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ORIGINAL RESEARCH ARTICLE

Assessment of Insulin Resistance and Pancreatic β-Cell Function Using Homeostatic Model Assessment Indices in Asian Indian Patients with Type 2 Diabetes Mellitus: A Case-Control Study

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Abstract

Type 2 diabetes mellitus (T2DM) is characterised by the dual pathophysiological abnormalities of insulin resistance and progressive pancreatic β-cell dysfunction. Asian Indians exhibit a distinctive metabolic phenotype characterised by increased central adiposity and heightened insulin resistance at lower body mass index (BMI) values compared with Western populations, contributing to disproportionately high susceptibility to T2DM. The Homeostatic Model Assessment (HOMA) provides a simple and validated method for quantifying insulin resistance (HOMA-IR) and β-cell secretory capacity (HOMA-β) from fasting glucose and insulin concentrations. The present hospital-based case-control study included 40 patients with T2DM and 40 age- and sex-matched non-diabetic controls recruited from a tertiary care centre in North India. Anthropometric measurements and biochemical parameters including fasting plasma glucose, fasting serum insulin, glycated haemoglobin (HbA1c), HOMA-IR, and HOMA-β were assessed and compared between groups. Diabetic subjects demonstrated significantly higher waist circumference (89.18±7.28 vs 81.50±8.34 cm, p=0.001), fasting plasma glucose (173.86±69.85 vs 92.56±16.80 mg/dL, p<0.001), fasting insulin (12.35±8.98 vs 7.33±4.98 mU/L, p=0.01), HbA1c (8.05±1.43 vs 5.12±0.55%, p=0.003), and HOMA-IR (5.20±3.87 vs 1.70±1.22, p=0.001) compared with non-diabetic controls. HOMA-β was significantly lower in diabetic subjects (53.36±51.34 vs 115.34±101.40, p=0.003), indicating impaired β-cell secretory function. BMI was higher in diabetic subjects, achieving borderline significance (25.28±4.14 vs 23.37±3.52 kg/m^2, p=0.05). These findings confirm the dual contribution of insulin resistance and β-cell dysfunction to T2DM pathogenesis in Asian Indians and support the utility of HOMA indices as practical, cost-effective tools for metabolic risk assessment in clinical and epidemiological settings.

Key words: Type 2 diabetes mellitus, insulin resistance, HOMA-IR, HOMA-β, β-cell dysfunction, Asian Indians, fasting insulin, glycated haemoglobin

1 | INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent metabolic disorders globally and represents a major public health burden, particularly in low- and middle-income countries. According to the International Diabetes Federation Atlas 2021, approximately 537 million adults worldwide are currently living with diabetes, and this number is projected to reach 783 million by 2045. (1) India bears a disproportionate share of this burden, harbouring over 74 million individuals with T2DM, and the disease onset in Asian Indians occurs nearly a decade earlier than in many Western populations. (1, 2)

The pathogenesis of T2DM involves two fundamen-

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tal and interrelated metabolic abnormalities: insulin resistance in peripheral tissues and progressive dysfunction of pancreatic β-cells. (3) Insulin resistance refers to the diminished biological response of insulin-sensitive tissues — primarily skeletal muscle, liver, and adipose tissue — to circulating insulin. This impairment results in reduced peripheral glucose uptake, increased hepatic glucose production, and compensatory hyperinsulinaemia. (4) Skeletal muscle accounts for approximately 70–80% of insulin-mediated postprandial glucose disposal, and reduced glucose transporter type 4 (GLUT-4) expression and translocation in myocytes represents a central cellular mechanism of peripheral insulin resistance. (5)

To compensate for insulin resistance, pancreatic β-cells initially increase insulin synthesis and secretion, a phenomenon termed compensatory hyperinsulinaemia. However, chronic exposure to glucotoxicity, lipotoxicity, oxidative stress, endoplasmic reticulum stress, and inflammatory cytokines progressively impairs β-cell function and reduces β-cell mass, ultimately leading to absolute insulin deficiency and clinical T2DM. (6, 7) Studies involving Asian Indian populations have demonstrated that β-cell decline may begin years before the clinical onset of T2DM and may progress more rapidly than in European populations. (8)

