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Association of Triglyceride–Glucose Index with Newly Diagnosed Hypertension in Patients With and Without Type 2 Diabetes Mellitus at a Tertiary Care Centre in Northern India

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ABSTRACT

Background: Hypertension and type 2 diabetes mellitus (T2DM) frequently coexist and share common pathophysiological mechanisms, particularly insulin resistance. The triglyceride–glucose (TyG) index, derived from fasting triglyceride and glucose levels, has emerged as a simple and reliable surrogate marker of insulin resistance. However, limited data are available from the Indian population evaluating the association between TyG index and newly diagnosed hypertension.

Objectives: To evaluate the association of the triglyceride–glucose index with newly diagnosed hypertension in patients with and without type 2 diabetes mellitus and to identify independent predictors of hypertension.

Methods: This cross-sectional observational study was conducted at a tertiary care centre in Northern India and included 164 adults with newly diagnosed hypertension. Demographic characteristics, lifestyle factors, anthropometric measurements, blood pressure, glycaemic parameters, lipid profile, renal parameters, inflammatory markers, and TyG index were assessed. Associations between TyG index and hypertension were analysed using bivariate analysis and multivariable logistic regression.

Results: The mean TyG index of the study population was 9.23 ± 0.25. Hypertensive participants had a significantly higher TyG index compared to normotensive individuals (9.24 ± 0.25 vs. 9.20 ± 0.20; $p = 0.031$). Hypertension was significantly associated with older age, obesity, increased waist–hip ratio, smoking, alcohol intake, hyperglycaemia, hypertriglyceridaemia, elevated hs-CRP levels, and diabetes mellitus. On multivariable logistic regression analysis, TyG index emerged as an independent predictor of newly diagnosed hypertension (adjusted OR 2.68; 95% CI: 1.72–4.18; $p < 0.001$), along with age, BMI, and diabetes mellitus.

Conclusion: The triglyceride–glucose index is independently associated with newly diagnosed hypertension and may serve as a simple, cost-effective marker for identifying individuals at high cardiovascular risk. Early identification of insulin resistance using TyG index may aid in targeted preventive strategies for hypertension and related metabolic disorders.

KEYWORDS: Triglyceride–glucose index, Hypertension, Type 2 diabetes mellitus, Insulin resistance, Dyslipidaemia.

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INTRODUCTION

Hypertension is a major public health challenge and a leading contributor to cardiovascular morbidity and mortality worldwide. According to the World Health Organization, hypertension affects more than 1.2 billion adults globally and is responsible for a substantial proportion of premature deaths due to cardiovascular diseases [1]. In India, the prevalence of hypertension has increased dramatically over the past few decades, driven by rapid urbanization, sedentary lifestyle, obesity, and changing dietary patterns [2]. Hypertension often coexists with metabolic disorders such as type 2 diabetes mellitus (T2DM), dyslipidaemia, and obesity, forming a complex network of cardiometabolic risk factors [3].

Insulin resistance plays a central role in the pathogenesis of both hypertension and T2DM. It contributes to endothelial dysfunction, increased sympathetic nervous system activity, renal sodium retention, and vascular smooth muscle proliferation, all of which promote elevated blood pressure [4,5]. The coexistence of hypertension and insulin resistance significantly increases the risk of atherosclerotic cardiovascular disease, chronic kidney disease, and cerebrovascular events [6].

Direct measurement of insulin resistance using the hyperinsulinaemic–euglycaemic clamp technique is considered the gold standard; however, its complexity and cost limit its use in routine clinical practice [7]. Alternative surrogate markers such as the homeostatic model assessment of insulin resistance (HOMA-IR) require insulin assays, which are not universally available and

may be expensive in low-resource settings [8]. Consequently, there has been growing interest in simpler and more accessible markers of insulin resistance.

The triglyceride–glucose (TyG) index, calculated using fasting plasma glucose and triglyceride levels, has emerged as a reliable and inexpensive surrogate marker of insulin resistance [9]. Several studies have demonstrated a strong correlation between TyG index and insulin resistance measured by the clamp technique and HOMA-IR [10,11]. The TyG index has also been associated with metabolic syndrome, T2DM, atherosclerosis, and cardiovascular events [12–14].

