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ORIGINAL RESEARCH

Obstructive sleep apnea affects the clinical outcomes of patients undergoing percutaneous coronary intervention

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Background: There is a paucity of evidence regarding the association between obstructive sleep apnea (OSA) and patients undergoing percutaneous coronary intervention (PCI) for coronary artery disease. We sought to investigate whether OSA affects the clinical outcomes of patients undergoing PCI.

Patients and methods: All enrolled individuals treated with PCI were evaluated for OSA by polysomnography. The primary end point was defined as major adverse cardiac events (MACEs) at 2 years, including cardiac death, myocardial infarction (MI), and/or target vessel revascularization.

Results: A total of 340 consecutive patients undergoing PCI were assigned to the OSA (n=152, apnea–hypopnea index ≥15) and non-OSA (n=188, apnea–hypopnea index <15) groups. The incidence of OSA in patients with coronary artery disease undergoing PCI was 44.7%. Patients in the OSA group had more three-vessel disease (34.9%), increased number of total implanted stents (3.3±2.0), and longer total stent length (83.8±53.1 mm) when compared to the non-OSA group (23.4%, P=0.020; 2.8±1.9, P=0.007; 68.7±48.4, P=0.010). After a median follow-up of 2 years, the incidence of MACEs was significantly higher in patients with OSA (25.0% vs 16.0%, P=0.038), mainly driven by the increased periprocedural MI (19.2% vs 11.2%, P=0.038) in the OSA group. By Cox regression multivariable analysis, the independent predictor of MACEs was OSA (hazard ratio: 1.962, 95% confidence interval: 1.036–3.717, P=0.039).

Conclusion: There was a high prevalence of moderate-to-severe OSA in patients undergoing PCI, and OSA was associated with significantly increased MACE rate, mainly due to the increase in periprocedural MI rate.

Keywords: coronary artery disease, percutaneous coronary intervention, myocardial infarction, obstructive sleep apnea

Introduction

Obstructive sleep apnea (OSA), which presents in ~9% of females and 24% of males, is characterized by repetitive upper airway obstruction in breathing during sleep and is usually associated with hypertension, arrhythmia, heart failure, insulin resistance, and cerebrovascular accident.[1,2] It has also been shown that the prevalence of OSA in patients with coronary artery disease (CAD) was two times more than that in those without CAD, with increased risk of mortality and myocardial infarction (MI), which was believed to be caused by hypoxia, enhanced inflammation, oxidative stress, and endothelial dysfunction.[1,3,4] So far, percutaneous coronary intervention (PCI) has been the crucial treatment for symptomatic CAD; however, there is a paucity of evidence regarding the association between OSA and patients undergoing PCI. In 89 consecutive patients

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with acute coronary syndrome (ACS) treated with PCI, the incidence of cardiac death, reinfarction, and target vessel revascularization (TVR) at 8-month follow-up was 23.5% in the OSA group, which was significantly higher than 5.3% in patients without OSA.[5] However, the advanced devices, especially the new generation of drug-eluting stent (DES), were not used in the small-scale study with short follow-up. Therefore, this prospective study is designed to address the long-term clinical impact of OSA in patients undergoing PCI.

Patients and methods

Study population

From October 2012 to April 2014, a total of 1,130 consecutive real-world patients who were treated with DES implantation at our center were considered as candidates for this study. Finally, 340 patients were included in the study according to the inclusion and exclusion criteria. Inclusion criteria were as follows: age >18 years and successful PCI in at least one major epicardial coronary artery. Exclusion criteria are shown in Figure 1. Of note, patients with OSA receiving any relevant treatments were excluded. The protocol was approved by the Institutional Ethics Committee of Nanjing First Hospital, and written informed consent was obtained from all patients.

