Original Article

Comparison of clinical, biochemical, and polysomnographic parameters between obese and nonobese obstructive sleep apnea

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ABSTRACT

Background: Obstructive sleep apnea (OSA) occurs in both obese and nonobese individuals. This study was designed to compare clinical, metabolic profile, and polysomnographic parameters among obese and nonobese OSA patients. **Material and Methods:** This cross-sectional retro-prospective study involved 148 OSA patients. OSA patients were classified as nonobese (body mass index [BMI] <27.5 kg/m^2) and obese (BMI $\geq$ 27.5 kg/m^2) to determine the influence of BMI on its risks, clinical, metabolic, and polysomnographic parameters. For statistical comparisons, continuous variables were analyzed by Student's *t*-test and categorical variables by Chi-square. **Results:** Of 148 patients, 106 patients were of a retrospective group and 42 in the prospective group. 116 patients were obese and 32 were nonobese with a mean BMI of 33.66 ± 5.3 versus. 25.17 ± 2.2 kg/m^2 respectively. Female sex (70.7% vs. 43.4%), larger neck circumference (37.99 ± 3.93 vs. 33.67 ± 5.5 cm), loud snoring (94.8% vs. 81.3%), excessive daytime sleepiness (53.4% vs. 9.4%), fatigability (94.8% vs. 75%), high Epworth Sleepiness Scale score (16% vs. 8%), and hypertension (77.6% vs. 46.9%) were significantly ($P < 0.05$) more common among obese OSA patients while as smoking and sedative use was more prevalent among nonobese OSA group. However, no significant difference in median apnea-hypopnea index and severity of OSA between obese and nonobese group was observed. At the same time, the median oxygen desaturation index was significantly higher in obese patients (26.1 vs. 12.7, $P = 0.005$). **Conclusion:** Nonobese OSA patients depicted less severe disease symptoms and thus require high index of suspicion for early identification due to associated cardiovascular risk.

KEY WORDS: Apnea–hypopnea index, obesity, obstructive sleep apnea, polysomnography

INTRODUCTION

Sleep-related breathing disorders (SRBDs) are being increasingly recognized as one of the major public health problems.[1] SRBDs are classified as Central sleep apnea syndrome, Obstructive sleep apnea syndrome (OSAS), hypoventilation/hypoxia syndrome, nonspecific/undefined sleep disorder.[2,3] OSA is the most common SRBD. OSA is

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defined as a disorder characterized by recurrent episodes of apnea or hypopnea due to a total or partial pharyngeal collapse and temporary upper airway obstruction during sleep, resulting in repeated episodes of hypoxemia and hypercapnia.[4] OSA associated with excessive daytime sleepiness is referred to as OSA syndrome. Global estimates using the American Academy of Sleep Medicine (AASM) criteria 2012 suggest, 936 million people worldwide have mild to severe OSA, and 425 million people having moderate to severe OSA, between the ages of 30 and 69 years.[5] In India, the reported prevalence of OSA varies from 7.5% to 13.5%.[6,7]

Well-defined risk factors for OSA include older age, male gender, obesity, craniofacial and upper airway abnormalities.[8] Other, risk factors include smoking, family history, and nasal congestion. The risk of OSA correlates well with body mass index (BMI). In one study, a 10 percent increase in weight was associated with a six-fold increase in risk of OSA.[9] Although obesity is risk factor for OSA, nonobese individuals are also at the risk. The reasons for narrowing of airway leading to OSA are different in nonobese when compared to obese patients. In obese people, the dominant mechanism is fat deposits in the upper respiratory tract narrowing the airway along with a decrease in muscle activity in this region, leading to hypoxic and apneic episodes, ultimately resulting in sleep apnea, whereas bony structural abnormalities may be the dominant contributing factors among nonobese patients.[10] There are various studies that compared the clinical and polysomnographic parameters among obese and nonobese subjects with OSA. However, studies comparing the risk factors, severity, clinical and metabolic parameters, and their correlation with polysomnographic parameters, among obese and nonobese subjects with OSA are scarce. The study was thus designed to understand the impact of BMI on OSA for better clinical recognition and therapeutic practice. The current study is the first to compare the metabolic profile including fasting plasma glucose, fasting lipid profile, glycated hemoglobin (HbA1c), liver function test (LFT) between obese and nonobese OSA groups.