Recent evidence suggests that elevated TyG index is linked to the development and progression of hypertension [15]. Insulin resistance, reflected by higher TyG values, may contribute to hypertension through impaired nitric oxide–mediated vasodilation, activation of the renin–angiotensin–aldosterone system, and increased inflammatory response [16,17]. Moreover, low-grade chronic inflammation, as indicated by elevated high-sensitivity C-reactive protein (hs-CRP), further amplifies vascular dysfunction and blood pressure elevation in insulin-resistant individuals [18].

Despite increasing global interest, data examining the relationship between TyG index and newly diagnosed hypertension in the Indian population remain limited. Given the high burden of undiagnosed insulin resistance, diabetes, and hypertension in India, identifying simple markers for early risk stratification is of paramount importance [19,20]. Evaluating the TyG index in patients with newly diagnosed hypertension may provide insights into underlying metabolic derangements and help guide early preventive and therapeutic interventions.

Therefore, the present study was undertaken to assess the association between triglyceride–glucose index and newly diagnosed hypertension in patients with and without type 2 diabetes mellitus at a tertiary care centre in Northern India, and to evaluate the independent predictors of hypertension in this population.

MATERIAL AND METHODS

This was a **hospital-based cross-sectional observational study** conducted in the Department of General Medicine at a tertiary care teaching hospital in Northern India. The study was carried out over a defined study period after obtaining approval from the Institutional Ethics Committee. All procedures were performed in accordance with the ethical standards laid down in the Declaration of Helsinki.

Study Population and Sample Size

A total of **164 adult patients** with **newly diagnosed hypertension**, with or without type 2 diabetes mellitus, attending the outpatient and inpatient services of the Department of General Medicine were enrolled in the study. Newly diagnosed hypertension was defined as blood pressure values meeting diagnostic criteria at first presentation, without prior history of antihypertensive treatment.

Inclusion Criteria

1. Adults aged **≥18 years**
2. Newly diagnosed cases of hypertension
3. Patients with or without previously diagnosed or newly detected type 2 diabetes mellitus
4. Patients willing to participate and provide written informed consent

Exclusion Criteria

1. Known cases of secondary hypertension
2. Patients already on antihypertensive therapy
3. Pregnant women
4. Patients with known chronic liver disease, chronic kidney disease (eGFR <60 ml/min/1.73 m²), acute infections, malignancy, or autoimmune disorders
5. Patients on lipid-lowering drugs, steroids, or medications affecting glucose or lipid metabolism
6. Critically ill patients

Data Collection

After obtaining informed consent, detailed clinical evaluation was performed. Data were collected using a predesigned and pretested proforma, which included demographic details (age, gender), lifestyle factors (smoking and alcohol intake), medical history, and physical examination findings.

Anthropometric Measurements

Body weight and height were measured using standard calibrated instruments, and **body mass index (BMI)** was calculated as weight in kilograms divided by height in meters squared (kg/m²). BMI was categorized as normal (<25 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²).

Waist and hip circumferences were measured using a non-stretchable measuring tape, and **waist–hip ratio (WHR)** was calculated. Increased WHR was defined as per standard cut-off values.

Blood Pressure Measurement

Blood pressure was measured using a calibrated sphygmomanometer with the patient in a seated position after at least five minutes of rest. Two readings were taken at an interval of five minutes, and the average of the two readings was recorded. Hypertension was classified according to standard guidelines into normotensive, stage 1 hypertension, and stage 2 hypertension categories.

Laboratory Investigations

After an overnight fast of at least 8–10 hours, venous blood samples were collected under aseptic precautions for the estimation of:

- Fasting blood glucose (FBG)
- Glycated hemoglobin (HbA1c)
- Lipid profile including triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol
- Serum creatinine
- High-sensitivity C-reactive protein (hs-CRP)

Estimated glomerular filtration rate (eGFR) was calculated using standard formulae.

Definition of Type 2 Diabetes Mellitus

Type 2 diabetes mellitus was defined based on fasting blood glucose ≥126 mg/dL and/or HbA1c ≥6.5%, or a prior diagnosis of diabetes with documented evidence.

