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Effects of sleep apnoea syndrome on severity of coronary artery disease in patients undergoing primary percutaneous coronary intervention

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With the new advances in discovering contributors of adverse health outcomes of cardiovascular disease, obstructive sleep apnoea has become an identifiable risk factor, implicating in development of coronary artery disease through many mechanisms….

Abstract

Background: Obstructive sleep apnoea (OSA) has been a recognized factor in many diseases, either alone or combined with other risk factors. Studies have emphasized on the important pathophysiological mechanisms that sleep apnoea and atherosclerotic cardiovascular diseases share in common.

Patients and methods: Two groups of patients were studied in prospective observational study January 2019 to July 2020 , using STOP-BANG score, first group (n: 25); were low-risk for the development of sleep apnoea, home sleep test (HST) was not done for them and the second group (n: 25); were high risk for development for sleep apnoea and HST was done for them. Comparison of both groups done in their general characteristics (as: age, BMI, presence of other risk factors for CAD, smoking) and severity of CAD that was identified during primary percutaneous coronary intervention (PCI). The high-risk group for whom we did HST, we identified the severity of sleep apnoea by apnoea- hypopnoea index (AHI) and mean minimum $SPO2_2$.

Results: The high-risk group had significant increased body mass index (BMI), with insignificant differences between the two groups regarding age, gender and smoking status. Hypertension was found frequently in the high-risk group but hyperlipidemia was more in the low risk group. Morbidity from coronary artery disease was comparable in the two groups (both having a mean follow up ejection fraction of 52 after 6 months). Importantly, Nearly half of high-risk patients (n: 12) had mild OSA (n: 12) and 28% had moderate OSA (n: 7) and severe OSA was found in 12% (n: 3) with a mean minimum SPO_2 of 85%.

Conclusions: Raised BMI is an important predictor of presence of sleep apnoea in patients with acute myocardial infarction.

Keywords: **A**pnoea hypopnoea index, **H**ome sleep test, **O**bstructive sleep apnoea, **P**rimary percutaneous coronary intervention

Introduction

Definition of sleep apnoea

Obstructive sleep apnoea is disorder of repetitive closure of pharynx during sleep. The closure of pharynx is either complete (causing apnoea) or partial (causing hypopnoea) [1].

A common definition of sleep apnoea syndrome is apnoea/hypopnoea index (AHI) of greater than 15. However, population based studies in the USA and Australia have found that 10–20% of middle-aged men have sleep apnoea according to this definition[2]. Also, there is poor correlation between AHI and symptoms of sleepiness. As in an epidemiological study the prevalence of excessive daytime sleepiness was found to be 41% in people with sleep disordered breathing, but this was not significantly greater than in those without sleep disordered breathing where the prevalence was 37%. In addition, many patients with major symptoms of sleepiness caused by sleep apnoea have an AHI of less than 15[3]. A definition, which incorporates apnoeas, hypopnoeas, upper air- way resistance, and symptoms of daytime sleepiness, has replaced the former definition[4].

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An apnoea, defined as cessation of airflow for at least 10 s, is classified as obstructive or central on the basis of presence or absence of respiratory effort. Hypopnoea is defined is either: substantial reduction in airflow (>50%), moderate reduction in airflow (<50%) with desaturation or moderate reduction of airflow (<50%) with electroencephalographic evidence of arousal[2-4].
Young and colleagues have demonstrated that 4% of men and 2% of women in a middle-aged North American population had symptoms of obstructive sleep apnoea and an AHI of greater than 5 events per hour of sleep. Prevalence estimates from studies with probability samples range, for OSA of at least mild severity (defined by AHI > 5), from 3 to 28%; for OSA of at least moderate severity (defined by AHI => 15), estimates range from 1 to 14%[5,6].

Risk factors of sleep apnoea are mainly: male gender, advancing age, obesity, menopausal status, black race, smoking and alcoholism. While the main clinical features of sleep apnoea include: witnessed snoring with or without gasping, non-restorative sleep, excessive daytime sleepiness and hypertension[7].