Percutaneous coronary intervention

All interventional procedures were performed in accordance with the current guidelines. Use of the type of DES, glycoprotein IIb/IIIa inhibitors, intravascular ultrasound, fractional flow reserve, and optical coherence tomography was at the operator's discretion. A loading dose of clopidogrel (300 mg) or ticagrelor (180 mg) was administered before the index procedure. Total creatine kinase, creatine kinase-myocardial band,

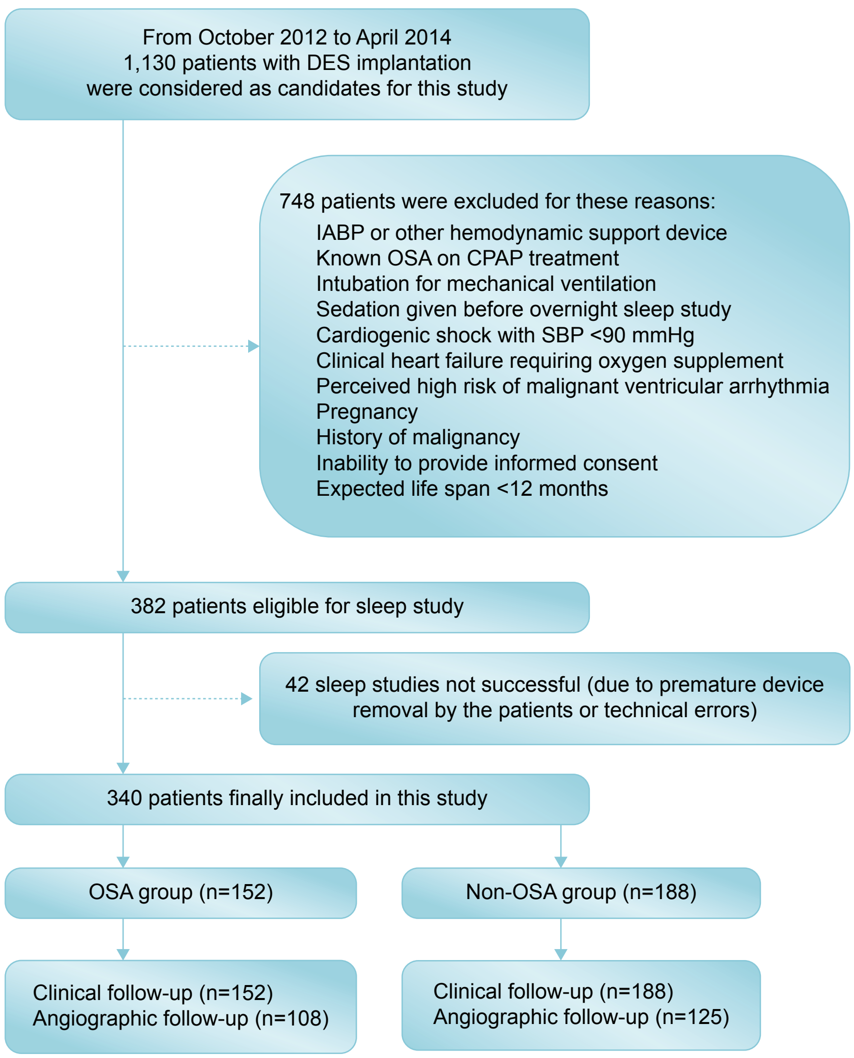

Figure 1 Flowchart of study design.

Abbreviations: CPAP, continuous positive airway pressure; DES, drug-eluting stent; IABP, intra-aortic balloon pump; OSA, obstructive sleep apnea; SBP, systolic blood pressure.

and troponin were dynamically measured until 72 hours postprocedure. After the intervention, all patients received 100 mg/d aspirin indefinitely and clopidogrel (75 mg/d) or ticagrelor (90 mg/d bid) for at least 12 months.

