

RESEARCH

Open Access



Clinical predictors of obstructive sleep apnea among residents of Sagamu local government area of Ogun State

S. O. Olalekan^{1*}, I. O. Osonuga¹, P. G. Okwute², O. E. Atekoja³, M. M. Adeyanju⁴, B. O. Adegbesan⁴, E. N. Ezima⁴, O. D. Odufejo¹, B. Tayo⁵, V. B. Edema¹ and D. D. Taiwo¹

Abstract

Background Obstructive sleep apnea (OSA) is the most commonly diagnosed sleep-associated pulmonary disorder in the world. So many risk factors have been attributed to OSA; however, conflicting results exist on how these factors contribute to OSA.

Purpose This study hypothesized that increasing the number of risk factors for OSA, as reflected in the STOP-BANG questionnaire, increases the probability of having OSA but also increases the likelihood of having other severe diseases such as hypertension in a Nigerian population and that the severity of OSA might be dependent on specific anthropometric indices.

Methods This study involved 110 male and female residents of Sagamu Local Government Area of Ogun State. Data was collected using a standardized instrument and the STOP-BANG questionnaire. Analysis involved descriptive statistics, parametric test of independent *t*-test to characterize data based on gender, and multinomial regressions to determine predictive factors of the various parameters of study on the severity of OSA.

Results The association of the male gender with higher risk of OSA was confirmed by this study. Diastolic blood pressure (DBP) and heart rate (HR) predicted the risk of OSA, with an increase in DBP and HR implying higher risk of OSA. Of the anthropometric tests examined in this study, body mass index (BMI), neck circumference (NC), and neck height ratio (NHtR) predicted the risk of OSA in comparing low risk and medium risk to high risk, an increase in BMI and NC implying a higher risk of OSA while an increase in NHtR implying a lower risk of OSA.

Introduction

Obstructive sleep apnea (OSA) is the most commonly diagnosed sleep-associated pulmonary disorder in the world [1]. OSA is characterized by repeated episodes of a total or partial closure of the upper airway during sleep, with a subsequent reduction of airflow resulting in intermittent hypoxia, hypercapnia, and increased frequency of awakenings [2]. OSA leads to lower quality of life by causing cognitive and neurobehavioral impairment, an inability to concentrate, memory loss, irritability, depression, fatigue, daytime somnolence, headache, an increased incidence of motor vehicle accidents, stroke, cardiovascular impairments, and subsequently increased morbidity and mortality [3–6].

*Correspondence:

S. O. Olalekan
samuel.olalekan@oouagoiwoye.edu.ng

¹ Department of Physiology, Olabisi Onabanjo University, Sagamu Campus, Sagamu, Ogun State, Nigeria

² Department of Physiology, Babcock University Teaching Hospital, Ilishan, Ogun State, Nigeria

³ Department of Nursing Science, Olabisi Onabanjo University, Sagamu Campus, Sagamu, Ogun State, Nigeria

⁴ Department of Biochemistry, Olabisi Onabanjo University, Sagamu Campus, Sagamu, Ogun State, Nigeria

⁵ Department of Medical Microbiology and Parasitology, Babcock University Teaching Hospital, Ilishan, Ogun State, Nigeria

Different factors, such as age, gender, obesity, craniofacial and oropharyngeal anatomical defects, ethnicity, endocrine factors, and personal habits such as smoking and alcohol consumption, have been implicated as risk factors [7–10]. Hormones such as estrogen and progesterone have been considered to play an important role in gender-defined anatomical differences in the pathogenesis of OSA since the prevalence of OSA increased in female after menopause [11, 12]. Likewise the anatomical distribution of fat could also explain the sex-dimorphic vulnerability of OSA, with fat being around the neck and waist in adult males and around the hips in adult females [13]. In a study, the relationship between obesity and OSA is complex with evidence of severity of OSA not correlating with the degree of obesity when assessed with the body mass index (BMI) [14].

Generally, the measures of obesity which have been employed and shown to better predict OSA include body mass index (BMI), neck circumference (NC), waist circumference (WC), waist–hip ratio (WHR), neck–height ratio, and waist-to-height ratio (WHtR) [15, 16]. However, results from studies on the relationship between these different anthropometric measures and OSA differed by age, gender, and ethnicity of the participants, as well as by environmental factors [8, 12, 17]. Obstructive sleep apnea (OSA) has also been associated with an increase in the prevalence and incidence of arterial hypertension and cardiovascular diseases (CVD) [18, 19]. In a study by Unnikrishnan et al. [20], systemic inflammation has been linked to hypoxia due to OSA leading to initiation and rapid progression of atherogenesis.

Despite the health implications, adequate attention has not been paid to OSA. It is recommended that a diagnosis of OSA should be established by laboratory-based or home-based polysomnography (PSG) [21]. Since it is not practicable to subject all patients with risk of OSA to PSG, it is useful to define its determinants. A useful tool for screening for OSA is STOP-BANG questionnaire as it has been found useful, simple, and validated [22]. We hypothesized that increasing the number of risk factors for OSA, as reflected in the STOP-BANG questionnaire, increases the probability of having OSA but also increases the likelihood of having other severe diseases such as hypertension in a Nigerian population situated in Sagamu area of Ogun State, Nigeria. Moreover, it has also been established that fat distribution rather than total body fat is a major contributor to OSA and also a determinant of cardiovascular risk [23, 24]. Therefore, we also hypothesized that the severity of OSA might be dependent on specific anthropometric indices. Hence, this present study seeks to examine how sociodemographic, anthropometric, and lifestyle factors predict the risk of

OSA using the STOP-BANG questionnaire in a Nigerian population.

Methodology

Study design

This study used an ex post facto research design; the parameters of interest were collected from the study subjects without any form of experimentation.