MATERIALS AND METHODS

This hospital-based prospective as well as retrospective observational study was conducted at the Department of Endocrinology and Department of Internal/Pulmonary medicine, SKIMS, a tertiary care referral institute located in North India. Retrospectively, data were collected from all the subjects who were diagnosed to have OSA from March 2019 to March 2020 and prospectively subjects were recruited from April 2020 to March 2021.

For the retrospective part of the study, data of patients who had already undergone Level 1 or Level 3 sleep study between March 2019 and March 2020 were retrieved from medical records maintained in the sleep laboratory of pulmonary medicine, and subjects who were found to have OSA (mild, moderate or severe) were included in the study. In subjects with confirmed OSA, data was retrieved with regards to:
1. Demographic profile (age, sex, and residential address)
2. Risk for OSA (STOP BANG questionnaire) and Epworth Sleepiness Scale (ESS) score
3. Any history of loud snoring, excessive daytime sleepiness, early morning headache and fatigability, and history of current smoking
4. History of any comorbidities like hypertension, Type 2 diabetes mellitus (T2DM), hypothyroidism, any heart disease (heart failure, coronary artery disease [CAD] and atrial fibrillation), nonalcoholic fatty liver disease (NAFLD), chronic kidney disease, acromegaly, or any endocrine disorder and history of use of drugs like tranquilizers/sedatives
5. Upper airway assessment by Mallampatti score
6. Anthropometry: Neck circumference in centimeters (cm), height in cm, weight in kilograms (kg), and BMI (kg/m²)
7. Sleep study results that include apnea-hypopnea index (AHI) and oxygen desaturation index (ODI) (only in level 3 study)
8. Available baseline investigation including blood glucose, lipid profile, liver function test (LFT), kidney function test (KFT), and HbA1c.

In prospective part of the study, patients presenting with clinical features of OSA to outpatient clinics of the institute were subjected to STOP BANG questionnaire and (ESS) questionnaire. Subjects with STOP BANGscore of ≥3 or ESS >9 were referred to the sleep laboratory to undergo level 3 sleep study after reverse transcription-polymerase chain reaction test (24 h prior to sleep study) for COVID-19 in view of the current ongoing COVID-19 pandemic. Sleep study was done in patient who was negative for COVID-19. Apart from demographic history, note was made of anthropometry, history of comorbidities, upper airway assessment by Mallampatti score, STOP BANG questionnaire, and ESS questionnaire. All subjects with confirmed OSA on level 1 or level 3 sleep study were included. Level 1 sleep study was performed as per the latest AASM guidelines,[11] using an Alice 6 computerized polysomnogram system (32 channel Philips Alice 6 Respironics) at the sleep laboratory. Thirty-two channels were used to document: Sleep stages (four-channel electroencephalogram, electrooculogram, chin electromyogram, electrocardiogram channel, airflow at nose and mouth (nasal thermistor and cannulae), chest and abdominal respiratory movement (respiratory impedance), oxygen saturation (pulse oximetry), snoring (microphone) and body position. Level 3 sleep study was done by ResMed ApneaLink Air portable device which has five channels that record information of respiratory effort, pulse, oxygen saturation, nasal flow and snoring. Apart from AHI, ODI is also obtained in Level 3 sleep study. OSA was diagnosed when a patient had AHI ≥15/h or if a patient with symptoms suggesting OSA had AHI ≥5/h of total sleep time[12] on either level 1 or level 3 sleep study [Figure 1]. Following this, next morning, 10 ml venous blood sample

was taken from all the patients in fasting state. Baseline investigations included fasting blood glucose, lipid profile, KFT, LFT, and glycated hemoglobin (HbA1c).

The statistical software, Statistical Package for Social Sciences (SPSS 21.0, IBM, New York, United States) was used to analyze the data. The Kolmogorov–Smirnov test was applied to test the normality of the data. The continuous variables have been shown in terms of descriptive statistics, i.e., mean and standard deviation for parameters which are normally distributed or by median and interquartile range for data nonnormally distributed. Categorical variables were defined in terms of frequency and percentage. Chi-square tests have been used to compare categorical variables. Student's independent *t*-test and Mann–Whitney test were used to compare the continuous variables for normally and nonnormally distributed data, respectively. All results have been described on 5% level of significance, i.e., $P < 0.05$ considered as statistically significant.

