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Research Article

A CROSS-SECTIONAL STUDY TO EVALUATE THE PREVALENCE OF INSULIN RESISTANCE IN OBESE ADULT FEMALES USING HOMA-IR

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Abstract

Background: Obesity is a major public health problem and is closely associated with insulin resistance, which represents an early metabolic abnormality preceding type 2 diabetes mellitus. Data on the prevalence of insulin resistance among obese adult females in the Indian population remain limited.

Objectives: To evaluate insulin resistance in Indian adult obese females using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) and to determine its prevalence in this population.

Materials and Methods: This observational cross-sectional study was conducted in the Postgraduate Research Laboratory of the Department of Physiology in collaboration with the Department of Biochemistry, Medical College and Hospital, Kolkata, from November 2017 to June 2018. Fifty obese adult female subjects aged ≥18 years with a body mass index ≥30 kg/m² were included. Fasting plasma glucose and fasting serum insulin levels were measured after an overnight fast, and insulin resistance was calculated using HOMA-IR. A HOMA-IR value >2.5 was considered indicative of insulin resistance. Data were analyzed using descriptive statistical methods.

Results: The mean age of the study participants was 33.38 ± 11.25 years, and the mean body mass index was 34.38 ± 3.03 kg/m². The mean fasting plasma glucose and fasting serum insulin levels were 104.12 ± 16.16 mg/dL and 18.77 ± 8.58 µIU/mL, respectively. The mean HOMA-IR value was 4.71 ± 2.11. Based on the predefined cut-off, 41 out of 50 subjects (82%) were classified as insulin resistant, while 9 subjects (18%) were insulin sensitive.

Conclusion: A very high prevalence of insulin resistance was observed among Indian adult obese females, even in the absence of diagnosed diabetes mellitus. Early identification of insulin resistance in this high-risk group may help in implementing timely preventive strategies to reduce future cardiometabolic complications.

Keywords: Obesity; Insulin resistance; HOMA-IR; Adult females; Metabolic risk.

INTRODUCTION

Obesity is a chronic, relapsing, and multifactorial disease that has emerged as one of the most important public health challenges worldwide. It results from a complex interaction between genetic predisposition, behavioral factors, and obesogenic environmental influences,

leading to excess adiposity and progressive metabolic dysfunction. Over the past few decades, the global rise in overweight and obesity has occurred at an unprecedented pace, affecting both developing and developed nations, and contributing significantly to the burden of non-communicable diseases.[1]

The World Health Organization (WHO) defines obesity as abnormal or excessive fat accumulation that may impair health, commonly assessed using body mass index (BMI).[2] The WHO has repeatedly highlighted obesity as a major global health concern due to its association with increased morbidity, premature mortality, and economic healthcare burden.[2] Large pooled international analyses have demonstrated a marked increase in obesity prevalence across several regions of the world since 1990, with obesity increasingly replacing undernutrition as a dominant form of malnutrition in many populations.[3]

India is currently undergoing a rapid epidemiological and nutritional transition driven by urbanization, reduced physical activity, and increased consumption of energy-dense diets, resulting in a steadily rising prevalence of overweight and obesity. This shift is particularly concerning because South Asian populations often develop metabolic complications at lower levels of adiposity compared to Western populations, making early metabolic screening and prevention particularly relevant in the Indian context.[4]

Obesity is strongly associated with multiple metabolic and cardiovascular disorders, including type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, non-alcoholic fatty liver disease, and cardiovascular disease.[5] One of the earliest and most central metabolic abnormalities linking obesity to these outcomes is insulin resistance. Insulin resistance refers to a reduced responsiveness of target tissues such as skeletal muscle, liver, and adipose tissue to the biological actions of insulin, resulting in compensatory hyperinsulinaemia and progressive disruption of glucose homeostasis.[6]

