Prevalence of obstructive sleep apnea among patients with coronary artery disease in Saudi Arabia

Siraj O. Wali [a,*], Muath A. Alsharif [a], Mohammed H. Albanji [a], Murad S. Baabbad [a], Haneen M. Almotary [a], Nabil Alama [b], Layth Mimish [b], Adil Alsulami [a], Muntasir M. Abdelaziz [c]

[a] Sleep Medicine and Research Center, King Abdulaziz University Hospital, Jeddah
[b] King Abdulaziz University Hospital, Jeddah
[c] Tameside Hospital, Ashton under Lyne OL6 9RW

[a,b] Saudi Arabia
[c] United Kingdom

Background: **Despite the association between obstructive sleep apnea (OSA) and coronary artery disease (CAD), few studies have investigated this issue in Saudi Arabia.**

Objectives: **This study aimed to identify the prevalence of OSA among CAD patients.**

Subjects and methods: **This was a cross-sectional (descriptive) study conducted at King Abdul-Aziz University Hospital in Jeddah, Saudi Arabia from April 2012 to December 2013. All consecutive patients referred to the cardiac catheterization lab for coronary angiography who exhibited evidence of CAD were included in this study. This study was conducted in two stages. During the first stage, each participant was interviewed individually. The administered interview collected data pertaining to demographics, comorbidities, and the STOP-BANG questionnaire score. The second stage of this study consisted of a diagnostic overnight polysomnography (PSG) of 50% of the subjects at high risk for OSA according to the STOP-BANG questionnaire.**

Results: **Among the patients with CAD (N = 156), 128 (82%) were categorized as high risk for developing OSA. PSG was conducted on 48 patients. The estimated prevalence of OSA in the study sample was 56.4%. Approximately 61% of the documented sleep apnea patients suffered from moderate to severe OSA.**

Conclusion: **This local study concurs with reports in the literature indicating that OSA is very common among CAD patients.**

Keywords: Sleep apnea, Coronary artery disease, Prevalence, Severity

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* Corresponding author at: College of Medicine, King Abdulaziz University, Director of Sleep Medicine and Research Center, Consultant in Pulmonary & Sleep Medicine, King Abdulaziz University Hospital, PO Box 21589, Jeddah 80215, Saudi Arabia. Tel.: +966 2 6408258; fax: +966 2 6408315.
E-mail address: sowali@kau.edu.sa (S.O. Wali).

P.O. Box 2925 Riyadh – 11461KSA
Tel: +966 1 2520088 ext 40151
Fax: +966 1 2520718
Email: sha@sha.org.sa
URL: www.sha.org.sa

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Introduction

Patients with obstructive sleep apnea (OSA) experience repetitive episodes of apnea or reduced airflow due to upper airway obstruction during sleep. OSA appears to be related to several cardiovascular diseases, even after adjustment for confounding variables. These diseases include systemic hypertension [1]; pulmonary hypertension, particularly in cases of comorbid daytime hypoxemia [2–6]; coronary artery disease (CAD) [7–9]; heart failure [9]; and nocturnal cardiac arrhythmia [10–12].

Furthermore, there is increasing evidence that severe OSA is associated with increased morbidity and mortality related to cardiovascular disease. In an observational study, patients with untreated severe OSA experienced a higher incidence of fatal and non-fatal cardiovascular events than healthy participants, patients with mild-moderate OSA, and even patients with severe but treated OSA [13]. In a prospective longitudinal epidemiological cohort study, Gottlieb et al. followed 1927 men and 2495 women aged 40 years or more who were free of coronary heart disease and heart failure at the time of recruitment [9]. All participants underwent baseline polysomnography (PSG) and were followed up for a median of 8.7 years. After adjustment for multiple risk factors, OSA was found to be a significant predictor of incident CAD in men younger than or equal to 70 years of age, but not in older men or in women of any age. Accordingly, the authors concluded that the association between OSA and the incidence of coronary heart disease in this sample was ambiguous [9].

Data from Saudi Arabia regarding OSA in stable CAD patients are scarce. Hence, the aim of our study was to identify its prevalence among CAD patients (confirmed by coronary angiography) attending a university hospital.

Subjects and methods

This was a cross-sectional (descriptive) study conducted at King Abdulaziz University Hospital in Jeddah, Saudi Arabia, from April 2012 to December 2013. Approval was obtained from the hospital research ethics committee prior to conducting the study. Each participant was required to sign a consent form.