RESULTS

A total of 148 patients were diagnosed to have OSA, of which 106 were in a retrospective group and 42 were in prospective group as shown in Figure 1. Among 148 patients with OSA, 116 (78.3%) were obese and 32 (21.7%) were nonobese with a mean BMI of 33.66 ± 5.3 versus 25.17 ± 2.2 kg/m², respectively. On comprising obese and nonobese OSA patients, there was no significant difference in mean age, early morning headache, Mallampati, and STOPBANG score [Table 1].
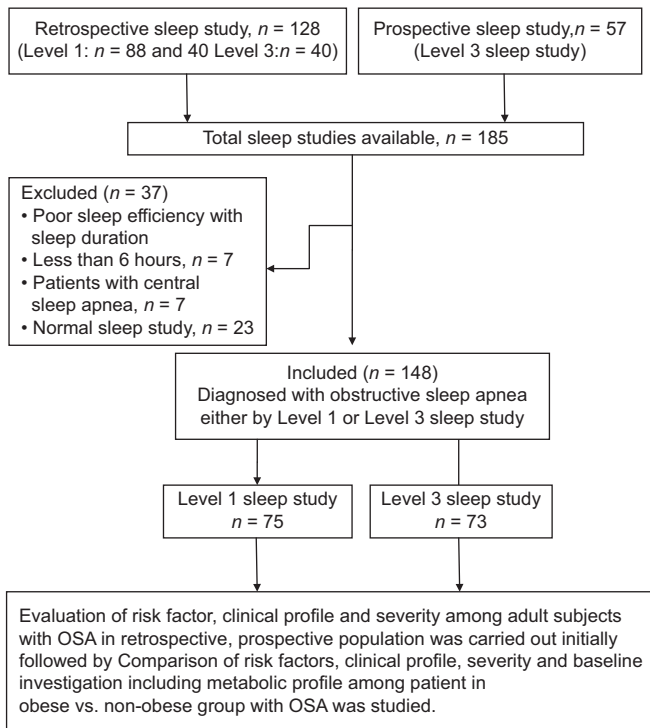
Figure 1: Consort chart describing the flow of subjects through the study

Characteristics such as male sex, smoking, and sedative use were more common in nonobese group than obese OSA group with significant differences between them. Female sex, larger neck circumference, loud snoring, excessive daytime sleepiness, fatigability, and high ESS score were characteristics more commonly found in obese OSA patients with a significant difference. Among comorbidities, only hypertension was significantly higher in the obese OSA (77.6% vs. 46.9%, $P = 0.01$) compared to nonobese OSA patients [Table 1].

Median ODI was significantly higher in obese patients as compared to nonobese patients with OSA (26.1 vs. 12.7, $P = 0.005$). However, no significant difference in median AHI and severity of OSA between the two groups was observed as shown in Tables 2 and 3. As shown in Table 4, there was no significant difference in metabolic profile (fasting plasma glucose, lipids, HbA1c, and liver function indices) between obese and nonobese OSA patients,

There was a significant positive correlation between AHI and neck circumference, ESS and STOPBANG score on level 1 polysomnography [Supplementary Table 1]. Whereas on level 3 polysomnography, it was BMI and STOPBANG score that showed correlation with AHI [Supplementary Table 2]. ODI had significant correlation

Table 1: Comparison of risk factors, clinical profile and evaluation tool parameters in obese and nonobese with obstructive sleep apnea

Patient characteristics	Obese group (*n*=116)	Nonobese group (*n*=32)	*P*
Age (years)#	52.12±10.77	52.00±10.87	0.95
BMI*	33.66±5.3	25.17±2.2	0.001
Male+	34 (29.3)	21 (65.6)	0.01
Females+	82 (70.7)	11 (34.4)	
Smokers, *n* (%)	5 (4.3)	5 (15.6)	0.02
NC*	37.99±3.93	33.67±5.5	0.001
Mallampati score			
Class 1	5	4	0.07
Class 2	53	18	
Class 3	47	10	
Class 4	11	0	
Loud snoring+	110 (94.8)	26 (81.3)	0.01
Excessive day time sleepiness+	62 (53.4)	3 (9.4)	0.001
Fatiguability+	110 (94.8)	24 (75)	0.001
Early morning headache+	24 (20.7)	4 (12.5)	0.29
Sedative use+	1 (0.86)	10 (31.25)	0.001
Hypertension+	90 (77.6)	15 (46.9)	0.001
T2DM+	38 (32.8)	6 (18.8)	0.12
Hypothyroidism+	33 (28.4)	9 (28.1)	0.97
CAD+	15 (12.9)	5 (15.6)	0.69
NAFLD+	15 (12.9)	1 (3.1)	0.18
CKD+	5 (4.3)	2 (6.3)	0.64
STOPBANG score#	5 (4-5)	4 (3-5)	0.081
ESS#	16 (8.25-18)	8 (6-9)	0.001