Health consequences
Hypertension: Apnoea and hypopnea episodes during sleep cause acute, transient blood pressure perturbations, inducing elevations of 30 mm Hg or more in mean arterial pressure. Davies and co workers assessed 24-hour ambulatory blood pressure in 45 persons with OSA and 45 persons without OSA, matched on several factors including age, BMI, alcohol and cigarette use, and heart disease. The patients with OSA had higher daytime and night-time blood pressures and demonstrated an attenuated night-time reduction in blood pressure compared with the matched participants without OSA[8, 9].

Cardiovascular consequences: Obstructive respiratory events cause temporary cardiovascular disturbances that may lead to long-term cardiovascular remodelling. In addition to chronically elevated blood pressure, a number of possible mechanisms by which OSA might affect cardiovascular function have been hypothesized, as vascular injury with acceleration of atherosclerosis due to episodic hypoxemia, chronic sympathetic hyperactivity, elevated fibrinogen and homocysteine, elevated pulmonary blood pressure. These alterations can lead to increased risk for right heart hypertrophy, heart failure and of plaque ruptures and subsequent cardiovascular or cerebrovascular events[10-13]. Coronary artery disease (CAD) is described as a gradual growth of plaques within the intima of the vessel that leads to subsequent lumen narrowing. The pathophysiological mechanisms of systemic inflammation (mediating the formation of pre-atherosclerotic lesions), oxidative stress, endothelial dysfunction and metabolic (leading to adipose tissue dysfunction) that occur in sleep apnoea can drive to atherosclerosis eventually. In Sleep Heart Health Study, the followed 4,422 subjects a median of 8.7 years, severe OSA predicted an increased risk of developing symptomatic CAD, but only in men aged 70 or younger1[14,15]

When comparing 1651 adults for 10 years, Marin and colleagues found that patients with severe OSA had increased risk of non-fatal cardiovascular disease compared with patients without OSA. Moreover, Yaggi and coworkers found that adults with AHI 5 and more had a two fold increased adjusted risk for stroke or death compared with non- OSA participants. Gami and coworkers found that AHI was independent predictor of death from cardiovascular disease from midnight to 6 am and found that CPAP was associated with reduced mortality[16].
Other consequences: other important consequences of sleep apnoea, which are out of the scope of this study; include mainly: disturbances in cognitive-behavioural function and increased incidence of road traffic and occupational accidents due to impaired concentration and daytime sleepiness[17'18].

Diagnostic criteria:
The American Association of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events, states that:
An obstructive apnea is scored if there is a drop in the respiratory effort signal by ≥90% of pre-event baseline, for ≥10 seconds and continued or increased inspiratory effort from chest and/or abdomen.
A hypopnea is defined when there is drop in the respiratory effort by ≥30% of pre-event baseline for ≥10 seconds, associated with oxygen desaturation by ≥3% (AASM criteria) .
A Respiratory Effort-Related Arousal (RERA) is scored if there is flattening of the inspiratory nasal pressure for

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≥10 seconds causing an arousal from sleep, but not meeting criteria for an apnea or hypopnea[47].
Apnea Hypopnea Index (AHI) or Respiratory Disturbance Index (RDI), if PSG is preformed, or Respiratory Event Index (REI) determines this if OCST is performed.

- AHI = number of Apnoeas+ Hypopnoeas/total sleep time
- RDI = number of Apneas+Hypopneas +RERAs/total sleep time
- REI = number of Apneas+Hypopneas/monitoring time
- AHI or REI <5/hour = normal (for adults); 5–14.9/hour = mild OSA; 15–29.9/hour = moderate OSA; and ≥30/hour = severe OSA[20].

