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Association of Obstructive Sleep Apnea with Aortic Dilatation in Hypertensive Men of Dominican Descent in an Outpatient Clinic

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Abstract

Obstructive sleep apnea (OSA) is a highly prevalent disorder, affecting up to 50% of middle-aged men and 23% of middle-aged women, with up to 80% of patients with OSA being undiagnosed. Several studies have shown an association between OSA and thoracic aortic dilatation/aneurysm; however, hypertension seems to play an important role in the incidence of aortic root dilatation and dissection. Thus, given the fact that OSA is also linked to hypertension, it remains unclear if the association between OSA and thoracic aortic aneurysms is causal or if the aortic dilatation is actually secondary to the OSA-induced hypertension. We conducted a retrospective cross-sectional study and we included a total of 295 male hypertensive patients of Dominican descent ≥ 45 years of age (145 patients diagnosed with OSA and 150 patients without OSA).There was a statistically significant higher percentage of patients with an aortic root size > 3.7 cm in the OSA group, as compared to the non-OSA group (41.37% vs. 26.66%; P=0.0097). Thus, our small retrospective, cross-sectional study supports a direct association between OSA and aortic dilatation, beyond the presence of HTN, in a male, hypertensive population of Dominican descent. Notwithstanding, further larger studies in diverse patient populations will be needed to definitely confirm this association and possibly establish its causal nature.

Keywords: Obstructive sleep apnea; Aortic dilatation; Hypertension; Dominican ethnicity

Introduction

Obstructive sleep apnea (OSA) is a highly prevalent disorder, affecting up to 50% of middle-aged men and 23% of middle-aged women [1], with up to 80% of patients with OSA being undiagnosed [2]. This condition is characterized by a repetitive partial or complete obstruction of the pharynx during sleep. Despite increasing breathing efforts, the upper airway collapse results in episodes of obstructive hypopnea or apnea affecting the sleep architecture and the whole body via instant and long-term mechanisms. The severity of sleep apnea is measured with the apnea/hypopnea index (AHI), which quantifies the number of times per hour an episode of apnea or hypopnea occurs. An AHI ≥ 5 is currently considered as diagnostic of OSA.

OSA has been shown to have a strong association with cardiovascular disease (CVD) [3-8] and it has been linked to a higher incidence of cardiovascular morbidity and mortality [9]. In addition, OSA is considered to be a causal factor in the pathogenesis of vascular dysfunction and hypertension (HTN) [10].

The Sleep Heart Health Study, in a cross-sectional analysis of > 6000 patients, showed that over 50% of individuals with OSA have HTN and revealed a linear relationship between systolic (SBP) and diastolic blood pressure (DBP) and OSA severity [11].

Several studies have shown an association between OSA and thoracic aortic dilatation/aneurysm [12,13]; however, hypertension seems to play an important role in the incidence of aortic root dilatation and dissection [14,15]. Thus, given the fact that OSA is also linked to hypertension [15,16], it remains unclear if the association between OSA and thoracic aortic aneurysms is causal or if the aortic dilatation is actually secondary to the OSA-induced hypertension. As it was mentioned above, there is an increased prevalence of OSA in the male population with a male to female ratio of 2:1 [1]. Moreover, the male population presents a higher prevalence of thoracic aortic aneurysms compared to the female population [17], as well as an increased prevalence of acute aortic dissection with a male to female ratio of 1.5:1 [18].

Given the above, our study was designed to specifically answer the question if there is a direct association between OSA and aortic root dilatation, beyond the presence of HTN, in men of Dominican descent, seen in an outpatient clinic.

Patients and Methods

From October 2016 to February 2017 we conducted a retrospective cross-sectional study and we included a total of 295 male hypertensive patients of Dominican descent ≥ 45 years of age (145 patients diagnosed with OSA and 150 patients without OSA).

In our study, a patient was considered as having HTN if BP was ≥ 140/90 mm Hg (obtained during the patient's visit to the clinic when the patient was enrolled in our study), and/or if the patient was being treated with antihypertensive medication(s).

The diagnosis of OSA was established in our study in patients with an AHI ≥ 5 by polysomnography.