*Mean±SD, +Frequency (%), #Median and IQR. CAD: Coronary artery disease, NAFLD: Nonalcohlic fatty liver diseases, CKD: Chronic kidney diseases, IQR: Interquartile range, SD: Standard deviation, BMI: Body mass index, ESS: Epworth Sleepiness Scale, T2DM: Type 2 diabetes mellitus, NC: Neck circumference, STOPBANG: Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference and Gender

with neck circumference, BMI, and ESS [Supplementary Table 3].

DISCUSSION

Obesity is an important established risk factor for OSA with obese patients being twice vulnerable than normal weight people.[11,12] Many cross-sectional studies show strong association between increased body weight and risk for OSA.[13] A study estimated that 58% of OSA cases arise due to BMI >25 kg/m2[14] However, South-East Asian ethnicity has been associated with increased risk of OSA and its development at significantly lower BMI than Caucasians,[15,16] implying that nonobese individuals are also at risk for OSA. In this study, among 148 adult subjects with OSA, major proportion of the patients was obese, though there was no difference in severity of OSA between the subgroups.

The current study showed a significant difference in gender among obese and nonobese groups which is in contrast to various other comparative studies[17,18] in both groups. We observed females as dominant gender in obese OSA category, which contradicts the study by Kumar

Table 2: Comparison of apnea hypopnea index and oxygen desaturation index in obese and nonobese with obstructive sleep apnea

Parameter	Obese (*n*=116)	Nonobese (*n*=32)	P
AHI#	27.05 (15.2-58.7)	24.2 (12.125-42.275)	0.321
ODI#	26.1 (15.925-43.325)	12.7 (8.275-23.175)	0.005

#Median and IQR. AHI: Apnea hypopnea index, ODI: Oxygen desaturation index, IQR: Interquartile range

Table 3: Comparison of severity of obstructive sleep apnea in obese and nonobese with obstructive sleep apnea

Severity of OSA	Obese, *n* (%)	Nonobese, *n* (%)	P
Mild (AHI 5-15)	28 (24)	9 (28)	0.62
Moderate (AHI ≥15-30)	37 (32)	12 (38)	
Severe (AHI ≥30)	51 (44)	11 (34)	

AHI: Apnea hypopnea index, OSA: Obstructive sleep apnea

Table 4: Comparison of metabolic profile in obese and nonobese with obstructive sleep apnea

Parameter	Mean±SD		P
	Obese (*n*=57)	Nonobese (*n*=16)	
Fasting plasma glucose (mg/dl)	112.19±42.66	104.18±34.14	0.49
Total cholesterol (mg/dl)	191.07±41.10	193.00±38.14	0.86
Triglycerides (mg/dl)	193.49±78.62	200.00±99.45	0.78
HDL (mg/dl)	44.86±14.30	42.43±5.15	0.51
LDL (mg/dl)	109.96±31.40	108.50±33.92	0.87
HbA1c (%)	6.15±1.29	5.75±0.81	0.24
Aspartate transaminase (IU/L)	37.32±20.48	37.61±17.65	0.95
Alanine transaminase (IU/L)	36.66±17.76	34.94±16.21	0.72
Alkaline phosphatase (IU/L)	115.63±31.14	110.56±28.95	0.56
Total protein (mg/dl)	7.54±0.62	7.80±0.49	0.13
Serum albumin (mg/dl)	4.17±0.56	4.38±0.49	0.72

SD: Standard deviation, HDL: High density lipoprotein, LDL: Low density lipoprotein, HbA1c: Hemoglobin A1c

et al.[19] where in males represented greater proportion of obese OSA patients. Older age is a known risk factor for OSA,[19,20] we observed the mean age of subjects in our study was 52 years which is in concordance with the study done by Ammar Ghanem *et al*.[17] in which they found only 1 patient out of 17 patients <50 years had OSA and stated that OSA is unlikely to occur in patients <50-year-old.[17] Further, characteristics such loud snoring, easy fatigability, excessive day time sleepiness as assessed by ESS score, presence of hypertension, and ODI were significantly higher in obese ($P < 0.05$) as compared to versus nonobese OSA group. Increase in neck circumference is considered as a risk factor for OSA progression among obese patients than lean ones.[21] Our results are in agreement with these previous studies[22,23] as large neck circumference was a common observation among study subject with OSA.