Diagnostic tests:
Classification of sleep tests made depending on the number of the channels monitored during the test. Polysomnography (PSG) or the in-lab attended sleep study, which is the gold standard test to diagnose OSA. Out-of-centre sleep testing (OCST) or Home Sleep Apnoea Test (HSAT) is an acceptable alternative, when PSG is not feasible. The HSAT does not measure sleep, as there is no EEG recording. The American association of sleep medicine (AASM) recommends HSAT in patients with high pre-test probability after comprehensive sleep evaluation. HSAT is not appropriate in patients with comorbid severe pulmonary disease, congestive heart failure, and neuromuscular disease. A negative or technically inadequate HSAT is followed by PSG in a symptomatic patient with high index of suspicion for OSA[19'20].

Treatment of sleep apnoea syndrome:
Nasal CPAP is the treatment of choice in OSA, although previously invasive treatments as tracheostomy were required. CPAP provides a pneumatic splint by delivering airway pressure above atmospheric pressure, in order to increase upper airway cross-sectional area and volume and to keep the upper airway open during sleep[21, 22].

Patients and methods

We conducted this prospective case control study at Cardiac Centre/ Erbil/ Kurdistan region/ Iraq, from January 2019 to July 2020. We studied a convenient sample of 50 patients that were admitted to the coronary care units and diagnosed as acute ST elevation myocardial infarction and undergoing primary percutaneous coronary intervention.

Inclusion criteria:

1. Patients with coronary artery disease diagnosed by primary percutaneous coronary intervention.
2. Both male and female.
3. Age less 55 years old in males and Age less than 65 years old in females
4. Accepted to participate in the study.

Exclusion criteria:

1. Having chronic respiratory disease.
2. Diabetes Miletus
3. Previously diagnosed coronary artery diseases
4. Family history of premature coronary artery disease

The variables of interest in the questionnaires were patients' code, age, body mass index (BMI) smoking or not, medical history; specifically hypertension, hyperlipidemia or both, severity of coronary artery disease as: one, two and three vessel disease and left main stenosis, follow up ejection fraction by echocardiogram after 6 months of PCI and mortality after one year. Regarding screening for sleep apnoea we used the STOP-BANG score[23]. Patients were divided into two groups first group or control group; were those identified as low risk for the development of sleep apnoea, home sleep test (HST) was not done for them and the second group or case group; were those identified as high risk for development for sleep apnoea and HST was done for them. We compared both groups in terms of differences in their general characteristics (such as: age, BMI, presence of other risk factors for CAD, smoking) and most importantly severity of CAD that was identified during primary percutaneous coronary intervention (PCI). The high-risk group for whom we did HST, we identified the severity of sleep apnoea by AHI and mean minimum SPO2 during the test.

Scores calculated from 0 to 2 can be classified as being at low risk for moderate to severe OSA. Those with a STOP-Bang score of 5 to 8 can be classified as being at high risk for moderate to severe OSA. In patients

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with a STOP-Bang score of 3 or 4, the specific combinations of positive items should be examined further to ensure proper classification. If a combination of a STOP score ≥ 2 plus (BMI > 35 kg/m2 or neck circumference > 40 cm) we considered it as high-risk group.

Table I: STOP BANG score

	Question	Yes or No
S	Do you Snore loudly?	
T	Do you feel Tired or sleepy during the day?	
O	Has anyone Observed apneas or choking during sleep?	
P	Do you have high Blood Pressure	
B	BMI > 35	
A	Age > 50	
N	Neck circumference ≥17 inches (men); ≥16 inches (women)	
G	Gender – male	

After explaining the study to the participants and taking medical history, we performed examination for them including general examination of weight and height and of vital signs as heart rate and blood pressure, with measurements of neck circumference (by wrapping a tape measure in mid neck just below the cricothyroid membrane with the shoulders relaxed and patient in upright position). Severity of CAD was already determined at PCI and follow up echocardiogram was done for all patients after 6 months.