Asian Indians possess a distinctive metabolic phenotype characterised by excess truncal and visceral adiposity, reduced lean muscle mass, lower adiponectin levels, heightened insulin resistance, and increased susceptibility to T2DM at lower BMI values compared with Caucasian populations. (9, 10) Epidemiological evidence consistently demonstrates that Asian Indians develop T2DM at BMI values 3–5 kg/m^2 lower than European counterparts, highlighting that body mass index alone inadequately captures metabolic risk in this population. (11) The concept of the ”thin-fat Indian” phenotype, wherein individuals exhibit low birth weight yet disproportionate truncal adiposity, further underscores the unique metabolic vulnerability of this ethnic group. (12)

Adipose tissue dysfunction, particularly visceral adiposity, plays a central role in mediating insulin resistance among Asian Indians. Visceral adipose tissue releases excess free fatty acids and pro-inflammatory adipokines including tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and resistin into the portal circulation, impairing hepatic insulin signalling and promoting gluconeogenesis. (13) Simultaneously, reduced adiponectin secretion — an anti-inflammatory, insulin-sensitising adipokine — contributes to systemic insulin resistance. Asian Indians exhibit constitutively lower adiponectin levels even at lower BMI values, further exacerbating their metabolic risk. (14)

Insulin resistance and β-cell dysfunction are quantified clinically using the Homeostatic Model Assessment (HOMA), originally described by Matthews et al. in 1985. (15) HOMA-IR, calculated as fasting insulin (μU/mL) $\times$ fasting glucose (mmol/L) / 22.5, estimates the degree of insulin resistance from basal steady-state glucose and insulin concentrations. HOMA-β, calculated as 20 $\times$ fasting insulin (μU/mL) / [fasting glucose (mmol/L) $-$ 3.5], provides an estimate of pancreatic β-cell secretory capacity. HOMA indices are derived from a mathematical model of glucose-insulin homeostasis and have been extensively validated against the euglycaemic hyperinsulinaemic clamp technique, the gold standard for insulin resistance measurement. (16) The advantages of HOMA include low cost, minimal invasiveness, and suitability for large-scale epidemiological studies.

Despite the established importance of HOMA indices in diabetes research, limited data are available on the simultaneous assessment of insulin resistance and β-cell function in well-characterised Asian Indian cohorts, particularly from North India. Furthermore, the relationship between anthropometric indices — particularly waist circumference as a marker of central adiposity — and HOMA-derived metabolic parameters warrants evaluation in this high-risk population.

The present study therefore aimed to evaluate insulin resistance (HOMA-IR) and β-cell secretory function (HOMA-β) in Asian Indian patients with T2DM and compare these parameters with matched non-diabetic controls, and to examine the association between central adiposity and insulin resistance in this cohort.

2 | MATERIALS AND METHODS

Study Design

A hospital-based case-control study was conducted in the Departments of Biochemistry and Endocrinology, SMS Medical College and Attached Hospitals, Jaipur, Rajasthan, India.

3 | ETHICAL APPROVAL AND INFORMED CONSENT

The study protocol was approved by the Institutional Ethics Committee, SMS Medical College and Attached Hospitals, Jaipur (Ref. No. 470/MC/EC/2022). Written informed consent was obtained from all participants prior to enrolment. The study was conducted in accordance with the Declaration of Helsinki.

Study Population

The study enrolled 80 participants comprising 40 patients with T2DM (case group) and 40 non-diabetic individuals (control group).

Inclusion Criteria

Case group: Adult patients aged 18–65 years with established T2DM diagnosed according to the American Diabetes Association (ADA) 2022 criteria; willing to provide written informed consent.

Control group: Age- and sex-matched adults aged 18–65 years with fasting plasma glucose <100 mg/dL and no known history of diabetes mellitus or impaired fasting glucose; willing to provide written informed consent.

Exclusion Criteria

Patients were excluded if they had type 1 diabetes mellitus, gestational diabetes, secondary causes of diabetes, active infection or systemic inflammatory disease, malignancy, severe hepatic or renal dysfunction (serum creatinine >2.0 mg/dL), were pregnant or lactating, or were receiving medications known to affect insulin sensitivity including glucocorticoids, thiazolidinediones, or pioglitazone.

