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Assessment of Insulin Resistance in Lean versus Obese Indian Adults with Type 2 Diabetes Mellitus

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Abstract:

Background: Insulin resistance (IR) is a central pathophysiological feature of type 2 diabetes mellitus (T2DM), and its severity is influenced by adiposity. Asian Indians exhibit higher visceral adiposity and metabolic susceptibility even at lower body mass index (BMI). This study aimed to compare insulin resistance between lean and obese Indian adults with T2DM and to identify clinical and biochemical predictors of IR.

Methods: A cross-sectional study was conducted among 110 adults with T2DM, divided into lean (BMI <25 kg/m², n=55) and obese (BMI ⩾25 kg/m², n=55) groups. Anthropometric measurements, blood pressure, fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), fasting insulin, and lipid profiles were recorded. Insulin resistance was assessed using the homeostatic model assessment of insulin resistance (HOMA-IR). Correlation and multivariable linear regression analyses were performed to determine predictors of HOMA-IR.

Results: Obese T2DM subjects exhibited significantly higher BMI (29.2 ± 2.8 vs. 22.4 ± 1.4 kg/m², p<0.001), waist circumference (103.5 ± 9.4 vs. 87.2 ± 7.5 cm, p<0.001), and systolic/diastolic blood pressures compared to lean subjects. Fasting insulin (20.4 ± 7.5 vs. 13.2 ± 6.0 µU/mL, p<0.001) and HOMA-IR (7.4 ± 2.3 vs. 4.6 ± 1.7, p<0.001) were significantly higher in obese participants. A greater proportion of obese subjects exhibited HOMA-IR >2.5 (92.7% vs. 72.7%, p=0.005). HOMA-IR correlated positively with BMI (r=0.471), waist circumference (r=0.505), waist–hip ratio (r=0.411), HbA1c (r=0.322), and triglycerides (r=0.378), and inversely with HDL-C (r=−0.209). Multivariable regression identified waist circumference (β=0.12, p=0.002), BMI (β=0.18, p=0.011), triglycerides (β=0.015, p=0.021), and HbA1c (β=0.25, p=0.023) as independent predictors of HOMA-IR.

Conclusion: Obese adults with T2DM exhibit significantly higher insulin resistance than lean counterparts, with central adiposity, BMI, dyslipidemia, and HbA1c as major determinants. These findings underscore the importance of targeting obesity and metabolic risk factors to mitigate insulin resistance and associated complications in Indian T2DM populations.

Keywords:

Type 2 diabetes mellitus, insulin resistance, HOMA-IR, obesity, lean diabetics

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Introduction

Type 2 diabetes mellitus (T2DM) is a major global health concern, with an estimated 537 million adults affected worldwide in 2021, a number projected to rise to 783 million by 2045 [IDF, 2021]. India alone accounts for more than 77 million adults with diabetes, making it the "diabetes capital" of the world [1]. T2DM is characterized by chronic hyperglycemia resulting from a combination of insulin resistance (IR) and progressive β-cell dysfunction [2]. While obesity has long been established as the predominant risk factor for insulin resistance and T2DM in Western populations, a significant proportion of patients in South and East Asia develop T2DM despite having a normal or near-normal body mass index (BMI), a phenotype commonly referred to as "lean T2DM" [3,4].

Insulin resistance plays a central role in the pathogenesis of T2DM. It is defined as a diminished ability of insulin to promote glucose uptake in skeletal muscle and adipose tissue and to suppress hepatic glucose production [5]. In obese individuals, IR is strongly linked to excess visceral adiposity, chronic low-grade inflammation, ectopic fat accumulation in the liver and pancreas, and dysregulated adipokine secretion [6]. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) is one of the widely used surrogate indices to quantify IR in clinical and epidemiological studies [7]. Higher HOMA-IR values are consistently reported in obese diabetics compared to lean diabetics, but the degree of difference varies across populations and studies [8].

