

Association Between Insulin Resistance and Obstructive Sleep Apnea in Non-Diabetic Obese Children

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Abstract

Objectives

Although several studies have demonstrated an independent association between obstructive sleep apnea (OSA) and insulin resistance, the evidence remains inconsistent. We aimed to investigate the relationship between the presence and severity of OSA and insulin resistance in non-diabetic obese children and to explore the potential role of Homeostasis Model Assessment- Insulin Resistance index (HOMA-IR) in identifying children at higher risk for OSA.

Methods

Obese children aged 6–18 years who underwent polysomnography were retrospectively analyzed. Patients with diabetes, neuromuscular disorders, craniofacial anomalies or those receiving treatment affecting insulin sensitivity were excluded. Age, sex, BMI, and oral glucose tolerance test (OGTT) results were extracted from medical records, and HOMA-IR was calculated. Patients were divided into two groups based on their obstructive apnea-hypopnea index (OAHI): <2 and ≥ 2 events/hour. ROC analysis was performed for HOMA-IR to determine the optimal cut-off value for identifying patients with OSA.

Results

A total of 90 obese children were enrolled, among whom 39 (43.3%) had OSA. The groups were comparable in terms of age and sex. Median HOMA-IR values were significantly higher in the OSA group ($p = 0.014$). Total OAHI showed moderately positive correlation with HOMA-IR ($\rho = 0.325$, $p = 0.002$), and baseline insulin levels measured during OGTT ($\rho = 0.323$, $p = 0.002$). In multiple linear regression analysis, HOMA-IR remained independently associated with OSA severity after adjustment for age, sex, BMI z-score, sleep efficiency, and pubertal status ($\beta = 0.691$, $p = 0.025$). ROC analysis identified a HOMA-IR cut-off of 3.39, with 72% sensitivity and 59% specificity for OSA.

Conclusion

HOMA-IR was independently associated with OSA severity in obese children, suggesting that insulin resistance may serve as a useful adjunct marker for identifying patients at higher risk and prioritizing PSG evaluation.

1. INTRODUCTION

Pediatric obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, leading to oxygen desaturation, arousals, and sleep fragmentation (1). It affects approximately 1–5% of children worldwide. Obesity is strongly associated with sleep disorders, particularly OSA, which is considerably more common in children with obesity, with reported prevalence rates ranging from 13% to 71% (2).

Obesity promotes the development and progression of OSA by increasing upper airway collapsibility, partly due to excessive fat deposition around the pharyngeal structures and alterations in upper airway mechanics (3). Beyond its mechanical effects, obesity is also associated with metabolic and hormonal alterations, including changes in insulin sensitivity. In this context, while obesity is widely recognized as a major contributor to OSA, accumulating evidence suggests that insulin resistance may also be associated with OSA independently of obesity (4, 5). Furthermore,

several studies have suggested that OSA severity, reflected by the apnea-hypopnea index (AHI), is independently associated with Homeostasis Model Assessment- Insulin Resistance index (HOMA-IR) (6–9). However, findings across studies remain inconsistent, with some failing to demonstrate an independent association (10).

The relationship between impaired glucose metabolism and OSA is likely bidirectional (11, 12). While OSA has traditionally been viewed as a contributor to insulin resistance through intermittent hypoxia, sympathetic activation, oxidative stress, and systemic inflammation (13), several studies have also reported that impaired glucose regulation may be associated with an increased risk of OSA. Proposed mechanisms include abnormalities in autonomic nervous system activity and impaired ventilatory control due to neuropathy or through effects of insulin resistance as well as inflammatory and oxidative stress-activated signaling pathways (11, 14, 15). Furthermore, autonomic dysfunction has been reported even in individuals with prediabetes and impaired glucose tolerance, suggesting that neural alterations may occur early in the course of metabolic impairment (16). However, this relationship between diabetes and OSA may be confounded by obesity, a well-established risk factor shared by both conditions.

Polysomnography (PSG) remains the gold standard for the diagnosis of OSA; however, its high cost and limited availability restrict access, particularly for the growing population of obese children (17). Therefore, there is a need for practical approaches to prioritize patients who would benefit most from PSG evaluation. In this context, metabolic markers such as HOMA-IR may offer a potential adjunctive tool for risk stratification and prioritization of PSG referral.

