Risk factors of left ventricular hypertrophy in obstructive sleep apnea

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Abstract. Obstructive sleep apnea (OSA) and left ventricular hypertrophy (LVH) are both related to major cardiovascular diseases. Previous studies have indicated that, compared with non-OSA, OSA is related to LVH with an odds ratio (OR) of 1.70 (95% CI: 1.44-2.00), particularly in patients with coronary artery disease. Meta-analysis has revealed that the severity of OSA is significantly associated with left ventricular mass compared with non-OSA controls. There is, however, limited data on the risk factors of LVH in patients with OSA. The present study aimed to assess the prevalence and clinical factors that are predictive of LVH in patients with OSA. A retrospective analysis of adult patients diagnosed with OSA who had undergone echocardiography was performed. LVH defined by echocardiography indicated an enlarged LV mass index. Clinical factors predictive of LVH were assessed using multivariate logistic regression analyses. An unadjusted OR and an adjusted OR with 95% confidence intervals (CI) were determined. During the study period, 130 patients met the study criteria, with an LVH prevalence of 27.69% (36 patients). The final predictive model of LVH comprised six factors: Age, sex, unrefreshed sleep, body mass index, systolic blood pressure and apnea-hypopnea index. Only age was independently associated with LVH, with an adjusted OR of 1.048 (95% CI: 1.002-1.096). The prevalence rate of LVH in patients with OSA was 27.69%. Older age was independently related to LVH in patients with OSA.

Introduction

Obstructive sleep apnea (OSA) is a condition that is commonly encountered in clinical practice but may be underestimated. The importance of OSA is that it has a high prevalence rate and is related to major cardiovascular diseases. The prevalence of OSA may be as high as 37% in males and 50% in females (1), and up to 67.5% in smokers (2). OSA has been reported to be related to hypertension, hypertensive crisis, impaired left ventricular ejection fraction and peripheral artery disease (3-7). The presence of OSA alongside these diseases has also been indicated to produce unfavorable cardiovascular disease outcomes.

Left ventricular hypertrophy (LVH) is a crucial predictor of cardiovascular diseases and mortality (8,9). A previous study indicated that LVH increased the risk of cardiovascular disease and coronary heart disease by 62 and 56%, respectively (8). LVH in hypertensive patients was also determined to be related to mortality [adjusted odds ratio (OR): 1.40; 95% confidence interval (CI): 1.08-1.81] (10).

As both OSA and LVH have been associated with major cardiovascular diseases, it may be worthwhile to evaluate LVH in patients with OSA (3-5,8). Previous studies have indicated that, compared with non-OSA, OSA is related to LVH with an OR of 1.70 (95% CI: 1.44-2.00), particularly in patients with coronary artery disease (11-15). According to one meta-analysis, the severity of OSA is significantly associated with the left ventricular mass compared to controls (15). However, there is limited data on the risk factors of LVH in patients with OSA. The present study aimed to evaluate the prevalence and clinical factors that are predictive of LVH in patients with OSA.

Patients and methods

Patients. The present study was a retrospective, analytical study conducted at Srinagarind Hospital, a university hospital of Khon Kaen University (Khon Kaen, Thailand). The inclusion criteria were adult patients with a diagnosis of OSA who had undergone an echocardiogram. Pregnant patients were excluded from the study. Those patients diagnosed with OSA between January 2011 and 2017 were included in the study. OSA was diagnosed by evidence of an apnea-hypopnea index of five events/hour or more by polysomnography type 3. The type 3 polysomnography was comprised of at least three channels of recording: Airflow, respiratory effort and blood oxygenation (16,17). This study was a part of an OSA and cardiovascular consequences project (7).

Eligible patients were reviewed for baseline characteristics, physical signs and laboratory results, including

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Key words: apnea-hypopnea index, age, prevalence

Table I. Baseline characteristics of patients with OSA categorized by presence of LVH.