Calculation of Triglyceride–Glucose (TyG) Index

The **TyG index**, a surrogate marker of insulin resistance, was calculated using the following formula:

TyG index=ln⁡(fasting triglycerides (mg/dL)×fasting glucose (mg/dL)2)\text{TyG index} = \ln \left(\frac{\text{fasting triglycerides (mg/dL)} \times \text{fasting glucose (mg/dL)}}{2}
\right)TyG index=ln(2fasting triglycerides (mg/dL)×fasting glucose (mg/dL))

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages.

Comparisons between hypertensive and normotensive groups were performed using appropriate statistical tests. Associations between categorical variables were analyzed using the chi-square test. Independent predictors of hypertension were identified using **multivariable logistic regression analysis**, and results were expressed as adjusted odds ratios (OR) with 95% confidence intervals (CI). A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 164 participants with newly diagnosed hypertension, with or without type 2 diabetes mellitus, were included in the study. The majority of the study population belonged to the age group of 55–64 years (37.8%), followed by 45–54 years (28.0%) and ≥65 years (19.5%), with a mean age skewed toward middle and older age groups. Males constituted 56.1% of the participants, while females accounted for 43.9%, indicating a slight male predominance. Lifestyle risk factor assessment revealed that 29.9% of participants were smokers and 40.9% reported alcohol consumption

Anthropometric evaluation showed a high burden of adiposity in the study population. The mean body mass index (BMI) was 29.1 ± 3.2 kg/m², with 59.1% of participants classified as obese and 33.5% as overweight. Only 7.3% had a normal BMI. The mean waist–hip ratio was 0.88 ± 0.06, with a significant proportion exhibiting increased central adiposity, highlighting the prevalence of obesity-related metabolic risk factors among newly diagnosed hypertensive individuals

The mean systolic blood pressure (SBP) was 141.6 ± 15.4 mmHg and the mean diastolic blood pressure (DBP) was 88.9 ± 9.8 mmHg. Nearly half of the participants (49.4%) were classified as having stage 2 hypertension, while 37.2% had stage 1 hypertension. Only 13.4% were normotensive at presentation, underscoring the predominance of clinically significant hypertension in the cohort

Regarding glycaemic status, 96.3% of participants were diabetic, and only 3.7% were non-diabetic. Fasting blood glucose levels showed that 43.3% had values ≥126 mg/dL, while 39.0% were in the impaired fasting glucose range (100–125 mg/dL). HbA1c analysis revealed that more than half of the participants (56.1%) had HbA1c levels ≥6.5%, reflecting poor long-term glycaemic control in a large proportion of the study population

Lipid profile analysis demonstrated significant dyslipidaemia. The mean triglyceride level was 168.4 ± 46.7 mg/dL, and 64.6% of participants had triglyceride levels ≥150 mg/dL. Low HDL cholesterol was observed in 54.3% of subjects, while 45.1% exhibited combined dyslipidaemia, indicating atherogenic lipid patterns commonly associated with insulin resistance and hypertension

Renal and inflammatory parameters showed a mean serum creatinine of 1.08 ± 0.21 mg/dL and a mean estimated glomerular filtration rate (eGFR) of 86.3 ± 18.9 ml/min/1.73m². The mean hs-CRP level was 1.12 ± 0.78 mg/dL, with elevated hs-CRP (≥1 mg/dL) observed in a substantial proportion of hypertensive participants, suggesting an underlying inflammatory milieu

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The mean triglyceride–glucose (TyG) index in the study population was 9.23 ± 0.25, ranging from 8.62 to 9.88. Hypertensive participants had a significantly higher mean TyG index compared to normotensive individuals (9.24 ± 0.25 vs. 9.20 ± 0.20; p = 0.031), indicating a positive association between insulin resistance and hypertension

Bivariate analysis revealed significant associations between new-onset hypertension and increasing age, smoking status, alcohol intake, higher BMI, increased waist–hip ratio, elevated fasting blood glucose, hypertriglyceridaemia, elevated hs-CRP levels, and higher TyG index values ($p < 0.05$). Gender did not show a statistically significant association with hypertension. Obese individuals and those with increased waist–hip ratio demonstrated a significantly higher prevalence of hypertension compared to their counterparts