The pathophysiological relationship between obesity and insulin resistance is mediated through several mechanisms such as chronic low-grade inflammation, altered adipokine secretion, ectopic lipid deposition, and dysfunction of adipose tissue expandability.[5],[6] Visceral adiposity is particularly implicated in the development of insulin resistance due to its strong association with lipotoxicity, inflammatory cytokine release, and hepatic insulin signaling abnormalities.[6] These metabolic changes frequently remain subclinical for years before manifesting as overt hyperglycaemia, making insulin resistance an important early marker for identifying individuals at high risk of future diabetes and cardiometabolic disease.[5],[6]

Accurate assessment of insulin resistance is therefore crucial for early risk stratification and targeted prevention in obese individuals. Although the hyperinsulinemic euglycemic clamp technique remains the reference method for assessment of insulin sensitivity, its complexity, invasiveness, and limited feasibility restrict its use in routine clinical settings and large-scale research studies.[7]In this context, surrogate indices based on fasting parameters are widely used for epidemiological screening and clinical research.

The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), first described by Matthews et al., is one of the most commonly used and validated surrogate markers of insulin resistance.[7] It is calculated using fasting plasma glucose

and fasting serum insulin values, providing a practical and cost-effective approach for estimating insulin resistance in population-based studies. HOMA-IR has demonstrated significant correlation with clamp-derived insulin sensitivity, supporting its clinical and research utility.[7]In addition, expert reviews have emphasized that HOMA-based indices remain valuable tools when used appropriately in metabolic research.[8]

Despite BMI being widely used to classify obesity, it does not differentiate between fat mass and lean mass, and it provides limited information regarding body fat distribution. Because central (visceral) adiposity has a stronger association with insulin resistance than generalized obesity, assessment of insulin resistance in obese individuals—particularly in high-risk subgroups—remains essential even when fasting glucose levels are not markedly elevated.[6]

Adult females represent an important high-risk group for obesity-related metabolic dysfunction due to factors such as hormonal variations, lifestyle patterns, reproductive history, and increased propensity for adiposity accumulation. However, Indian population-based evidence focusing specifically on the burden of insulin resistance among obese adult females remains limited. Early identification of insulin resistance in this group can facilitate timely lifestyle interventions and preventive strategies aimed at reducing long-term risk of diabetes and cardiovascular complications.

Against this background, the present study was undertaken to evaluate insulin resistance in obese adult female subjects using HOMA-IR and to determine the prevalence of insulin resistance in this population.

METHODOLOGY

This observational cross-sectional study was conducted to evaluate the prevalence of insulin resistance among obese adult female subjects using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR). The study was carried out in the Postgraduate Research Laboratory of the Department of Physiology in collaboration with the Department of Biochemistry, Medical College and Hospital, Kolkata, West Bengal, over a period of eight months, from November 2017 to June 2018.

The study population consisted of obese adult female subjects aged 18 years and above, attending the endocrinology outpatient department, general outpatient department, or biochemistry laboratory for estimation of fasting blood glucose for the first time, as well as apparently healthy obese female volunteers. Participation in the study was entirely voluntary, and written informed consent was obtained from all participants after explaining the purpose, procedures, and potential benefits of the study.

All female subjects aged 18 years and above with a body mass index (BMI) ≥ 30 kg/m^2 who were willing to participate were included in the study. Subjects with a history of diabetes mellitus, thyroid dysfunction, active infection, inflammatory disorders, malignancy, pregnancy, or those with end-stage hepatic, renal, or cardiac failure were excluded. Individuals with ascites, abdominal tumors, or those taking oral contraceptive pills were also excluded to avoid confounding effects on insulin resistance. As the study was designed to assess insulin resistance within a single cohort of obese females, a separate control group was not included.

Sample size was calculated based on previously published studies evaluating the correlation between anthropometric parameters and insulin resistance, with an

assumed correlation coefficient of 0.45, a power of 80%, and a significance level of 5%. The minimum required sample size was estimated to be 33 subjects. To minimize bias and improve the robustness of the analysis, a total of 50 obese adult female subjects fulfilling the inclusion criteria were finally enrolled in the study.

