

Obstructive Sleep Apnea as a Risk Factor for Ischemic Heart Disease

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Abstract

Background: Obstructive sleep apnea (OSA) is increasingly recognized as a potential risk factor for ischemic heart disease (IHD), but its role remains underexplored. This study investigated the association between OSA and IHD using a case-control design.

Objectives: The objective of this study was to establish OSA as a risk factor for developing IHD.

Methods: A total of 122 participants were recruited and categorized into two groups: patients with a recent history of IHD (< 7 days) (wIHD group, n = 61) and controls without IHD but with hypertension (cIHD group, n = 61). The STOP-Bang questionnaire was used to assess OSA risk, and statistical analyses, including odds ratio (OR), relative risk (RR), and attributable risk (AR), were performed. A significance level of 0.05 was adopted for all statistical analyses.

Results: Patients in the wIHD group had significantly higher STOP-Bang scores than the control group ($p < 0.01$), with an AR of 71.929% and an OR of 40.078. Body mass index showed a significant positive correlation with OSA risk ($p = 0.0016$, $r = 0.396$). The OR/RR was calculated at 11.25 (> 1). The level of statistical significance was found to be 0.05.

Conclusion: This study identifies OSA as an important risk factor for IHD, supporting the need for routine OSA screening in cardiovascular risk assessment. Larger studies are needed to further explore this relationship

Keywords: Myocardial Ischemia; Obstructive Sleep Apnea; Case-Control Studies; Risk Factors.

Introduction

Ischemic heart disease (IHD) has been widely and repetitively studied in medical science from morphological and clinical perspectives. However, the correlation of IHD or ischemia as a pathological process with obstructive sleep apnea (OSA) has long been neglected from the aspects of research in clinical medicine until recently. Studies have already shown OSA to be related with chronic heart failure. The pathogenesis of this phenomenon has been investigated to be fat deposition on the upper body, including visceral fat, tongue, and throat.¹ Moreover, fluid translocation from the lower extremities to the neck has been associated with vascular congestion and edema of the pharyngeal area, and these morphological changes contribute to exacerbation of decompensation in heart failure.² From the pathophysiology point of view, micro RNAs play a vital role in development of OSA.³ In the development of

IHD, the role of micro RNAs has been widely discussed in molecular biology, although a significant lack of understanding of the signaling pathways of pathogenesis persists.⁴ In a study by Nir Peled et al., an increased chance of nocturnal ischemic events was noted in patients with OSAs, indicating towards a viable but less understood correlation between the two conditions.⁵ One of the most vital mechanisms might be embedded in the availability of oxygen during different phases of the sleep cycle. Multiple studies using the ventilation hood method have shown that oxygen availability drastically decreases during the rapid eye movement (REM) phase of the sleep cycle.⁶ Cardiologists and clinicians of other specialties have widely observed that the highest number of myocardial infarctions and most massive myocardial infarctions happen during early morning hours,⁷ implying that these cardiovascular events are more likely to happen during the REM phase of sleep. This is a nested case-control study conducted in the department of cardiology at the City Clinic Hospital No. 31 named after academician G.M Savelyova, in Moscow, Russian Federation. The study aimed to understand the correlation between IHD and OSA by assessing the risk of developing OSA in patients with IHD in a timeframe of less than one week after stabilization. This study is unique in its nature as a case-control study; it determines the risk OSA predisposes to IHD, validating the exposure and incidence relation between two factors. The Central Illustration displays a synopsis of the findings.

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Central Illustration: Obstructive Sleep Apnea as a Risk Factor for Ischemic Heart Disease



Risk of IHD development in OSA

Results of a case-control study

EXPERIMENTAL GROUP

93.44% developed OSA
6.55% did not develop OSA

CONTROL GROUP

26.22% developed OSA
73.77% did not develop OSA

RR = 3.56 OR = 40.07 OR/RR > 1

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Obstructive Sleep Apnea as a Risk Factor for Ischemic Heart Disease Synopsis of risk of developing ischemic heart diseases in obstructive sleep apnea. IHD: ischemic heart disease; OR: odds ratio; OSA: obstructive sleep apnea; RR: relative risk.