The home sleep test (HST)

After taking written informed consent from the participant, the sleep study was performed nearly one month after the diagnosis of acute myocardial infarction using the home sleep study device Alice NightOne Philips Respironics. The device consists of 3 sensors: a belt with integrated buckle design, a disposable Nasal Cannula and a pulse oximeter. The device works by connecting the elastic belt to the testing device and wearing it to the patients mid chest. Then the nasal cannula is connected to the device and under the patients nose and oximeter probe attached to the patient's fingertip. There are green lights on the device that act as indicators to ensure the accurate testing has been completed prior to returning to the sleeps services. The device should work for minimum 3 hours of sleep. Then when the device retuned, it was connected to the computer and test result shown through SleepwareG3 program. The results were scored by this software and interpreted by the sleep specialist. The specialist used AHI scale to count episodes of apnoea and hypopneas (which must last at least more than 10 seconds) in each hour of sleep. Through these findings the high-risk patients where ranked as having: no sleep apnoea, mild, moderate or severe sleep apnoea.

Ethical considerations:

We had our study approval from scientific research committee of the Kurdistan Board of Medical specialities and we took permission from authorities in Cardiac Centre in Erbil. While interviewing the patients we took verbal consent and from those who underwent sleep test we took written informed consent.

Statistical analysis:

We analysed our data using the SPSS software and we compared the means of both groups using the students' "t" test and the value of statistical significance was set as $p<0.05$.

Results

In our study we compared two groups of patients. Of these; twenty-five patients were ranked as low risk for development of sleep apnoea with a STOP-BANG score of less than 2 and we used them as a control and compared them to the high-risk group who scored 5 and more (also 25 patients):

The general characteristics of the two groups where as follows:

The mean age of the low risk and high-risk groups were 52.84 and 52.24 years respectively with statistically insignificant differences (p: 0.755). While there was a statistically significant difference between the two groups with regards to mean BMI (35.60 in high risk vs. 29.32 in low risk group), there were insignificant differences in terms of gender and smoking status (16 males and 9 females were high risk and 17 males and 8 females were low risk, 16 low risk and 4 high risk patients were smokers, as illustrated in table 2:

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Table II: General characteristics of the study groups

Variables	Study group	N	Mean	Std. Deviation	P
Age	High risk	25	52.84	6.434	0.755
	Low risk	25	52.24	7.073	
Body mass index	High risk	25	35.60	2.972	0.001
	Low risk	25	29.32	3.288	
Stop bang score	High risk	25	5.76	.597	0.001
	Low risk	25	2.88	.726	

p: 0.765			N	%	Total
Gender		High risk	16	48.5%	33
	Male	Low risk	17	51.5%	
	Female	High risk	9	52.9%	17
		Low risk	8	47.1%	

p: 0.564		N		%	Total
Smoker	No	High risk	11	55.0%	20
		Low risk	9	45.0%	
	Yes	High risk	14	46.7%	30
		Low risk	16	53.3%	

With regards to medical diseases, hypertension was found in 10 high risk and 8 low risk patients but hyperlipidemia was more frequent in the low risk (10 patients) than the high risk (6 patients). Though these differences were not significant (p: 0.664).

Table III: Past medical history of the study groups

p: 0.664		Study group High risk	Low risk	Total
Medical HX	no disease	6	4	10
		60.0%	40.0%	100.0%
	hypertension	10	8	18
		55.6%	44.4%	100.0%
	hyperlipidemia	6	10	16
		37.5%	62.5%	100.0%
	combined	3	3	6

After primary percutaneous coronary intervention (PCI) the severity of coronary artery disease in high risk and low risk groups were as shown in table 4 as more severe CAD i.e. three vessel disease and left main stenosis were found more frequently in the high risk group. Still these differences were not empowered statistically (p: 0.264):

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Table IV: Severity of CAD among the study groups