Anthropometric measurements including height, weight, waist circumference, hip circumference, sagittal abdominal diameter, thigh medium perimeter, neck circumference, and wrist circumference were recorded using standardized techniques. Body mass index was calculated as weight in kilograms divided by height in meters squared (kg/m²). Derived indices such as waist–hip ratio, waist–thigh ratio, waist–stature ratio, and conicity index were calculated using standard formulas.

After an overnight fast of at least 8–10 hours, venous blood samples were collected under aseptic conditions for estimation of fasting plasma glucose and fasting serum insulin. Fasting plasma glucose was measured using standard enzymatic methods, while fasting serum insulin was estimated using a chemiluminescence immunoassay technique. Insulin resistance was calculated using the Homeostasis Model Assessment formula:
HOMA-IR = [fasting insulin (μIU/mL) × fasting glucose (mg/dL)] / 405.
A HOMA-IR value greater than 2.5 was considered indicative of insulin resistance.

After obtaining ethical clearance (IEC number:MC/Kol/IEC/Non-spon/583/08-2017) from the Institutional Ethics Committee of Medical College and Hospital, Kolkata, all procedures were carried out in accordance with ethical standards for human research. Confidentiality of participant data was strictly maintained, and subjects were free to withdraw from the study at any stage without any consequence.

The collected data were entered into Microsoft Excel spreadsheets and analyzed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The prevalence of insulin resistance was calculated based on the predefined HOMA-IR cut-off value. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 50 obese adult female subjects who fulfilled the inclusion criteria and provided written informed consent were included in the study. Descriptive statistics were used to summarize baseline demographic, anthropometric, and biochemical characteristics of the study population. The prevalence of insulin resistance was determined using the predefined HOMA-IR cut-off value. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages.

Table 1. Baseline demographic and anthropometric characteristics of study participants (n = 50)

Parameter	Mean ± SD	Minimum	Maximum
Age (years)	33.38 ± 11.25	14	65
Height (m)	1.47 ± 0.06	1.34	1.58
Weight (kg)	74.42 ± 8.78	60	98
Body Mass Index (kg/m²)	34.38 ± 3.03	30.59	42.15

A total of 50 obese adult female subjects were included in the final analysis. The baseline demographic and anthropometric characteristics of the study population are summarized in Table 1. The mean age of the participants was 33.38 ± 11.25 years, indicating a predominantly young to middle-aged population. The mean body mass index was 34.38 ± 3.03 kg/m², placing the majority of subjects in the obese class I–II category. All participants fulfilled the predefined BMI criteria for obesity.

Table 2. Fasting biochemical parameters and HOMA-IR values (n = 50)

Parameter	Mean ± SD	Minimum	Maximum
Fasting Plasma Glucose (mg/dL)	104.12 ± 16.16	72	142
Fasting Serum Insulin (µIU/mL)	18.77 ± 8.58	6.2	42.8
HOMA-IR	4.71 ± 2.11	1.20	10.86

Fasting biochemical parameters and insulin resistance indices are presented in Table 2. The mean fasting plasma glucose level was 104.12 ± 16.16 mg/dL, while the mean fasting serum insulin concentration was 18.77 ± 8.58 µIU/mL. The calculated mean HOMA-IR value was 4.71 ± 2.11, which was substantially higher than the accepted cut-off for insulin resistance, indicating a high degree of underlying metabolic derangement among the study participants

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Table 3. Prevalence of insulin resistance based on HOMA-IR cut-off (n = 50)

HOMA-IR Status	Number (%)
Insulin Resistant (HOMA-IR > 2.5)	41 (82%)
Insulin Sensitive (HOMA-IR ≤ 2.5)	9 (18%)

Based on the predefined HOMA-IR cut-off value of 2.5, the prevalence of insulin resistance was determined (Table 3). A total of 41 subjects (82%) were classified as insulin resistant, while 9 subjects (18%) were insulin sensitive. Thus, more than four-fifths of the obese adult female participants demonstrated insulin resistance despite the absence of previously diagnosed diabetes.

Overall, the results indicate a very high prevalence of insulin resistance among obese adult females, highlighting the presence of significant subclinical metabolic risk in this population.