The phenotype of lean T2DM is particularly common in Asian countries. Studies indicate that up to 20–30% of Asian Indian patients with T2DM have a BMI <25 kg/m², yet they exhibit features of insulin resistance comparable to or greater than those seen in obese individuals [9]. This paradox has been attributed to higher body fat percentage, increased visceral fat, and reduced muscle mass in Asians even at lower BMI thresholds [10]. Furthermore, lean T2DM patients often display earlier β-cell dysfunction and a greater likelihood of requiring insulin therapy within 5 years of diagnosis compared to their obese counterparts [11]. Some evidence also suggests that lean diabetics have poorer glycemic control and a higher risk of complications due to delayed recognition of the condition [12].

Despite these observations, there remains a lack of clarity regarding the extent and determinants of insulin resistance among lean versus obese adults with T2DM. Few studies have systematically compared these groups using standardized methods, and the findings have been inconsistent. For example, some reports suggest that obese diabetics demonstrate markedly higher IR due to adiposity-driven mechanisms [13], whereas others have shown that lean diabetics exhibit comparable or even greater IR, likely due to ectopic fat deposition and sarcopenia [14]. These inconsistencies underscore the need for more comparative data, particularly in the Indian context, where lean T2DM is highly prevalent.

Therefore, the present study aimed to compare insulin resistance between lean and obese adults with T2DM using validated indices, thereby contributing to a better understanding of the heterogeneity of T2DM. This knowledge may aid in tailoring individualized therapeutic strategies—emphasizing weight reduction and insulin sensitizers in obese diabetics, and β-cell preservation in lean diabetics.

Material and methods

Study Design and Setting

This hospital-based, cross-sectional comparative study was conducted in the Department of Medicine at a tertiary care teaching hospital located in North India. The study was carried out over a period of twelve months, from March 2024 to March 2025. Ethical clearance was obtained from the Institutional Ethics Committee and all participants provided written informed consent before enrolment.

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Study Population

Adult patients with a confirmed diagnosis of type 2 diabetes mellitus (T2DM) were recruited from the outpatient and inpatient services of the hospital. The diagnosis of T2DM was established based on the American Diabetes Association (ADA) 2022 criteria, which included fasting plasma glucose ⩾126 mg/dL, 2-hour plasma glucose ⩾200 mg/dL during an oral glucose tolerance test, HbA1c ⩾6.5%, or random plasma glucose ⩾ 200 mg/dL in a patient with classical symptoms of hyperglycemia. For the purpose of comparison, participants were stratified into two groups on the basis of body mass index (BMI) using Asia-Pacific guidelines: lean diabetics (BMI <25 kg/m²) and obese diabetics (BMI ⩾25 kg/m²).

Inclusion and Exclusion Criteria

Participants aged between 18 and 65 years with a duration of diabetes of at least one year and receiving stable antidiabetic therapy for the preceding three months were eligible for inclusion. Patients with type 1 diabetes mellitus, secondary forms of diabetes, chronic kidney disease (eGFR <60 ml/min/1.73 m²), chronic liver disease, thyroid or adrenal disorders, active infections, or those on medications known to influence insulin sensitivity such as corticosteroids and thiazolidinediones were excluded. Pregnant and lactating women were also not included in the study.

Sample Size

The sample size was calculated using the formula for comparison of two means, with assumptions drawn from previous literature that demonstrated a mean difference of 1.0 in HOMA-IR values between lean and obese diabetic groups, a standard deviation of 1.5, a power of 80%, and an alpha error of 5%. The minimum sample size was estimated at 45 subjects in each group [8]. To compensate for potential dropouts or incomplete data, 55 subjects were recruited per group, giving a total sample of 110 participants.

