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Original Article

The influence of timing of polysomnography on diagnosis of obstructive sleep apnea in patients presenting with acute myocardial infarction and stable coronary artery disease

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ABSTRACT

Background: We aimed to determine if timing of polysomnography (PSG) influences the diagnosis of obstructive sleep apnea (OSA) in acute myocardial infarction (AMI) or stable coronary artery disease (CAD).

Methods: A total of 160 patients admitted with AMI or stable CAD were consecutively recruited for either in-hospital ($n = 80$) or postdischarge ($n = 80$) PSG.

Results: The median time from admission to PSG for the in-hospital and postdischarge groups was 1 day and 17 days, respectively ($P < .001$). Overall, 59 patients (36.9%) were diagnosed with OSA (apnea–hypopnea index [AHI] $\geqslant 15$), and they were more likely to have diabetes mellitus (DM), hypertension, hyperlipidemia, chronic renal failure, and a greater body mass index (BMI) ($P < .05$ for all). The diagnosis of OSA was significantly higher ($P = .037$) in patients who had a PSG performed as an inpatient than those who had a PSG as an outpatient. There was a significant interaction between clinical presentation and the effect of PSG timing on the diagnosis of OSA ($P = .003$). For the patients presenting with AMI but not those with stable CAD, in-hospital PSG was an independent predictor of OSA (adjusted odds ratio, 3.84 [95% confidence interval, 1.42–10.41]; $P = .008$).

Conclusion: The timing of PSG influenced the diagnosis of OSA in patients who presented with AMI but not in those who presented with stable CAD.

1. Introduction

Obstructive sleep apnea (OSA) is characterized by recurrent upper airway collapse and intermittent apnea or hypopnea episodes during sleep. Apart from causing disruptive snoring and excessive daytime somnolence, there is evidence that OSA is associated with adverse effects on cardiovascular physiology [1–3]. Epidemiologic studies have shown that the prevalence of OSA was significantly higher in patients with cardiovascular disease than in the general population [4,5] and that untreated OSA leads to untoward cardiovascular events [6–8]. The results from these studies highlight the importance of diagnosing concomitant OSA in patients presenting with cardiovascular disease [9].

Overnight polysomnography (PSG) is a simple and noninvasive investigation for diagnosing OSA. However, the optimal time to perform PSG for patients admitted with coronary heart disease has not been well-defined. There have been a few studies describing how PSG performed at different times seemed to have affected the results. In a study of 18 patients admitted to the coronary care unit for a variety of cardiac conditions, the prevalence of OSA reduced from 56% during the acute phase to 18% at 6-week follow-up [10]. Likewise, in 28 patients presenting with acute coronary syndrome and diagnosed with OSA during hospitalization, repeat PSG at 6-month follow-up showed resolution of OSA in most patients [11]. This finding suggests that the timing of PSG may influence the diagnosis of OSA. However, these studies were too small to be conclusive, and the lower prevalence of OSA at the repeat PSG could be due to measures taken by the patients when receiving the diagnosis after the first PSG. Moreover, the small sample size precluded analysis based on different clinical presentations.

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Determining the optimal timing of PSG in patients is crucial in correctly identifying patients with OSA, and therefore optimizing resource allocation. In our study, we sought to determine if the timing of PSG affected the diagnosis of OSA. We also evaluated if the relationship differed for patients presenting with acute myocardial infarction (AMI) vs stable coronary artery disease (CAD).

2. Materials and methods

2.1. Study design and patient population

Our study was a single-center study conducted at a university-affiliated hospital. We prospectively recruited 160 consecutive patients who had undergone coronary angiography and intervention for either AMI ($n = 80$) or stable CAD ($n = 80$). Recruited patients were scheduled to undergo a PSG during in-hospital or postdischarge period in a 1:1 ratio. We first recruited 80 consecutive patients (AMI [$n = 40$]; stable CAD [$n = 40$]) in a parallel manner for in-hospital PSG, followed by recruiting another 80 consecutive patients (AMI [$n = 40$]; stable CAD [$n = 40$]) in a parallel manner for postdischarge PSG. The recruitment began in December 2007 and ended in February 2012. Exclusion criteria were known OSA, intubation for mechanical ventilation, sedation, high risk for malignant ventricular arrhythmia, cardiogenic shock, previous coronary artery bypass surgery, and inability to give informed consent. The study was approved by the local institutional review board and all patients provided written informed consent prior to participation.

For the in-hospital PSG group, all of the polysomnograms were done in a cardiology ward before hospital discharge. For the post-discharge PSG group, all of the polysomnograms were scheduled within 4 weeks after hospital discharge and conducted at patients' homes [12]. The PSG appointment was given to the patients on hospital discharge.