Study area

The study was conducted in Sagamu Local Government Area of Ogun State, Nigeria, on the following criteria.

Criteria for selection

The subjects for this study were selected by non-probability convenient sampling technique.

Inclusion criteria: Male and female residents of Sagamu Local Government Area of Ogun State and its environs within the age range of 18–90 years.

Exclusion criteria: Participants must be found to be free from known cases of gross anemia, gross clinical abnormalities of the vertebral column, thoracic cage, neuromuscular diseases, chronic bronchitis, emphysema, bronchial asthma, tuberculosis, and malignancy. Subjects who had undergone vigorous exercise and abdominal or chest surgery were excluded from the study.

Method of data collection

Data was collected using a standardized instrument, the STOP-BANG questionnaire [24]. Ten research assistants were trained on the objective of the study, the ethics of research, and modality of the data collection from the questioning to measurement of various variables of interest. The purpose and methodology of this study was carefully explained to the subjects and after which interested subjects were asked to give their consent. The research assistants conducted an interviewer-administered questionnaire survey regarding sociodemographic information and lifestyle factors, as well as sleep-related questions including the STOP-BANG questionnaire [24]. Ethical approval for this study was obtained from the Health Research Ethics Committee, Olabisi Onabanjo University Teaching Hospital, Sagamu, with an approval number OOUTH/HREC/658/2023AP.

Instrumentation

Severity of risk to OSA was classified as low, moderate, and high risk based on their STOP-BANG score with 0–2 scored as low risk, 3–4 scored as moderate risk, and 5–8 scored as high risk.

Measurements of height, weight, body mass index (BMI), body adiposity index (BAI), waist circumference (WC), hip circumference (HC), waist hip ratio (WHR),

waist height ratio (WHtR), neck circumference (NC), neck height ratio (NHtR), and blood pressure (BP) were taken by trained research assistants.

Data analysis

Descriptive statistics and a parametric test of independent *t*-test were used to characterize data based on gender, while multinomial regressions were used to determine predictive factors of the various parameters of the study on the severity of OSA.

Results

In Table 1, the sociodemographic and lifestyle characteristics of the study subjects were presented. The subjects were aged between 18 and 90 years with most of them within the age bracket of 41–50 years. It can also be deduced that the percentage of those with low risk of OSA decreased from 80.00% in age bracket of 18–20 years to 25.00% in age bracket 71–80 years. The only person in age bracket 81–90 years had a low risk of OSA.

In terms of gender, there was almost an equal distribution between the male and female genders. The females and males that had low risk of OSA were 67.92% and 47.54%, respectively.

In terms of lifestyle habits, most (86.80%) of the study subjects were non-smokers with about 54.90% of them with a low risk of OSA while 75.00% of those who smoke had low risk of OSA. Most of them were non-consumer of alcohol (43.00%) and a relatively lesser number being occasional consumers of alcohol (38.60%). Those that drink regularly, drink occasionally, and those that do not drink with low risk of OSA were 42.85%, 58.70%, and 59.25%, respectively.

In terms of their nature of job, most of the subjects had jobs with moderate activity (43.00%) and a smaller percentage having jobs that are physically exhausting (38.60%). Those that have physically exhausting, moderate, and sedentary jobs with low risk of OSA were 48.88%, 61.66%, and 66.67%, respectively.

From Table 2, it can be deduced that females had a lower risk of OSA across all age groups except the age brackets of 21–30 years. Between ages 81 and 90 years, females were not represented in this study. Also between age bracket of 51–80 years, males had medium to high risk of OSA.

From Table 3, the anthropometric characteristics of the study population was presented based on their risk of OSA, most of them had a waist circumference above 90 cm (37.70%); those with 1–70 cm, 71–80 cm, 81–90 cm, and above 90 cm with low risk of OSA were 83.33%, 67.74%, 53.57%, and 42.11%, respectively.

A higher percentage of the subject subjects had a waist hip ratio of less than 1 (88.60%); those with waist hip ratio of <1 and >1 with low risk of OSA were 59.43% and 25.00%, respectively.

The study samples who had a waist height ratio <1 were 95.6%; those with waist height ratio of <1 and >1 with a low risk of OSA were 58.03% and 0.00%, respectively.

The study samples who had a neck height ratio between >20 cm/m were 86.00%; those with neck height ratio of <20 cm/m and >20 cm/m with low risk of OSA were 68.18% and 54.34%, respectively.

In this study, most of the subjects had a body adiposity index more than 25% (65.80%); those with BAI of <10%, 10–25%, and >25% with low risk of OSA were 57.14%, 53.33%, and 58.44%, respectively.

Most of the subjects had a BMI between 18.50 and 24.90 (40.40%); those with BMI of <18.50, 18.50–24.90, 25.00–29.90, and >29.90 with low risk of OSA were 50.00%, 63.26%, 80.95, and 36.11%, respectively.

From Table 4, it can be deduced that for the different categories of waist circumference, females had the lowest risk to OSA. Males have a higher risk of OSA compared whether the WHR is <1 or >1. For waist height ratio, females have a lower risk of OSA when the ratio is <1 only. Males have a lower risk of OSA when the neck height ratio is <20 cm/m; otherwise, females have a lower risk of OSA. When the body adiposity index is less than 10.00%, females have a lower risk of OSA, when the BAI is between 10 and 25%, males have a lower risk of OSA, but when the BAI is >25%, females have a lower risk of OSA compared to males. Females despite their BMI had a lower risk of OSA compared to males.