Multivariable logistic regression analysis identified the TyG index as an independent predictor of new-onset hypertension, with an adjusted odds ratio (OR) of 2.68 (95% CI: 1.72–4.18; $p < 0.001$). Increasing age (OR 1.04), higher BMI (OR 1.18), and the presence of diabetes mellitus (OR 2.09) were also independently associated with hypertension after adjusting for confounding variables

Table 1. Age Distribution of Study Population (n = 164)

Age Group (years)	n	%
25–34	6	3.7
35–44	18	11.0
45–54	46	28.0
55–64	62	37.8
≥65	32	19.5

Table 2. Gender Distribution

Gender	n	%
Male	92	56.1
Female	72	43.9

Table 3. Lifestyle Risk Factors

Variable	Yes n (%)	No n (%)
Smoking	49 (29.9)	115 (70.1)
Alcohol Intake	67 (40.9)	97 (59.1)

Table 4. Anthropometric Profile

Parameter	Mean ± SD	Range
BMI (kg/m²)	29.1 ± 3.2	22.8–36.9
Waist–Hip Ratio	0.88 ± 0.06	0.72–1.02

Table 5. BMI Category Distribution

BMI Category	n	%
Normal (<25)	12	7.3
Overweight (25–29.9)	55	33.5
Obese (≥30)	97	59.1

Table 6. Blood Pressure Distribution

Parameter	Mean ± SD	Min–Max
SBP (mmHg)	141.6 ± 15.4	118–182
DBP (mmHg)	88.9 ± 9.8	72–112

Table 7. Classification of Blood Pressure

BP Category	n	%
Normotensive	22	13.4
Stage 1 HTN	61	37.2
Stage 2 HTN	81	49.4

Table 8. Glycaemic Status of Participants

Variable	n	%
Diabetes Mellitus	158	96.3
Non-Diabetic	6	3.7

Table 9. Fasting Blood Glucose Categories

FBG (mg/dL)	n	%
<100	29	17.7
100–125	64	39.0
≥126	71	43.3

Table 10. HbA1c Distribution

HbA1c (%)	n	%
<5.7	24	14.6
5.7–6.4	48	29.3
≥6.5	92	56.1

Table 11. Lipid Profile

Parameter (mg/dL)	Mean ± SD
Triglycerides	168.4 ± 46.7
Total Cholesterol	196.2 ± 32.1
HDL	47.8 ± 8.4
LDL	132.6 ± 24.9

Table 12. Atherogenic Dyslipidemia Pattern

Lipid Abnormality	n	%
TG ≥150 mg/dL	106	64.6
HDL <40 (M) / <50 (F)	89	54.3
Combined Dyslipidemia	74	45.1

Table 13. Renal & Inflammatory Parameters

Parameter	Mean ± SD
Serum Creatinine (mg/dL)	1.08 ± 0.21
eGFR (ml/min/1.73m²)	86.3 ± 18.9
hs-CRP (mg/dL)	1.12 ± 0.78

Table 14. Distribution of TyG Index

Parameter	Value
Mean TyG Index	9.23 ± 0.25
Minimum	8.62
Maximum	9.88

Table 15. Association of TyG Index with Hypertension

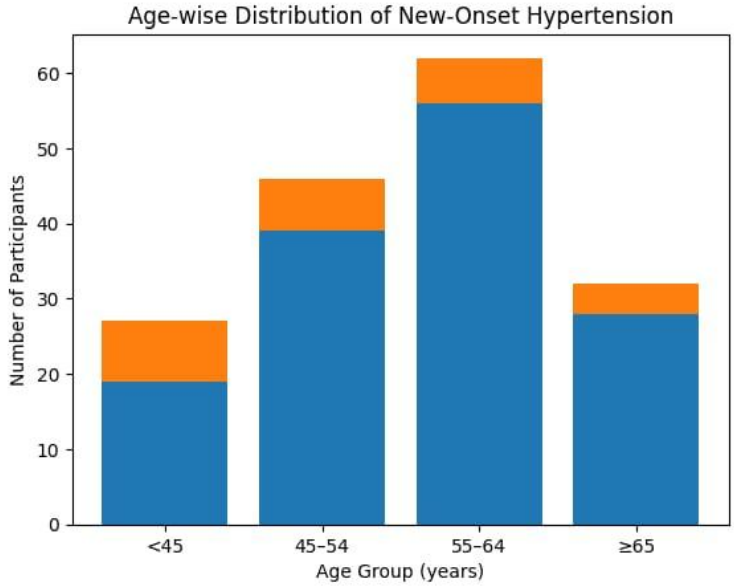
Group	Mean TyG ± SD	p-value
Hypertensive	9.24 ± 0.25	
Normotensive	9.20 ± 0.20	**0.031***

