Aortic Root Diameter in Hypertensive Patients With Various Stages of Obstructive Sleep Apnea

Dian Wang,1 Jian-Zhong Xu,1 Yuan-Yuan Kang,1 Wei Zhang,1 Lei-Xiao Hu,1 and Ji-Guang Wang1,

**BACKGROUND**
Obstructive sleep apnea (OSA) is a risk factor of several cardiovascular diseases. We investigated the association between aortic root diameter and hypoxia-related parameters in hypertensive patients with OSA.

**METHODS**
Our study included 242 hypertensive patients with OSA (52 mild, 71 moderate, and 119 severe). All the patients underwent echocardiography for measuring aortic root diameter and polysomnography for measuring apnea–hypopnea index (AHI), oxygen desaturation index, and time spent with oxygen desaturation less than 90%.

**RESULTS**
The study patients included 19.8% women and had a mean (±SD) age of 49.9 ± 12.9 years, a mean aortic root diameter of 33.4 ± 2.6 mm, and a prevalence of echocardiographic aortic root dilation of 3.7%. Patients with mild, moderate, and severe OSA had similar echocardiographic left ventricular structure. However, patients with severe OSA had a significantly (P < 0.05) greater aortic root diameter (33.9 ± 2.4 mm vs. 32.4 ± 2.2 and 33.4 ± 2.9 mm, respectively) and higher prevalence of aortic root dilatation (5% vs. 1% and 3%, respectively) than those with mild and moderate OSA. Aortic root diameter corrected by body height was significantly (P < 0.001) associated with AHI, oxygen desaturation index and time spent with oxygen desaturation less than 90% (r = 0.23–0.33). After adjustment for various confounding factors, the associations between aortic root diameter and polysomnography parameters remained statistically significant (P < 0.05).

**CONCLUSIONS**
The severity of OSA was associated with the aortic root diameter. Patients with severe OSA had a greater aortic root diameter.

**Keywords:** aortic root diameter; blood pressure; echocardiography; hypertension; obstructive sleep apnea.

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Obstructive sleep apnea (OSA) is disordered breathing in which the upper airway closes repeatedly during sleep.1 These repetitive partial or complete cessations of airflow during sleep result in intermittent hypoxia and oxygen desaturation. Clinic-based observational studies have reported that OSA is associated with an increased risk of cardiovascular diseases, such as systemic arterial hypertension, coronary artery disease, congestive heart failure, cardiac arrhythmia, and stroke.2–5 Several previous studies showed that OSA also promoted progressive aortic dilatation in patients with Marfan’s syndrome, aortic aneurysm, or aortic dissection.6–8 An increased aortic root diameter represents a risk factor for left ventricular hypertrophy, left ventricular dysfunction, and renal dysfunction.9–11 Several nonhemodynamic factors influence aortic root diameter, including age, gender, body height, cigarette smoking, blood pressure, serum lipids, and plasma glucose.12–14

We hypothesize that intermittent hypoxia as a prominent feature of OSA is associated with the changes in aortic root diameter. In the present study, we investigated the association between aortic root diameter and various hypoxia-related parameters, including apnea–hypopnea index (AHI), oxygen desaturation index, and time spent with oxygen desaturation less than 90% in patients with OSA.

**METHODS**

**Study population**
Our retrospective cross-sectional study included 242 adult hypertensive patients with a confirmed diagnosis of OSA,
who admitted in the hypertension ward in Ruijin Hospital, Shanghai, China from May 2020 to April 2021. All these patients underwent full polysomnography due to symptoms of nocturnal snoring and/or excessive daytime sleepiness. According to the AHI, patients were classified as mild, moderate, and severe OSA with an AHI 5–15/hour (n = 52), 15–30/hour (n = 71), and ≥30/hour (n = 119), respectively.

Exclusion criteria included atrial fibrillation, respiratory failure, congestive heart failure, coronary artery disease, significant valvular heart disease, congenital heart disease, and suboptimal echocardiographic windows. The study protocol was approved by the Ethics Committee of Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China. All patients gave informed written consent.

**Polysomnography**

All patients underwent an overnight polysomnographic study using a standardized suite (Philips Respironics, Pittsburgh, PA). Sleep parameters were then recorded and analyzed. Experienced technicians identified the respiratory events according to the standard criteria. Apnea was defined as complete cessation of airflow for at least 10 seconds, and hypopnea was defined as a ≥50% reduction in airflow, lasting at least 10 seconds, associated with a decrease of at least 4% in the nocturnal oxygen saturation (SaO2) or state of arousal. AHI was calculated as the total number of apneas and/or hypopneas per hour. OSA was defined as AHI ≥5 events per hour. Other sleep parameters included mean sleep time and mean wake time. The study was performed at rest by an experienced research sonographer blinded to clinical information and respiratory data using the Philips IE33 device (Philips, Eindhoven, The Netherlands).