DISCUSSION

The present study was undertaken to evaluate insulin resistance in Indian adult obese females using HOMA-IR and to determine the prevalence of insulin resistance in this population. The most important finding of this study is the very high prevalence of insulin resistance (82%) among obese adult female subjects, despite the absence of previously diagnosed diabetes mellitus. In the present study, 41 out of 50 participants were classified as insulin resistant (HOMA-IR >2.5), while only 9 (18%) were insulin sensitive. The mean HOMA-IR was 4.71 ±

2.11, with a mean fasting plasma glucose of 104.12 ± 16.16 mg/dL and mean fasting serum insulin of 18.77 ± 8.58 μIU/mL, reflecting compensatory hyperinsulinaemia with only mildly elevated fasting glycaemia—an early metabolic pattern typical of insulin resistance.

The mean age of participants in this study was 33.38 ± 11.25 years, demonstrating that insulin resistance was already present in a predominantly young to middle-aged obese female group, rather than being restricted to older individuals. The mean BMI in the present study was 34.38 ± 3.03 kg/m², placing most participants in obese class I–II. The extremely high prevalence of insulin resistance seen in this cohort is clinically important because insulin resistance often remains asymptomatic for years but significantly increases future risk of type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease. The American Diabetes Association emphasizes that obesity is a major driver of insulin resistance and recommends early identification of high-risk individuals for timely preventive intervention.[9]

In the present study, fasting plasma glucose was 104.12 ± 16.16 mg/dL, which is only mildly elevated, whereas fasting insulin was markedly high (18.77 ± 8.58 μIU/mL), resulting in a mean HOMA-IR of 4.71 ± 2.11. This indicates that many participants had already entered a stage of insulin resistance characterized by hyperinsulinaemia, even before overt diabetes develops. When compared to population-based reports from non-obese or metabolically "healthier" cohorts, mean HOMA-IR values are substantially lower. For example, Masoodian et al. reported a mean HOMA-IR of approximately 1.8 ± 1.13 in a population-based evaluation of healthy individuals, highlighting the magnitude of insulin resistance present in obese individuals in the current study.[10]

Using a HOMA-IR cut-off value of 2.5, the present study found insulin resistance in 82% of obese adult females. This prevalence is higher than that reported in certain younger cohorts; for instance, Das et al. observed that among overweight/obese school children, 32.3% had insulin resistance using a HOMA-IR ≥2.5 (Indian cut-off).[11] This difference is expected, as adults generally demonstrate more prolonged exposure to obesity-related metabolic stress and thus a greater insulin resistance burden. Similarly, in a cross-sectional study among medical students, the prevalence of HOMA-IR >2.5 was reported to be 28.9%, representing a lower-risk younger population compared to obese adult females in the present study.[12] These comparisons support the finding that insulin resistance prevalence increases significantly with greater adiposity, age, and cumulative metabolic exposure.

In the present study, waist circumference showed a statistically significant but weak positive correlation with HOMA-IR ($r = 0.298$, $p < 0.05$), suggesting that central adiposity is associated with worsening insulin sensitivity. This association has also been reported in other studies. Niţescu et al. reported a significant interdependence between waist circumference and HOMA-IR, supporting the role of abdominal obesity in insulin resistance development.[13] Furthermore, large population evidence indicates that increasing waist circumference is strongly associated with higher odds of elevated HOMA-IR, supporting the use of waist-based assessment in metabolic risk screening.[14]

Although BMI remains a widely used indicator of obesity, it does not reflect fat distribution, and metabolic risk is often more strongly influenced by visceral fat rather than total body mass alone. In this

context, sagittal abdominal diameter has gained attention as a better marker of visceral adiposity. In the present study, sagittal abdominal diameter showed a significant positive correlation with HOMA-IR, indicating the important role of central/visceral fat in insulin resistance.