All consecutive patients referred to the cardiac catheterization lab for elective coronary angiography were eligible for this study. Only those with demonstrated CAD based on coronary angiography were recruited. Patients who refused or were unable to participate were excluded. This study was conducted in two stages. During the first stage, trained physicians interviewed each participant individually. The administered questionnaire collected data pertaining to patient demographics, comorbidities, the STOP-BANG questionnaire score, and coronary angiographic findings (positive or negative for CAD). The second stage of the study consisted of a diagnostic overnight PSG. Due to limitation of budget, 50% of the subjects – categorized into the high-risk OSA group according to the STOP-BANG questionnaire – were randomly selected for PSG to confirm OSA. A systematic random sampling technique was followed by choosing odd numbers on the sample list.

Abbreviations

AASM	American Academy of Sleep Medicine
ACS	Acute Coronary Syndrome
AHI	Apnea-Hypopnea Index
BMI	Body Mass Index
CAD	Coronary Artery Disease
IHD	Ischemic Heart Disease
MI	Myocardial Infarction
OSA	Obstructive Sleep Apnea
PSG	Polysomnography
SPSS	Statistical Package for Social Sciences

Instruments

The STOP-BANG questionnaire [14] screens for symptoms of OSA and has been validated for individuals with a mean age of 57 ± 16 years. It was originally intended for use in a preoperative setting because untreated OSA is associated with increased post-operative complications and longer hospital stays [15]. This questionnaire consists of four yes/no and fill-in-the-blank questions represented by the mnemonic 'STOP BANG' as follows: S (Do you Snore loudly?), T (Do you often feel Tired, fatigued, or sleepy during the daytime?), O (Has anyone Observed you to stop breathing during sleep?), and P (Do you have or are you being treated for high blood Pressure?). To improve the accuracy of the scale, B (body mass index or BMI), A (age), N (neck circumference), and G (gender) are recorded. The STOP-BANG questionnaire is scored as follows: for the first four yes/no questions, each positive response is assigned one point. An additional point is added for each of the following conditions: a

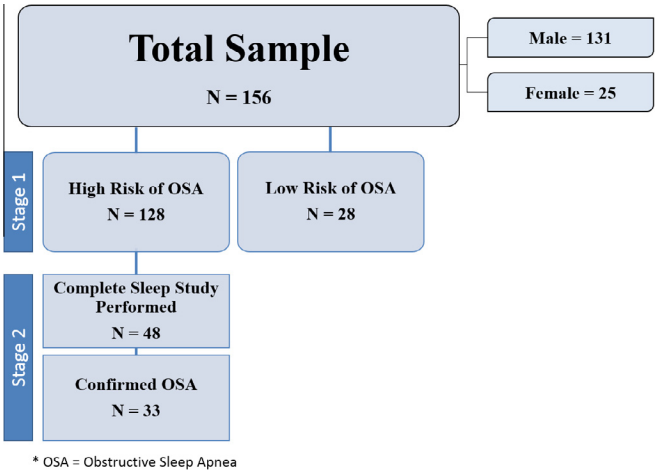

Figure 1. The outline of the study groups and stages.

BMI of more than 35 kg/m^2, an age of 50 years or more, a neck circumference of more than 40 cm, and male gender. A total score of three or more places the individual at a high risk for OSA [15].

PSG: This procedure was performed either at the patient's home using type 2 devices (SOMNOtouch, SOMNOmedics; and Embletta® X100, Natus) or the sleep laboratory using type 1 devices (SOMNOscreen EEG 32, SOMNOm edics; SomnoStar® z4 sleep system). For tests performed at home, 10 channels and surface leads were used to continuously monitor the following physiological parameters during sleep: nasal pressure, nasal and oral airflow, respiratory effort (thoracic and abdominal expansion), pulse rate, oxygen saturation, and body position. Type 2 devices were also used to perform electroencephalography and electrooculography. The in-center devices were equipped with 22 channels and leg sensors and were used to continuously monitor nasal pressure, nasal and oral airflow (thermocouple), snoring, respiratory effort (thoracic and abdominal expansion), oxygen saturation, pulse rate, and to perform electroencephalography, electrooculograp hy, electromyography, and electrocardiography. The PSG data were scored manually according to the American Academy of Sleep Medicine (AASM) 2012 scoring manual [16]. Apnea was defined as the absence of airflow for at least 10 s, and hypopnea was defined as a discernible reduction in airflow associated with a reduction in oxygen saturation by at least 3% from the baseline and/or subsequent arousal. The apnea-hypopnea index (AHI) was defined as the average number of apnea and hypopnea events per hour of sleep. OSA was defined as an AHI score of $\geqslant$5 accompanied by symptoms or signs of disturbed sleep [17].