Sleep study

All overnight sleep studies for eligible patients were conducted using the Embletta Gold standardized level-3 portable diagnostic system (Natus Medical Inc., Ontario, Canada) 7–10 days after the index PCI procedure,[6,7] which included nasal airflow, thoracoabdominal movements, arterial oxygen saturation, snoring episodes, limb movement, electrocardiogram, and body position. All sleep studies were reviewed by an independent sleep specialist according to the guidelines,[8] and the poor quality of sleep tracings were excluded from the final analysis of the sleep data. Two independent sleep technologists with registered polysomnographic technologist credentials manually analyzed all studies. The respiratory events of sleep study were performed in accordance with the American Association of Sleep Medicine Guidelines.[8] The primary measurement of the sleep study was the apnea–hypopnea index (AHI), which "was used to stratify patients" into the OSA (AHI ≥15) and non-OSA groups (AHI <15).[9]

Study end points and definitions

The primary end point was the rate of major adverse cardiac events (MACEs) at 2 years, including cardiac death, MI, and/or TVR. The safety end point was the occurrence of stent thrombosis (ST). All deaths were considered cardiac in origin unless a noncardiac cause was confirmed clinically or at autopsy. MI was diagnosed in accordance with the Society for Cardiovascular Angiography and Interventions definition.[10] Target lesion revascularization, TVR, and ST were defined according to the Academic Research Consortium definition.[11]

Follow-up

Clinical follow-up was performed either by telephone or through a clinical office visit at 1 month, 6 months, and 12 months, and every year thereafter. Repeat coronary angiography was scheduled at 12 months after the indexed procedure unless clinical reasons indicated earlier. An independent committee that was blinded to the study assessed all clinical events.

Quantitative coronary angiography

Quantitative coronary angiographic analysis (QCA) at baseline, post-stenting, and at follow-up was performed offline using edge detection techniques (CAAS II, Version 5.0; Pie Medical, Maastricht, the Netherlands) by an independent core laboratory (China Cardiovascular Research Foundation, Beijing, People's Republic of China). QCA variables included reference vessel diameter, minimal lumen diameter, acute gain, late lumen loss, and net gain, which were defined in our previous study.[12]

Statistical analysis

The distribution of continuous variables was assessed by the Kolmogorov–Smirnov test. Categorical variables were expressed as frequencies or percentages and compared using chi-square statistics or Fisher's exact test. Continuous variables were summarized as mean ± SD or median and compared using Student's *t*-test (for normal data) and Mann–Whitney *U*-test (for non-normally distributed variables). Survival curves were generated by the Kaplan–Meier method and compared using the log-rank test. Hazard ratios (HR) are presented along their 95% confidence interval (CI). Univariable and multivariable Cox proportional hazard models were applied to identify potential factors that correlated with MACEs. The variables showing statistical significance or a trend ($P<0.1$) in univariable Cox model were subjected to multivariable analysis with a forced-entry method. A *P*-value <0.05 was considered statistically significant, except for *P*-value <0.01 in univariable analysis to minimize the probability of type I error. All analyses were performed using the statistical program SPSS 16.0 (SPSS Inc., Chicago, IL, USA).

Results

Baseline clinical and sleep characteristics

A total of 340 patients (average age: 64.69±10.38 years) with DES implantation were finally enrolled and assigned to the OSA (n=152, 44.7%) and non-OSA (n=188, 55.3%) groups stratified by the AHI. Baseline clinical and sleep characteristics are shown in Table 1. None of the 340 study patients had received treatments for OSA. Ten percent of the individuals were admitted with stable angina and the remaining with ACS. Patients in the OSA group were more likely to be males (78.9%) and had greater body mass index (BMI; 25.4±2.9 kg/mm^2) and larger left ventricular end diastolic diameter (LVDd; 50.6±6.8 mm), when compared to the non-OSA group (68.6%, $P=0.032$; 24.5±3.1 kg/mm^2, $P=0.007$; 48.2±4.6 mm, $P=0.003$). The median AHI of 340 enrolled patients was 13.8, and the median time for SpO_2 level <90% was 16.3 minutes in the OSA group, which was significantly higher than the >5.1 minutes in the non-OSA group ($P<0.001$).