Method

In this study, patients hospitalized in the cardiology department of City Clinic Hospital No. 31, Moscow with a recent history of IHD not exceeding 7 days were included. Patients included were diagnosed with ST-segment elevation myocardial infarction, non-ST-segment elevation myocardial infarction, stable angina, and unstable angina. The clinical data of these patients were compared with patients without IHD in their anamnesis. For assessment of risk of developing OSA, the STOP-Bang score questionnaire was used, and patients were interviewed individually by the authors with signed consent. In this questionnaire, subjects are assessed on the following eight criteria: snoring, tiredness or fatigability, observed stopped breathing or choking during sleep, arterial blood pressure, body mass index (BMI) > 35 kg/m², age older than 50 years, neck size measured around Adam's apple or the laryngeal prominence of the thyroid cartilage, and sex (with male gaining one point). On the score system, 0 to 2 indicates a low risk of developing OSA; 3 to 4 indicates an intermediate risk of developing OSA; and 5 to 8 indicates a high risk of developing OSA.⁸ A retrospective study and comparison of individual patient histories including anamnesis and comorbidities was also carried out simultaneously.

Statistical analysis

A total of 122 patients (N = 122) were assessed in this study, as calculated statistical power was observed to be greater than 99.99% with this sample size, and the sample size taken is greater than in previously conducted studies on the

topic. In order to conduct a case-control study, the patients were divided into two equal groups: 61 patients with recent history of IHD were grouped and denominated "wIHD," and the control group consisted of 61 patients without any history of IHD but with chronic arterial hypertension, denominated "cIHD." Chronic arterial hypertension was chosen as a baseline characteristic as it is a comorbidity factor and, in this case, a dependent variable. Continuous variables that follow a normal distribution are presented as the mean plus or minus the standard deviation. On the other hand, continuous variables without a normal distribution are presented as the median and interquartile range (IQR). Categorical variables are expressed as the count and corresponding percentage. Normality of continuous variables was evaluated using the Shapiro-Wilk test. Differences in proportions between groups were assessed with the two-proportion Z test. Receiver operating characteristic (ROC) curve analysis was conducted to determine the diagnostic accuracy of the STOP-Bang score. Relationships between continuous variables were examined using Pearson's correlation coefficient and simple linear regression. Associations between categorical variables were tested with the chi-square test. All statistical analyses were carried out in STATSOFT Statistica version 13.3,⁹ adopting a significance level (alpha) of 0.05. There were no missing data for the variables analyzed.

Results

This case-control study obtained statistically significant results.

The STOP-Bang score was selected as the primary screening tool after analyzing the study model based on ROC curve analysis. As illustrated in Figure 1, the area under the curve was 0.974, indicating that the STOP-Bang score has excellent diagnostic accuracy for identifying participants with IHD. The ROC curve demonstrates that higher STOP-Bang scores are strongly associated with an increased likelihood of belonging to the IHD group.

Among both groups, age and sex were equally distributed, as shown in Table 1.

Age was normally distributed ($p > 0.05$, Shapiro-Wilk test); therefore, results are represented as mean $\pm$ standard deviation above. Considering Table 1 and the data obtained, the proportion of subjects exposed (p_1) was $(57/61) = 0.934$, and the proportion of subjects in the control group (p_2) was $(16/61) = 0.262$. Using the two-proportion Z test, effect size or Cohen's h was estimated to be 1.46. The level of significance or two-tailed alpha value was estimated to be 0.05, and the

ratio of cases to controls was 1:1. The statistically observed power thus obtained was greater than 0.999. Hence, the sample size of 122 was adequate, with negligibly low chance of type II error (false negative).

Among the participants of the wIHD group, a significant 31.14% had high risk of developing OSA; 60% had intermediate risk of developing OSA; and only 6.45% showed low or no risk of developing OSA according to the STOP-Bang score. Statistical analysis showed that patients recruited in the wIHD group had 2 points more on the STOP-Bang score than the patients recruited in the cIHD group, as shown in Figure 2, with probability value found to be less than 0.01 ($p < 0.05$).

It has to be taken into account that the patients recruited in this study were from middle and older age groups with minimum age being 50 years and maximum being 90 years; the average age of the recruited patients was 69.27 years. The median STOP-Bang score for the wIHD group was 6, with an IQR of 5 to 7, and the score for the cIHD group was