Table 16. Association of Diabetes Status with Hypertension

Diabetes	Hypertensive n (%)	Normotensive n (%)	p-value
Yes	136 (86.1)	22 (13.9)	**0.041***
No	6 (100)	0 (0)	

Table 17. Association of Demographic and Lifestyle Factors with New-Onset Hypertension

Variable	Category	Hypertensive n (%)	Normotensive n (%)	p-value
Age (years)	<45	19 (70.4)	8 (29.6)	
	45–54	39 (84.8)	7 (15.2)	
	55–64	56 (90.3)	6 (9.7)	
	≥65	28 (87.5)	4 (12.5)	**0.032***
Gender	Male	81 (88.0)	11 (12.0)	
	Female	61 (84.7)	11 (15.3)	0.53
Smoking	Yes	46 (93.9)	3 (6.1)	
	No	96 (83.5)	19 (16.5)	**0.048***
Alcohol intake	Yes	63 (94.0)	4 (6.0)	
	No	79 (81.4)	18 (18.6)	**0.021***



Graph No. 1: Agewise distribution

Table 18. Association of Anthropometric Parameters with New-Onset Hypertension

Variable	Category	Hypertensive n (%)	Normotensive n (%)	p-value
BMI	Overweight (25–29.9)	54 (80.6)	13 (19.4)	
	Obese (≥30)	88 (90.7)	9 (9.3)	**0.034***
Waist–Hip Ratio	Normal	38 (77.6)	11 (22.4)	
	Increased	104 (90.4)	11 (9.6)	**0.018***

Table 19. Association of Glycaemic and Lipid Parameters with New-Onset Hypertension

Variable	Category	Hypertensive n (%)	Normotensive n (%)	p-value
Diabetes Mellitus	Yes	136 (86.1)	22 (13.9)	**0.041***
	No	6 (100)	0 (0)	
Fasting Blood Glucose	≥126 mg/dL	67 (94.4)	4 (5.6)	
	<126 mg/dL	75 (80.6)	18 (19.4)	**0.012***
Triglycerides	≥150 mg/dL	98 (92.5)	8 (7.5)	
	<150 mg/dL	44 (75.9)	14 (24.1)	**0.006***

Table 20. Association of Inflammatory Marker with New-Onset Hypertension

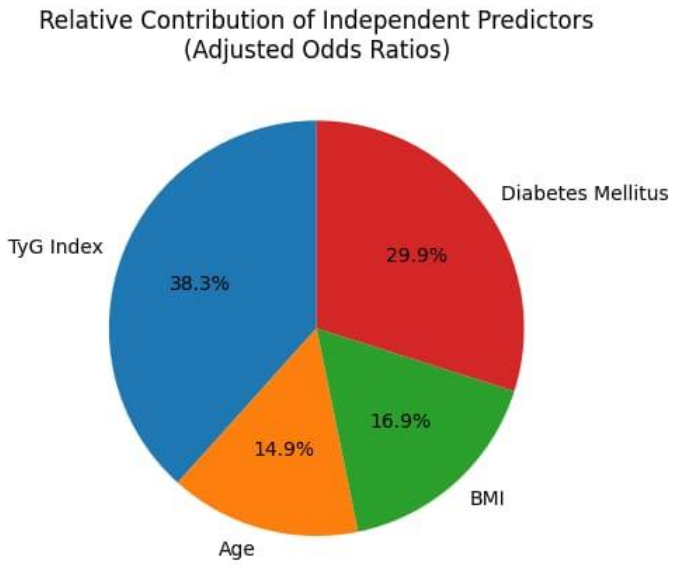
Variable	Category	Hypertensive n (%)	Normotensive n (%)	p-value
hs-CRP	≥1 mg/dL	84 (91.3)	8 (8.7)	
	<1 mg/dL	58 (80.6)	14 (19.4)	**0.041***