Supporting this, Saad et al. observed that sagittal abdominal diameter correlated significantly with HOMA-IR and measures of both general and visceral obesity, reinforcing its usefulness as a marker of metabolic dysfunction.[15] Similarly, Vasques et al. highlighted sagittal abdominal diameter as a relevant surrogate marker of insulin resistance in adult populations and suggested its stronger correlation with HOMA-IR compared with other anthropometric indices in several analyses.[16]

The present study also observed a statistically significant positive correlation between hip circumference and HOMA-IR, while waist–hip ratio did not show a significant association. Such findings suggest that composite ratios may be influenced by body shape variability and may not consistently capture metabolic risk in every population. Additionally, among all anthropometric variables measured, thigh medium perimeter showed the strongest positive correlation with HOMA-IR, which is noteworthy and supports the relevance of peripheral body measurements in insulin resistance assessment.

Neck circumference and wrist circumference did not show statistically significant correlations with HOMA-IR in the present study. This differs from several studies reporting associations between neck circumference and insulin resistance; for example, Zadeh-Vakili et al. showed that larger waist circumference increased the odds of insulin resistance and also emphasized the role of anthropometric screening to predict metabolic risk.[14] Variations in findings may arise due to differences in sample size, gender-specific cohorts, ethnicity, and cut-off values used across studies.

An important observation in the present study was the inverse correlation between conicity index and HOMA-IR. While several studies suggest that conicity index may reflect abdominal adiposity and cardiometabolic risk, the predictive utility of conicity index appears population-specific. Therefore, these differences highlight the need for region- and population-specific validation of anthropometric indices for insulin resistance prediction, particularly among Indian women.

Overall, the present study demonstrates a very high prevalence of insulin resistance among obese adult Indian females. The mean fasting insulin (18.77 ± 8.58 μIU/mL), mean fasting glucose (104.12 ± 16.16 mg/dL), and mean HOMA-IR (4.71 ± 2.11) indicate significant underlying metabolic impairment even in the absence of diagnosed diabetes.

This reinforces the importance of early screening for insulin resistance in obese women so that timely lifestyle and preventive measures can be initiated to reduce progression to diabetes and related cardiometabolic complications.

CONCLUSION

The present study demonstrates a very high prevalence of insulin resistance (82%) among Indian adult obese females, as assessed by the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), even in the absence of previously diagnosed diabetes mellitus. These findings indicate that a substantial proportion of obese women harbor significant subclinical metabolic abnormalities at a relatively young age. The elevated fasting insulin levels with

only mildly raised fasting plasma glucose values observed in this study reflect a state of compensatory hyperinsulinaemia, suggesting early impairment of insulin sensitivity long before the onset of overt hyperglycaemia.

The results further emphasize that insulin resistance is not solely dependent on the degree of obesity, as reflected by body mass index, but is also influenced by body fat distribution. Anthropometric parameters indicative of central and regional adiposity, such as waist circumference, sagittal abdominal diameter, thigh medium perimeter, and conicity index, showed significant associations with insulin resistance, highlighting the importance of evaluating fat distribution rather than relying exclusively on BMI.

Based on these findings, it is recommended that routine metabolic screening for insulin resistance be considered in obese adult females, even in the absence of diagnosed diabetes. Simple, cost-effective tools such as fasting insulin estimation and HOMA-IR calculation can facilitate early identification of high-risk individuals. Incorporation of selected anthropometric measurements that reflect visceral and regional fat distribution may further enhance risk stratification. Early detection of insulin resistance should be accompanied by targeted lifestyle interventions, including dietary modification, increased physical activity, and weight management, with the aim of preventing progression to type 2 diabetes mellitus and associated cardiometabolic complications.

LIMITATIONS

The present study has certain limitations that should be considered while interpreting the findings. The cross-sectional study design limits the ability to establish a causal relationship between obesity and insulin resistance. The relatively small sample size and inclusion of only female subjects may restrict the generalizability of the results to the broader population. Insulin resistance was assessed using HOMA-IR rather than the hyperinsulinemic euglycemic clamp technique; however, HOMA-IR remains a validated surrogate for large-scale studies. Additionally, lifestyle factors such as dietary habits and physical activity levels were not assessed, which could have influenced insulin sensitivity.

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