Anthropometric Measurements

Height was measured to the nearest 0.1 cm using a stadiometer with the subject standing erect without footwear. Body weight was recorded to the nearest 0.1 kg using a calibrated digital weighing scale. BMI was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the mid-point between the lower costal margin and the iliac crest using a non-elastic tape measure at the end of normal expiration. Hip circumference was measured at the widest point of the greater trochanters. Waist-to-hip ratio (WHR) was calculated accordingly. All measurements were performed in duplicate by a single trained observer.

Biochemical Investigations

Fasting venous blood samples were collected after a minimum overnight fast of eight hours. Fasting plasma glucose was estimated by the glucose oxidase-peroxidase method. Fasting serum insulin was measured by electrochemiluminescence immunoassay (ECLIA). Glycated haemoglobin (HbA1c) was determined using high-performance liquid chromatography (HPLC). Serum creatinine was estimated using the modified Jaffe's kinetic method. All assays were performed on validated automated analysers in the central laboratory of SMS Medical College.

Assessment of Insulin Resistance and β-Cell Function

Insulin resistance and β-cell secretory function were assessed using HOMA indices as described by Matthews et al.: (15)

HOMA-IR = [Fasting insulin (μU/mL) $\times$ Fasting glucose (mmol/L)] / 22.5

HOMA-β = [20 $\times$ Fasting insulin (μU/mL)] / [Fasting glucose (mmol/L) $-$ 3.5]

Fasting plasma glucose values in mg/dL were converted to mmol/L by dividing by 18.

Statistical Analysis

Data were analysed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation (SD). The Shapiro-Wilk test was used to assess the normality of distribution of continuous variables. Normally distributed variables were compared between groups using the independent samples Student's t-test. Non-normally distributed variables including HOMA-IR, HOMA-β, and fasting insulin were compared using the Mann-Whitney U test. Pearson correlation analysis was

performed to assess relationships between waist circumference, BMI, and HOMA indices within the diabetic group. Statistical significance was considered at $p<0.05$ (two-tailed).

4 | RESULTS

Anthropometric Characteristics

Anthropometric parameters of diabetic and non-diabetic subjects are presented in Table 1. Diabetic subjects demonstrated significantly higher waist circumference compared with non-diabetic controls (89.18±7.28 vs 81.50±8.34 cm, p=0.001). BMI was higher in the diabetic group, reaching borderline statistical significance (25.28±4.14 vs 23.37±3.52 kg/m^2, p=0.05). Height, weight, and WHR did not differ significantly between the groups Table 1

Table 1. Anthropometric parameters in diabetic and non-diabetic subjects (values expressed as mean ± SD)

Parameter	Diabetic (n=40)	Non-Diabetic (n=40)	p-value
Height (m)	1.62±0.09	1.64±0.09	0.33
Weight (kg)	67.36±15.33	63.32±11.17	0.25
BMI (kg/m^2)	25.28±4.14	23.37±3.52	0.05
Waist circumference (cm)	89.18±7.28	81.50±8.34	0.001
WHR	0.91±0.07	0.88±0.05	0.09

BMI: body mass index; WHR: waist-to-hip ratio; SD: standard deviation. Statistical significance at p<0.05.

Biochemical and HOMA Parameters

Biochemical and HOMA parameters are presented in Table 2 Fasting plasma glucose was markedly elevated in diabetic subjects compared with controls (173.86±69.85 vs 92.56±16.80 mg/dL, p<0.001). Fasting serum insulin was significantly higher in the diabetic group (12.35±8.98 vs 7.33±4.98 mU/L, p=0.01), as was HbA1c (8.05±1.43 vs 5.12±0.55%, p=0.003). HOMA-IR was significantly elevated in diabetic subjects (5.20±3.87 vs 1.70±1.22, p=0.001), indicating substantially greater insulin resistance. In contrast, HOMA-β was significantly lower in the diabetic group (53.36±51.34 vs 115.34±101.40, p=0.003), indicating impaired pancreatic β-cell secretory function. Serum creatinine did not differ significantly between groups, confirming comparable renal function Table 2

Table 2. Biochemical parameters and HOMA indices in diabetic and non-diabetic subjects (values expressed as mean ± SD)

Parameter	Diabetic (n=40)	Non-Diabetic (n=40)	p-value
Serum creatinine (mg/dL)	1.14±0.65	1.08±1.98	0.79
Fasting plasma glucose (mg/dL)	173.86±69.85	92.56±16.80	<0.001
Fasting serum insulin (mU/L)	12.35±8.98	7.33±4.98	0.01
HbA1c (%)	8.05±1.43	5.12±0.55	0.003
HOMA-IR	5.20±3.87	1.70±1.22	0.001
HOMA-β	53.36±51.34	115.34±101.40	0.003

HOMA-IR: homeostatic model assessment of insulin resistance; HOMA-β: homeostatic model assessment of β-cell function; HbA1c: glycated haemoglobin; SD: standard deviation. Statistical significance at p<0.05.