The aortic root size was measured in all patients by transthoracic echocardiography. Increased aortic size, based on transthoracic echocardiography, was considered in those patients with an aortic root diameter > 3.7 cm.

Patients with bicuspid aortic valve, Marfan syndrome, history of valve replacement or history of coronary artery bypass surgery were excluded from the study.

Statistical analysis was performed using the Fisher's Exact Test. Two-tailed P-values were used and a P-value < 0.05 was considered statistically significant.

Results

There were 145 male hypertensive patients diagnosed with OSA and 150 male hypertensive patients without OSA. The mean SPB and DBP in the OSA group were 126.88 mm Hg and 79.55 mm Hg, respectively. The mean SPB and DBP in the non-OSA group were 133.07 mm Hg and 83.08 mm Hg, respectively. The blood pressure was well controlled with therapy (< 140/90 mm Hg) in 95 of the 145 patients in the OSA group and in 70 of the 150 patients in the non-OSA group. The mean aortic root size was 3.61 cm in the OSA group and 3.50 cm in the non-OSA group. There was a statistically significant higher percentage of patients with an aortic root size > 3.7 cm in the OSA group, as compared to the non-OSA group (41.37% vs. 26.66%; P=0.0097). In the subgroup of patients with controlled BP, the presence of an aortic root size > 3.7 cm was again significantly more frequent in the OSA group, as compared to the non-OSA group (41.05% vs. 22.85%; P=0.0190). In the subgroup of patients with not well controlled BP, the presence of an aortic root size > 3.7 cm was again more frequent in the OSA group, as compared to the non-OSA group (42.0% vs. 30.0%); however, probably due to the smaller number of patients, the difference did not reach statistical significance.

Discussion

In our study, which included 295 hypertensive men of Dominican descent, we observed a statistically significant higher prevalence of aortic dilatation (defined as aortic root size > 3.7 cm) in patients with OSA. More specifically, aortic dilatation was present in 41.37% of patients in the OSA group vs. 26.66% of patients in the non-OSA group (P= 0.0097) despite a somewhat lower mean SBP and DBP in the OSA group, as compared to the non-OSA group (126.88 mm Hg and 79.55 mm Hg vs. 133.07 mm Hg and 83.08 mm Hg, respectively).

Although several studies have linked OSA to an increased incidence of thoracic aortic dilatation/aneurysms [12,13], the fact that OSA is also linked to hypertension [15,16], which seems to play an important role in the incidence of aortic root dilatation and dissection [14,15], brings up the question if the association between OSA and thoracic aortic dilatation is causal or if the aortic dilatation is secondary to OSA-induced HTN.

In our study, we actually demonstrated a statistically significant, direct association between OSA and aortic dilatation, beyond the presence of HTN.

Several mechanisms may be responsible for the observed association between OSA and aortic dilatation. These may include nocturnal negative intra thoracic pressure surges leading to mechanical stretching of the aorta and ultimately aortic distension, arousal-induced reflex sympathetic activation with subsequent hypertension, as well as intermittent hypoxia associated with autonomic nervous system activation and consequently increased oxidative stress [19].

The results of our study are in agreement with those of an earlier prospective, cross-sectional study, which demonstrated that patients with OSA had a significantly greater thoracic aortic size than those without OSA, whereas there was no significant independent relationship between blood pressure/hypertension and thoracic aortic size [20]. Furthermore, in a recent prospective, observational study, it was shown that moderate to severe OSA seemed to increase the rate of expansion of thoracic aortic aneurysms at the level of sinus of Valsalva, whereas Continuous Positive Airway Pressure (CPAP) appeared to be an effective counter measure [21].

In conclusion, our small retrospective, cross-sectional study supports a direct association between OSA and aortic dilatation, beyond the presence of HTN, in a male hypertensive population of Dominican descent. Notwithstanding, further larger studies in diverse patient populations will be needed to definitely confirm this association and possibly establish its causal nature.

Finally, among the limitations of our study is the relatively small number of subjects, which prevented a statistically sound multivariate analysis, as well as the use of only one imaging method (transthoracic echocardiogram) and the measurement of the aortic size only at the aortic root level.

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