The primary aim of this study was to investigate the relationship between HOMA-IR and obstructive AHI (OAHI) in obese non-diabetic children, while the secondary aim was to evaluate a potential HOMA-IR cut-off value for identifying patients at higher risk of OSA.

2. METHODS

2.1. Study Design and Population

This retrospective cross-sectional study was conducted at the Sleep Laboratory of the Pediatric Pulmonology Department of our tertiary referral centre between 2018 and 2024. Children aged 6–18 years with a BMI $\geq$ 95th percentile who underwent overnight PSG and had available anthropometric measurements and laboratory data for HOMA-IR were included in the study. Patients diagnosed with diabetes mellitus, neuromuscular disorders, genetic syndromes or craniofacial abnormalities, as well as those receiving medications affecting insulin sensitivity, were excluded.

Detailed clinical history, physical examination findings, and pubertal assessment documented by a pediatric endocrinologist were reviewed. Children were classified into prepubertal and pubertal groups. Pubertal onset was defined as a testicular volume $\geq$ 4 mL in boys and the onset of breast development in girls. Age, sex, anthropometric measurements (weight, height, BMI), and laboratory data were extracted from the medical records. Laboratory data obtained within 9 months of the PSG were included. Patients were divided into two groups based on their obstructive AHI (OAHI) values as < 2 events/hour and ≥ 2 events/hour.

Ethical approval was obtained from the ethics committee of our university (Reference number: GO 22/138).

2.2. Polysomnography and Sleep Evaluation

All children underwent type 1 PSG (Alice 6, Philips, USA). Parameters recorded included electroencephalogram (4 channels, parietal and occipital), electrooculography (2 channels, right and left), electromyography (tibialis anterior

and submandibular), electrocardiogram, oronasal airflow sensor, chest and abdominal movement sensor, body position sensor, snoring microphone, capnometer and a pulse oximeter.

PSG variables considered for analyses included total sleep time, sleep efficiency, arousal index, sleep stages, OAH, rapid-eye movement (REM) OAH and non-REM OAH. PSG data were scored by pediatric sleep medicine specialists using the American Academy of Sleep Medicine guidelines (18). Hypopneas were scored when there was a $\geq 30\%$ reduction in the amplitude of the nasal pressure signal lasting for at least two breaths, associated with either an arousal or a $\geq 3\%$ oxygen desaturation. Apneas were defined as a $\geq 90\%$ reduction in airflow lasting at least two breaths. The OAH was defined as the total number of obstructive apnea and hypopnea events per hour of sleep. Clinically significant OSA was classified as OAH ≥ 2 events/hour.

2.3. Biochemical analysis

All participants underwent a standard oral glucose tolerance test (OGTT), and HbA1c levels were measured in all participants. Blood glucose and insulin levels were assessed at baseline (0 minutes) and 120 minutes after glucose administration. Venous blood samples were obtained after a 12-hour overnight fast to measure baseline blood glucose and insulin levels. These parameters are referred to as baseline glucose, baseline insulin, 120-min glucose, and 120-min insulin. HOMA-IR was calculated using the standard formula: fasting insulin [$\mu\text{IU/mL}$] $\times$ fasting blood glucose [mg/dL]/405 (19).

2.4. Statistical Methods

Statistical analyses were performed using SPSS statistical software, version 22 (IBM Corp., Armonk, NY, USA). Normally distributed continuous variables were analyzed using Student's t-test and expressed as mean $\pm$ standard deviation (SD). Non-normally distributed continuous variables were analyzed using Mann-Whitney U test and expressed as median (with 25th -75th percentile, interquartile range-IQR). The variables, which are suitable for normal distribution, were assessed by visual (histogram and probability graphics), and analytic methods (Kolmogorov-Smirnov/Shapiro-Wilk tests). Categorical variables were presented as percentages (%) and analyzed using Fisher's exact test or Chi-square test (with or without continuity correction). Spearman correlation test was used to evaluate the association between age, BMI, total OAH and laboratory data. To assess the independent predictors of OAH, variables with a p value ≤ 0.25 in univariate analysis were entered into the multiple regression model as covariates. In addition, covariates with known associations based on previous studies were also included in the multiple regression model. Multicollinearity among variables included in the regression model was assessed using variance inflation factor (VIF) and tolerance values. Log transformation was applied to HOMA-IR and total OAH values prior to analysis because of their non-normal distribution, in order to improve data normality and ensure the validity of regression model assumptions. Receiver operating characteristic (ROC) analysis was performed to determine the diagnostic performance and optimal cut-off value for HOMA-IR in order to distinguish OAH < 2 and OAH ≥ 2 groups. A priori power analysis ($\alpha = 0.05$, power = 0.80) indicated a required sample size of 30 participants. The power of our study was 0.99. Values of p < 0.05 were considered statistically significant.