Table 1 Baseline clinical and sleep characteristics of the OSA and non-OSA groups

Characteristics	OSA (n=152)	Non-OSA (n=188)	*P*-value
Age, years	65.6±10.3	64.0±10.4	0.170
Males, n (%)	120 (78.9)	129 (68.6)	0.032
BMI, kg/m^2	25.4±2.9	24.5±3.1	0.007
Hypertension, n (%)	113 (74.3)	132 (70.2)	0.399
Hyperlipidemia, n (%)	107 (70.9)	124 (66.0)	0.336
Diabetes, n (%)	50 (32.9)	45 (23.9)	0.067
Stroke, n (%)	15 (9.9)	25 (13.3)	0.329
Smoking status, n (%)			0.083
Current smoker	49 (32.2)	70 (37.2)	
Former smoker	18 (11.8)	10 (5.3)	
eGFR, mL/min/1.73 m^2	105.7±28.8	126.0±30.2	0.358
Previous MI, n (%)	23 (15.1)	18 (9.6)	0.118
Stable angina, n (%)	17 (11.2)	17 (9.0)	0.513
Unstable angina, n (%)	105 (69.1)	130 (69.1)	0.989
STEMI, n (%)	10 (6.6)	20 (10.6)	0.189
NSTEMI, n (%)	11 (7.2)	10 (5.3)	0.465
CHF, n (%)	24 (15.8)	21 (11.2)	0.211
LVEF, %	59.2±10.6	61.5±8.2	0.067
LVDd, mm	50.6±6.8	48.2±4.6	0.003
Previous PCI, n (%)	33 (21.7)	32 (17.0)	0.274
Previous CABG, n (%)	2 (1.3)	2 (1.1)	0.830
PAD, n (%)	4 (2.7)	2 (1.2)	0.319
Sleep study characteristics			
AHI	30.9 (21.8, 43.5)	8.4 (3.9, 11.9)	<0.001
Average SpO_2, %	93.3 (92.1, 94.3)	93.9 (92.8, 94.8)	0.001
Lowest SpO_2, %	82.0 (79.0, 85.0)	87.0 (82.0, 88.0)	<0.001
Time SpO_2 <90%, minutes	16.3 (5.7, 47.2)	5.1 (0.8, 26.9)	<0.001
Time SpO_2 <80%, minutes	0.4 (0.1, 1.4)	0.2 (0.0, 0.4)	0.041

Note: Continuous variables were summarized as mean ± standard deviation for normal data or median (quartile) for non-normally distributed variables.
Abbreviations: AHI, apnea–hypopnea index; BMI, body mass index; CABG, coronary artery bypass grafting; CHF, congestive heart failure; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; LVDd, left ventricular end diastolic diameter; MI, myocardial infarction; NSTEMI, non-STEMI; OSA, obstructive sleep apnea; PAD, peripheral artery disease; PCI, percutaneous coronary intervention; ST, stent thrombosis; STEMI, ST-segment elevation MI.

Lesions and procedural characteristics

Table 2 shows that patients in the OSA group had more three-vessel disease (34.9%), increased number of total implanted stents (3.3±2.0), and larger total stent length (83.8±53.1 mm) when compared to the non-OSA group (23.4%, *P*=0.020; 2.8±1.9, *P*=0.007; 68.7±48.4, *P*=0.010). As a result, the OSA group had higher procedural time (64.0±49.7 minutes vs 52.8±39.6 minutes, *P*=0.026).

OSA and cardiovascular events

After a median follow-up of 2 years, there were 38 (25.0%) composite MACEs in the OSA group and 30 (16.0%) in the non-OSA group (*P*=0.038), which were mainly driven by the more periprocedural MI (19.2%) in the OSA group compared to the non-OSA group (11.2%, *P*=0.038; Table 3, Figure 2). By Cox regression multivariable analysis, the independent predictor of MACEs was OSA (HR: 1.962, 95% CI: 1.036–3.717, *P*=0.039; Table 4). There were no significant differences in QCA between the OSA and non-OSA groups (Table 5).

Subgroup analysis

Figure 3 shows the results of subgroup analysis for the associations between OSA and risk of MACEs in patients with DES implantation at 2 years. The incidence of composite MACEs in the OSA group was more remarkable among male patients aged ≤75 years, with BMI >25 kg/m^2, hyperlipidemia, and ACS.