From Table 5, most of the study population showed normal cardiovascular variables with most of them having a systolic blood pressure below 140 mmHg (74.60%); those with SBP with <80, 81–90, 91–100, and >100 have percentage low risk of OSA as 81.13%, 48.15%, 43.75%, and 16.67%, respectively. Most of the subjects had diastolic blood pressure below 100 mmHg (89.50%); those with SBP with <110, 110–120, 121–140, and >140 have percentage low risk of OSA as 67.86%, 80.95%, 50.00%, and 37.93%, respectively. Most of the subjects had a heart rate between 60 and 100 beats/min (87.60%); those with HR <60, 60–100, and >100 with low risk of OSA were 80.00%, 57.00%, and 50.00%, respectively.

From Table 6, in predicting the risk of OSA by age, comparing subjects with low risk to those with high risk of OSA it can be deduced that as age increases the risk to having low risk of OSA decreases by 0.919 times, whereas by comparing subjects with moderate risk to those with high risk of OSA that as age increases the risk to OSA decreases by 0.955 times.

Table 1 Sociodemographic and lifestyle characteristics of the study subjects by risk of OSA

| | Parameters | | Total percentage (N = 110) | Percentage by risk of OSA |
|---|----------------------------|-----------------------|-------------------------------|---|
| 1 | Age | 18–20 | 17.50 | Low risk—80.00% Medium risk—20.00% High risk—0.00% |
| | | 21–30 | 12.30 | Low risk—71.42% Medium risk—21.42% High risk—7.14% |
| | | 31–40 | 24.60 | Low risk—53.57% Medium risk—42.85% High risk—3.57% |
| | | 41–50 | 22.80 | Low risk—53.84% Medium risk—42.30% High risk—3.84% |
| | | 51–60 | 14.00 | Low risk—37.50% Medium risk—43.75% High risk—18.75% |
| | | 61–70 | 5.30 | Low risk—33.33% Medium risk—33.33% High risk—33.33% |
| | | 71–80 | 2.60 | Low risk—25.00% Medium risk—50.00% High risk—25.00% |
| | | 81–90 | 0.90 | Low risk—100.00% Medium risk—0.00% High risk—0.00% |
| 2 | Gender | Female | 46.50 | Low risk—67.92% Medium risk—30.18% High risk—1.88% |
| | | Male | 53.50 | Low risk—47.54% Medium risk—40.98% High risk—11.47% |
| 3 | Smoking status | Yes | 13.20 | Low risk—75.00% Medium risk—25.00% High risk—0.00% |
| | | No | 86.80 | Low risk—54.90% Medium risk—37.25% High risk—7.84% |
| 4 | Alcohol consumption status | Regularly | 18.40 | Low risk—42.85% Medium risk—35.71% High risk—21.43% |
| | | Occasionally | 38.60 | Low risk—58.70% Medium risk—35.95% High risk—4.34% |
| | | Never | 43.00 | Low risk—59.25% Medium risk—35.19% High risk—5.56% |
| 5 | Nature of job | Physically exhausting | 38.60 | Low risk—48.88% Medium risk—44.44% High risk—6.66% |
| | | Moderate activity | 43.00 | Low risk—61.66% Medium risk—31.66% High risk—6.66% |
| | | Sedentary | 18.40 | Low risk—66.67% Medium risk—22.22% High risk—11.11% |

Table 2 Risk of the study subjects to OSA across age groups by gender

| Parameter | | Percentage by risk of OSA | |
|-----------|-------|---------------------------|--|
| Age | 18–20 | Female | Low risk—91.66% Medium risk—8.33% High risk—0.00% |
| | | Male | Low risk—71.43% Medium risk—28.57% High risk—0.00% |
| 21–30 | 21–30 | Female | Low risk—50.00% Medium risk—50.00% High risk—0.00% |
| | | Male | Low risk—75.00% Medium risk—16.66% High risk—8.33% |
| 31–40 | 31–40 | Female | Low risk—60.00% Medium risk—40.00% High risk—0.00% |
| | | Male | Low risk—46.15% Medium risk—46.15% High risk—7.70% |
| 41–50 | 41–50 | Female | Low risk—60.00% Medium risk—40.00% High risk—0.00% |
| | | Male | Low risk—50.00% Medium risk—43.75% High risk—6.25% |
| 51–60 | 51–60 | Female | Low risk—66.67% Medium risk—33.33% High risk—0.00% |
| | | Male | Low risk—0.00% Medium risk—57.14% High risk—42.86% |
| 61–70 | 61–70 | Female | Low risk—66.67% Medium risk—33.33% High risk—0.00% |
| | | Male | Low risk—0.00% Medium risk—66.67% High risk—33.33% |
| 71–80 | 71–80 | Female | Low risk—50.00% Medium risk—50.00% High risk—0.00% |
| | | Male | Low risk—0.00% Medium risk—100.00% High risk—0.00% |
| 81–90 | 81–90 | Female | – |
| | | Male | Low risk—100.00% Medium risk—0.00% High risk—0.00% |

In predicting the risk of OSA by gender in the study subjects, it can be deduced with reference to the male gender the risk of having a low risk as to having high risk of OSA is 15.268 times, whereas the risk of having moderate risk of OSA as against high risk for a female subject as to a male subject is 6.288 times.

In predicting the risk of OSA by smoking status, it can be deduced with reference to those that smoke that there

is low risk of OSA for a non-smoker compared to having a high risk is reduced by $1.489E - 007$ times, whereas the risk to moderate risk of OSA for a non-smoker compared to having a high risk is reduced by $3.419E - 007$ times.

In predicting the risk of OSA by nature of alcohol consumption, it can be deduced with reference to those that drink regularly that there is low risk of OSA for a non-drinker compared to having a high risk being 6.514 times and those that drink occasionally being 9.721 times to having low risk as to having high risk, whereas the risk to moderate risk of OSA for a non-consumer of alcohol compared to having a high risk being 9.446 times and those that drink occasionally being 9.186 times to having low risk as to having high risk.