3. RESULTS

A total of 90 obese children (mean age 13.31 ± 2.99 years) were included in the study, all of whom were of Caucasian. Forty-four (48.8%) were male. The median BMI and mean BMI z-score of the cohort were 32.26 ($29.25\text{--}38.14$) kg m^{-2} and 2.96 ± 0.77 , respectively. Fifty-one patients (56.7%) had total OAH < 2 events/h (non-OSA group), whereas 39 patients (43.3%) had a total OAH ≥ 2 events/h (OSA group). The groups with and without OSA were comparable in terms of age and sex, with no statistically significant differences observed between the groups. Patients with OSA had significantly higher BMI and BMI z-scores compared to those without OSA (p = 0.045 and p = 0.003, respectively).

As expected, total OAHl, REM OAHl, and non-REM OAHl values were significantly higher in the OSA group in comparison to the non-OSA group (Table 1).

Table 1
Demographic, polysomnographic and laboratory characteristics of the participants

	Total (n = 90)	OAH1 < 2 (n = 51)	OAH1 ≥ 2 (n = 39)	p
Demographic data				
Age, years (mean ± SD)	13.31 ± 2.99	12.90 ± 2.88	13.84 ± 3.09	0.141
BMI, kg·m ⁻² (median, IQR)	32.26 (29.25–38.14)	31.07 (27.30–36)	35.43 (30.32–41.35)	0.045
BMI z-score (mean ± SD)	2.96 ± 0.77	2.82 ± 0.70	3.15 ± 0.82	0.003
Sex, male (n, %)	44 (48.8%)	25 (49%)	19 (48.7%)	0.977
Polysomnographic data				
REM % (mean ± SD)	12.19 ± 7.30	13.11 ± 7.11	10.98 ± 7.44	0.171
N1% (median, IQR)	4.20 (2.20–7.20)	3.50 (1.90–5.80)	4.70 (2.60–8.80)	0.027
N2% (mean ± SD)	52.52 ± 13.18	51.23 ± 13.43	54.20 ± 12.84	0.294
N3% (mean ± SD)	29.06 ± 11.80	29.50 ± 12.18	28.48 ± 11.41	0.689
Total OAH1 (median, IQR)	1.35 (0.5-5.0)	0.7 (0.3–1.1)	6.3 (3.8–20)	< 0.001
REM OAH1 (median, IQR)	2.6 (0-11.50)	1.4 (0-3.5)	12.5 (1.5–24)	< 0.001
Non-REM OAH1 (median, IQR)	0.7 (0.2–3.55)	0.2 (0-0.6)	3.9 (2.2–16)	< 0.001
Sleep efficiency, %, (median, IQR)	57.90 (43.60–70)	56.85 (43.75–69.20)	60.50 (43.10–74.50)	0.901
Arousal index, per hour (median, IQR)	4.60 (2.75–7.85)	3.80 (2.20–5.25)	7.90 (3.80–13.30)	< 0.001
Biochemical data				
Oral glucose tolerance test				
Baseline glucose, mg/dL (mean ± SD)	83.72 ± 10.12	82.54 ± 9.23	85.25 ± 11.11	0.211
120-min glucose, mg/dL (median, IQR)	104 (90.50-114.50)	99 (88–110)	109.50 (90.75-125.25)	0.048
Baseline insulin, µIU/ml (mean ± SD)	20.41 ± 12.92	18.26 ± 13.55	23.23 ± 11.61	0.071
120-min insulin, µIU/ml (median, IQR)	95.68 (52.34-175.83)	87.60 (48.78–152.50)	126.56 (65.37-191-32)	0.113
HOMA-IR (median, IQR)	3.56 (2.37–5.62)	3.22 (2.13–4.44)	4.59 (2.41–7.04)	0.014
HbA1c (median, IQR)	5.40 (5.22–5.67)	5.40 (5.30–5.60)	5.50 (5.20–5.80)	0.242
<i>The data are presented as mean ± SD, median (IQR), or n (%).</i>				
<i>OAH1: obstructive apnea-hypopnea index; BMI: body mass index; HOMA-IR: homeostatic model assessment - insulin resistance; HbA1c: glycated hemoglobin</i>				