A strong association between cigarette smoking and OSA has been reported with smokers 2.5 fold more likely to develop OSA than nonsmokers.[24] A study on Wisconsin cohort revealed current smokers to be three times more likely to develop OSA than former or nonsmokers[24,25] while as another study reported no significant difference among obese and nonobese OSA group with respect to smoking.[17] We observed the current smoking was significantly higher ($P < 0.05$) among nonobese OSA group as compared to obese.

OSA exposes the cardiovascular system to cycles of hypoxia, exaggerated negative intra thoracic pressure, and arousals which in turn, subdues myocardial contractility and causes upsurge in blood pressure, heart rate, systemic inflammation, activate platelets, and impair vascular endothelial function thus leading to CAD.[26] This study did not find any significant difference in proportion of patients with CAD in patients between obese and nonobese OSA group as was found in previous study.[27] On the other hand, hypertension was the most common co-morbidity found in patients with OSA and it was found more in obese OSA group (n = 90,77.6%), these findings are concordant with various other studies.[28-30]

Insomnia-related complaints are predominant among more than 50% of the OSA patients which warrants the use of sedatives to ameliorate the severity of insomnia.[31,32] Here, we report significantly higher use of sedatives for sleeping in nonobese OSA group (31.25%) as compared to obese OSA group, which is in concordance with previous studies studies.[33] In addition, loud snoring was also predominant among obese OSA group (94.8%) than in nonobese OSA group (81.3%). This finding is concordant with study done by Kumar *et al*.[19] where patients in obese OSA group (58.33%) were found to have significant loud snoring when compared to nonobese OSA group (17.39%). Excessive day time sleepiness is also found to be significantly more in patients of obese OSA (53.4%) group than in nonobese OSA group (9.4%) as assessed by ESS. The median ESS score in patients of obese OSA group differed significantly when compared to median

ESS score in nonobese OSA group. This finding was in contrast to various studies,[18,19,33] where no difference was found in the ESS score between obese and nonobese OSA group. STOP BANG questionnaire is validated to screen for risk of OSA.[34] In our study, we observed no significant difference of median STOPBANG score between groups of OSA patients under study.

Exposure of healthy people to sleep deprivation or intermittent hypoxia increases glucose intolerance, insulin resistance (IR),[3,35] and activity of the sympathetic nervous system.[36] OSA is a well-recognized risk factor for IR and T2DM independent of BMI.[37] In cross-sectional studies, involving 2656 participants, OSA patients displayed greater IR and higher prevalence of hyperglycemia and T2DM.[38,39] In this study, we observed higher proportion of patients with T2DM in patients of obese OSA group than in nonobese OSA group as reported previous in various studies.[39,40] Studies have shown higher prevalence of clinical hypothyroidism in obese OSA patients[40] and our study depicted the similar results.

In addition, no significant difference in the median AHI between the obese and nonobese OSA subgroups, which is contrast to the AHI results found in several other studies[17] where higher AHI is found in obese OSA subjects. These results may be attributed to the presence of moderate to severe OSA in our study population. In contrast to AHI, there is a significant difference ($P = 0.005$) in median ODI between patients in obese and nonobese OSA group, which was similarly found in other studies.[18,41] This finding of significant difference in ODI and no significant difference between AHI between patients of Obese OSA and nonobese OSA group is in concord with a retrospective study done by Ling *et al.*[42] who reported significant oxygen desaturation in obese subjects.