Clinical and Anthropometric Assessment

A detailed history regarding demographic characteristics, duration of diabetes, family history, comorbidities, and treatment modalities was obtained through structured interviews. Physical examination included measurement of height using a wall-mounted stadiometer and weight using a calibrated digital weighing scale. Body mass index was calculated as weight in kilograms divided by height in meters squared (kg/m²). Waist circumference was measured at the midpoint between the lower margin of the last rib and the iliac crest using a non-stretchable tape, while hip circumference was measured at the widest portion of the buttocks. Waist–hip ratio was subsequently derived. Blood pressure was recorded in the sitting position using a mercury sphygmomanometer after five minutes of rest, and the mean of two readings was taken for analysis.

Laboratory Investigations

All participants underwent blood sampling after an overnight fast of 8–10 hours. Fasting plasma glucose (FPG) was measured by the glucose oxidase–peroxidase method, and fasting insulin levels were determined by chemiluminescent immunoassay. Glycated hemoglobin (HbA1c) was analyzed using high-performance liquid chromatography (HPLC). Lipid profile including total cholesterol, high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), and triglycerides was assessed by enzymatic colorimetric methods. Renal function tests (serum creatinine, blood urea nitrogen) and liver function tests (AST, ALT, total bilirubin) were also performed to exclude confounding systemic diseases.

Assessment of Insulin Resistance

The degree of insulin resistance was quantified using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated from fasting insulin and fasting glucose levels using the formula:

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HOMA-IR=Fasting insulin (μU/mL)×Fasting glucose (mg/dL)/405

A higher HOMA-IR value indicated greater insulin resistance. For descriptive purposes, a HOMA-IR threshold of >2.5 was considered suggestive of significant insulin resistance in accordance with earlier studies conducted in Asian populations.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Normality of data was tested using the Kolmogorov–Smirnov test. Differences in continuous variables between lean and obese diabetic groups were compared using the independent samples t-test for normally distributed data and the Mann–Whitney U test for skewed data. Categorical variables were analyzed using the chi-square test or Fisher's exact test, as appropriate. Correlation between HOMA-IR and anthropometric indices (BMI, waist circumference, waist–hip ratio) was assessed using Pearson correlation coefficients. A p-value <0.05 was considered statistically significant.

Results

The mean age of participants was comparable between lean and obese T2DM groups (52.4 ± 8.9 vs. 53.1 ± 8.3 years, p=0.625). The sex distribution was also similar, with males accounting for 58.2% in the lean group and 63.6% in the obese group (p=0.556). The mean duration of diabetes did not differ significantly (6.8 ± 4.1 vs. 7.2 ± 4.3 years, p=0.578). A family history of diabetes was present in 36.4% of lean and 43.6% of obese subjects (p=0.492). However, hypertension was significantly more prevalent in the obese group (50.9% vs. 30.9%, p=0.044). In terms of treatment, most participants were on OHA only, but a higher proportion of obese diabetics required combined OHA and insulin therapy compared to lean diabetics (36.3% vs. 21.8%, p=0.089) (Table 1).

Table 1. Baseline demographic and clinical characteristics of study participants.

Variable	Lean T2DM (n=55)	Obese T2DM (n=55)	p-value
	Frequency (%)/mean ± SD		
Age (years)	52.4 ± 8.9	53.1 ± 8.3	0.625
Gender			
Male	32 (58.2%)	35 (63.6%)	0.556
Female	21 (41.8%)	20 (36.4%)	
Duration of diabetes (years)	6.8 ± 4.1	7.2 ± 4.3	0.578
Family history of diabetes	20 (36.4%)	24 (43.6%)	0.492
Hypertension	17 (30.9%)	28 (50.9%)	0.044
Treatment modality			
OHA only	37 (67.3%)	31 (56.4%)	0.224
Insulin only	6 (10.9%)	4 (7.3%)	0.551
OHA + Insulin	12 (21.8%)	20 (36.3%)	0.089

