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Original Research Article

Proportion of insulin resistance in overweight and obese children of the age group 5-15 years using the homeostasis model assessment of insulin resistance index

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ABSTRACT

Background: Childhood obesity is increasingly recognized as a major public health concern and is associated with insulin resistance, the underlying mechanism behind many components of metabolic syndrome. Early detection of insulin resistance may provide an opportunity for timely intervention. The homeostasis model assessment of insulin resistance (HOMA-IR) is a simple and validated index used to quantify insulin resistance.
Methods: We conducted a hospital-based cross-sectional study among 165 children attending the outpatient department or admitted under the Department of Paediatrics at a tertiary care centre in southern India. Detailed history, clinical examination, anthropometric measurements, and laboratory investigations were performed. Insulin resistance was assessed using HOMA-IR, with a value ≥2.5 considered indicative of insulin resistance.
Results: Insulin resistance (HOMA-IR≥2.5) was present in 124 (75.2%) children. Significant associations were observed between insulin resistance and acanthosis nigricans, and a positive family history of type 2 diabetes mellitus. Although 78.2% of children had normal fasting blood glucose levels, a substantial proportion exhibited insulin resistance. Mean total cholesterol and triglyceride levels were 180.2±25.9 mg/dl and 105±34.5 mg/dl, respectively, and both showed significant positive correlations with HOMA-IR (r=0.417 and r=0.511, respectively; p<0.01).
Conclusions: Insulin resistance is highly prevalent among overweight and obese children and may precede overt abnormalities in blood glucose. Reliance on fasting blood glucose alone may underestimate the burden of metabolic dysfunction. Early identification using HOMA-IR may facilitate timely lifestyle interventions during a potentially reversible stage and help prevent future metabolic complications.

Keywords: HOMA IR index, Insulin resistance, Acanthosis nigricans, Diabetes, Metabolic syndrome

INTRODUCTION

Childhood obesity has emerged as one of the most important public health challenges worldwide and is now recognized as a chronic, multifactorial disease with significant health consequences.[1] Recent estimates from the World Health Organization indicate that more than 390 million children and adolescents aged 5-19 years were living with overweight or obesity.[2] This reflects obesity as a rapidly escalating global burden. India is experiencing a dual burden of malnutrition, with undernutrition coexisting alongside a rapidly increasing prevalence of childhood overweight and obesity. The most recent prevalence study in Kerala revealed that 23% of school-aged children are reported to be overweight or obese.[3] The recent Indian Academy of Pediatrics (IAP) Guidelines emphasize that childhood obesity should not be viewed merely as excessive weight gain but as a chronic disease requiring early identification and comprehensive evaluation because of its strong

association with type 2 diabetes mellitus, metabolic dysfunction-associated steatotic liver disease (MASLD), hypertension, dyslipidemia, polycystic ovarian syndrome, obstructive sleep apnea, and cardiovascular disease.[4]

Insulin resistance is the central pathophysiological mechanism linking excess adiposity to this obesity related metabolic complications. In the early stages of obesity, compensatory hyperinsulinemia maintains normal glucose homeostasis despite declining insulin sensitivity, allowing significant metabolic abnormalities to remain clinically silent for several years. Consequently, fasting plasma glucose often remains within the normal range until β-cell compensation begins to fail, making it an insensitive marker for identifying early metabolic dysfunction in children with obesity. Early recognition of insulin resistance therefore provides a critical opportunity for timely lifestyle interventions that can halt or even reverse the progression toward overt diabetes and other cardiometabolic disorders.

Although the hyperinsulinemic-euglycemic clamp remains the reference standard for measuring insulin sensitivity, its invasive nature, technical complexity, cost, and limited feasibility make it unsuitable for routine clinical practice or large epidemiological studies. Consequently, surrogate indices have been developed, among which the HOMA-IR is the most widely used and validated. First described by Matthews et al HOMA-IR estimates insulin resistance using fasting plasma glucose and fasting insulin concentrations and has demonstrated good correlation with the reference standard in both clinical and research settings.[5] Its simplicity, reproducibility, and cost-effectiveness make it particularly suitable for screening and assessing insulin resistance in children with overweight and obesity.

Despite the increasing burden of childhood obesity in India, data on the prevalence of insulin resistance among overweight and obese children remain limited, particularly from South India. Differences in body composition, visceral adiposity, and insulin sensitivity suggest that population specific data are essential. Since children with similar body mass index may exhibit markedly different degrees of insulin resistance, anthropometric assessment alone may not adequately identify those with early metabolic abnormalities. Therefore, the present study was undertaken to determine the proportion of insulin resistance using HOMA-IR among overweight and obese children. Early identification of insulin resistance may facilitate timely intervention and reduce the future burden of obesity-related metabolic disease.