Table 21. Association of TyG Index (Exposure Variable) with New-Onset Hypertension

TyG Index	Hypertensive	Normotensive	p-value
Mean ± SD	9.24 ± 0.25	9.20 ± 0.20	**0.031***

Table 22. Multivariable Logistic Regression Showing Independent Associations

Variable	Adjusted OR	95% CI	p-value
TyG Index	**2.68**	1.72–4.18	**<0.001***
Age	1.04	1.01–1.07	0.011
BMI	1.18	1.05–1.32	0.004
Diabetes Mellitus	2.09	1.12–3.88	0.019



Graph 2: Multivariable Logistic Regression Showing Independent Associations

DISCUSSION

Hypertension and type 2 diabetes mellitus (T2DM) frequently coexist and represent major contributors to cardiovascular morbidity and mortality worldwide, particularly in developing countries such as India [1,2]. Insulin resistance is a central pathophysiological mechanism linking these two conditions and plays a crucial role in the development of endothelial dysfunction, vascular stiffness, and sympathetic overactivity [3,4]. The present study evaluated the association between the triglyceride–glucose (TyG) index, a simple surrogate marker of insulin resistance, and newly diagnosed hypertension in patients with and without T2DM.

In the present study, the mean TyG index was 9.23 ± 0.25, and hypertensive participants had a significantly higher TyG index compared to normotensive individuals (9.24 ± 0.25 vs. 9.20 ± 0.20; p = 0.031). This finding supports earlier evidence that elevated TyG index reflects increased insulin resistance, which contributes to blood pressure elevation even at early stages of hypertension [5,6]. Furthermore, multivariable logistic regression analysis revealed that the TyG index was an independent predictor of newly diagnosed hypertension (adjusted OR 2.68; 95% CI: 1.72–4.18; p < 0.001), independent of age, BMI, and diabetes status. Similar associations between TyG index and hypertension have been reported in population-based and hospital-based studies across different ethnic groups [7–9].

Age emerged as an important determinant of hypertension in the present study, with a higher prevalence observed among individuals aged 55–64 years. Increasing age was independently associated with hypertension, which is consistent with previous epidemiological studies demonstrating age-related arterial stiffening, endothelial dysfunction, and cumulative metabolic stress [10,11]. Although a slight male predominance was observed, gender was not significantly associated with hypertension, suggesting that metabolic risk factors outweigh sex-related differences in this cohort [12].

Obesity and central adiposity were highly prevalent among the study participants and showed significant associations with new-

onset hypertension. Both increased BMI and waist–hip ratio were strongly linked to hypertension, and BMI remained an independent predictor on regression analysis. Excess adiposity promotes insulin resistance through adipokine imbalance, chronic inflammation, and activation of the renin–angiotensin–aldosterone system, thereby contributing to elevated blood pressure [13–15].

Lifestyle factors such as smoking and alcohol consumption were significantly associated with hypertension on bivariate analysis. Smoking induces oxidative stress and endothelial injury, while alcohol intake increases sympathetic activity and impairs baroreceptor sensitivity [16,17]. Although these factors did not retain independent significance after adjustment, their contribution to the overall cardiometabolic risk burden cannot be overlooked.

A striking finding of the present study was the high prevalence of diabetes mellitus among newly diagnosed hypertensive patients. Diabetes mellitus was independently associated with hypertension (adjusted OR 2.09; $p = 0.019$). Chronic hyperglycaemia contributes to vascular inflammation, arterial stiffness, renal sodium retention, and impaired nitric oxide-mediated vasodilation, all of which promote hypertension [18,19].

Dyslipidaemia, particularly hypertriglyceridaemia, was common in the study population and showed a significant association with hypertension. Elevated triglyceride levels are a hallmark of insulin resistance and play a direct role in endothelial dysfunction and atherogenesis [20]. Since triglycerides are a core component of the TyG index, this finding further supports the biological plausibility of TyG as an integrative marker linking metabolic dysfunction with hypertension [21].