3.1. Comparison of metabolic variables between OSA and non-OSA groups

The comparison of metabolic variables between OSA and non-OSA groups is presented in Table 1. There was no significant difference between the groups in terms of baseline glucose ($p = 0.211$), baseline insulin ($p = 0.071$), 120-min insulin ($p = 0.113$) or HbA1c levels ($p = 0.242$). However, 120-min glucose level and HOMA-IR were significantly higher in the OSA group in comparison to the non-OSA group ($p = 0.048$ and $p = 0.014$, respectively).

3.2. Correlation between polysomnographic and metabolic variables

Correlation analysis demonstrated that total OAHl was moderately positively associated with HOMA-IR ($\rho = 0.325$, $p = 0.002$), and baseline insulin levels ($\rho = 0.323$, $p = 0.002$). In addition, HOMA-IR showed significant positive correlations with both REM OAHl and non-REM OAHl ($\rho = 0.241$, $p = 0.022$ and $\rho = 0.287$, $p = 0.006$, respectively).

As expected, HOMA-IR demonstrated a strong positive correlation with baseline insulin level ($\rho = 0.980$, $p < 0.001$), and a moderate positive correlation with BMI z-score ($\rho = 0.427$, $p < 0.001$). BMI z-score was also significantly correlated with baseline insulin level ($\rho = 0.445$, $p < 0.001$), and age ($\rho = 0.291$, $p = 0.005$) (Table 2).

Table 2
The Correlation Analysis Between Polysomnographic and Metabolic Data

Spearman's rho	Age	BMI z-score	REM OAHl	Non-REM OAHl	Total OAHl	HOMA-IR	Baseline glucose*	Baseline insulin*	120-min glucose*	120-min insulin*
Age	1									
BMI z-score	.291	1								
REM OAHl	.042	.130	1							
Non-REM OAHl	.123	.120	.198	1						
Total OAHl	.106	.165	.515	.861	1					
HOMA-IR	.323	.427	.241	.287	.325	1				
Baseline glucose*	.098	.077	.033	.085	.129	.442	1			
Baseline insulin*	.322	.445	.251	.289	.323	.980	.281	1		
120-min glucose*	.329	.180	.178	.099	.173	.304	.043	.281	1	
120-min insulin*	.364	.307	.118	.190	.190	.631	– .085	.669	.618	1

OAHl: obstructive apnea-hypopnea index; BMI: body mass index; HOMA-IR: homeostatic model assessment - insulin resistance; REM: rapid eye movement

*Oral glucose tolerance test-derived measurements

Bold values indicate statistical significance.

3.3. Multiple Regression Analyses of Factors Associated with OAHl

Univariate linear regression analysis revealed a mild positive association between total OAHl and BMI z-score ($\beta = 0.212$, $p = 0.034$), a strong positive association with HOMA-IR ($\beta = 0.845$, $p = 0.002$), and a mild negative association with sleep efficiency ($\beta = -0.009$, $p = 0.041$) (Table 3). The positive association between HOMA-IR and total OAHl remained significant after adjustment for age, sex, BMI z-score, pubertal status, and sleep efficiency in the multiple linear regression model ($\beta = 0.691$, 95% CI: 0.087–1.294, $p = 0.025$). None of the other variables, including age, sex, BMI z-score, pubertal status, and sleep efficiency, were significantly associated with OAHl in multiple linear regression analysis (Table 3).

Table 3
Independent predictors of total AHI at the multiple regression analysis

Variable	β	95% CI	p
Age	0.020	-0.057, 0.097	0.611
Sex	-0.143	-0.455, 0.169	0.364
BMI z-score	0.116	-0.125, 0.358	0.340
Pubertal status	-0.145	-0.707, 0.416	0.608
Sleep efficiency	-0.008	-0.016, 0.000	0.054
HOMA-IR	0.691	0.087, 1.294	0.025
<i>OAHl: obstructive apnea-hypopnea index; BMI: body mass index; CI: confidence interval; HOMA-IR: homeostatic model assessment - insulin resistance; OR: odds ratio</i>			
Bold values indicate statistical significance.			