Statistical analysis

The data were analyzed using Statistical Package for Social Sciences (SPSS) software, version 18. The Chi-square test was utilized to test for the association and/or difference between the categorical variables. Yates' correction was applied when appropriate. Student's t-test was applied to compare the means of the continuous variables (age, weight and BMI) between the two groups. Spearman's correlation coefficient was applied to test for the linear association between AHI and age, weight, and BMI. A p value of less than 0.05 was considered to be statistically significant. A scatterplot was generated to display the relationship between BMI and AHI.

Results

A total of 156 patients (84% males) with demonstrated CAD based on coronary angiography were recruited for this study. Table 1 shows the demographic data and comorbidities of the study population. The outline of the study stages and groups is summarized in Fig. 1.

Based on the STOP-BANG questionnaire, patients with CAD (n = 156) were classified into the high-risk or low-risk OSA group, as shown in Fig. 2. The characteristics of both groups are summarized in Table 2.

Those at high risk for developing OSA (128 patients) were eligible to participate in the second phase of this study: a PSG test to confirm OSA

Table 1. Demographic data and characteristics of the study population (N = 156).

	N (%)
Gender	
Female	25 (16)
Male	131 (84)
Smoking status	
Current smoker	55 (35.3)
Former smoker	27 (17.3)
Non-smoker	74 (47.4)
Medical disease	
DM	94 (60.3)
HTN	96 (61.5)
GERD	47 (30.1)
COPD	16 (10.3)
Stroke	8 (5.1)

DM: diabetes mellitus; GERD: gastro-esophageal reflux disease. COPD: chronic obstructive pulmonary disease; HTN: hypertension.

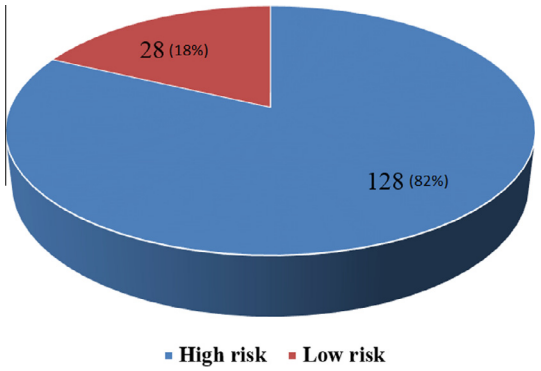

Figure 2. Classification of Coronary Artery Disease patients based on Obstructive Sleep Apnea risk using the STOP-BANG questionnaire (n = 156).

Table 2. Comparison of high and low risk groups for Obstructive Sleep Apnea.

	High risk N = 128	Low risk N = 28	P-value
	Mean (SD)	Mean (SD)	
Age (years)	57.41 (10.47)	49.54 (10.58)	<0.001**
BMI (kg/m^2)	30.8 (8.09)	25.3 (5.7)	<0.001**
	N (%)	N (%)	P-value
Gender			0.29
Female	22 (17.2%)	3 (10.7%)	
Male	106 (82.8%)	25 (89.3%)	
DM	84 (65.6%)	10 (35.7%)	0.004**
HYPT	87 (68%)	9 (32.1%)	0.001**
Stroke	7 (5.5%)	1 (3.6%)	0.56
GERD	40 (31.2%)	7 (25%)	0.34
COPD	14 (10.9%)	2 (7.1%)	0.43
Current smoker	40 (31.2%)	15 (53.6%)	0.072
Former smoker	25 (19.5%)	2 (7.1%)	0.072

BMI: body mass index; DM: diabetes mellitus; HTN: hypertension; GERD: gastro-esophageal reflux disease; COPD: chronic obstructive pulmonary disease.
** *P* value <0.05 is considered statistically significant.

Table 3. Characteristics of participants at high risk of Obstructive Sleep Apnea who did polysomnography compared to those who did not.