Inflammation also appears to play a contributory role in the pathogenesis of hypertension. Elevated hs-CRP levels were significantly associated with hypertension in the present study, indicating the presence of low-grade chronic inflammation. Inflammatory mediators exacerbate insulin resistance, impair vascular reactivity, and accelerate arterial stiffness, thereby increasing blood pressure [22,23].

Several recent large-scale studies in 2025 have reinforced the **relationship between the TyG index and hypertension.** A cross-sectional analysis involving 4,028 subjects demonstrated that individuals in the highest TyG quartile had significantly higher odds of hypertension compared with those in the lowest quartile, regardless of whether hypertension was defined by 140/90 mmHg or the newer 130/80 mmHg criteria (OR 2.87 and 2.93, respectively), indicating a clear dose–response relationship between TyG index and blood pressure elevation [23]. Similarly, in a national study of 10,405 middle-aged and older adults, the TyG index was found to mediate a significant portion of the association between exposure to air pollution and elevated hypertension risk, suggesting that insulin resistance may be a key biological pathway linking environmental stressors to blood pressure regulation [24]. Furthermore, research among patients with established hypertension has shown that higher TyG index levels are not only associated with the *presence* of coronary heart disease but also with its *severity*, with those in the highest TyG tertile exhibiting nearly fourfold greater odds of CHD and significantly higher Gensini scores compared with the lowest tertile [25].

These recent findings align closely with the present study's results, in which a higher TyG index was independently associated with newly diagnosed hypertension after adjustment for age, BMI, and diabetes mellitus. The consistency between our findings and those of other 2025 studies supports the hypothesis that the TyG index is a robust marker of metabolic dysfunction and cardiovascular risk across diverse populations. Furthermore, by demonstrating that elevated TyG index predicts both hypertension and coronary disease severity, these studies underscore the potential value of TyG as a **cost-effective screening tool** for early cardiometabolic risk stratification in routine clinical practice.

Overall, the present study demonstrates that the TyG index integrates multiple metabolic abnormalities—including insulin resistance, dyslipidaemia, obesity, hyperglycaemia, and inflammation—that collectively contribute to the development of hypertension. Given that the TyG index is derived from routinely available laboratory parameters, it represents a practical and cost-effective tool for early identification of individuals at high cardiometabolic risk, especially in resource-limited settings [26,27].

CONCLUSION

The present study demonstrates a significant and independent association between the triglyceride–glucose (TyG) index and newly diagnosed hypertension.A higher TyG index reflects underlying insulin resistance, which plays a pivotal role in the development of hypertension.The TyG index remained an independent predictor of hypertension even after adjusting for age, BMI, and diabetes mellitus.Obesity, central adiposity, dysglycaemia, dyslipidaemia, and systemic inflammation were commonly observed among hypertensive individuals, highlighting the multifactorial metabolic basis of hypertension.Given its simplicity, low cost, and reliance on routinely available laboratory parameters, the TyG index may serve as a practical screening tool for early identification of individuals at high cardiometabolic risk.Early detection of insulin resistance using the TyG index may facilitate timely preventive and therapeutic interventions, thereby reducing long-term cardiovascular complications.

LIMITATIONS OF THE STUDY

1. The cross-sectional design limits the ability to establish a causal relationship between TyG index and hypertension.
2. The study was conducted at a single tertiary care centre, which may limit generalizability to the wider population.

3. A large proportion of participants had coexisting diabetes mellitus, which may have influenced the strength of association.
4. Direct measures of insulin resistance such as HOMA-IR or hyperinsulinaemic–euglycaemic clamp were not used for comparison.
5. Long-term follow-up was not performed to assess the predictive value of TyG index for incident hypertension or cardiovascular outcomes.