3.3. ROC analysis of HOMA-IR for predicting obstructive sleep apnea

ROC analysis was performed to evaluate the ability of HOMA-IR to predict the presence of OSA (Fig. 1). The area under the curve (AUC) was 0.652 (95% CI: 0.534–0.769, $p = 0.014$), indicating modest discriminatory performance. ROC analysis identified an optimal HOMA-IR cut-off value of > 3.39 for predicting OSA ($\text{OAHl} \geq 2$ events/h), with a sensitivity of 72% and a specificity of 59%. Using this cut-off value, the positive and negative predictive value for identifying OSA were 57.1%, and 73.2%, respectively.

4. DISCUSSION

In this study, we demonstrated that insulin resistance, as assessed by HOMA-IR, is independently and strongly associated with OSA as well as its severity in obese children, as reflected by total OAHl measured by the gold standard PSG. Our study extends previous findings by not only demonstrating an independent association between HOMA-IR and OAHl, but also exploring the potential role of HOMA-IR as a marker for identifying high-risk patients and prioritizing PSG evaluation.

The etiological mechanisms contributing to the increased risk of OSA in children with obesity remain incompletely understood (20). Obesity is a shared risk factor for both OSA and impaired glucose metabolism. Although obesity plays an important role in the development and severity of OSA and diabetes, growing evidence suggests that the association between OSA and diabetes is independent of obesity (21). This is further supported by findings demonstrating a higher prevalence of OSA not only in patients with type 2 diabetes, which is commonly associated with obesity, but also in those with type 1 diabetes compared with the general population (22). Importantly, our findings suggest that the association between HOMA-IR and OSA severity extends beyond the influence of obesity alone. Consistent with this, HOMA-IR, but not BMI, remained independently associated with OAHl in our multiple regression analysis.

Numerous studies have suggested that OSA may contribute to the development of insulin resistance, with AHI reported as an independent predictor of HOMA-IR (6, 23–25). In these studies, intermittent hypoxia and sleep fragmentation associated with OSA, along with consequent sympathetic activation and systemic inflammation, are

considered key pathophysiological mechanisms underlying insulin resistance and the subsequent development of diabetes (13, 26).

A recent study published in 2024, involving 483 adult patients without diabetes, demonstrated an independent association between HOMA-IR and AHI after adjusting for covariates, highlighting the importance of considering OSA evaluation in individuals with insulin resistance, even in the absence of known diabetes (25). Moreover, emerging evidence suggests a complex and bidirectional relationship between OSA and metabolic dysfunction (27). While OSA can impair glucose metabolism, chronic inflammation and oxidative stress associated with insulin resistance may potentiate OSA-related inflammatory pathways, establishing a self-perpetuating cycle that aggravates both conditions. Building on this, our study further explores the potential of HOMA-IR by evaluating a cut-off value to help identify children at higher risk for OSA. This approach was motivated by the need for a simple and practical parameter to aid in prioritizing PSG evaluation in obese children.

Correlation analyses further support the link between insulin resistance and OSA. Polysomnographic comparisons based on HOMA-IR levels demonstrated significantly higher REM, non-REM, and total OAHl values in children with elevated HOMA-IR levels, highlighting the association between insulin resistance and the severity of respiratory events.

Although HOMA-IR cut-off values have been established for several metabolic conditions, no such threshold exists for OSA. Although the discriminatory performance of HOMA-IR was modest, the identified threshold may still provide clinical utility for risk stratification in settings where PSG availability is limited.

Sleep efficiency, defined as the ratio of total sleep time to time in bed, was relatively low in our cohort compared with previous studies. This may reflect reduced total sleep time, which has consistently been reported to be lower in children with obesity than in their normal-weight counterparts (20). Short sleep duration has been associated with an increased risk of obesity, supported by studies involving leptin, ghrelin, and orexin that highlight the role of sleep loss in body weight regulation. Because sleep efficiency was lower in our study population, it was included in the multiple regression model to account for its potential confounding effect. The association between HOMA-IR and OAHl remained significant after adjustment for sleep efficiency.