	PSG group N 48	No PSG group N 80	P-value
	Mean (SD)	Mean (SD)	
Age (Years)	57.9 (9.7)	57.1 (10.9)	0.387
BMI (kg/m^2)	30.7 (9.0)	30.9 (7.6)	0.189
	N (%)	N (%)	P-value
Gender			
Female	8 (16.6%)	14 (17.5%)	0.904
Male	40 (83.4%)	66 (82.5%)	
Smoking status			
Current smoker	16 (33.3%)	24 (30%)	0.337
Former smoker	12 (25%)	13 (16.3%)	
Non-smoker	20 (41.7%)	43 (53.7%)	
Medical Conditions			
DM	31 (64.6%)	53 (66.3%)	0.848
HTN	33 (68.8%)	54 (67.5%)	0.883
GERD	16 (33.3%)	24 (30%)	0.694
COPD	6 (12.5%)	8 (10%)	0.661
Stroke	4 (8.3%)	3 (3.8%)	0.270

DM: diabetes mellitus; GERD: gastro-esophageal reflux disease. COPD: chronic obstructive pulmonary disease; HTN: hypertension.

(Fig. 1). The plan was to perform PSG on 50% of the high-risk group. Unfortunately, only 48 patients (37.5%) agreed to undergo this procedure. Nevertheless, the high-risk group of OSA patients who had undergone PSG compared to those who had not were similar based on demographic data, smoking status, and medical conditions (Table 3).

Among the high-risk patients with CAD, 68.8% suffered from OSA. Based on the assumption that all of the low-risk patients were free of significant sleep apnea, the prevalence of OSA in our study population of CAD patients was estimated to be 56.4%. Two thirds of these confirmed sleep apnea patients were of moderate and severe degree based on AHI (Fig. 3).

Discussion

In this study, we demonstrated that the prevalence of OSA in patients suffering from CAD is significantly higher than reported in the general population [18,19]. According to the STOP-BANG questionnaire, 82% of our CAD patients were considered to be at significantly high risk for developing OSA compared to only 33–39% of individuals in the general Saudi population [18,19]. However, based on PSG, the estimated prevalence of OSA among the CAD patients was 56.4%. These results suggest that these two conditions are closely correlated in Saudi Arabia, which

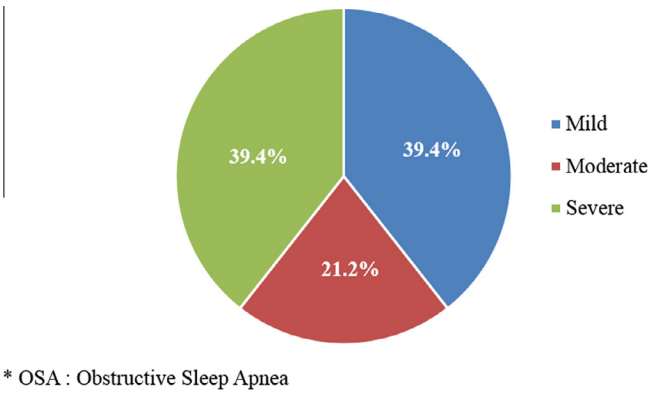

Figure 3. Pie chart displaying the severity of Obstructive Sleep Apnea according to Apnea Hypopnea Index.

is in accordance with several previous studies conducted elsewhere.

The higher prevalence of OSA in CAD patients compared with the general population has been demonstrated previously by many international studies [20–31]. The prevalence of OSA among patients with CAD has been reported to be as high as 69% compared to 5–15% among the general population, emphasizing the association between these two conditions [31,32,33]. Mooe et al. [20] reported the presence of OSA (AHI > 10) in 37% of 142 patients with stable angina pectoris and angiographically confirmed CAD. Similarly, Peker et al. [21] reported that the prevalence of OSA was 30% among admitted patients with acute CAD. Furthermore, in a relatively recent prospective cohort study, PSG was performed on 89 acute coronary syndrome (ACS) patients who underwent percutaneous coronary intervention (53 with acute myocardial infarction (MI) and 36 with unstable angina) [27]. In this study, OSA (defined as an AHI $\geqslant$ 10) was detected in 51 patients (57%), similar to our findings [27]. In another study, OSA was reported in 63.7% (422 out of 662) of revascularized CAD patients [28]. Among ischemic cardiomyopathy patients, on the other hand, Oldenburg et al stated that 36% were documented to suffer from OSA [29]. More recently, Tan et al. reported that 32 (34.4%) of 93 CAD patients exhibited moderate to severe OSA [30]. This wide variation in the prevalence of OSA among CAD patients can be partially explained by differences in the methodologies and study populations assessed in these studies. It is believed that studies reporting relatively low prevalence rates may underestimate the actual frequency of OSA among CAD patients. Recently, Konecny et al. [31] emphasized the under-recognition of OSA among patients admitted with acute CAD. The primary finding of this study was the low rate (12%) of documented or suspected OSA among 789 patients hospitalized for acute MI based on chart reviews. These findings conflicted with the high prevalence of OSA (69%) among 74 MI patients who underwent prospective evaluations using PSG. Based on this study, it is tempting to postulate that the association between OSA and CAD has been under-reported and under-diagnosed [31].