METHODS

Study design and setting

A hospital-based cross-sectional study was conducted over a period of one year (July 2023 to July 2024) in the Department of Paediatrics, Sree Gokulam Medical College and Research Foundation, Venjaramoodu, Kerala. Children attending both outpatient and inpatient services were screened for eligibility.

Study population and sample size

The study included overweight and obese children aged 5-15 years, classified according to the Indian Academy of Pediatrics BMI growth charts. Based on a prevalence of insulin resistance of 37.8% reported by Rashmi Ranjan Das et al. and using the formula $4PQ/D^2$ with a relative error of 20%, the calculated sample size was 165.[6]

Inclusion and exclusion criteria

Children aged 5-15 years who were overweight (BMI >85th to <95th percentile) or obese (BMI ≥95th percentile) were included. Children with congenital hypothyroidism, chromosomal anomalies, genetic syndromes associated with obesity, chronic infections, malignancies, Cushing's syndrome, or those receiving long-term corticosteroid therapy were excluded.

Sampling technique and data collection

Consecutive eligible children were enrolled until the required sample size was attained. After obtaining informed consent, demographic details, family history of type 2 diabetes mellitus, and the presence of acanthosis nigricans were recorded using a validated semi-structured questionnaire. Anthropometric measurements including weight, height, and BMI were obtained.

Following an overnight fast of 8-10 hours, 5 mL of venous blood was collected for estimation of fasting blood glucose, fasting insulin, total cholesterol, and triglycerides. Fasting blood glucose was measured by the hexokinase method, while fasting insulin was estimated using chemiluminescence immunoassay. Insulin resistance was assessed using the HOMA-IR, calculated as:

HOMA-IR= (Fasting plasma glucose [mg/dl] × fasting serum insulin [μIU/ml])/405

A HOMA-IR value ≥2.5 was considered indicative of insulin resistance.

Statistical analysis

Data were entered into Microsoft excel and analyzed using SPSS version 20. Quantitative variables were expressed as mean±standard deviation, while categorical variables were expressed as proportions. Pearson's correlation coefficient was used to assess the association of serum triglyceride and total cholesterol levels with HOMA-IR. A $p<0.05$ was considered statistically significant.

Ethical considerations

Institutional ethics committee approval was obtained prior to commencement of the study. Written informed consent was obtained from the parents or primary caregivers of all participants. Confidentiality was maintained throughout the study, and the findings were used solely for scientific and academic purposes.

RESULTS

A total of 165 overweight and obese children aged 5-15 years were included in the study.

Females constituted 55.8% (n=92) and males 44.2% (n=73). Most participants belonged to the 5-10-year age group (74.5%, n=123), and 78.2% (n=129) were obese (Table 1).

The mean HOMA-IR of the study population was 3.6±1.7, with a median value of 3.29 (IQR 2.5-4.37). Insulin resistance (HOMA-IR ≥2.5) was present in 124 children (75.2%) (Table 2 and Figure 1).

Fasting blood glucose was normal in 78.2% of participants, impaired in 15.2%, and in the diabetic range in 6.7%. Mean HOMA-IR increased significantly across these categories ($F=76.35$, $p<0.01$) (Table 3). The mean total cholesterol and triglyceride levels were 180.2±25.9 mg/dL and 105±34.5 mg/dL, respectively. HOMA-IR showed significant positive correlations with total cholesterol ($r=0.417$, $p<0.01$) and triglycerides ($r=0.511$, $p<0.01$) (Table 4 and Figure 2).

Acanthosis nigricans present in 17.0% of children. Insulin resistance was observed in 96.4% of those with acanthosis nigricans, compared with 70.8% of those without acanthosis nigricans ($\chi^2=8.18$, $p=0.004$) (Table 5). A positive family history of type 2 diabetes mellitus was present in 50.3% of participants. Children with a positive family history had a significantly higher prevalence of insulin resistance than those without (79.5% vs. 70.7%; $\chi^2=10.78$, $p=0.001$) (Table 5).

Table 1: Baseline characteristics of the study participants (n=165).

Characteristics	N (%)
Gender	
Male	73 (44.2)
Female	92 (55.8)
Age (in years)	
5-10	123 (74.5)
11-15	42 (25.5)
Weight	
Overweight	36 (21.8)
Obese	129 (78.2)

Table 2: HOMA-IR distribution and proportion of insulin resistance.