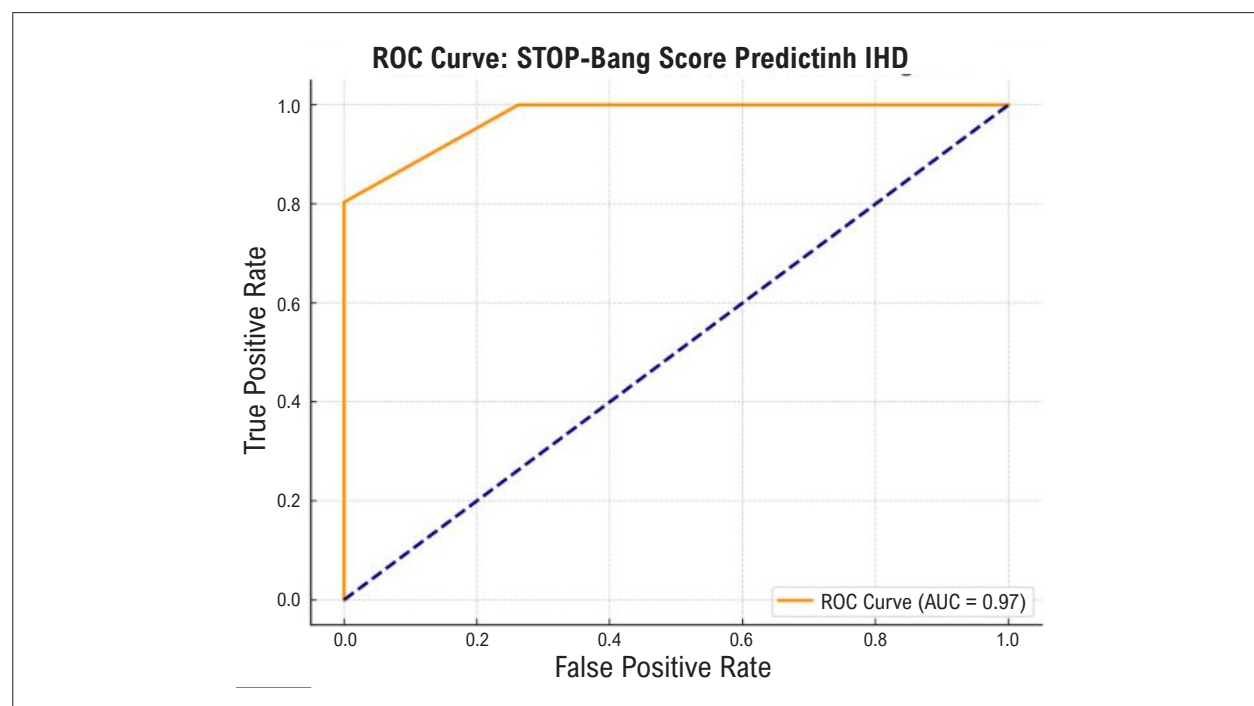


Figure 1 – ROC curve analysis for the STOP-Bang score's diagnostic effectiveness. AUC: area under the curve; IHD: ischemic heart disease; ROC: receiver operating characteristic.

Table 1 – Age and sex distribution of both participant groups

Groups	Sex	Age (years) $\pm$ SD	
wIHD	36 male (59%)	69.3 $\pm$ 11.58	67.6 $\pm$ 11.31
	25 female (41%)		71.76 $\pm$ 11.7
cIHD	36 male (59%)	69.4 $\pm$ 6.33	65.8 $\pm$ 4.88
	25 female (41%)		74.4 $\pm$ 4.46

cIHD: control group without ischemic heart disease; SD: standard deviation; wIHD: group with ischemic heart disease.