Factors	No LVH (n=94)	LVH (n=36)	P-value
Age, years	48 (35-56)	56 (41-65)	0.005
Female sex	36 (38.3)	25 (69.4)	0.001
OSA symptoms			
Snoring	62 (96.9)	21 (95.5)	0.754
Duration of snoring, years	8 (3-10)	10 (4-19)	0.372
Witnessed apnea	29 (72.5)	13 (81.3)	0.495
Nocturia, times/night	2 (1-3)	2 (1-3)	0.375
Morning headache	22 (53.7)	9 (60.0)	0.672
Unrefreshed sleep	33 (86.8)	8 (61.5)	0.047
EDS	51 (85.0)	14 (87.5)	0.801
Lifestyle habits			
Previous alcohol drinking	14 (31.8)	6 (31.6)	0.985
Current alcohol drinking	6 (13.6)	6 (31.6)	0.096
Previous smoking	9 (20.0)	4 (22.2)	0.844
Current smoking	4 (8.9)	0 (0)	0.204
Comorbid diseases			
DM	25 (26.6)	11 (30.6)	0.652
GERD	34 (41.0)	14 (46.7)	0.588
Allergic rhinitis	19 (44.2)	10 (62.5)	0.211
OSA consequences			
Hypertension	74 (78.7)	31 (86.1)	0.339
Stroke	5 (5.3)	5 (13.9)	0.101
Coronary artery disease	5 (5.3)	4 (11.1)	0.244
Heart failure	3 (3.2)	5 (13.9)	0.023
Atrial fibrillation	1 (1.1)	0 (0)	0.534
Other arrhythmias	5 (5.3)	0 (0)	0.158
CPAP treatment	43 (45.7)	20 (55.6)	0.334

Values are expressed as n (%) or median (interquartile range). Total numbers of patients in both groups may not be 94 and 36 due to missing data. OSA, obstructive sleep apnea; LVH, left ventricular hypertrophy; EDS, excessive daytime sleepiness; DM, diabetes mellitus; GERD, gastroesophageal reflux disease; CPAP, continuous positive airway pressure machine.

echocardiography. Patients were categorized into two groups by LVH. LVH was defined by using left ventricular mass index (LMVI) calculated by the Cube formula 2D linear method: $LVMI=\{0.8x1.04x[(IVS + LVID + PWT)^3-LVID^3] + 0.6\}$/body surface area, with an LVH cut-off point of >115 g/m^2 and 95 g/m^2 in males and females, respectively (18). IVS is the interventricular septum; LVID the LV internal diameter and PWT the inferolateral wall thickness.

Statistical analysis. All cardiovascular events were reported as the prevalence. Patients with or without LVH were compared regarding basic characteristics, symptoms and signs of OSA, polysomnography, comorbidities, cardiovascular events, current medications, laboratory findings and echocardiography. For numerical factors, the Wilcoxon rank-sum or Student's t-test was used to analyze differences between the LVH and non-LVH groups. The categorical factors of these two groups were compared by either Fisher's exact test or the Chi-square test where appropriate. In the first step of the logistic regression analysis, a univariate logistic regression analysis was applied to determine an unadjusted OR for each factor for evidence of LVH. Factors that either had a P-value of <0.20 according to univariate logistic regression analysis or were clinically significant were selected for the subsequent multivariate, binary logistic regression analysis. Results were presented as unadjusted OR and adjusted OR with 95% CI. The final predictive model was evaluated by the Hosmer-Lemeshow test. Statistical analyses were performed using STATA software, version 10.1 (StataCorp LP).

Results

Patient characteristics. A total of 130 patients were enrolled in the present study. Of these, 36 patients had LVH (27.69%). There were four significantly different factors between those patients with and without LVH in terms of baseline characteristics (Table I). The LVH group had an older age (56 years

Table II. Physical signs of patients with obstructive sleep apnea categorized by presence of LVH.

Factor	No LVH (n=94)	LVH (n=36)	P-value
BMI, kg/mm^2	28.49 (24.89-35.59)	30.0 (26.60-34.36)	0.405
SBP, mmHg	141 (130-153)	141 (130-151)	0.924
DBP, mmHg	86 (79-95)	82.5 (76-90)	0.103
Torus palatinus	8 (25.8)	2 (25.0)	0.963
Torus mandibularis	5 (16.7)	3 (37.5)	0.199
Tonsil enlargement	10 (27)	0 (0)	0.064
Tonsillectomy	1 (3.4)	0 (0)	0.594
Retrognathia	5 (18.5)	3 (37.5)	0.261
Mallampati class			0.615
1	3 (4.9)	1 (3.8)	
2	23 (37.7)	7 (26.9)	
3	27 (44.3)	12 (46.1)	
4	8 (13.1)	6 (23.1)	
Macroglossia	31 (75.6)	12 (75.0)	0.962
Dentures	2 (3.4)	1 (4.8)	0.787
Neck circumference, cm	42 (38-45.5)	39 (37-42)	0.113