Variables	Value
Mean±SD	3.6±1.7
Median (IQR)	3.29 (2.5-4.37)
Range	1.3-12.9
IR (HOMA-IR ≥2.5)	124 (75.2%)

Table 3: Association between fasting blood glucose categories and HOMA-IR.

Fasting blood glucose category	N (%)	Mean HOMA-IR±SD	P value
Normal (<100 mg/dl)	129 (78.2)	3.09±1.07	<0.001*
Impaired fasting glucose (100-125 mg/dl)	25 (15.2)	4.58±1.11	
Diabetic range (≥126 mg/dl)	11 (6.7)	7.48±2.47	

*One-way ANOVA ($F=76.35$).

Table 4: Lipid profile and correlation with HOMA-IR.

Variables	Mean±SD	Correlation coefficient (r)	P value
Total cholesterol (mg/dl)	180.2±25.9	0.417	<0.001
Triglycerides (mg/dl)	105.0±34.5	0.511	<0.001

*Pearson's correlation coefficient.

Table 5: Association of acanthosis nigricans and family history of type 2 diabetes mellitus with insulin resistance (HOMA-IR ≥2.5).

Variables	Category	Insulin resistance present N (%)	Insulin resistance absent N (%)	P value
Acanthosis nigricans	Present (n=28)	27 (96.4)	1 (3.6)	0.004
	Absent (n=137)	97 (70.8)	40 (29.2)	
Family history of type 2 diabetes mellitus	Present (n=83)	66 (79.5)	17 (20.5)	0.001
	Absent (n=82)	58 (70.7)	24 (29.3)	

*Chi-square test.

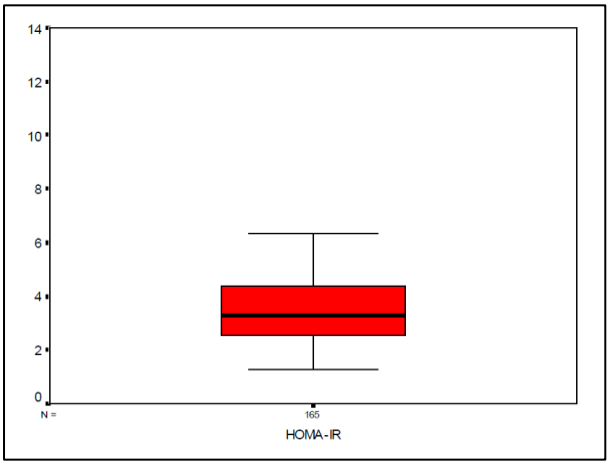

Figure 1: Box plot of HOMA-IR.

The box plot demonstrates that the median HOMA-IR was 3.29 (IQR: 2.5-4.37), with the majority of observations lying above the diagnostic cut-off of 2.5. The distribution was mildly right-skewed, with a few participants exhibiting markedly elevated HOMA-IR values.

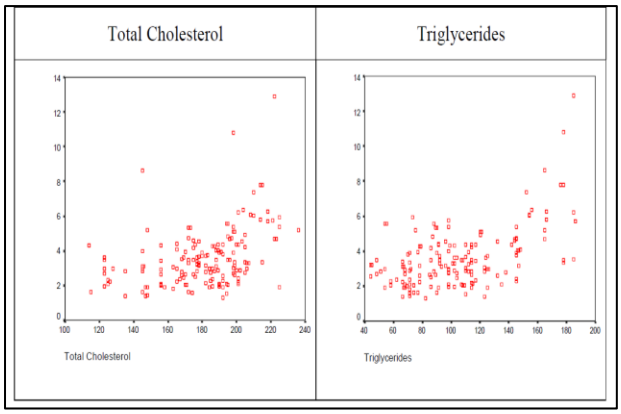

Figure 2: Scatter plots of HOMA-IR vs total cholesterol and triglycerides.

Figure 2 demonstrates a positive linear relationship between HOMA-IR and serum lipid parameters. HOMA-IR showed a moderate positive correlation with total cholesterol ($r=0.417$, $p<0.001$) and triglycerides ($r=0.511$, $p<0.001$), indicating that increasing insulin resistance was associated with higher lipid levels.

DISCUSSION

In this hospital-based cross-sectional study involving 165 overweight and obese children aged 5-15 years, we observed a high proportion of insulin resistance as assessed by HOMA-IR. Insulin resistance was significantly associated with acanthosis nigricans, positive family history of type 2 diabetes mellitus, and elevated fasting blood glucose. Serum triglycerides and total cholesterol also showed significant positive correlations with HOMA-IR.