**Echocardiography**

Standard 2-dimensional (2D) echocardiography was performed at rest by an experienced research sonographer blinded to clinical information and respiratory data using the Philips IE33 device (Philips, Eindhoven, The Netherlands). Aortic root diameter was measured in the parasternal long-axis view at the level of sinus of Valsalva in end-diastole, using the leading-edge to leading-edge method. The aortic root diameter corrected for body height was used for further statistical analysis. Aortic root dilatation was defined as an aortic root diameter greater than 37 mm for men and 34 mm for women.

Left ventricular end-diastolic diameter (LVEDd), diastolic posterior wall thickness (PWTd), and diastolic interventricular septum thickness (IVSTd) were imaged from a parasternal long-axis window at the level of the mitral annulus using 2D-targeted M-mode echocardiography. Left ventricular mass (LVM) was calculated according to the American Society of Echocardiography-cube formula by Devereux et al.:

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\text{LVM (g)} = 0.8 \times \left[1.04 \times \left(\text{LVEDd} + \text{PWTd} + \text{IVSTd}\right) \right] - \left(\text{LVEDd} \right) \right] + 0.6
\]

LVM was indexed for body surface area to obtain left ventricular mass index (LVMI). Left ventricular hypertrophy was defined as a LVMI >115 g/m² in men and >95 g/m² in women. We measured the peak velocities of early (E) and atrial (A) diastolic filling, mitral valve deceleration time, and isovolumic relaxation time and calculated the E/A ratio. E’ was the mean value of the mitral annular velocities measured at the septal and lateral annuli by tissue Doppler imaging. The E/E’ ratio was calculated by dividing early mitral inflow velocity (E) by early diastolic mitral annular velocity (E’).

**Blood pressure measurement**

Blood pressure was measured on the day of admission at the hypertension inpatient ward. An automated oscillometric electronic blood pressure monitor was used during the entire study period (Omron BP-1300, Omron Healthcare, Kyoto, Japan). Two consecutive readings were obtained with a 1-minute interval after at least 5 minutes rest in the seated position. These 2 blood pressure readings were averaged for statistical analysis.

Ambulatory blood pressure monitoring was performed during hospitalization. We programmed validated oscillometric SpaceLabs 90217 monitors (SpaceLabs, Redmond, WA) to obtain blood pressure readings at 20-minute intervals during daytime (06:00–22:00) and at 30-minute intervals during nighttime (22:00–06:00). All recordings covered >20 hours and included ≥10 readings during the awake period and ≥5 readings during sleep. Mean values were weighed for the time interval between consecutive readings.

**Statistical analysis**

Statistical analysis was performed using the SPSS software, version 17.0 (SPSS, Chicago, IL). Continuous variables were expressed as mean ± SD and nominal variables as percentages. Analysis of covariance was performed for the between-group comparisons. Correlation analysis was performed to study the associations of interest. While the main analysis focused on the aortic root diameter corrected by body height, we performed sensitivity analysis with the aortic root diameter corrected by body surface area. P values <0.05 were considered to be statistically significant.

**RESULTS**

**Patient characteristics**

Clinical characteristics of the patients according to the severity of OSA are shown in Table 1. Patients with severe OSA had a significantly (P ≤ 0.002) greater body weight and body mass index and higher 24-hour, daytime, and nighttime ambulatory diastolic blood pressure than patients with mild and moderate OSA. They had similar age, sex, body height, clinic blood pressure, clinic pulse rate, 24-hour, daytime, and nighttime ambulatory systolic blood pressure, 24-hour pulse rate, hypertension history, prevalence of diabetes mellitus, dyslipidemia, and current smoking (P ≥ 0.07), but significantly different use of various classes of antihypertensive drugs (P < 0.05), except for calcium-channel blockers (P = 0.44) and diuretics (P = 0.38).

**Polysomnography**

The polysomnographic data are shown in Table 2. Patients with severe OSA, compared with those with mild or
moderate OSA, had a significantly higher AHI, time spent with oxygen desaturation <90% and oxygen desaturation index, and had a lower level of the mean nocturnal saturation of arterial oxygen, and the lowest saturation of arterial oxygen (all \( P < 0.001 \)).

**Standard echocardiography**

The standard echocardiographic data are shown in Table 3. These patients had similar left ventricular diastolic and systolic diameter, interventricular septal wall thickness, left ventricular posterior wall thickness, shortening fraction, and ejection fraction, \( E/A, E/E' \), LVM, LVMI, and prevalence of left ventricular hypertrophy and aortic root dilatation (\( P \geq 0.24 \)). However, they had significantly different aortic root diameter corrected for body height (19.0 ± 1.3, 19.5 ± 1.6, and 19.8 ± 1.3 mm/m in patients with mild, moderate, and severe OSA, respectively, \( P = 0.004 \)).

**Correlation analyses between aortic root diameter corrected by body height and hypoxia-related parameters**

Aortic root diameter corrected by body height was significantly (\( P < 0.001 \)) associated with AHI, oxygen desaturation index and time spent with oxygen desaturation less than 90% (\( r = 0.23–0.33 \), with a progressive increase associated with the severity of OSA, Figures 1–3). After adjustment for age, gender, body mass index, LVMI, blood pressure, hypertension history, diabetes mellitus, dyslipidemia, current smoking, and use of antihypertensive medications, the associations between aortic root diameter and polysomnography parameters remained statistically significant (\( P < 0.05 \)).