Discussion

The major finding of the current study was that the high prevalence of moderate-to-severe OSA was found in patients with DES implantation, and OSA was associated with significantly increased 2-year composite MACE rate, mainly due to the increment of periprocedural MI rate.

In the current study, we found that 44.7% of patients with CAD undergoing PCI had moderate-to-severe OSA, which was similar to 48.3% and 57% in the current literature.[5,13] With chronic intermittent hypoxemia, reoxygenation, and hypercapnia in patients with OSA, there is increased

Table 2 Angiographic and procedural characteristics of the OSA and non-OSA groups

Characteristics	OSA (n=152)	Non-OSA (n=188)	*P*-value
Angiographic characteristics			
Number of diseased vessels			0.017
One-vessel disease	47 (30.9)	84 (44.7)	0.010
Two-vessel disease	52 (34.2)	60 (31.9)	0.654
Three-vessel disease	53 (34.9)	44 (23.4)	0.020
CTO lesion			0.507
LAD	11 (7.2)	7 (3.7)	
LCX	5 (3.3)	6 (3.2)	
RCA	12 (7.9)	10 (5.3)	
>1 CTO	1 (0.7)	2 (1.1)	
Needing rotablation	2 (1.3)	1 (0.5)	0.442
Thrombus-containing lesions	7 (4.7)	7 (3.7)	0.673
Procedural characteristics			
Transradial, n (%)	148 (97.4)	184 (97.9)	0.549
IIb/IIIa inhibitor used, n (%)	18 (12.1)	18 (10.7)	0.688
IVUS assessment, n (%)	31 (20.8)	29 (17.2)	0.407
FFR assessment, n (%)	24 (16.1)	37 (21.9)	0.191
OCT assessment, n (%)	9 (6.0)	8 (4.7)	0.605
DES used, n (%)	152 (100)	188 (100)	NS
Total implanted stents	3.3±2.0	2.8±1.9	0.007
Mean stent diameter, mm	3.0±0.4	3.0±0.4	0.279
Total stent length, mm	83.8±53.1	68.7±48.4	0.010
Complete revascularization, n (%)	96 (63.2)	132 (70.2)	0.169
Final TIMI grade 3, n (%)	150 (98.7)	187 (99.5)	0.193
Procedural time, minutes	64.0±49.7	52.8±39.6	0.026
Contrast volume, mL	211.0±108.1	190.2±89.3	0.053

Notes: Continuous variables were summarized as mean ± standard deviation for normal data or median (quartile) for non-normally distributed variables. Categorical variables were expressed as percentages.
Abbreviations: CTO, chronic total occlusion; DES, drug-eluting stent; FFR, fractional flow reserve; IVUS, intravascular ultrasound; LAD, left anterior descending artery; LCX, left circumflex artery; MI, myocardial infarction; NS, nonsignificant; OCT, optical coherence tomography; OSA, obstructive sleep apnea; RCA, right coronary artery; TIMI, thrombolysis in MI.

sympathetic activation, induction of oxidative stress, vasoconstriction, and provocation of inflammation leading to endothelial dysfunction and atherosclerosis.[1,14,15] Therefore, it is not surprising that the high prevalence of OSA was found in these patients with CAD undergoing PCI. In addition, the current study showed that patients with OSA were more likely to be males and had a significantly greater BMI, which was consistent with published data in the Randomized Intervention With Continuous Positive Airway Pressure in Coronary Artery Disease and Sleep Apnea trial,[16] suggesting that age, male, and BMI were predictors of OSA. Moreover, compared to patients without OSA, patients with OSA were more likely to undergo multivessel PCI and had an increased number of DESs implanted during PCI, which might be explained by chronic hypoxia, enhanced inflammation, oxidative stress, and endothelial dysfunction.[1,3]

The association between OSA and adverse cardiac events has been explored in general population in several studies.[3,4,17] The Sleep Heart Health Study enrolling 1,927 men and 2,495 women free of CAD showed that OSA was a significant predictor of incident CAD, MI, revascularization procedure, or cardiovascular death after a median of 8.7-year follow-up.[4] In another long-term study including 1,436 patients, OSA increased the risk of coronary events or cardiovascular death, and OSA was an independent risk factor for cardiovascular events, including MI.[3] Similar negative effects of OSA could be found in patients with ACS.[11,18,19] In 168 consecutive patients with unstable angina or non-ST elevation acute MI, OSA had higher risk of cardiovascular complications during the acute phase of ACS.[11] Overall, the current data indicated that OSA has been associated with increased risk of adverse cardiac events in general population or patients with ACS.