Anthropometric and blood pressure measurements are summarized in Table 2. Obese T2DM subjects had significantly higher body weight (78.3 ± 8.5 vs. 60.5 ± 6.1 kg, p<0.001), BMI (29.2 ± 2.8 vs. 22.4 ±

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1.4 kg/m², p<0.001), and waist circumference (103.5 ± 9.4 vs. 87.2 ± 7.5 cm, p<0.001). Waist–hip ratio was also elevated in the obese group (0.97 ± 0.06 vs. 0.88 ± 0.05, p<0.001). Systolic (135.4 ± 14.2 vs. 127.6 ± 12.1 mmHg, p=0.011) and diastolic blood pressure (83.1 ± 8.4 vs. 78.9 ± 7.2 mmHg, p=0.015) were significantly higher in obese subjects, reflecting greater cardiovascular risk burden (Table 2).

Table 2. Anthropometric and hemodynamic parameters.

Variable	Lean T2DM (n=55)	Obese T2DM (n=55)	p-value
	Frequency (%)/mean ± SD		
Weight (kg)	60.5 ± 6.1	78.3 ± 8.5	<0.001
BMI (kg/m²)	22.4 ± 1.4	29.2 ± 2.8	<0.001
Waist circumference (cm)	87.2 ± 7.5	103.5 ± 9.4	<0.001
Waist–Hip ratio	0.88 ± 0.05	0.97 ± 0.06	<0.001
SBP (mmHg)	127.6 ± 12.1	135.4 ± 14.2	0.011
DBP (mmHg)	78.9 ± 7.2	83.1 ± 8.4	0.015

Fasting plasma glucose was marginally higher in obese diabetics compared to lean diabetics (154.3 ± 32.5 vs. 142.7 ± 28.7 mg/dL, p=0.056). Mean HbA1c levels were elevated in both groups, with a trend toward poorer glycemic control in the obese group (8.4 ± 1.3% vs. 8.0 ± 1.2%, p=0.078). Fasting insulin levels were significantly higher in obese participants (20.4 ± 7.5 vs. 13.2 ± 6.0 µU/mL, p<0.001), reflecting greater insulin resistance. Obese subjects also demonstrated a more atherogenic lipid profile, with higher total cholesterol (208.7 ± 36.3 vs. 186.2 ± 34.6 mg/dL, p=0.002), LDL-C (132.5 ± 32.6 vs. 114.5 ± 29.8 mg/dL, p=0.001), and triglycerides [210 (160–270) vs. 160 (120–210) mg/dL, p=0.002], alongside significantly lower HDL-C levels (35.5 ± 7.4 vs. 39.7 ± 8.3 mg/dL, p=0.011) (Table 3).

Table 3. Glycemic and lipid profile of study participants.

Variable	Lean T2DM (n=55)	Obese T2DM (n=55)	p-value
	median (IQR)/mean ± SD		
FPG (mg/dL)	142.7 ± 28.7	154.3 ± 32.5	0.056
HbA1c (%)	8.0 ± 1.2	8.4 ± 1.3	0.078
Fasting insulin (µU/mL)	13.2 ± 6.0	20.4 ± 7.5	<0.001
Total cholesterol (mg/dL)	186.2 ± 34.6	208.7 ± 36.3	0.002
LDL-C (mg/dL)	114.5 ± 29.8	132.5 ± 32.6	0.001
HDL-C (mg/dL)	39.7 ± 8.3	35.5 ± 7.4	0.011
Triglycerides (mg/dL)	160 (120–210)	210 (160–270)	0.002

The mean HOMA-IR score was substantially higher in obese subjects compared to lean subjects (7.4 ± 2.3 vs. 4.6 ± 1.7, p<0.001). Additionally, a greater proportion of obese diabetics had HOMA-IR >2.5, indicating insulin resistance (92.7% vs. 72.7%, p=0.005). This confirms the stronger degree of insulin resistance among obese individuals with T2DM (Table 4).

Table 4. Insulin resistance indices in lean and obese T2DM.