Graphical Representation of HOMA Indices

The comparative distribution of HOMA-IR and HOMA-β between diabetic and non-diabetic subjects is illustrated in Figure 1 HOMA-IR values were markedly higher in diabetic subjects, whilst HOMA-β values were substantially lower, visually demonstrating the inverse relationship between insulin resistance and β-cell function in T2DM. Figure 2

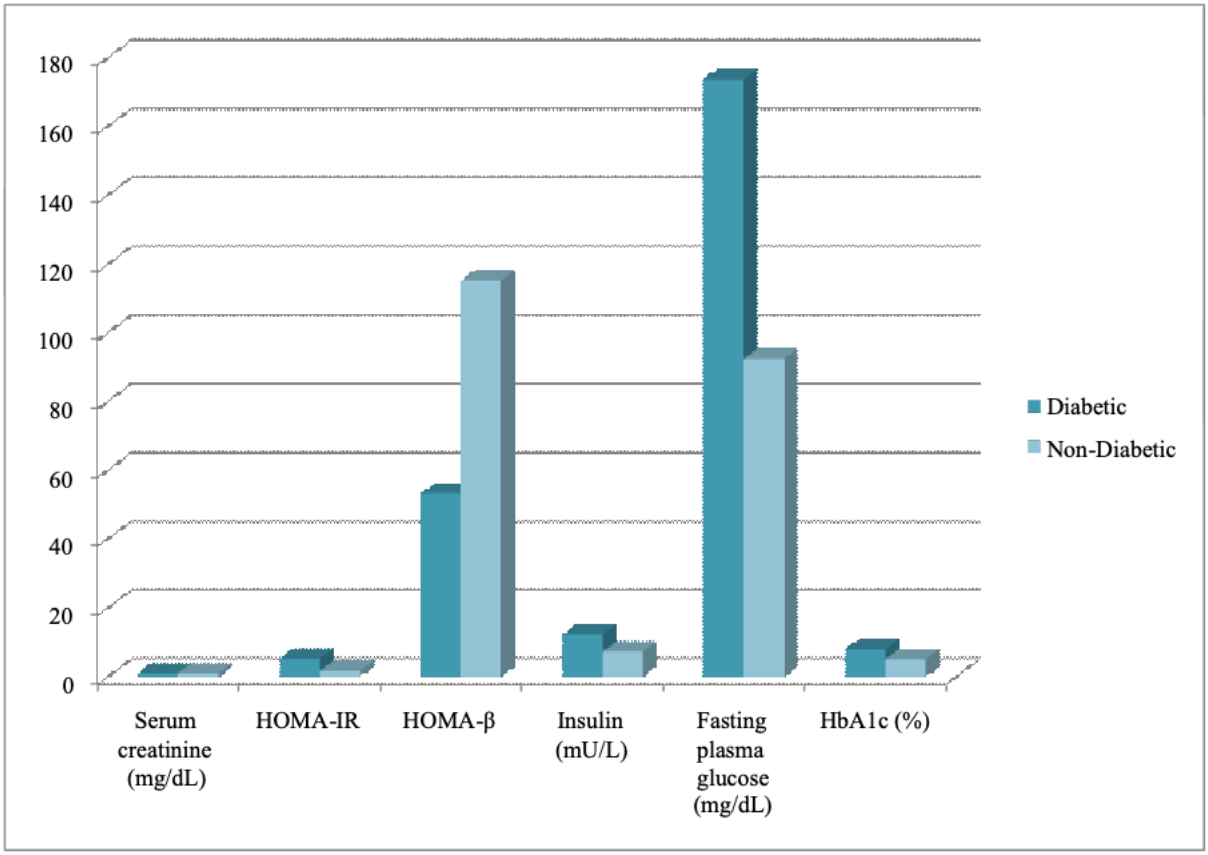

Fig. 1: Comparison of HOMA-IR and HOMA-β values between Asian Indian patients with type 2 diabetes mellitus (T2DM, n=40) and non-diabetic controls (n=40). Data expressed as mean ± standard deviation. *p<0.05 vs non-diabetic controls. HOMA-IR: homeostatic model assessment of insulin resistance; HOMA-β: homeostatic model assessment of β-cell function.

