syndr Relat Disord.* 2008 Sep;6(4):299-304.
5. Park HM, Lee HS, Lee YJ, Lee JH. The triglyceride-glucose index is a more powerful surrogate marker for predicting the prevalence and incidence of type 2 diabetes mellitus than the homeostatic model assessment of insulin resistance. *Diabetes Metab J.* 2021 Oct;45(5):733-746.
6. Pham T, Bui L, Giovannucci E, Hoang M, Tran B, Chavarro JE, et al. Prevalence of obesity and abdominal obesity and their association with metabolic-related conditions in Vietnamese adults: an analysis of Vietnam STEPS survey 2009 and 2015. *Lancet Reg Health West Pac.* 2023;39:100859.
7. Dang AK, Thi Le LT, Pham NM, Nguyen DQ, Thi Nguyen HT, Dang SC, et al. An upward trend of dyslipidemia among adult population in Vietnam: Evidence from a systematic review and meta-analysis. *Diabetes Metab Syndr Clin Res Rev.* 2025; doi: 10.1016/j.dsx.2024.103171.
8. WHO Consultation on Obesity & World Health Organization. Obesity: preventing and managing the global epidemic: report of a WHO consultation. World Health Organization; 2000.

9. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. *Diabetes Care*; 2022 Jan 1;45(Supplement_1):S17-S38.

10. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. Brussels: IDF; 2006.

11. Ashwell M, Gibson S. A proposal for a primary screening tool: 'Keep your waist circumference to less than half your height'. *BMC Med*. 2014;12:207. doi: 10.1186/s12916-014-0207-1

12. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285(19):2486–2497. doi: 10.1001/jama.285.19.2486.

13. Lim S, Meigs JB. Links between ectopic fat and vascular disease in humans. *Arterioscler Thromb Vasc Biol*; 2014 Aug;34(8):1820-6.