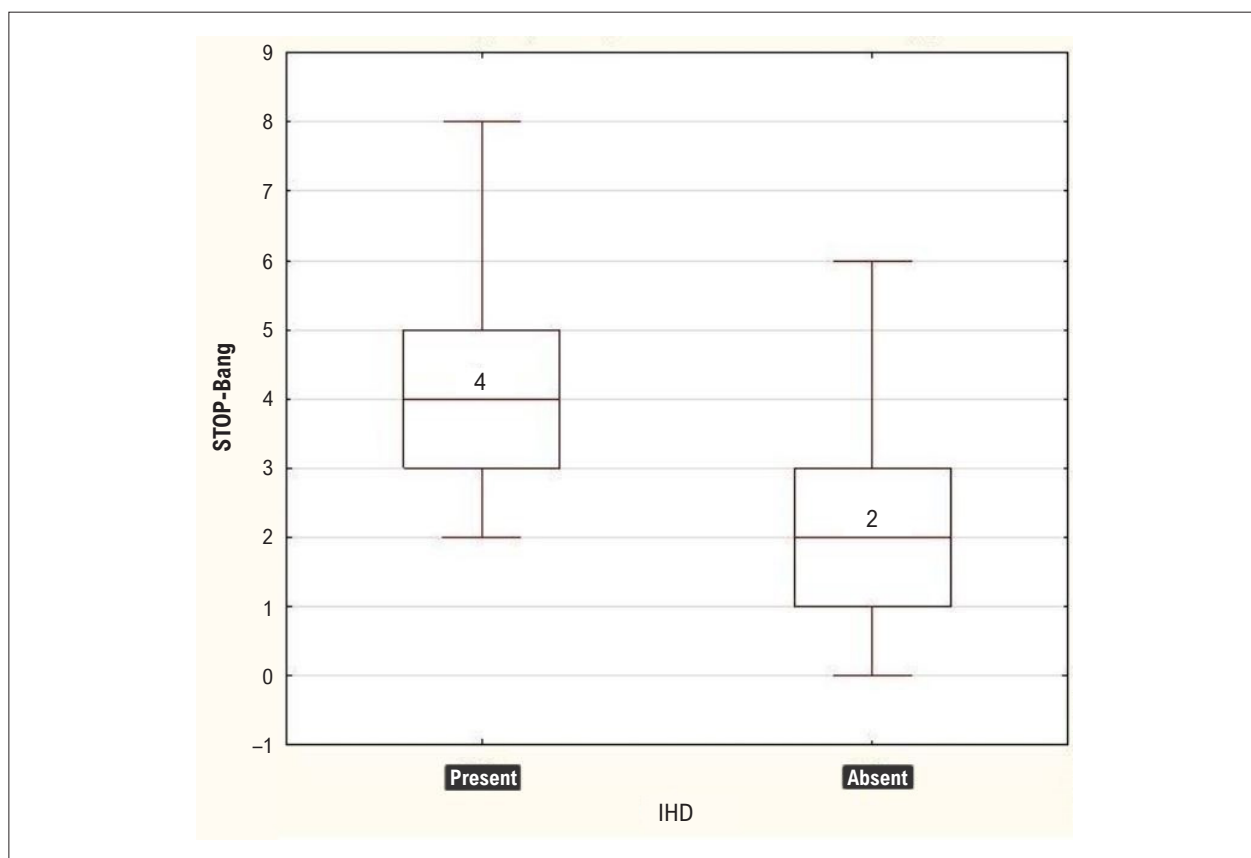


Figure 2 – Box and whisker plot showing STOP-Bang score and IHD. The horizontal line inside the box (—) indicates the median; the box (•) indicates 25% to 75% of the interquartile range; the horizontal line inside the box indicates the median, and the horizontal whiskers extend to the minimum and maximum values.

4 with IQR of 3 to 5. On linear regression model, a graphical correlation of the equation $y = -0.035x + 6.543$ with $R^2 = 0.104$ between age and STOP-Bang score was observed, as shown in Figure 3.

Further comorbidity factors were analyzed for development of detailed understanding of the pathology and its correlations. BMI was found to have a contributory relationship as a factor of influence among the participants in the wIHD group. It had a statistically significant probability value of 0.0016 ($p < 0.05$) with a Pearson's correlation coefficient of 0.396 ($r = 0.396$).

This case-control study thus effectively established the association between IHD and risk of developing OSA. Moreover, it demonstrated the correlation of increased BMI as a comorbid factor, as shown in Figure 4. The wIHD group had median BMI of 29.80 kg/m² with IQR 27.4 to 32.1, and the cIHD group had median BMI of 26.3 kg/m² with IQR 24.5 to 29.1.

For further validation of the study, risk factors were statistically analyzed in the wIHD and cIHD groups. Participants from both groups were classified according to STOP-Bang score, as shown in Table 2.

On the comparing the data, it was found that the attributed risk (AR) was 0.672; relative risk (RR) was 3.562; and attributed

risk percentage was 71.929% for the development of OSA in patients with IHD. On further analysis, the OR was found to be 40.078 at a confidence interval of 95% with a Z-score of 6.219. The ratio between OR and RR (OR/RR) was thus calculated to be 11.25 (> 1). Hence, the study has successfully demonstrated that OSA is strongly associated with IHD and may contribute to the risk of developing it.

A chi-square test was performed based on the data in Table 2. The calculated expected count is displayed in Table 3.

The chi-square statistic was calculated at 54.57; degree of freedom was found to be 1. Thus, a significant p value of 1.50×10^{-13} was obtained here.