5 | DISCUSSION

The present case-control study evaluated insulin resistance and pancreatic β-cell function using HOMA indices in Asian Indian patients with T2DM and compared these parameters with matched non-diabetic controls. The principal findings were significantly elevated HOMA-IR and markedly reduced HOMA-β in diabetic subjects, confirming the simultaneous presence of enhanced insulin resistance and impaired β-cell secretory capacity in this population.

Insulin Resistance in T2DM

Insulin resistance is widely recognised as a cardinal and often the earliest metabolic abnormality in the natural history of T2DM. (3) The substantially elevated HOMA-IR values observed among diabetic subjects in the present study (5.20±3.87 vs 1.70±1.22 in controls, p=0.001) are consistent with the well-documented insulin resistance characteristic of Asian Indian T2DM. This finding aligns with numerous previous reports from Indian populations. Mohan and Deepa demonstrated markedly elevated insulin resistance indices in Asian Indian T2DM patients and emphasised the important contribution of adiposity and visceral fat accumulation to insulin resistance in this ethnic group. (17) Similarly, studies from the Chennai Urban Rural Epidemiology Study (CURES) cohort have consistently reported higher insulin resistance in urban Asian Indians compared with rural counterparts, implicating lifestyle and dietary factors as important modifiers. (18)

At the molecular level, insulin resistance in T2DM involves multiple interrelated defects. Impaired tyrosine phosphorylation of insulin receptor substrate proteins (IRS-1 and IRS-2), reduced activation of phosphatidylinositol 3-kinase (PI3K)/Akt signalling, and decreased GLUT-4 translocation to the cell surface collectively impair insulin-stimulated

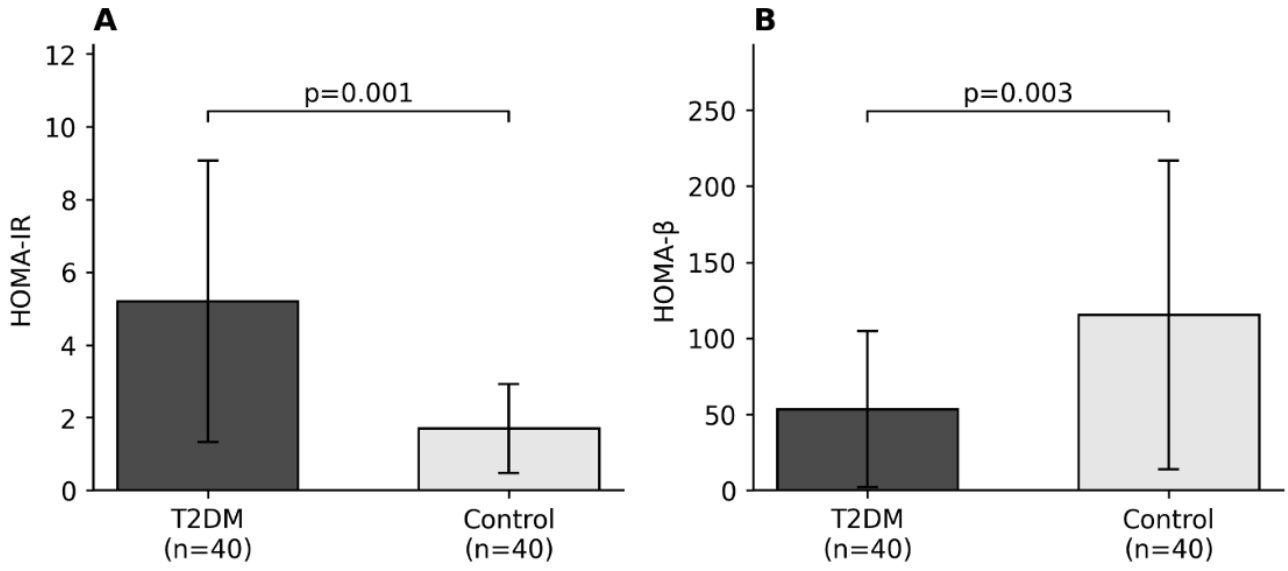

Fig. 2: Grouped bar chart: X-axis — HOMA-IR and HOMA-β; Y-axis — mean values; two bars per parameter representing Diabetic and Non-Diabetic groups; error bars representing ±SD; asterisks indicating p<0.05