Whether OSA adversely affected the prognosis of patients with stent placement for CAD has not been established, and there is a deficiency of evidence regarding the association between OSA and patients undergoing PCI. In 105 patients admitted with ST-segment elevation MI undergoing stent placement, severe OSA carried a negative prognostic impact for these patients after 18-month follow-up.[20] Another study which enrolled 89 consecutive patients with ACS treated with PCI showed that the incidence of cardiac death, reinfarction,

Table 3 Clinical outcomes in the OSA and non-OSA groups

Outcomes	OSA (n=152)	Non-OSA (n=188)	*P*-value
In-hospital, n (%)			
Cardiac death	0	0	NS
Periprocedural MI	29 (19.2)	21 (11.2)	0.038
Spontaneous MI	0	0	NS
TLR	0	0	NS
CABG	0	0	NS
TVR	0	0	NS
MACEs	0	0	NS
ST	0	0	NS
At 1 year, n (%)			
Cardiac death	1 (0.7)	1 (0.5)	0.880
Periprocedural MI	29 (19.2)	21 (11.2)	0.038
Spontaneous MI			
STEMI	0	0	NS
NSTEMI	2 (1.3)	1 (0.5)	0.442
TLR	5 (3.3)	5 (2.7)	0.773
CABG	1 (0.7)	0	0.265
TVR	7 (4.6)	5 (2.7)	0.334
MACEs	37 (24.3)	27 (14.4)	0.019
ST	2 (1.3)	2 (1.1)	0.830
At 2 years, n (%)			
Cardiac death	3 (2.0)	1 (0.5)	0.220
Periprocedural MI	29 (19.2)	21 (11.2)	0.038
Spontaneous MI			
STEMI	0	0	NS
NSTEMI	2 (1.3)	1 (0.5)	0.442
TLR	6 (3.9)	8 (4.3)	0.887
CABG	1 (0.7)	0	0.265
TVR	8 (5.3)	9 (4.8)	0.841
MACEs	38 (25.0)	30 (16.0)	0.038
ST	3 (2.0)	2 (1.1)	0.660

Abbreviations: CABG, coronary artery bypass grafting; MACEs, major adverse cardiac events; MI, myocardial infarction; NS, nonsignificant; NSTEMI, non-ST segment elevation MI; OSA, obstructive sleep apnea; ST, stent thrombosis; STEMI, ST segment elevation MI; TLR, target lesion revascularization; TVR, target vessel revascularization.

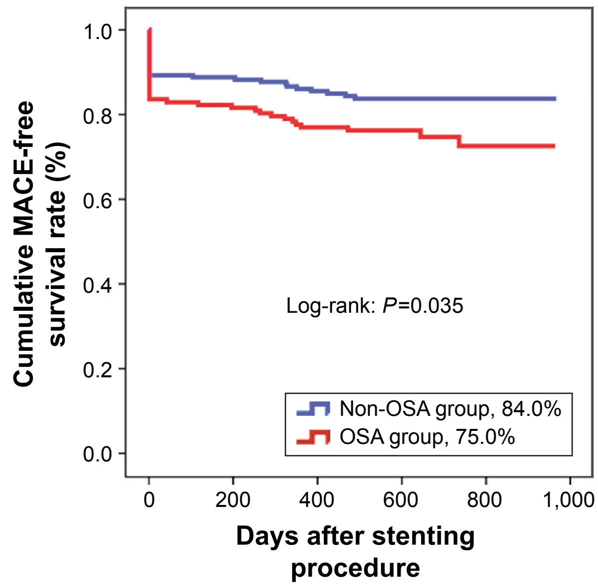

Figure 2 MACE-free survival rate at 2 years.
Note: Freedom from MACEs between the OSA group (red line) and non-OSA group (blue line) at 2-year follow-up.
Abbreviations: MACEs, major adverse cardiac events; OSA, obstructive sleep apnea.