Values are expressed as n (%) or median (interquartile range). Total numbers of patients in both groups may not be 94 and 36 due to missing data. OSA, obstructive sleep apnea; LVH, left ventricular hypertrophy; BMI, body mass index; SBP, systolic blood pressure, DBP, diastolic blood pressure.

Table III. Laboratory results of patients with obstructive sleep apnea categorized by presence of LVH.

Factor	No LVH (n=94)	LVH (n=36)	P-value
AHI, events/h	18.5 (10-39)	20 (14-34)	0.405
HbA1c, %	6.2 (5.8-7.1)	6.1 (5.4-7)	0.201
BUN, mg/dl	12.6 (9.7-16)	14 (9.4-16.2)	0.671
Cr, mg/dl	0.9 (0.7-1.1)	0.8 (0.7-1.0)	0.483
UACR, mg/d	11.5 (5-53)	25 (4-102)	0.614
Cholesterol, mg/dl	198 (171-230)	178 (152-192)	0.090
TAG, mg/dl	126 (101-172)	110 (93-148)	0.299
HDL, mg/dl	49.5 (41-57)	46 (40-54)	0.206
LDL, mg/dl	130 (104-163)	114.5 (106-150.5)	0.626
EF (Teichholz)	67.45 (65.59-72.0)	66.45 (62.84-72.49)	0.195

Values are expressed as median (interquartile range). Total numbers of patients in both groups may not be 94 and 36 due to missing data. LVH, left ventricular hypertrophy; AHI, apnea-hypopnea index; HbA1c, glycated hemoglobin; BUN, blood urea nitrogen; Cr, creatinine; UACR, urine albumin creatinine ratio; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TAG, triglyceride; HDL, high-density lipoprotein-cholesterol; LDL, low-density lipoprotein-cholesterol; EF, ejection fraction.

vs. 48 years), a larger proportion of female patients (69.4% vs. 38.3%), fewer patients with unrefreshed sleep (61.5% vs. 86.8%) and more patients with heart failure (13.9% vs. 3.2%) than the non-LVH group. The LVH group also had higher proportions of patients with consequences of OSA. A total of 63 patients (48.46%) were treated with a continuous positive airway pressure machine (CPAP) with a slightly higher proportion of CPAP treatment in the LVH group than the non-LVH group (55.6% vs. 45.7%; P=0.334).

Predictive factors for LVH. Regarding physical signs and laboratory results (Tables II and III), there were no significant differences between the two groups, even for the apnea-hypopnea index (20 events/h vs. 18.5 events/h; P=0.405). A total of six factors were identified using the final, multivariate predictive model of LVH (Table IV). Age was the only independent factor associated with LVH, with an adjusted OR of 1.048 (95% CI: 1.002-1.096). The predictive model had a strong goodness of fit, as the Hosmer-Lemeshow χ^2 value

Table IV. Factors associated with left ventricular hypertrophy in patients with obstructive sleep apnea by logistic regression analysis.

Factors	Unadjusted OR (95% CI)	P-value	Adjusted OR (95% CI)	P-value
Age (increment by 1 year of age)	1.045 (1.012-1.078)	0.007	1.048 (1.002-1.096)	0.037
Female sex	3.662 (1.609-8.331)	0.002	2.544 (0.837-7.731)	0.100
Unrefreshed sleep	0.528 (0.216-1.289)	0.161	0.502 (0.166-1.519)	0.223
Body mass index (increment by 1 kg/m^2)	1.017 (0.976-1.059)	0.407	1.055 (0.987-1.127)	0.111
Systolic blood pressure (increment by 1 mmHg)	1.000 (0.982-1.020)	0.923	1.004 (0.979-1.030)	0.741
Apnea-hypopnea index (increment by 1 event/h)	0.993 (0.975-1.012)	0.493	0.991 (0.965-1.016)	0.492
Snoring	0.722 (0.328, 1.589)	0.419	Not included	
Morning headache	1.295 (0.389, 4.307)	0.673	Not included	
Hypertension	1.675 (0.577, 4.865)	0.343	Not included	
Macroglossia	0.967 (0.254, 3.686)	0.962	Not included	

OR, odds ratio.

was 4.97 (P=0.761). A cutoff of 40 years of age or more gave sensitivity of 80.56% and specificity of 34.04%.