Discussion

Sleep medicine being a comparatively newer arena of discussion and discourse in medical science, the correlation between IHD and development of OSA is a neglected topic in clinical research. Very few studies have been conducted in this regard to date. In patients prescribed continuous positive airway pressure (CPAP) treatment for OSA, the non-compliant subjects with severe and moderate OSA were observed to have a higher risk of fatal and non-fatal cardiovascular events than those treated with CPAP and healthy individuals.¹⁰ The

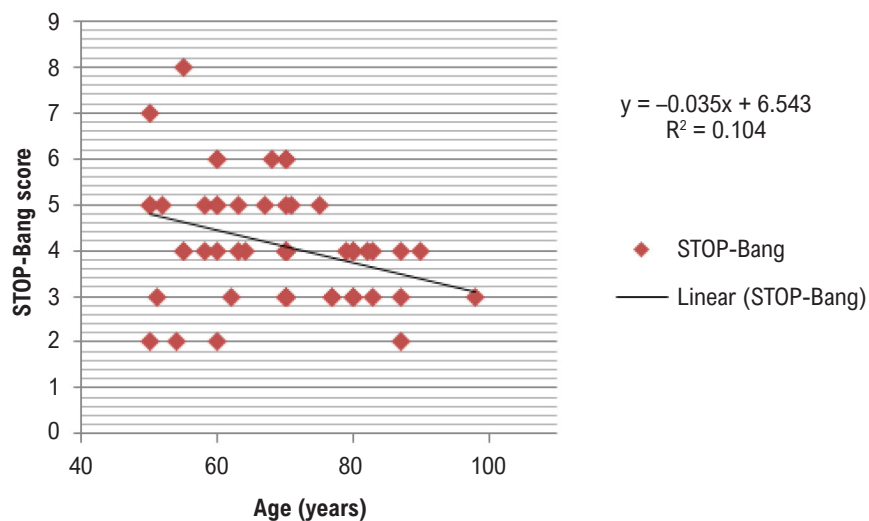


Figure 3 – Age-related STOP-Bang score distribution in the group of patients with ischemic heart disease

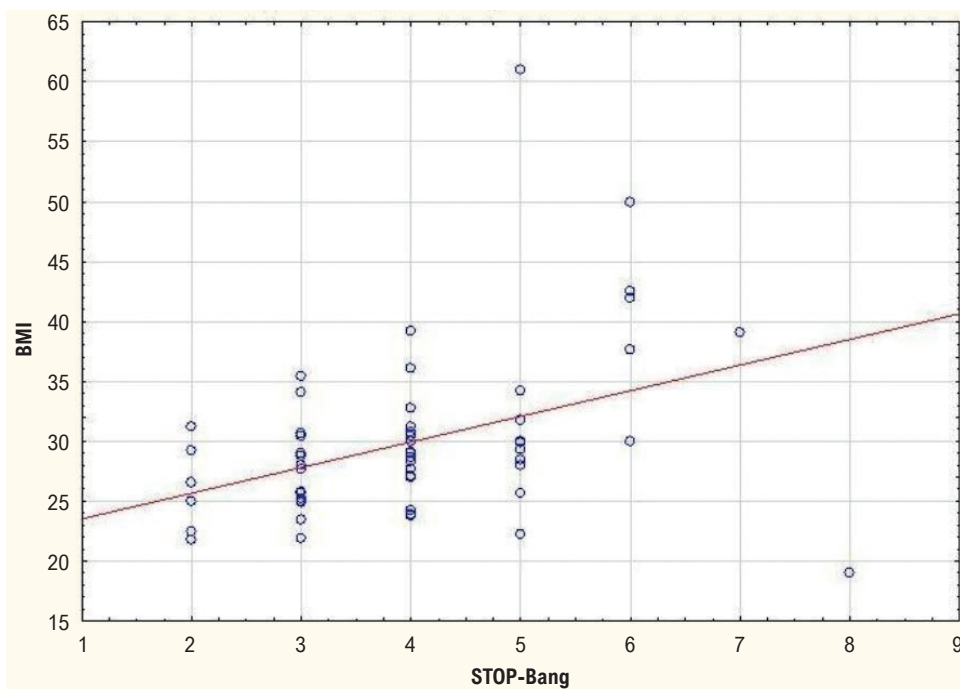


Figure 4 – Line graph demonstrating correlation between BMI and STOP-Bang score. BMI: body mass index.