In predicting the risk of OSA by nature of job of the subject, it can be deduced that with reference to those that engage in sedentary jobs that there is low risk of OSA for a those with physically exhausting jobs to having a high risk being 3.531 times and those that have moderate activity jobs being 1.019 times to having low risk as to having high risk, whereas the risk to moderate risk of OSA for those with physically exhausting jobs compared to having a high risk being 10.434 times and those that with moderate activity jobs being 1.847 times to having low risk as to having high risk of OSA.

From Table 7, in predicting the risk of OSA by waist circumference, it can be deduced that as WC increases, the tendency to having low risk decreases by a factor of 0.987, whereas the tendency of having moderate risk as to having high risk of OSA increases by a factor of 1.005.

In predicting the risk of OSA by BMI, it can be deduced that as BMI increases, the tendency to having low risk as to having high risk of OSA decreases by 0.936, whereas the tendency of having moderate risk to having high risk of OSA decreases by a factor of 0.962.

In predicting the risk of OSA by BAI, it can be deduced that as BAI increases, the tendency of having low risk as to having high risk of OSA increases by 1.039, whereas the tendency of having moderate risk to having high risk of OSA decreases by a factor of 0.995.

In predicting the risk of OSA by WHR, it can be deduced that as WHR increases, the tendency of having low risk as to having high risk of OSA increases by 2.570, whereas the tendency of having moderate risk to having high risk of OSA decreases by a factor of 0.349.

In predicting the risk of OSA by WHtR, it can be deduced that as WHtR increases, the tendency of having low risk as to having high risk of OSA decreases by 0.162 units, whereas the tendency of having moderate risk to having high risk of OSA increases by a factor of 4.845.

In predicting the risk of OSA by NHtR, it can be deduced that as NHtR increases, the tendency of having low risk as to having high risk of OSA increases by 3.414

Table 3 Anthropometric characteristics of the study subjects by risk of OSA

| Parameters | | | Total percentage (N= 110) | Percentage by risk of OSA |
|------------|----------------------|-------------|---------------------------|---|
| 1 | Waist circumference | 61–70 cm | 10.50 | Low risk—83.33% Medium risk—16.67% High risk—0.00% |
| | | 71–80 cm | 26.30 | Low risk—67.74% Medium risk—29.03% High risk—3.22% |
| | | 81–90 cm | 25.50 | Low risk—53.57% Medium risk—39.28% High risk—7.14% |
| | | Above 90 cm | 37.70 | Low risk—42.11% Medium risk—45.61% High risk—12.28% |
| 2 | Waist hip ratio | < 1 | 88.60 | Low risk—59.43% Medium risk—34.91% High risk—5.66% |
| | | > 1 | 11.40 | Low risk—25.00% Medium risk—50.00% High risk—25.00% |
| 3 | Waist height ratio | < 1 | 95.6 | Low risk—58.03% Medium risk—34.82% High risk—7.14% |
| | | > 1 | 4.40 | Low risk—0.00% Medium risk—100.00% High risk—0.00% |
| 4 | Neck height ratio | < 20 cm/m | 14.00 | Low risk—68.18% Medium risk—31.82% High risk—0.00% |
| | | > 20 cm/m | 86.00 | Low risk—54.34% Medium risk—36.95% High risk—8.69% |
| 5 | Body adiposity index | < 10% | 6.10 | Low risk—57.14% Medium risk—42.85% High risk—0.00% |
| | | 10–25% | 28.10 | Low risk—53.33% Medium risk—40.00% High risk—6.67% |
| | | > 25% | 65.80 | Low risk—58.44% Medium risk—33.76% High risk—7.79% |
| 6 | Body mass index | < 18.50 | 9.60 | Low risk—50.00% Medium risk—37.50% High risk—12.50% |
| | | 18.50–24.90 | 40.40 | Low risk—63.26% Medium risk—34.70% High risk—2.04% |
| | | 25.00–29.90 | 21.10 | Low risk—80.95% Medium risk—9.52% High risk—9.52% |
| | | > 29.90 | 28.90 | Low risk—36.11% Medium risk—52.77% High risk—11.11% |

units, whereas the tendency of having moderate risk to having high risk of OSA increases by a factor of 2.684.

From the foregoing only BMI, NC, and NHtR are the only anthropometric indices that adequately predicted the risk of OSA in comparing low risk and medium risk

to high risk as reference with an increase in BMI and NC implying a higher risk of OSA while an increase in NHtR implying a lower risk of OSA.

From Table 8, in predicting the risk of OSA by SBP, it can be deduced that as SBP increases, the tendency of

Table 4 Risk of the study subjects to OSA using different anthropometric indices by gender