and TVR at 8-month follow-up was 23.5% in the OSA group, significantly higher than 5.3% in patients without OSA.[5] However, the new generation of DES was not used in this small-scale study with short follow-up. Our data confirm this previous evidence and add some novel findings. First, to the best of our knowledge, this is the first study to demonstrate increased periprocedural MI rate of OSA after DES implantation in patients with CAD. Second, a great many angiographic and procedural variables, indicating anatomic severity for coronary disease and the potentially important confounding factors, were included and adjusted in this study. Third, subgroup analysis was conducted to identify a subset of patients undergoing PCI for whom an unfavorable effect of OSA was remarkable. Finally, the differences in techniques, lesion complexity, and DES types used may be impact factors attributed to the discrepancy in several results. However, results from a previous study[5] and ours demonstrated a negative effect of OSA on composite MACE rate in patients undergoing PCI for CAD.

Table 4 Univariate and multivariate analyses for MACEs

Variables	Univariate analysis; HR (95% CI)	*P*-value	Multivariate analysis; HR (95% CI)	*P*-value
Age	0.999 (0.976, 1.022)	0.916		
Male	1.235 (0.705, 2.163)	0.461		
BMI	1.051 (0.980, 1.126)	0.163		
Hypertension	0.940 (0.558, 1.584)	0.817		
Current smoker	1.244 (0.765, 2.022)	0.379		
Diabetes mellitus	0.845 (0.488, 1.463)	0.548		
Ejection fraction	0.976 (0.952, 1.002)	0.069	0.985 (0.955, 1.015)	0.315
hsCRP	0.998 (0.971, 1.026)	0.875		
OSA	1.627 (1.007, 2.628)	0.047	1.962 (1.036, 3.717)	0.039
Total implanted stents	1.069 (0.953, 1.199)	0.253		
Multivessel disease	1.453 (0.869, 2.432)	0.155		

Note: Multivariate analysis data is not shown for several variables because these variables did not enter the multivariate model for their nonsignificant results in the univariate analysis.
Abbreviations: BMI, body mass index; CI, confidence interval; HR, hazard ratio; hsCRP, high-sensitivity C-reactive protein; MACEs, major adverse cardiac events; OSA, obstructive sleep apnea.

Table 5 Quantitative coronary analysis of the OSA and non-OSA groups

Variables	OSA (231 lesions)	Non-OSA (277 lesions)	*P*-value
Lesion length, mm	35.22±19.18	31.82±19.45	0.048
Preprocedure			
Reference diameter, mm	2.83±0.46	2.85±0.49	0.621
Minimal lumen diameter, mm	1.03±0.56	1.05±0.61	0.647
Diameter stenosis, %	64.21±18.87	63.46±20.29	0.669
Postprocedure			
Reference diameter, mm	2.96±0.45	2.96±0.46	0.961
Minimal lumen diameter, mm	2.59±0.46	2.56±0.51	0.453
Diameter stenosis, %	12.71±6.21	13.74±9.69	0.165
Acute gain, mm	1.56±0.56	1.51±0.61	0.283
At follow-up			
Reference diameter, mm	2.95±0.43	2.97±0.46	0.700
Minimal lumen diameter, mm	2.41±0.61	2.40±0.61	0.796
Diameter stenosis, %	18.83±14.79	19.86±13.95	0.492
Late loss, mm	0.19±0.40	0.21±0.33	0.604
Net gain, mm	1.37±0.66	1.33±0.67	0.523
Restenosis, %	12 (6.6)	7 (3.5)	0.165

Notes: Continuous variables were summarized as mean ± standard deviation for normal data or median (quartile) for non-normally distributed variables. Categorical variables were expressed as percentages.
Abbreviation: OSA, obstructive sleep apnea.