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Measure	Lean T2DM (n=55)	Obese T2DM (n=55)	p-value
	Frequency (%)/mean ± SD		
HOMA-IR	4.6 ± 1.7	7.4 ± 2.3	<0.001
% with HOMA-IR > 2.5	40 (72.7%)	51 (92.7%)	0.005

Insulin resistance correlated positively with BMI (r=0.471, p<0.001), waist circumference (r=0.505, p<0.001), waist–hip ratio (r=0.411, p<0.001), HbA1c (r=0.322, p=0.002), and triglyceride levels (r=0.378, p<0.001). A significant inverse correlation was observed with HDL-C (r=−0.209, p=0.004). These results emphasize the role of central adiposity and dyslipidemia as contributors to insulin resistance (Table 5).

Table 5. Correlation of insulin resistance (HOMA-IR) with clinical and biochemical parameters.

Variable	Pearson 'r'	p-value
BMI	0.471	<0.001
Waist circumference	0.505	<0.001
Waist–Hip ratio	0.411	<0.001
HbA1c	0.322	0.002
Triglycerides	0.378	<0.001
HDL-C	−0.209	0.004

Waist circumference emerged as the strongest independent predictor (β=0.12, p=0.002), followed by triglyceride levels (β=0.015, p=0.021) and BMI (β=0.18, p=0.011). HbA1c also demonstrated a modest but significant association (β=0.25, p=0.023). HDL-C was inversely associated with HOMA-IR (β=−0.05, p=0.034). Age did not show any significant effect (p=0.427). The overall model explained 42% of the variance in HOMA-IR (Adjusted R^2=0.42, p<0.001) (Table 6).

Table 6. Multiple linear regression analysis of predictors of insulin resistance.

Variable	β coefficient (SE)	Standardized β	p-value
BMI (kg/m²)	0.18 (0.07)	0.21	0.011
Waist circumference (cm)	0.12 (0.04)	0.29	0.002
HbA1c (%)	0.25 (0.11)	0.17	0.023
Triglycerides (mg/dL)	0.015 (0.006)	0.22	0.021
HDL-C (mg/dL)	−0.05 (0.02)	−0.18	0.034
Age (years)	0.02 (0.03)	0.06	0.427

Discussion

This study aimed to compare insulin resistance between lean and obese adults with type 2 diabetes mellitus (T2DM) and to identify clinical and biochemical predictors of insulin resistance in an Indian cohort. Our results demonstrate a significantly higher degree of insulin resistance among obese diabetics compared to lean diabetics, highlighting the impact of obesity and central adiposity on metabolic derangements.

Demographics and baseline characteristics were broadly comparable between the two groups with

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respect to age, sex distribution, and duration of diabetes, which minimizes confounding by these factors. Hypertension was significantly more prevalent in the obese group (50.9% vs. 30.9%, p=0.044), consistent with the well-established association between obesity, insulin resistance, and increased cardiovascular risk [15]. Treatment patterns reflected common Indian clinical practice, with the majority of both groups on oral hypoglycemic agents (OHA only) and a higher proportion of obese patients requiring combined OHA and insulin therapy (36.3% vs. 21.8%, p=0.089), likely due to higher insulin resistance and associated glycemic challenges. These findings align with previous hospital-based Indian studies by Mathur et al., and Varghese et al., where obese diabetics often require combination therapy earlier in the course of disease [16,17].

Anthropometric and hemodynamic measurements revealed significant differences between lean and obese diabetics. Obese subjects exhibited markedly higher BMI, waist circumference, and waist–hip ratio, indicating greater overall and central adiposity. Elevated systolic and diastolic blood pressures in the obese group further support the coexistence of metabolic syndrome features. These observations corroborate Indian population data by Gupta et al., demonstrating that central obesity is a key driver of insulin resistance and cardiovascular risk, even at relatively lower BMI values compared to Western populations [18].