| Parameters | | | | Percentage by risk of OSA | | |
|------------|---------------------|-----------------|---|---|---|---|
| 1 | Waist circumference | 61–70 cm | Female | Low risk—100.00% Medium risk—0.00% High risk—0.00% | | |
| | | | Male | Low risk—66.67% Medium risk—33.33% High risk—0.00% | | |
| | | 71–80 cm | Female | Low risk—83.33% Medium risk—16.67% High risk—0.00% | | |
| | | | Male | Low risk—62.50% Medium risk—33.33% High risk—4.17% | | |
| | | 81–90 cm | Female | Low risk—84.62% Medium risk—15.38% High risk—0.00% | | |
| | | | Male | Low risk—31.25% Medium risk—56.25% High risk—12.50% | | |
| | Above 90 cm | Female | Low risk—50.00% Medium risk—46.43% High risk—3.57% | | | |
| | | Male | Low risk—33.33% Medium risk—40.00% High risk—26.67% | | | |
| | 2 | Waist hip ratio | < 1 | Female | Low risk—72.92% Medium risk—25.00% High risk—2.08% | |
| | | | | Male | Low risk—50.00% Medium risk—41.07% High risk—8.93% | |
| | | | > 1 | Female | Low risk—20.00% Medium risk—80.00% High risk—0.00% | |
| | | | | Male | Low risk—20.00% Medium risk—40.00% High risk—40.00% | |
| 3 | | | Waist height ratio | < 1 | Female | Low risk—70.59% Medium risk—27.45% High risk—1.96% |
| | | | | | Male | Low risk—49.15% Medium risk—38.98% High risk—11.86% |
| | > 1 | Female | | Low risk—0.00% Medium risk—100.00% High risk—0.00% | | |
| | | Male | | Low risk—0.00% Medium risk—100.00% High risk—0.00% | | |
| 4 | Neck height ratio | < 20 cm/m | Female | Low risk—55.56% Medium risk—44.44% High risk—0.00% | | |
| | | | Male | Low risk—71.43% Medium risk—28.57% High risk—0.00% | | |
| | | > 20 cm/m | Female | Low risk—70.46% Medium risk—27.27% High risk—2.27% | | |
| | | | Male | Low risk—44.44% Medium risk—42.59% High risk—2.96% | | |

Table 4 (continued)

| Parameters | | | Percentage by risk of OSA | |
|------------|-----|-----------|---------------------------|---|
| 5 | BAI | < 10% | Female | Low risk—75.00% Medium risk—25.00% High risk—0.00% |
| | | | Male | Low risk—33.33% Medium risk—66.67% High risk—0.00% |
| | | 10–25% | Female | Low risk—33.33% Medium risk—66.67% High risk—0.00% |
| | | | Male | Low risk—55.17% Medium risk—34.48% High risk—10.34% |
| | | > 25% | Female | Low risk—69.57% Medium risk—28.26% High risk—2.17% |
| | | | Male | Low risk—41.38% Medium risk—44.83% High risk—13.79% |
| 6 | BMI | < 18.5 | Female | Low risk—100.00% Medium risk—0.00% High risk—0.00% |
| | | | Male | Low risk—42.86% Medium risk—42.86% High risk—14.28% |
| | | 18.5–24.9 | Female | Low risk—76.92% Medium risk—23.08% High risk—0.00% |
| | | | Male | Low risk—55.88% Medium risk—41.18% High risk—2.94% |
| | | 25–29 | Female | Low risk—85.71% Medium risk—7.14% High risk—7.14% |
| | | | Male | Low risk—71.43% Medium risk—14.28% High risk—14.28% |
| | | > 29 | Female | Low risk—47.83% Medium risk—52.17% High risk—0.00% |
| | | | Male | Low risk—15.38% Medium risk—53.85% High risk—30.77% |

having low risk as to having high risk of OSA decreases by 0.973 units, whereas the tendency of having moderate risk to having high risk of OSA remains unchanged.

In predicting the risk of OSA by DBP, it can be deduced that as DBP increases, the tendency of having low risk as to having high risk of OSA decreases by 0.948 units, whereas the tendency of having moderate risk to having high risk of OSA decreases by 0.948.

In predicting the risk of OSA by HR, it can be deduced that as HR increases, the tendency of having low risk as to having high risk of OSA decreases by 0.928 units,

whereas the tendency of having moderate risk to having high risk of OSA decreases by 0.963.

DBP and HR adequately predicted the risk of OSA, with an increase in DBP and HR implying higher risk of OSA.

Discussion

Association of male gender with risk of OSA has been described. In this study, the prevalence of low risk of OSA is higher in women compared to men and appears to rise with age as females have a lower risk

Table 5 Cardiovascular indices of the study subjects by risk of OSA

| | Parameters | | Total percentage (N=110) | Percentage by risk of OSA |
|---|--------------------------|---------|--------------------------|---|
| 1 | Systolic blood pressure | < 110 | 24.60 | Low risk—67.86% Medium risk—32.14% High risk—0.00% |
| | | 110–120 | 18.40 | Low risk—80.95% Medium risk—14.29% High risk—4.76% |
| | | 121–140 | 31.60 | Low risk—50.00% Medium risk—47.22% High risk—2.78% |
| | | > 140 | 25.40 | Low risk—37.93% Medium risk—41.38% High risk—20.69% |
| 2 | Diastolic blood pressure | < 80 | 48.20 | Low risk—81.13% Medium risk—26.42% High risk—37.75% |
| | | 81–90 | 27.20 | Low risk—48.15% Medium risk—48.15% High risk—3.70% |
| | | 91–100 | 14.10 | Low risk—43.75% Medium risk—50.00% High risk—6.25% |
| | | > 100 | 10.50 | Low risk—16.67% Medium risk—50.00% High risk—33.33% |
| 3 | Heart rate | < 60 | 5.30 | Low risk—80.00% Medium risk—20.00% High risk—0.00% |
| | | 60–100 | 87.60 | Low risk—57.00% Medium risk—36.00% High risk—7.00% |
| | | > 100 | 7.10 | Low risk—50.00% Medium risk—37.50% High risk—12.50% |

of OSA across all age groups except the age brackets of 21–30 years. This finding does not support the role of hormones such as estrogen and progesterone being responsible for increasing the risk of OSA by age as the finding does not suggest this since there is no bell-shaped relationship in the risk of OSA of females across age [25]. The anatomical distribution of fat could be used to explain the sex-dimorphic vulnerability of OSA, with fat being around the neck and waist in adult males and around the hips in adult females [13]. Anatomical differences in the pharyngeal and upper airway structure may contribute partially to the higher susceptibility to OSA in males compared to females [26, 27]. In this study, it was discovered that for every year increase in age, having a low risk of OSA decreases by a factor of 8.1% (OR=0.919). This increased risk with age can be attributed to age-related anatomical changes in the pharynx which lead to increased upper airway collapsibility [28, 29]. Another study confirms that age difference in the risk of OSA is independent of BMI and gender [30].