2.2. Definition of AMI and stable CAD

AMI was defined as typical chest pain and more than 1-mm ST-segment elevation on a standard 12-lead electrocardiography. As a standard treatment at our institution, all AMI patients were treated with primary percutaneous coronary intervention (PCI). We followed the American College of Cardiology/American Heart Association guideline for primary PCI (door-to-balloon time) to be performed within 90 min after admission [13]. In our institution, the door-to-balloon time for primary PCI in 2007 and 2008 was 72 min [14]. Stable CAD was defined as symptomatic angina, angina-equivalent or silent ischemia with normal baseline cardiac enzyme levels (creatine kinase [CK]-MB $\leqslant 6$ μg/L or troponin I <0.03 μg/L.

2.3. Overnight PSG

All PSG studies were performed using a portable diagnostic device (Embletta Gold, Natus Medical Inc., Canada) suitable for both hospital and home use; it also has been validated against a full laboratory PSG [15]. Standard parameters were measured including nasal airflow (nasal cannula), thoracoabdominal movements (inductive respiratory bands), arterial oxygen saturation (pulse oximetry), snoring episodes derived from the integrated pressure transducer, limb movement, electrocardiogram and body position (continuous actigraphy).

Outputs from the portable diagnostic device were analyzed by two investigators with no knowledge of the clinical characteristics of the patients. An apneic episode was defined as a cessation of airflow for more than 10 s, and hypopnea was defined as a reduction of airflow of more than 50%, lasting more than 10 s. An event also was considered hypopnea if airflow reduction did not reach 50% but was associated with arterial oxygen desaturation of more than 3%. Apneas were classified as obstructive if paradoxical thoracoabdominal movement was observed and as central if there was no thoracoabdominal movement. For the purpose of our study, the sleep-disordered breathing of interest was OSA; therefore, patients with predominantly central sleep apnea were excluded for analysis. The apnea–hypopnea index (AHI) was calculated as the number of apneic plus hypopneic episodes per hour of recording time; the start of recording was considered the point when respiration settled to a rhythmic stable pattern, and the end of recording was either the waking time recorded by the subject or the point when the thoracoabdominal tracings became disturbed consistent with wakefulness [10]. The recruited patients were classified into OSA (AHI $\geqslant 15$) vs non-OSA (AHI <15).

2.4. Demographic and clinical data collection

The following demographic and clinical data were collected from consenting patients: age, gender, ethnicity, height, weight, body mass index (BMI), blood pressure, heart rate and rhythm, cardiovascular risk factors (e.g., diabetes mellitus [DM], hypertension, smoking, hyperlipidemia, family history of premature CAD), concomitant medical history (e.g., previous PCI, coronary artery bypass surgery, myocardial infarction, stroke, chronic renal impairment), infarct location (e.g., anterior, nonanterior, unknown), time from admission to PSG, angiographic parameters, peak level of cardiac enzymes (AMI patients only), and discharge medications (e.g., aspirin, thienopyridine, β blocker, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, lipid-lowering therapy).

2.5. Statistical analysis

Univariate association between OSA and other patient-related risk factors, such as gender, ethnicity, cardiovascular risk factors, concomitant conditions, PSG timing by clinical presentation, discharge medications, and angiographic characteristics of the culprit lesions were evaluated using the Fisher exact test. For continuous covariates such as age and BMI, the difference in means between groups was compared using an independent sample t test. We further explored the relationship between OSA diagnosis and the PSG timing, adjusting for potential confounding factors, such as DM, BMI, and hyperlipidemia using multivariable logistic regression analysis. We included an interaction term in the model involving PSG timing and clinical presentation. All statistical analyses were performed using STATA version 12 (StataCorp LP, College Station, TX, USA), assuming a two-sided test with a 5% level of significance.

3. Results

3.1. Study profile and patient characteristics

The study profile is shown in Fig. 1. A total of 160 consecutive patients presenting with coronary heart disease were recruited and scheduled to undergo in-hospital ($n = 80$) or postdischarge ($n = 80$) PSG. The demographic and clinical characteristics of the patients are shown in Table 1 stratified by timing of sleep study. Most of the patients were men. The ethnic composition of the recruited patients was consistent with the overall ethnic composition of Singapore.

All of the PSG studies were successful and the tracings were available for analysis for all of the recruited patients. The investigators who analyzed the tracings were blinded to the clinical presentation and angiographic and procedural details of the patients. The distribution of AHI in the entire patient cohort is shown in Fig. 2.