glucose uptake in skeletal muscle and adipose tissue. (5) Activation of serine kinases including c-Jun N-terminal kinase (JNK) and inhibitor of nuclear factor-kappa B kinase (IKKβ) by inflammatory mediators further disrupts insulin signalling pathways. (19)

Excess visceral fat, highly prevalent among Asian Indians, releases large quantities of free fatty acids and pro-inflammatory cytokines including TNF-α and IL-6 into the portal circulation, further impairing hepatic insulin sensitivity and promoting gluconeogenesis. (13, 20)

The elevated fasting insulin concentrations observed in diabetic subjects in the present study (12.35±8.98 mU/L vs 7.33±4.98 mU/L in controls, p=0.01) reflect compensatory hyperinsulinaemia in response to insulin resistance. However, the concurrent reduc-tion in HOMA-β indicates that this compensatory mechanism is already failing in established T2DM, consistent with the concept of progressive β-cell exhaustion. (7)

Pancreatic β-Cell Dysfunction

The significantly lower HOMA-β values observed in diabetic subjects (53.36±51.34 vs 115.34±101.40 in controls, p=0.003) provide quantitative

evidence of substantially impaired pancreatic β-cell secretory capacity. β-cell dysfunction is now recognised as an early and essential contributor to T2DM pathogene-sis, and not merely a late consequence of disease. (6) Longitudinal studies including the UK Prospective Diabetes Study (UKPDS) demonstrated that β-cell function is already reduced by approximately 50%at the time of T2DM diagnosis and continues to decline progressively despite pharmacological treat-ment (21)

Multiple mechanisms contribute to β-cell failure in T2DM. Glucotoxicity — chronic exposure of β-cells to elevated glucose concentrations — impairs insulin gene transcription through oxidative stress and downregulation of transcription factors includ-ing pancreatic and duodenal homeobox-1 (Pdx-1) and musculoaponeurotic fibrosarcoma oncogene homologue A (MafA). (6) Lipotoxicity, arising from excess circulating free fatty acids and intracellu-lar ceramide accumulation, induces mitochondrial dysfunction, endoplasmic reticulum stress, and β-

cell apoptosis. (22) Chronic low-grade inflammation, mediated by IL-1β, TNF-α, and interferon-gamma, further impairs β-cell function and promotes β-cell death through NF-κB activation. (23)

Emerging evidence suggests that Asian Indians may exhibit accelerated β-cell decline compared with European populations. (8) This may partially reflect genetic predisposition, as studies have identified population-specific susceptibility variants in genes regulating β-cell development and function — including TCF7L2, KCNJ11, and ABCC8 — in Indian cohorts. (24)

The reduced β-cell reserve capacity in Asian Indians may also be compounded by early-life nutritional programming, low birth weight, and intrauterine growth restriction — factors collectively described under the "thrifty phenotype" hypothesis. (12)

Central Adiposity and Metabolic Risk

The significantly higher waist circumference observed in diabetic subjects (89.18±7.28 vs 81.50±8.34 cm, p=0.001) underscores the importance of central adiposity as a determinant of insulin resistance in Asian Indians. Waist circumference is a more sensitive anthropometric indicator of visceral fat accumulation than BMI and demonstrates stronger correlations with HOMA-IR and metabolic syndrome components. (25) The World Health Organization and South Asian expert panels have recommended lower waist circumference thresholds for Asian populations — ≥90 cm for men and ≥80 cm for women — reflecting their predisposition to visceral adiposity at lower levels of total adiposity. (26)

The relatively modest BMI difference between diabetic and non-diabetic subjects in the present study (25.28±4.14 vs 23.37±3.52 kg/m^2, p=0.05) yet markedly greater waist circumference difference supports the concept that central adiposity, rather than generalised obesity, represents the predominant anthropometric determinant of T2DM risk in Asian Indians. (11) This finding is consistent with the adipose tissue overflow hypothesis, which proposes that constitutionally limited subcutaneous adipose storage capacity in Asian Indians promotes preferential visceral fat accumulation, ectopic lipid deposition, and insulin resistance even at lower BMI levels. (27)