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REFERENCES

1. World Health Organization. Hypertension. Geneva: WHO; 2023.
2. Gupta R, Gaur K, Ram CVS. Emerging trends in hypertension epidemiology in India. J Hum Hypertens. 2019;33(8):575–587.
3. Reaven GM. Insulin resistance, hypertension, and coronary heart disease. J Clin Hypertens. 2011;13(4):269–273.
4. Ferrannini E, Cushman WC. Diabetes and hypertension: the bad companions. Lancet. 2012;380(9841):601–610.
5. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Metab Syndr Relat Disord. 2010;8(4):299–304.
6. Simental-Mendía LE, Guerrero-Romero F. The triglyceride–glucose index is a useful biomarker to identify insulin resistance. J Clin Endocrinol Metab. 2018;103(2):389–395.
7. Lee SB, Ahn CW, Lee BK, et al. Association between triglyceride–glucose index and arterial stiffness in Korean adults. Cardiovasc Diabetol. 2018;17(1):41.
8. Sánchez-Íñigo L, Navarro-González D, Fernández-Montero A, et al. The TyG index as a predictor of incident hypertension. Hypertension. 2016;67(2):283–289.
9. Zhang M, Wang B, Liu Y, et al. Triglyceride–glucose index and the risk of hypertension. J Hypertens. 2021;39(3):494–501.
10. Franklin SS, Gustin W, Wong ND, et al. Hemodynamic patterns of age-related changes in blood pressure. Circulation. 1997;96(1):308–315.
11. Safar ME, London GM. Arterial and venous compliance in sustained essential hypertension. Hypertension. 1987;10(2):133–139.
12. Chow CK, Teo KK, Rangarajan S, et al. Prevalence, awareness, treatment, and control of hypertension. Lancet. 2013;382(9898):959–968.
13. Hall JE, do Carmo JM, da Silva AA, et al. Obesity-induced hypertension. Circ Res. 2015;116(6):991–1006.
14. Landsberg L, Aronne LJ, Beilin LJ, et al. Obesity-related hypertension: pathogenesis. Hypertension. 2013;61(2):257–265.
15. Kotsis V, Stabouli S, Papakatsika S, et al. Mechanisms of obesity-induced hypertension. Hypertens Res. 2010;33(5):386–393.
16. Virdis A, Giannarelli C, Neves MF, et al. Smoking and hypertension. Curr Pharm Des. 2010;16(23):2518–2525.
17. Briasoulis A, Agarwal V, Messerli FH. Alcohol consumption and the risk of hypertension. J Clin Hypertens. 2012;14(11):792–798.
18. De Boer IH, Bangalore S, Benetos A, et al. Diabetes and hypertension: a position statement. Diabetes Care. 2017;40(9):1273–1284.
19. Schram MT, Stehouwer CD. Vascular complications of diabetes. Diabetologia. 2015;58(1):1–3.
20. Sarwar N, Danesh J, Eiriksdottir G, et al. Triglycerides and cardiovascular disease. Circulation. 2007;115(4):450–458.
21. Low S, Khoo KCJ, Irwan B, et al. The role of triglyceride glucose index in insulin resistance. Clin Chim Acta. 2018;485:312–318.
22. Ridker PM. Inflammation, C-reactive protein, and cardiovascular disease. Circulation. 2003;107(3):363–369.
23. Libby P. Inflammation in atherosclerosis. Nature. 2002;420(6917):868–874.
24. Hao H, Geng H, Ma M. *Association Between Triglyceride–Glucose Index and Hypertension According to Different Diagnostic Criteria.* **Int J Gen Med**. 2025; eCollection 2025: DOI:10.2147/IJGM.S499194.
25. Li X, Zhang Y, et al. *Triglyceride–Glucose Index Mediated the Relationship Between Air Pollution and Hypertension in Middle-Aged and Older Adults.* **J Gerontol A Biol Sci Med Sci**. 2025;80(7):glaf114. DOI:10.1093/gerona/glaf114.
26. Wu L, Bao X, Xu J, et al. *The Triglyceride–Glucose Index Positively Associates with the Prevalence and Severity of Coronary Heart Disease in Patients with Hypertension.* **Sci Rep**. 2025;15(1):19571. DOI:10.1038/s41598-025-03948-y.
27. Mazidi M, Kengne AP, Katsiki N, et al. Triglyceride–glucose index and cardiovascular mortality. Atherosclerosis. 2020;297:51–57.
28. Wang A, Tian X, Zuo Y, et al. Change in triglyceride–glucose index and incident hypertension. Cardiovasc Diabetol. 2022;21(1):65.