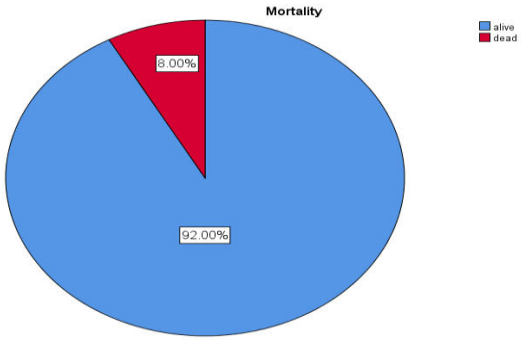
p: 0.264		Study group High risk	Low risk	Total
Severity of coronary artery disease	one vessel disease	2	6	8
		25.0%	75.0%	100.0%
	two vessel disease	11	13	24
		45.8%	54.2%	100.0%
	three vessel disease	4	2	6
		66.7%	33.3%	100.0%
	left main stenosis	8	4	12
		66.7%	33.3%	100.0%
Total		25	25	50

Follow up echocardiography of the patients of both groups after 6 months showed a mean Ejection Fraction (EF) of (52.08 vs. 52.28) of the high risk and low risk groups respectively which was also insignificant (p: 0.940):

Table V: Follow up echocardiography of the study groups

Variables	Study group	Mean	Std. Deviation	P
Ejection fraction	High risk	52.08	9.473	0.940
	Low risk	52.28	9.330	

The one year mortality from CAD of our study population was 8% (4 deaths out of 50 cases) which may not directly correlate with the being low risk or high risk for sleep apnoea in which there were 3 deaths from the high risk and 1death from the low risk group but insignificant statistically:



FigureI: Study population mortality

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Table VI: Mortality of the study groups

P: 0.609			Study group High risk	Low risk	Total
Mortality	Alive		22	24	46
			47.8%	52.2%	100.0%
	Dead		3	1	4
			75.0%	25.0%	100.0%
Total			25	25	50
			50.0%	50.0%	100.0%

After performing the sleep test for the high risk group, they had a mean AHI of 14.9 which means upper limit of mild sleep apnoea and a mean minimum SPO_2 of 85.4%:

Table VII: The home sleep test

	N	Range	Minimum	Maximum	Mean	Std. Deviation
AHI	25	39.60	4.40	44.00	14.9920	11.56964
Minimum spo2	25	18	73	91	85.40	5.515

Nearly half of high-risk patients had mild OSA (n: 12) and 28% had moderate OSA (n: 7) and severe OSA was found in 12% (n:3) as shown in the figure2:

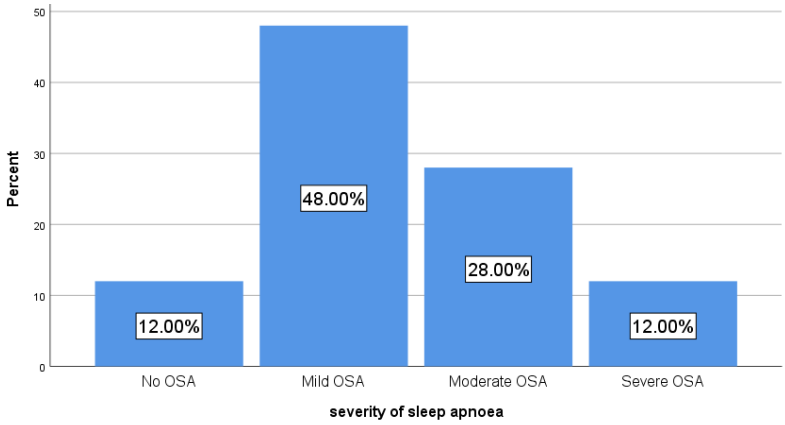

Figure II: Severity of obstructive sleep apnoea in the high-risk group

Discussion

It has been found that obstructive sleep apnoea poses a significant impact on public health with a prevalence rate of 9% in women and 25% in men and it has been implicated as a serious factor for increasing the risk of development of many medical conditions, of these group cardiovascular disorders are most commonly encountered namely; hypertension, atherosclerotic coronary artery disease and stroke particularly in middle aged adults. Moreover, sleep apnoea can lead to poor performances at work and increased risk of road traffic accidents due to daytime somnolence and poor concentration[3].