In this study, BMI, NC, and NHtR are the anthropometric indices that adequately predict the risk of OSA in comparing low and medium risk to high risk, an increase in BMI implying a higher risk of OSA while an increase in NHtR implying a lower risk of OSA. In a study by Awopeju et al. [31], BMI, WC, hip circumference, and waist-to-height ratio performed similarly in predicting high risk of OSA. Cardiovascular variables of DBP and HR predict the severity of OSA; this is like other studies that associated OSA with an increase in the prevalence and incidence of arterial hypertension and cardiovascular diseases (CVD) [18, 19]. Individuals suffering from OSA exhibit a greater occurrence of isolated diastolic hypertension [32]. This is potentially due to tachycardia and shortening of cardiac diastole. Hypertension, in turn, may be brought on by OSA through hypoxemia and hypercapnia, which lead to systemic inflammation and oxidative stress. This process results in the generation of increased levels of endothelin-1 and decreased production of nitric oxide in endothelial cells, increasing arterial peripheral resistance and elevating blood

Table 6 Risk of OSA by sociodemographic and lifestyle factors

| | Variables by risk of OSA | Sub-variables | Odds ratio | 95% confidence interval | Sig |
|------------------------|--------------------------|-----------------------|------------|-------------------------|--------|
| Age | | | | | |
| 1 | Low risk–high risk | | 0.919 | 0.864–0.979 | 0.008 |
| | Moderate risk–high risk | | 0.955 | 0.899–1.015 | 0.142 |
| Gender | | | | | |
| 2 | Low risk–high risk | Female | 15.268 | 1.253–185.982 | 0.033 |
| | | Male | Reference | – | – |
| | Moderate risk–high risk | Female | 6.288 | 0.158–76.33 | 0.149 |
| | | Male | Reference | – | – |
| Current smoking status | | | | | |
| 3 | Low risk–high risk | No | 1.489E–007 | 2.697E–008–8.221E–008 | 0.001 |
| | | Yes | Reference | – | – |
| | Moderate risk–high risk | No | 3.419E–007 | 3.4197E–007–3.419E–007 | – |
| | | Yes | Reference | – | – |
| Alcohol consumption | | | | | |
| 4 | Low risk–high risk | Never | 6.514 | 0.490–86.667 | 0.156 |
| | | Occasionally | 9.721 | 0.792–119.321 | 0.075 |
| | | Regularly | Reference | – | – |
| | Moderate risk–high risk | Never | 9.446 | 0.714–125.060 | 0.088 |
| | | Occasionally | 9.186 | 0.739–114.243 | 0.085 |
| | | Regularly | Reference | – | – |
| Nature of job | | | | | |
| 5 | Low risk–high risk | Physically exhausting | 3.531 | 0.121–102.894 | 0.478 |
| | | Moderate activity | 1.019 | 0.055 | 19.055 |
| | | Sedentary | Reference | – | – |
| 5 | Moderate risk–high risk | Physically exhausting | 10.434 | 0.353–308.561 | 0.175 |
| | | Moderate activity | 1.847 | 0.097–35.006 | 0.683 |
| | | Sedentary | Reference | – | – |

Table 7 Risk of OSA by anthropometric factors

| | Variables by risk of OSA | Odds ratio | 95% confidence interval | Sig |
|----------------------|--------------------------|------------|-------------------------|-------|
| Waist circumference | | | | |
| 1 | Low risk–high risk | 0.987 | 0.956–1.020 | 0.443 |
| | Moderate risk–high risk | 1.005 | 0.975–1.036 | 0.734 |
| Body mass index | | | | |
| 2 | Low risk–high risk | 0.936 | 0.804–1.091 | 0.398 |
| | Moderate risk–high risk | 0.962 | 0.826–1.121 | 0.622 |
| Body adiposity index | | | | |
| 3 | Low risk–high risk | 1.039 | 0.936–1.153 | 0.470 |
| | Moderate risk–high risk | 0.995 | 0.897–1.104 | 0.923 |
| Waist hip ratio | | | | |
| 4 | Low risk–high risk | 2.570 | 0.024–277.378 | 0.693 |
| | Moderate risk–high risk | 0.349 | 0.003–47.784 | 0.675 |
| Waist height ratio | | | | |
| 5 | Low risk–high risk | 0.162 | 0.001–115.127 | 0.587 |
| | Moderate risk–high risk | 4.845 | 0.005–4436.919 | 0.650 |
| Neck height ratio | | | | |
| 6 | Low risk–high risk | 3.414 | 0.065–179.027 | 0.543 |
| | Moderate risk–high risk | 2.684 | 0.050–114.112 | 0.627 |

Table 8 Risk of OSA by cardiovascular variables

| | Variables by risk of OSA | Odds ratio | 95% confidence interval | Sig |
|--------------------------|--------------------------|------------|-------------------------|-------|
| Systolic blood pressure | 1 Low risk–high risk | 0.973 | 0.930–1.019 | 0.247 |
| | Moderate risk–high risk | 1.000 | 0.960–1.042 | 0.983 |
| Diastolic blood pressure | 2 Low risk–high risk | 0.939 | 0.856–1.030 | 0.182 |
| | Moderate risk–high risk | 0.948 | 0.866–1.038 | 0.247 |
| Heart rate | 3 Low risk–high risk | 0.928 | 0.861–0.999 | 0.047 |
| | Moderate risk–high risk | 0.963 | 0.895–1.035 | 0.305 |

pressure [33–35]. Additionally, subjects with OSA have significantly higher renin generation, which is induced by efferent renal sympathetic nerve activation. This effect results in elevated plasma angiotensin-II and aldosterone, ultimately leading to increased vasoconstriction and sodium-water retention, respectively [35], both of which contribute to higher blood pressure levels.