Study limitations

The current study has several limitations. First, none of the 340 study patients had received treatments for OSA, and it was not possible to evaluate the effects of continuous positive airway pressure treatment on cardiovascular outcome. Second, there were 42 sleep studies that were not successful due to premature device removal by the patients or technical errors, and it might not be possible to conduct a repeat sleep study in consideration of the short hospitalization period in patients after PCI. Third, patients were not tested

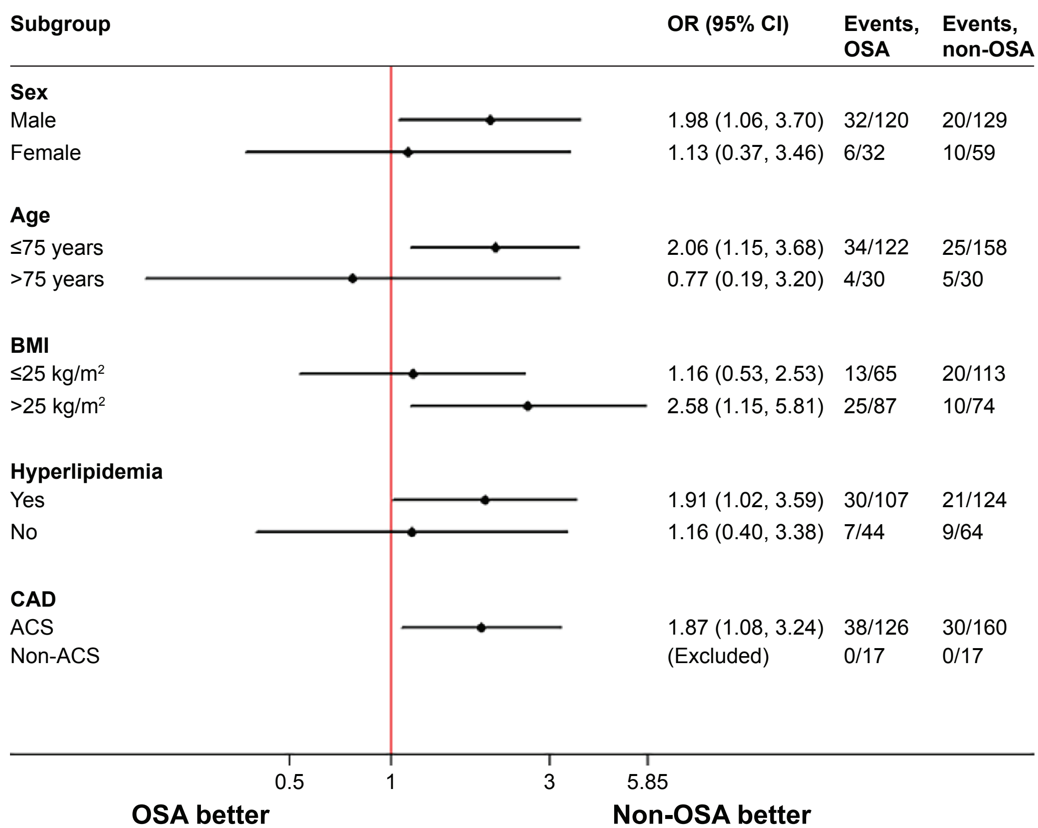

Figure 3 Forest plots of 2-year MACE rate in the prespecified subgroups.
Note: Data in the Events columns presented as number of events/total number of patients.
Abbreviations: ACS, acute coronary syndrome; BMI, body mass index; CAD, coronary artery disease; CI, confidence interval; MACEs, major adverse cardiac events; OR, odds ratio; OSA, obstructive sleep apnea.

for sleep data again in the follow-up. Thus, there was lack of information regarding post-discharge sleep data and the timing of cardiac events during the follow-up.

Conclusion

The current study showed a high prevalence of moderate-to-severe OSA in patients with DES implantation for CAD, and OSA was associated with significantly increased 2-year composite MACE rate, mainly due to the increment of periprocedural MI rate.

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Disclosure

The authors report no conflicts of interest in this work.

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