HbA1c as a Marker of Chronic Glycaemic Exposure

The substantially elevated HbA1c values in diabetic subjects (8.05±1.43 vs 5.12±0.55%, p=0.003) indicate poor long-term glycaemic control and confirm significant chronic hyperglycaemia. HbA1c represents the gold standard for assessment of glycaemic control over the preceding two to three months and is an important predictor of microvascular and macrovascular complications. (28) The mean HbA1c of 8.05% observed in the diabetic group is above the ADA treatment target of <7.0%, indicating suboptimal glycaemic control in this cohort and highlighting an important area for clinical intervention.

Utility of HOMA Indices in Clinical Practice

HOMA-IR and HOMA-β, as described by Matthews et al., (15) and subsequently updated as HOMA2, (16) provide practical surrogate measures of insulin resistance and β-cell function from simple fasting measurements. Numerous validation studies have demonstrated strong correlations between HOMA-IR and insulin-clamp-derived measures of insulin sensitivity ($r = -0.73$ to -0.87), (16) supporting the use of HOMA as a reliable surrogate in clinical and epidemiological settings. In resource-constrained settings such as India, HOMA indices offer significant practical advantages, enabling metabolic risk stratification without the need for expensive or invasive procedures.

The inverse relationship between HOMA-IR and HOMA-β observed in the present study — elevated insulin resistance accompanied by reduced β-cell function — is consistent with the hyperbolic relationship between insulin sensitivity and β-cell secretory capacity described by Kahn et al. (29) This relationship indicates that healthy β-cells compensate for insulin resistance by augmenting insulin secretion, and that breakdown of this compensatory axis is a defining pathological event in the transition from normoglycaemia to T2DM.

6 | LIMITATIONS

The present study has several limitations that should be acknowledged. The sample size of 40 subjects per group, whilst sufficient for the statistical comparisons performed, limits the generalisability of findings. The cross-sectional design precludes assessment of temporal changes in insulin resistance and

β-cell function or establishment of causal relationships. Insulin assay methodology, including inter-assay variability, may influence absolute HOMA values and limit direct comparison with studies using different assay platforms. Additional parameters including body fat percentage, visceral fat quantification by imaging, adipokine levels, and inflammatory biomarkers would have provided a more comprehensive characterisation of the metabolic phenotype. Furthermore, the study cohort was recruited from a single tertiary care centre in North India, and findings may not be fully representative of diverse Indian populations.

7 | CONCLUSION

Asian Indian patients with T2DM demonstrate significantly greater insulin resistance and substantially impaired pancreatic β-cell function compared with non-diabetic individuals, as evidenced by markedly elevated HOMA-IR and reduced HOMA-β values. Significantly higher waist circumference in the diabetic group, despite only borderline difference in BMI, highlights the pivotal role of central adiposity in mediating insulin resistance in this population. HbA1c values indicate suboptimal glycaemic control in the study cohort, underscoring the clinical need for early and effective metabolic management. HOMA indices represent practical, cost-effective, and minimally invasive tools for metabolic assessment and hold considerable utility for identifying high-risk individuals in resource-limited settings such as India. Future longitudinal studies incorporating larger cohorts, body composition analysis, and multi-omics characterisation of adipose tissue may further elucidate the molecular mechanisms underlying insulin resistance and β-cell dysfunction in Asian Indians and facilitate development of precision preventive and therapeutic strategies.

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Author Contributions

Neha Sharma: Conceptualisation, data collection, manuscript drafting and revision. Rati Mathur: Supervision, critical review, and intellectual content. Sandeep Mathur: Clinical expertise, endocrinological assessment, and manuscript approval. All authors read and approved the final manuscript.

Conflict of Interest

The authors declare no conflict of interest.

8 | FUNDING

No external funding was received for this study.

Ethics Approval

The study was approved by the Institutional Ethics Committee, SMS Medical College and Attached Hospitals, Jaipur, Rajasthan, India (Ref. No. 470/MC/EC/2022). Written informed consent was obtained from all participants.

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