Our study focused on clinical predictor of OSA in a general population; however, several studies have investigated predictors of obstructive sleep apnea (OSA) in various populations, identifying both common and distinct risk factors. Xu et al. [36] focused on patients with hypertrophic cardiomyopathy (HCM) and identified older age, male gender, higher BMI, hypertension, and left ventricular outflow tract obstruction as significant predictors of OSA, aligning with the broader understanding of age, male gender, and BMI as key risk factors. Conversely, Cadavid et al. [37] explored OSA predictors in nonobese individuals, finding age and male gender as the only independent predictors, challenging the traditional profile of older, obese, and frequent snorers. Peruvemba et al. [38] emphasized the utility of clinical parameters such as age, sex, and neck circumference in identifying OSA risk, while Jonassen et al. [39] proposed a prediction score based on age, snoring, breathing cessations, BMI, and hypertension for severe OSA.

Our study contributes insights into OSA prediction, diverging from some previous studies by highlighting the significance of anatomical differences in fat distribution and upper airway structure. We found that males exhibit higher vulnerability to OSA due to fat accumulation around the neck and waist, with age-related changes in the pharynx exacerbating risk. Our observations challenge the notion that hormonal factors alone account for lower OSA prevalence in females, suggesting anatomical differences play an equally important role. Comparing with prior research, while age consistently emerges as a

predictor, our focus on anatomical factors offers nuanced understanding of gender differences in OSA vulnerability. Overall, our findings underscore the multifaceted nature of OSA risk factors, emphasizing the importance of comprehensive approaches to prediction. Like most questionnaire-based studies, overestimation or underestimation may be an issue, but it is hoped that the findings of this study will add to the body of knowledge on this subject.

Conclusion

Obstructive sleep apnea can be caused by several risk factors such as age, gender, obesity, craniofacial and oropharyngeal anatomical defects, and personal habits such as smoking and alcohol consumption. Likewise, certain anthropometric measures have also been revealed to be a cause of obstructive sleep apnea; measures like BMI and NHTr have a high predictive value to cause OSA. To reduce the incidence of OSA, it is recommended that the identified anthropometric properties should be monitored over time and measures should be put in place to ensure good respiratory health.

Acknowledgements

Not applicable.

Authors' contributions

S.O.O. and I.O.O. conceptualized the study; S.O.O., I.O.O., P.G.O., O.E.A., M.M.A., and B.O.A. wrote the manuscript. E.N.E., P.G.O., E.V.B., O.D.O., D.D.T., and B.T. edited and checked the manuscript. All authors contributed to the article and approved the submitted version. All authors have read and agreed to the published version of the manuscript.

Funding

Not applicable.

Availability of data and materials

All data generated or analyzed during this study are included in this article.

Declarations

Ethics approval and consent to participate

Approval for this study was obtained from the Health Research Ethics Committee, Olabisi Onabanjo University Teaching Hospital with ethical clearance number OOUTH/HREC/658/2023AP.

Competing interests

The authors declare no conflict of interest.

Received: 28 December 2023 Accepted: 22 May 2024

Published: 24 June 2024

References

- Tufik S, Santos-Silva R, Taddei JA, Bittencourt LR (2010) Obstructive sleep apnea syndrome in the Sao Paulo Epidemiologic Sleep Study. *Sleep Med* 11(5):441–446
- Spicuzza L, Caruso D, Di Maria G (2015) Obstructive sleep apnoea syndrome and its management. *Ther Adv Chronic Dis* 6:273–285
- Vaessen TJ, Overeem S, Sitskoorn MM (2015) Cognitive complaints in obstructive sleep apnea. *Sleep Med Rev* 19:51–58

4. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP et al (2009) Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 5(3):263–276
5. Ljunggren M, Byberg L, Theorell-Haglöw J, Lindahl B, Michaëlsson K, Lindberg E (2016) Increased risk of heart failure in women with symptoms of sleep-disordered breathing. *Sleep Med* 17:32–37
6. Drager LF, Lorenzi-Filho G (2012) CPAP for obstructive sleep apnea and the metabolic syndrome. *N Engl J Med* 366(10):964
7. Dempsey J, Veasey S, Morgan B, O'Donnell C (2010) Pathophysiology of sleep apnea. *Physiol Rev* 90:47–112
8. Ahmad AN, McLeod G, Al Zahrani N, Al Zahrani H (2019) Screening for high risk of sleep apnea in an ambulatory care setting in Saudi Arabia. *Int J Environ Res Public Health* 16:459
9. Salvador J, Iriarte J, Silva C, Gomez-Ambrosi J, Diez-Caballero A et al (2004) El síndrome de apneas obstructivas del sueño en la obesidad: Un conspirador en la sombra. *Rev Med Univ Navarra* 48:55–62
10. Sharma H, Sharma SK (2008) Overview and implications of obstructive sleep apnoea. *Indian J Chest Dis Allied Sci* 50:137–150
11. Popovic RM, White DP (1998) Upper airway muscle activity in normal women: influence of hormonal status. *J Appl Physiol* 84:1055–1062
12. Bixler EO, Vgontzas AN, Lin HM et al (2001) Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med* 163:608–613
13. Folsom AR, Stevens J, Schreiner PJ, McGovern PG (1998) Body mass index, waist/hip ratio, and coronary heart disease incidence in African Americans and whites. Atherosclerosis Risk in Communities Study Investigators. *Am J Epidemiol* 148:1187–1194
14. Serafini FM, MacDowell Anderson W, Rosemurgy AS, Strait T, Murr MM (2001) Clinical predictors of sleep apnea in patients undergoing bariatric surgery. *Obes Surg* 11:28–31
15. Magalhães EI, Sant'Ana LF, Priore SE et al (2014) Waist circumference, waist/height ratio, and neck circumference as parameters of central obesity assessment in children. *Rev Paul Pediatr*. 32(3):273–281
16. Cho JH, Choi JH, Suh JD et al (2016) Comparison of anthropometric data between Asian and Caucasian patients with obstructive sleep apnea: a meta-analysis. *Clin Exp Otorhinolaryngol* 9(1):1–7
17. Mabchour AEL, Delisle H, Vilgrain C et al (2015) Specific cut-off points for waist circumference and waist-to-height ratio as predictors of cardio-metabolic risk in Black subjects: a cross-sectional study in Benin and Haiti. *Diabetes Metab Syndr Obes* 8:513–523
18. Fuchs FD, Martinez D (2015) Obstructive sleep apnoea should be deemed a cardiovascular disease. *Heart* 101(16):1261–1262
19. Cai A, Wang L, Zhou Y. Hypertension and obstructive sleep apnea. *Hypertens Res*. 2016. LID - <https://doi.org/10.1038/hr.2016.11>.
20. Unnikrishnan D, Jun J, Polotsky V (2015) Inflammation in sleep apnea: an update. *Rev Endocr Metab Disord* 16:25–34
21. Qaseem A, Dallas P, Owens DK, Starkey M, Holty JE, Shekelle P (2014) Clinical Guidelines Committee of the American College of Physicians. Diagnosis of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. *Ann Intern Med* 161:210–20
22. Chung F, Abdullah HR, Liao P (2016) STOP-Bang questionnaire: a practical approach to screen for obstructive sleep apnea. *Chest* 149:631–638
23. Okorodudu DO, Jumean MF, Montori VM et al (2010) Diagnostic performance of body mass index to identify obesity as defined by body adiposity: a systematic review and meta-analysis. *Int J Obes* 34(5):791–799
24. Ledoux S, Coupaye M, Essig M et al (2010) Traditional anthropometric parameters still predict metabolic disorders in women with severe obesity. *Obesity* 18(5):1026–1032
25. Ayub S, Won CH (2019) Obstructive sleep apnea in women. *Journal of sleep medicine* 16(2):75–80
26. Schellenberg JB, Maislin G, Schwab RJ (2000) Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures. *Am J Respir Crit Care Med* 162:740–748
27. Schwab J (1999) Sex differences and sleep apnoea. *Thorax* 54:284–285
28. Malhotra A, Huang Y, Fogel R et al (2006) Aging influences on pharyngeal anatomy and physiology: the predisposition to pharyngeal collapse. *Am J Med*. 119(1):72.e9-72.e14. <https://doi.org/10.1016/j.amjmed.2005.01.077>
29. Schwab RJ (2003) Pro: sleep apnea is an anatomic disorder. *Am J Respir Crit Care Med* 168:270–271
30. Eikermann M, Jordan AS, Chamberlin NL et al (2007) The influence of aging on pharyngeal collapsibility during sleep. *Chest* 131(6):1702–1709. <https://doi.org/10.1378/chest.06-2653>
31. Awopeju OF, Fawale MB, Anu S, Salami OT, Adewole OO (2020) The risk of obstructive sleep apnea and its association with indices of general and abdominal obesity in a Nigerian family practice clinic: a cross-sectional study. *Alex J Med* 56(1):14–20. <https://doi.org/10.1080/20905068.2019.1711304>
32. Baguet JP, Hammer L, Levy P, Pierre H, Rossini E, Mouret S, Ormezzano O, Mallion JM, Pepin JL (2005) Night-time and diastolic hypertension are common and underestimated conditions in newly diagnosed apnoeic patients. *J Hypertens* 23:521–527
33. Garvey JF, Taylor CT, McNicholas WT (2009) Cardiovascular disease in obstructive sleep apnoea syndrome: the role of intermittent hypoxia and inflammation. *Eur Respir J* 33:1195–1205
34. Atkeson A, Yeh SY, Malhotra A, Jelic S (2009) Endothelial function in obstructive sleep apnea. *Prog Cardiovasc Dis* 51:351–362
35. Cai A, Wang L, Zhou Y (2016) Hypertension and obstructive sleep apnea. *Hypertens Res* 39:391–395. <https://doi.org/10.1038/hr.2016.11>
36. Xu H, Wang J, Yuan J, Guo C, Hu F, Yang W, Song L, Luo X, Liu R, Cui J, Liu S (2021) Clinical predictors of the presence of obstructive sleep apnea in patients with hypertrophic cardiomyopathy. *Sci Rep* 11(1):13528
37. Cadavid JC, Ganesan M, Braitman L (2009) Clinical predictors of obstructive sleep apnea in nonobese individuals: are we heading towards universal screening? *Chest*. 136(4_MeetingAbstracts):59S-g. <https://doi.org/10.7860/JCDR/2012/2247.4051>
38. Peruvemba HL, Thazhepurayil R, Ponneduthamkuzhi J, Chetambath R (2012) Clinical prediction of obstructive sleep apnea (OSA) in a tertiary care setting. *J Clin Diagn Res* 6(5):833–837. <https://doi.org/10.7860/JCDR/2012/2247.4051>
39. Jonassen TM, Bjorvatn B, Saxvig IW, Eagan TML, Lehmann S (2022) Clinical information predicting severe obstructive sleep apnea: a cross-sectional study of patients waiting for sleep diagnostics. *Respir Med* 193:106860. <https://doi.org/10.1016/j.rmed.2022.106860>

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.