The current study is the first to compare the co-morbidities such as NAFLD, CAD, and metabolic profile including fasting plasma glucose, fasting lipid profile, HbA1c, LFT between obese and nonobese OSA groups and it was found that there was no significant difference between them. Comparative studies reported till date on patients in obese and nonobese OSA group are retrospective nature, while our study has both retrospective as well as prospective components. Apart from these strengths, there are certain limitation of this study that includes, polysomnography (Level 1 sleep study) could not be done in all patients. About one-third in retrospective part and all patients in prospective part, were diagnosed with OSA based on Level 3 sleep study due to the current COVID-19 pandemic, upper airway soft tissue enlargement assessment using only Mallampatti score and neck circumference, were done which could have been inferior to cephalometric radiography, computed tomography, magnetic resonance imaging and fluoroscopy. Some part of the study involves patients recruited retrospectively in whom complete baseline investigations were missing. Another limitation was there may be a selection bias in this study as most patients recruited and diagnosed with OSA are obese. This being a hospital-based study done in Kashmiri population, so results may not be generalizable to other population.

CONCLUSION

Nonobese OSA patients have similar clinical severity of the disorder as that of obese ones, however, there seems more metabolic complications (Hypertension) among the obese group. However, the frequency of OSA in nonobese is comparatively less among normal weight people. In addition, smoking is an independent risk factor for OSA.

Conflicts of interest
There are no conflicts of interest.

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Supplementary Table 1: The correlation between apnea-hypopnea index and various clinical, evaluation tool parameters and metabolic profile on level 1

Parameter studied	r	Level of significance
Age	−0.125	0.301
NC	0.265	0.025*
BMI	0.212	0.076
ESS scale	0.276	0.020*
STOPBANG	0.302	0.011*
FBS	−0.156	0.512
Serum total cholesterol	0.290	0.215
Triglycerides	0.345	0.137
HDL	−1.75	0.460
LDL	0.158	0.506
HbA1c	−0.03	0.869
Hb (%)	0.03	0.900
HCT	−0.018	0.941
SGOT	−0.108	0.650
SGPT	−0.120	0.615

*Correlation is significant at the 0.05 level (two-tailed), **Correlation is significant at the 0.01 level (two-tailed). BMI: Body mass index, ESS: Epworth Sleepiness Scale, HDL: High density lipoprotein, LDL: Low density lipoprotein, HbA1c: Hemoglobin A1c, NC: Neck circumference, FBS: Fasting blood sugar, HCT: Hematocrit, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, STOPBANG: Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference and Gender

Supplementary Table 2: Depicts the correlation between apnea-hypopnea index and various clinical, evaluation tool parameters and metabolic profile on level 3

Parameter studied	r	Level of significance
Age	0.07	0.526
NC	0.218	0.057
BMI	0.267	0.019*
ESS score	0.04	0.683
STOPBANG score	0.293	0.01*
FBS	0.225	0.105
Total cholesterol	−0.028	0.842
Triglycerides	−0.073	0.605
HDL	−0.035	0.804
LDL	0.013	0.924
HbA1c	0.200	0.151
Hb (%)	−0.57	0.687
HCT	0.04	0.75
SGOT	−0.117	0.405
SGPT	−0.018	0.898

*Correlation is significant at the 0.05 level (two-tailed), **Correlation is significant at the 0.01 level (two-tailed). BMI: Body mass index, ESS: Epworth Sleepiness Scale, HDL: High density lipoprotein, LDL: Low density lipoprotein, HbA1c: Hemoglobin A1c, NC: Neck circumference, FBS: Fasting blood sugar, HCT: Hematocrit, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, STOPBANG: Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference and Gender

Supplementary Table 3: Correlation between oxygen desaturation index between apnea-hypopnea index and various clinical, evaluation tool parameters and metabolic profile on level 3

Parameters	r	Level of significance
Age	0.033	0.775
NC	0.240	0.0.037*
BMI	0.285	0.0.013*
ESS scale	0.262	0.02*
STOPBANG score	0.173	0.136
FBS	0.238	0.087
Serum total cholesterol	−0.154	0.171
Triglycerides	−0.90	0.520
HDL	0.045	0.750
LDL	−0.071	0.614
HbA1c	0.216	0.120
Hb (%)	−0.08	0.571
HCT	0.015	0.917
SGOT	−0.2.86	0.038*
SGPT	−0.207	0.138

*Correlation is significant at the 0.05 level (two-tailed), **Correlation is significant at the 0.01 level (two-tailed). BMI: Body mass index, ESS: Epworth Sleepiness Scale, HDL: High density lipoprotein, LDL: Low density lipoprotein, HbA1c: Hemoglobin A1c, NC: Neck circumference, FBS: Fasting blood sugar, HCT: Hematocrit, SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, STOPBANG: Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference and Gender