Biochemical profiles revealed a trend toward higher fasting plasma glucose and HbA1c in obese diabetics, although not statistically significant. However, fasting insulin levels were significantly higher in the obese group (20.4 ± 7.5 vs. 13.2 ± 6.0 µU/mL, p<0.001), suggesting pronounced insulin resistance. Lipid analysis showed an atherogenic profile in obese patients, with higher total cholesterol, LDL-C, triglycerides, and lower HDL-C, reflecting the typical “diabetic dyslipidemia” observed in Indian T2DM populations [19,20]. These metabolic derangements likely contribute to the higher HOMA-IR observed in obese subjects.

Insulin resistance indices demonstrated a significantly higher mean HOMA-IR in obese diabetics (7.4 ± 2.3) compared to lean diabetics (4.6 ± 1.7, p<0.001). Furthermore, 92.7% of obese subjects exhibited HOMA-IR >2.5 compared to 72.7% of lean subjects (p=0.005), underscoring the strong influence of obesity on insulin resistance. These findings are consistent with previous studies in India by Uppal et al., and Bhor et al., which report HOMA-IR values ranging from 4–5 in lean diabetics and 6–8 in obese diabetics [21,22]. International study by Song et al., also support this pattern, confirming that adiposity, particularly central obesity, is a principal determinant of insulin resistance [23].

Correlation analysis revealed positive associations between HOMA-IR and BMI (r=0.471), waist circumference (r=0.505), waist–hip ratio (r=0.411), HbA1c (r=0.322), and triglycerides (r=0.378), while HDL-C correlated inversely (r=−0.209). These results highlight the dual contribution of both general and central adiposity to insulin resistance, as well as the interplay with glycemic control and dyslipidemia. Similar correlations have been reported in other Indian studies by Garg et al., and Faraz et al., which indicate that waist circumference is often a stronger predictor of insulin resistance than BMI alone [24,25].

Multivariable regression analysis identified waist circumference as the strongest independent predictor of HOMA-IR (β=0.12, p=0.002), followed by BMI, triglycerides, and HbA1c. HDL-C demonstrated a negative association with HOMA-IR. Age was not a significant predictor. The adjusted R^2 of 0.42 indicates that these factors collectively explain a substantial proportion of variability in insulin resistance. This aligns with the pathophysiological understanding that visceral adiposity contributes to insulin resistance via increased free fatty acid flux, chronic low-grade inflammation, and adipokine dysregulation [26]. Elevated triglycerides and low HDL-C further exacerbate insulin resistance by

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promoting lipotoxicity and endothelial dysfunction [27].

Collectively, these findings emphasize that in Indian adults with T2DM, obesity—particularly central adiposity—is the primary driver of insulin resistance. Lean diabetics, although relatively less insulin resistant, still exhibit significant HOMA-IR elevation, reflecting the unique "Asian Indian phenotype" characterized by higher visceral fat, impaired beta-cell function, and metabolic susceptibility even at lower BMI [28]. The study underscores the need for targeted interventions focusing on weight reduction, central obesity management, and metabolic optimization in both lean and obese T2DM patients.

Limitations

This study provides a detailed, comparative evaluation of insulin resistance using HOMA-IR in an Indian population while controlling for age, sex, and diabetes duration. Limitations include the cross-sectional design, relatively small sample size, and lack of direct measures of visceral adiposity such as MRI or CT-based fat quantification.

Conclusion

Obese adults with type 2 diabetes mellitus exhibit significantly higher insulin resistance compared to lean counterparts, as reflected by elevated HOMA-IR and fasting insulin levels. Central adiposity, BMI, dyslipidemia, and HbA1c are independent predictors of insulin resistance, highlighting the metabolic burden associated with obesity. These findings emphasize the importance of early identification and targeted management of obesity and associated metabolic risk factors to improve glycemic control and reduce long-term cardiovascular and microvascular complications in Indian T2DM populations.

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