The proportion of insulin resistance observed in our study was considerably higher than that reported by Rashmi Das et al among children from Odisha.[6] However, Singh et al observed insulin resistance in 77% of obese Indian adolescents, which is comparable to our findings.[7] Tandon et al also demonstrated increasing HOMA-IR values with obesity and metabolic syndrome among urban Indian adolescents.[8] Various international studies have demonstrated a high prevalence of insulin resistance among overweight and obese children. Weiss et al from the United States, in their landmark study involving obese children and adolescents, showed that the prevalence of metabolic syndrome increased with increasing insulin resistance and severity of obesity.[9] Similarly, Keskin et al from Turkey demonstrated that HOMA-IR is a reliable method for assessing insulin resistance in obese children and reported a high prevalence of insulin resistance in this population.[10]

One of the most important observations in our study was that despite 78.2% of children having normal fasting blood glucose levels, insulin resistance was present in 75.2% of the study population. Nogueira-de-Almeida et al similarly reported that abnormalities in blood glucose are uncommon in children despite the presence of insulin resistance.[11] In the early stages of insulin resistance, compensatory hyperinsulinemia maintains normal blood glucose levels despite reduced tissue responsiveness to insulin. Abnormal blood glucose and diabetes become evident only after progressive β-cell dysfunction. Thus, insulin resistance may represent the “base of the iceberg”, while clinically apparent manifestations of metabolic syndrome and diabetes form the “tip”, becoming evident much later in life.

Another noteworthy observation was the presence of early dyslipidemia in our cohort. The mean total cholesterol and triglyceride levels were 180.2±25.9 mg/dL and 105±34.5 mg/dl, respectively. Furthermore, HOMA-IR demonstrated significant positive correlations with total cholesterol and triglycerides. Similar findings have been reported by Kostovski et al who observed significant associations between insulin resistance and lipid abnormalities in obese children and adolescents.[12] Nogueira-de-Almeida et al also demonstrated that hypertriglyceridemia is closely associated with insulin resistance in overweight children.[11] Increased lipolysis and free fatty acid flux to the liver in insulin-resistant states promote hepatic triglyceride synthesis and very-low-density lipoprotein production, thereby explaining the close relationship between insulin resistance and dyslipidemia. These findings suggest that metabolic abnormalities in lipid metabolism may precede overt abnormalities in glucose metabolism.

Acanthosis nigricans showed a strong association with insulin resistance in our study. Stuart et al described the

association between acanthosis nigricans and hyperinsulinemia, while Kong et al subsequently demonstrated its utility as a clinical marker of insulin resistance in overweight children.[13,14]

Children with a positive family history of type 2 diabetes mellitus had a significantly higher prevalence of insulin resistance. Similar findings have been reported by Sinaiko et al who demonstrated the contribution of genetic predisposition and familial clustering to insulin resistance and metabolic syndrome.[15] This emphasizes the importance of targeted screening among children with a strong family history of diabetes.

The major strength of our study lies in the assessment of insulin resistance and its relationship with readily identifiable clinical and biochemical markers using HOMA-IR, a simple and reproducible index suitable for routine clinical practice. However, several limitations should be considered. Being a single-center cross-sectional study, causal relationships could not be established and the findings may not be generalizable to the wider population. HOMA-IR, although widely used, remains a surrogate marker rather than the gold-standard hyperinsulinemic euglycemic clamp. Pubertal status, dietary habits, and physical activity were not evaluated and could have influenced insulin sensitivity. Longitudinal multicentric studies with larger sample sizes are required to establish age-specific HOMA-IR cut-offs and evaluate long-term metabolic outcomes in Indian children.

CONCLUSION

Our findings demonstrate that insulin resistance is highly prevalent among overweight and obese children and is significantly associated with dyslipidemia, acanthosis nigricans, and a positive family history of type 2 diabetes mellitus. An important observation was that although elevated fasting blood glucose was significantly associated with insulin resistance, the majority of children with insulin resistance had normal fasting blood glucose levels, suggesting that metabolic abnormalities begin much earlier than overt diabetes. These findings indicate that relying solely on fasting blood glucose may fail to identify children at risk. Early recognition of insulin resistance using simple indices such as HOMA-IR may provide an opportunity for timely lifestyle interventions at a stage when the process is still potentially reversible. Identifying these children early could help reduce the future burden of type 2 diabetes mellitus and other complications related to metabolic syndrome.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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