Conversely, CAD has also been frequently found in OSA patients. Pecker et al. conducted a long-term clinic-based observational study. Over seven years, the investigators reported CAD in 16.2% of patients with OSA compared with 5.4% of snorers without OSA [34]. Furthermore, several subsequent studies have demonstrated that patients with untreated severe OSA exhibit an increased incidence of fatal and nonfatal cardiovascular events (including MI and ACS) compared with those without sleep apnea or even with those with untreated mild to moderate OSA [7–9,13,35]. However, such an association suggests but does not demonstrate a causal relationship.

Nevertheless, data from Saudi Arabia on the association between OSA and CAD are scarce. BaHammam et al. examined the prevalence of OSA among patients presenting with acute coronary artery syndrome [36]. They reported that 56% of the study population (50 patients) exhibited OSA based on PSG according to an AHI > 10, and their conditions persisted even at six months after the initial acute event [36].

Although the total number of patients who underwent PSG in our study was relatively small, approximately 40% of those with sleep apnea were classified as severe based on the AHI criteria (Fig. 3). These findings may be consistent with the aforementioned association between untreated severe OSA and the development of adverse cardiovascular events. In addition, the

reverse may also be true: that CAD may be associated with a worsening of sleep-disordered breathing. This hypothesis was demonstrated in a prospective cohort study of 2721 individuals without CAD who were followed up for a mean of five years. PSG was performed at baseline and after five years [37]. Those who had experienced MI exhibited an increase in the AHI of a mean of 6.37 respiratory events per hour of sleep compared to an increase of only 2.71 events among those who had not experienced MI [37].

The most important risk factors for OSA include advanced age, male gender, and obesity [11,32]. In our study, age and obesity prevalence were significantly higher in the high-risk OSA group than in the low-risk OSA group (Table 2). However, the small number of females in our study may account for the lack of a significant gender difference between these two groups. In addition, although smoking may increase the risk or worsen the symptoms of OSA [38], this result was not confirmed in our study, which was likely due to the relatively small sample size.

OSA has been closely associated with adverse clinical outcomes, including hypertension [1] and diabetes mellitus [39], which were clearly observed in our study, as both hypertension and diabetes were observed more frequently in the high-risk OSA group than in the low-risk OSA group. Furthermore, OSA has been associated with an increased risk of ischemic stroke [40]. However, the small number of patients who had experienced stroke in our sample may explain the lack of a significant difference between these two groups (Table 2).

This link between CAD and OSA is not surprising because both share common risk factors, such as obesity, male gender, increased age, and smoking status [42]. The worldwide obesity pandemic, particularly in Saudi Arabia, has actually increased the risk of both conditions [43]. Nevertheless, it has been postulated that the correlation between CAD and OSA is independent of these risk factors. Indeed, Peker et al. studied 182 men with suspected OSA who were free of other risk factors for Ischemic Heart Disease (IHD), such as hypertension and diabetes mellitus [43]. These authors found that 36.7% of individuals with OSA developed CAD compared to 6.6% of subjects without OSA ($p < 0.001$), suggesting that the association between these two conditions is independent of other risk factors, including obesity [43].

Our study contained some limitations, primarily involving the sample size and the performance of a single center rather than a multi-center study. Another limitation of our study was that not all patients underwent PSG to confirm OSA. However, the sample underwent a confirmatory test was not different in terms of demographic parameters, smoking status, and medical conditions from the whole high risk group (Table 3). In addition, unlike other previous studies, which utilized the Berlin questionnaire, we used the STOP-BANG questionnaire to determine the risk for OSA. The systematic review performed by Abrishami et al. did not reach a definitive conclusion regarding the most accurate questionnaire to use for OSA screening. However, they recommended the STOP-BANG questionnaire due to its high-quality methodology and reasonably accurate results [44].

In conclusion, our study demonstrated that many patients with CAD are either at risk for developing OSA or already suffer from OSA. This correlation may have serious consequences since OSA exacerbates CAD. Therefore, clinicians should be aware of the coexistence of these two conditions in Saudi Arabian patients and should endeavor to address both conditions simultaneously. Although CAD alone is not an indication for a diagnostic evaluation of OSA, the presence of this condition should prompt the clinician to consider whether there are other signs or symptoms of OSA that indicate a diagnostic evaluation.

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