Discussion

In the cohort of the present study >1/4 of patients with OSA had LVH. Elderly patients with OSA were at risk for LVH.

The prevalence of LVH in patients with OSA in the present study was lower than that in previous reports. Previous studies determined that the prevalence of LVH in patients was OSA was 59-88% (12-14). The different prevalence rate of LVH in the present study was due to its different study population. The present study enrolled consecutive patients with OSA, while the other three were performed in specific OSA populations, including middle-aged males with hypertension (59%), patients with coronary artery disease (86%) and patients with severe OSA (88%). The results of the present study may provide data for a more general population.

Even though previous studies identified predictors for LVH in patients with OSA (13,14,19,20), the present study added that age was another factor related to LVH in patients with OSA after adjusting for severity, gender, OSA symptoms and systolic blood pressure. There are several proposed mechanisms by which LVH affects OSA: Ventricular pressure overload, increasing sympathetic drives, loss of vagal heart rate regulation and systemic vasculature remodeling (13,21). Increasing age is a risk factor for both OSA and LVH, regardless of blood pressure level (22,23). In addition, up to 80% of individuals with OSA have been indicated to go undiagnosed for a decade (24,25). These factors may result in an increased risk of LVH and cardiovascular consequences in elderly patients, regardless of systolic blood pressure level or other factors. Even though sex was a significant factor according to univariate logistic regression analysis, it was not significant in the multivariate analysis. These findings indicate that sex was not an independent predictor. It may be that certain confounders such as hypertension, which may influence both OSA and LVH or sex, was not strong enough to be significant compared to other factors. As the present study considered clinical factors that are predictive of LVH in patients with OSA, the results may be applied in resource-limited facilities, where clinicians may use these predictors to evaluate high-risk patients for further referral.

There are certain limitations to this study. First, the cohort was from a single center in a university hospital setting. In addition, the sample size was small (n=130). Furthermore, the present study only enrolled patients who underwent echocardiography. This may result in selection bias. Other aspects of OSA (CPAP purchasing, CPAP compliance), other cardiovascular risk factors (diabetes, hypertension, dyslipidemia, albuminuria, exercise) and full results of cardiac ultrasound (LV end-diastolic dimension, interventricular septal thickness at end-diastole, posterior wall thickness at end-diastole or right ventricular systolic pressure) were not determined (26-33). In addition, the present study did not evaluate the physiological characteristics of patients with OSA, such as upper airway gain or arousal threshold (34). Finally, even though echocardiography is not routinely performed in patients with OSA, the present results had a statistical power of 80.85%. This was traced back by using a comparison of mean age between both groups by STATA software. However, further prospective studies may be required to evaluate the consequences of LVH in patients with OSA and to confirm the results of the predictive model. A further prospective study will be conducted to validate this predictive model. CPAP therapy has demonstrated beneficial outcomes in reducing LVH and improving LV function (35), but the present study did not evaluate the effects of CPAP on LVH as it is not the study objective. However, 48.46% of

patients in the present study were treated with CPAP. Further studies are required to confirm the results of the present study with a prospective cohort design using consecutive patients with OSA in a multicenter setting, including all levels of hospital from primary care hospitals to referral hospitals.

In conclusion, the prevalence rate of LVH in patients with OSA in the present study was 27.69%. Older age was independently related to LVH in patients with OSA.

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Availability of data and materials

The datasets used and/or analyzed during the present study are available from the corresponding author upon reasonable request.

Authors' contributions

SK designed the study, analyzed and interpreted the data and wrote the manuscript. TS, PL, JC and WB interpreted the data. SS and KS confirm the authenticity of all the raw data, participated in data analysis and interpretation and prepared the manuscript. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The study protocol was approved by the Khon Kaen University Ethics Committee for Human Research (approval no. HE641504).

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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