In the present study we found BMI was significantly higher in the high-risk group, were as other features differences as gender, age and smoking status were insignificant. In a polish study by Filip M[24], similarly they found higher BMI in the sleep apnoea group but older ages.

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We found hypertension more frequently in the high-risk group but hyperlipidemia less. Eugene et al[25] found that undiagnosed sleep apnoea syndrome may be associated with systemic hypertension in many middle- and older-aged men by comparing them to normotensive men at same age and weight groups.
Although in this study we found the low-risk group having hyperlipidemia (although few in number) more frequently, on the contrary; Savransky V[26] showed that chronic intermittent hypoxia (that is commonly found in severe OSA) leads to hypercholesterolemia and lipid peroxidation, in the absence of obesity, and that the degree of metabolic impairment is dependent on the severity of the hypoxic stimulus.
On the other hand, we observed more severe coronary artery disease among the high-risk group (such us left main stenosis and three vessel disease) but comparable follow up ejection fractions of the two groups. Nakashima et al[27] investigated the association of SDB and infarct size after AMI based on cardiovascular magnetic resonance and left ventricular angiography, respectively. They found less myocardial salvage, smaller reduction in infarct size and less recovery of left ventricular function in patients with AHI ≥15 events/h after 3 months and after 21 days, respectively. These findings reinforce that SDB contributes to impaired outcome after AMI.
After one year of follow up, we found higher mortality in the high-risk group (3cases) compared to the low risk group (1 case). Findings of some other studies are controversial, as Mohananey et al[28] identified a group of OSA patients with diagnosis of MI from a nationwide sample. The cohort included 1 850 625 patients with STEMI, of whom 24 623 patients (1.3%) had documented OSA. The prevalence of male sex, smoking, chronic pulmonary disease, depression, hypertension, known history of CAD, dyslipidaemia, obesity and renal failure was higher in OSA patients. Interestingly, OSA patients had significantly lower in-hospital mortality, but longer hospital stays. In contrast, other studies have emphasised a negative prognostic impact of OSA on acute coronary syndrome as Barbé et al[29] when including 213 OSA patients (AHI >15 events/h) and comparing them with 218 controls. Patients with OSA showed higher prevalence of arterial hypertension, higher BMI and lower percentage of smokers. After adjusting for these parameters, OSA was associated with higher peak troponin levels in plasma and longer stay in the coronary care unit. They also concluded that higher AHI was associated with increased number of the diseased vessels.
Finally, when performing the home sleep test for the high-risk group, we found nearly half (48%) of high-risk patients had mild OSA (n: 12) and 28% had moderate OSA (n: 7) and severe OSA was found in 12% (n: 3). Our result are consistent with findings of Ludka[30] who observed when using a threshold of AHI ≥ 5 events/hour, sleep apnoea was present in 65.7% of patients after acute MI. mild sleep apnoea was present in 32.6%, moderate in 20.4% and severe in 12.7%.
The major limitations of current study were: first, sample size was small, as we might have lost follow-up, had invalid sleep tests or patients refusal to complete the study. Second: some studies have observed that there might be a transient worsening of sleep disordered breathing in the acute phase of MI and then returns back to previous levels. However, the sleep test was performed after nearly one month from the diagnosis of MI and the patient were clinically stable.
The points that we regard them strength points are that this study is the first one in Iraq focusing on sleep apnoea in patients with coronary artery disease, and it was an opportunity to discuss and bring lights on the presence and implications of sleep apnoea among the patients.

Conclusions

We concluded that raised BMI (>35) is an important predictor of presence of sleep apnoea in patients with acute myocardial infarction and the finding of severe sleep apnoea among high-risk individuals is not uncommon.

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