Continuous positive airway pressure (CPAP) therapy for OSA has been associated with improved glycemic control and insulin sensitivity in patients with diabetes (28). Conversely, effective diabetes management may also reduce OSA severity. Notably, antidiabetic therapies such as glucagon-like peptide-1 (GLP-1) receptor agonists and sodium-glucose co-transporter 2 inhibitors have been shown to improve both metabolic parameters and OSA severity (29, 30), with randomized trials demonstrating significant reductions in AHI (31, 32). The beneficial effects of these treatments on AHI are likely driven by weight loss; however, their anti-inflammatory effects on the upper airway mucosa are thought to confer additional therapeutic advantages.

Several limitations should be acknowledged. First, the retrospective cross-sectional design precludes causal inference. Second, the single-center setting and modest sample size may limit generalizability of our findings. Additionally, sleep efficiency was relatively low in our cohort. However, sleep efficiency was included as a covariate in the multiple regression model to account for its potential confounding effect, and adjustment for sleep efficiency did not alter the observed association between HOMA-IR and OSA severity. Another limitation is that a single HOMA-IR cut-off value was derived for the entire study population despite the well-recognized influence of pubertal status on insulin resistance. Although pubertal status was incorporated into the multiple analysis to account for this potential confounding effect, the optimal HOMA-IR threshold for identifying OSA may differ between prepubertal and pubertal children. Puberty-specific cut-off values may provide better discrimination, but larger cohorts are needed to define

these thresholds reliably. Furthermore, the discriminatory performance of HOMA-IR was modest, suggesting that it should be considered an adjunctive rather than a stand-alone screening tool for OSA. To create a more homogeneous study population, only children aged 6–18 years were included in the analysis. Consequently, our findings may not be generalizable to younger children. Future multicenter studies with larger sample sizes and broader age ranges are needed to validate the proposed HOMA-IR threshold and further clarify the relationship between insulin resistance and OSA across different pediatric age groups. Despite these limitations, our study integrates detailed polysomnographic and metabolic data in a well-characterized cohort of obese children. The consistent association between HOMA-IR and OSA observed across group comparisons, correlation analyses and multivariate models supports the robustness of our findings.

This study has several strengths. It addresses a clinically relevant question by evaluating the relationship between insulin resistance and OSA in obese children, a population at high risk for both conditions. All participants underwent overnight polysomnography, the gold standard for OSA diagnosis, ensuring accurate case classification. The use of multiple regression analysis allowed adjustment for important confounders, including age, sex, BMI z-score, pubertal status, and sleep efficiency. Furthermore, the identification of a practical HOMA-IR threshold may have clinical utility in prioritizing obese children for PSG in resource-limited settings. Finally, the integration of detailed metabolic and polysomnographic data provides additional insight into the relationship between insulin resistance and OSA in pediatric obesity.

In conclusion, insulin resistance is independently associated with OSA severity in obese children and may represent a useful adjunctive marker for identifying high-risk individuals. Incorporating metabolic profiling into the evaluation of pediatric OSA could improve risk stratification, particularly in resource-limited settings.

Abbreviations

AHI: apnea-hypopnea index

BMI: body mass index

CPAP: continuous positive airway pressure

GLP-1: glucagon-like peptide-1

HOMA-IR: homeostasis Model Assessment- Insulin Resistance index

OAHI: obstructive AHI

OSA: obstructive sleep apnea

OGTT: oral glucose tolerance test

PSG: polysomnography

REM: rapid-eye movement

ROC: receiver operating characteristic

SD: standard deviation

VIF: variance inflation factor

Declarations

Ethics approval and consent to participate: The study protocol was approved by the Ethics Committee of Hacettepe University (Approval No: GO 22/138) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the parents or legal guardians of all participants, and assent was obtained from children when appropriate.

Consent for publication: All authors have read and approved the final manuscript and consent to its publication.

Availability of data and material: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: All authors declare that they have no conflict of interest.

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Authors' contributions: BS conceived and designed the study, performed the statistical analysis, interpreted the data, and drafted the manuscript. DA, MAE, HID and BS contributed to patient recruitment and data collection. DV, NEO, NG, AO, EY, DD, and UO supervised the study and contributed to study design, interpretation of the findings and critical revision of the manuscript. All authors reviewed and approved the final manuscript.