SAPAMI study demonstrated that OSA was a significant finding in patients after acute myocardial infarction. The SAPAMI study also found that, using apnea-hypopnea index (AHI)¹¹ of greater than 5 events per hour, 65.7% had sleep apnea.¹² Considering ischemia as a pathological process of inadequate end-organ oxygen supply,¹³ OSA has been found to be a comorbid factor in ischemic stroke and transient

ischemic attacks in the United States of America and a significant contributing factor to silent cerebral infarctions in Japan.¹⁴ The discussion of correlation between OSA and IHD is incomplete without recognizing the circadian rhythm and physiological changes during the sleep cycle. In genetically modified animal models, factors like light intensity, small molecules that maintain circadian rhythm,

Table 2 – Distribution of participants in wIHD and cIHD groups according to STOP-Bang score

	Participants with STOP-Bang score 3 to 8 points	Participants with STOP-Bang score 0 to 2 points	Total participants (N)
wIHD	57 (93.40%)	4 (6.60%)	61
cIHD	16 (26.20%)	45 (73.80%)	61
Totals	73	49	122

cIHD: control group without ischemic heart disease; wIHD: group with ischemic heart disease.

Table 3 – Expected count according to chi-square test

Group	STOP-Bang score 3 to 8	STOP-Bang score 0 to 2
wIHD	36.5	24.5
cIHD	36.5	24.5

cIHD: control group without ischemic heart disease; wIHD: group with ischemic heart disease.

therapeutic drugs, and diet contributed to transition and translation of circadian rhythm. The aging process has also been considerably regarded as a contributing factor in myocardial ischemia-reperfusion injury and heart failure.¹⁵ The paradoxical phase of sleep or REM phase plays a considerable role in understanding the relation between OSA and IHD. According to electroencephalography studies, during the REM phase of sleep, the body undergoes many physiological changes. Onset of arrhythmia, increase in brain temperature and cerebral blood flow, muscle atonia,¹⁶ and a rapid rise in blood pressure were noticed.¹⁷ Lung volumes are decreased during REM and between REM and non-REM phases of sleep; end-expiratory volume is decreased significantly, even if on CPAP therapy.¹⁸ Moreover, patients with non-dipping blood pressure have been linked to development of ischemic diseases including IHD and transient ischemic attacks, and interleukin-2 mediated systemic inflammation development.^{19,20} In case of ischemic changes, the volumetric oxygen consumption by skeletal muscles is permanently decreased,²¹ along with increased higher baseline oxygen saturation, decreased lower plateau in oxygen saturation, lowered oxygen saturation under curve, prolonged time to reach oxygen saturation plateau, and prolonged time to increase oxygen saturation due to abnormal microvascular reperfusion.²² Hence, these considerations, added to the results of the present case-control study, establish an association between IHD and risk of developing OSA. Further research in this field with a longitudinal study is very likely to establish OSA as a risk factor of IHD. Being associated with IHD, patients with established OSA can be screened for probable IHD, or OSA might be used as a determinant of developing IHD with the help of artificial intelligence based on machine learning models. This study shows AR of 71.929%; smoking has a similar AR for the occurrence of myocardial infarction. Therefore, with further study, OSA can be added to risk stratification for IHD or acute coronary syndrome.²³ Risk

stratification might be done by using OSA, defined in stages from mild to moderate, which can be added to scales like the Systematic Coronary Risk Evaluation 2 (SCORE2),²⁴ after further large-scale evaluation.

Limitations

The study was conducted with minimal resources and over a short duration of 6 months. The study has indicated that OSA is positively related with risk of developing IHD; however, in order to establish it as a risk factor for IHD, it is necessary to conduct a longitudinal study among a sufficiently large population. Although the control group was isolated from risk factors including smoking and alcohol consumption, medications were not stopped. Polysomnography was not conducted to diagnose OSA, but STOP-Bang was used to determine higher probability of OSA.

Conclusion

IHD is associated with development of OSA. This case-control study has effectively established this association with statistically viable data. The risk of developing OSA after an IHD event in the wIHD group was 2 points higher on the STOP-Bang score and was statistically significant, with a probability value less than 0.01 ($p < 0.05$). It was additionally found that, on occurrence of IHD, AR was 71.929%. Hence, it is essential in the clinical research of cardiology and sleep medicine to recognize OSA as a factor of importance in risk assessment of IHD and other ischemic processes. From the point of view of future research, a large-scale population study on the topic addressed in this study should be conducted.

Author Contributions

Conception and design of the research: Samajpaty S, Zhuravlov AK. Acquisition of data: Samajpaty S, Dmitrii E, Sergeivich IY. Analysis and interpretation of the data: Samajpaty S, Dmitrii E. Statistical analysis: Sergeivich IY. Writing of the manuscript: Samajpaty S.

Critical revision of the manuscript for intellectual content: Zhuravlov AK.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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