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References

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BMI: body mass index

CPAP: continuous positive airway pressure

GLP-1: glucagon-like peptide-1

HOMA-IR: homeostasis Model Assessment- Insulin Resistance index

OAHI: obstructive AHI

OSA: obstructive sleep apnea

OGTT: oral glucose tolerance test

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Figures

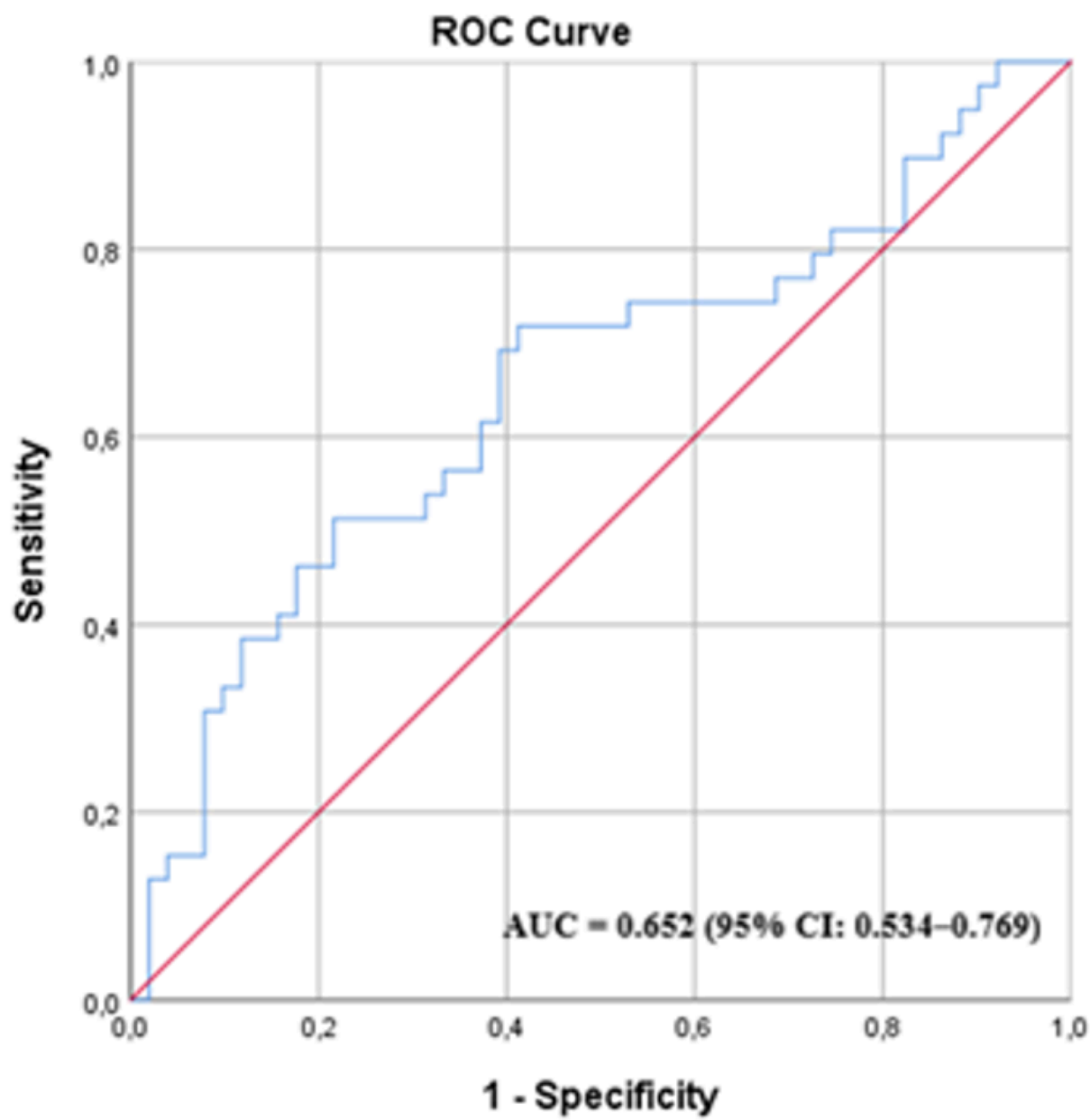


Figure 1
ROC Analysis of HOMA-IR Value for Predicting Obstructive Sleep Apnea Syndrome