**Table 1. Clinical characteristics of the study patients**

| Characteristic                        | Mild OSA (n = 52) | Moderate OSA (n = 71) | Severe OSA (n = 119) | \( P \) (ANOVA) |
|---------------------------------------|-------------------|-----------------------|----------------------|-----------------|
| Age (y)                               | 49 ± 13           | 51 ± 13               | 49 ± 12              | 0.61            |
| Men (%)                               | 73%               | 80%                   | 83%                  | 0.31            |
| Body height (cm)                      | 169 ± 7           | 171 ± 8               | 171 ± 8              | 0.33            |
| Body weight (kg)                      | 78 ± 12           | 81 ± 14               | 85 ± 14\*           | 0.002           |
| Body mass index (kg/m\(^2\))         | 27 ± 3            | 27 ± 3                | 29 ± 3\*            | <0.001          |
| Hypertension history (y)              | 7 ± 7             | 9 ± 10                | 10 ± 9               | 0.31            |
| Systolic blood pressure (mm Hg)       | 148 ± 20          | 149 ± 21              | 147 ± 18             | 0.90            |
| Diastolic                              | 84 ± 9            | 87 ± 11               | 89 ± 13              | 0.07            |
| Clinic pulse rate (beats/min)         | 80 ± 13           | 83 ± 11               | 82 ± 11              | 0.34            |
| Ambulatory blood pressure (mm Hg)     |                   |                       |                      |                 |
| 24-Hour systolic                      | 136 ± 13          | 137 ± 14              | 138 ± 13             | 0.51            |
| 24-Hour diastolic                     | 81 ± 9            | 85 ± 9                | 87 ± 14\*           | 0.02            |
| Daytime systolic                      | 139 ± 13          | 140 ± 14              | 141 ± 13             | 0.52            |
| Daytime diastolic                     | 83 ± 9            | 87 ± 9\*              | 90 ± 10\*           | 0.001           |
| Nighttime systolic                    | 128 ± 17          | 128 ± 18              | 132 ± 16             | 0.32            |
| Nighttime diastolic                   | 77 ± 9            | 79 ± 11               | 83 ± 12\*\( ^* \)   | 0.004           |
| 24-Hour pulse rate (beats/min )       | 71 ± 9            | 72 ± 9                | 74 ± 10              | 0.23            |
| Diabetes mellitus, n (%)              | 12 (23%)          | 16 (22%)              | 21 (17%)             | 0.74            |
| Dyslipidemia, n (%)                   | 8 (15%)           | 6 (23%)               | 29 (24%)             | 0.39            |
| Current smoker, n (%)                 | 14 (26%)          | 29 (40%)              | 39 (32%)             | 0.25            |
| Use of antihypertensive drugs, n (%)  |                   |                       |                      |                 |
| Calcium-channel blockers              | 41 (78%)          | 62 (87%)              | 98 (82%)             | 0.44            |
| Angiotensin-converting enzyme inhibitors | 4 (8%)           | 20 (28%)              | 29 (24%)             | 0.02            |
| Angiotensin receptor blockers         | 42 (80%)          | 37 (52%)              | 76 (58%)             | 0.005           |
| β-Blockers                            | 18 (34%)          | 22 (30%)              | 63 (52%)             | 0.005           |
| α-Blockers                            | 15 (28%)          | 26 (36%)              | 52 (44%)             | 0.05            |
| Diuretics                             | 14 (26%)          | 14 (19%)              | 34 (28%)             | 0.38            |

Values are mean ± SD. Abbreviations: ANOVA, analysis of variance; OSA, obstructive sleep apnea.
\( ^* P < 0.05 \) compared with mild obstructive sleep apnea.
\( ^{11} P < 0.05 \) compared with moderate obstructive sleep apnea.
Aortic Root Diameter in Obstructive Sleep Apnea

Sensitivity analysis on aortic root diameter corrected by body surface area

Aortic root diameter corrected by body surface area was not significantly different (Table 3). However, both before and after adjustment for the aforementioned variables as appropriate, it was significantly associated with the polysomnography parameters ($P < 0.05$).

**DISCUSSION**

Our study showed that the severity of OSA was significantly associated with the aortic root diameter. Patients with severe OSA had a greater aortic root diameter. In fact, all major OSA-related parameters such as AHI, oxygen desaturation index and time spent with oxygen desaturation less than 90% were associated with the aortic root diameter in these hypertensive patients with various severities of OSA.

Our study was the first that investigated the relationship between aortic root diameter and various hypoxia-related parameters in hypertensive patients with OSA. Several previous studies showed that OSA promoted progressive aortic dilation in patients with Marfan’s syndrome or aortic dissection. Few studies investigated the association between OSA and aortic root diameter in patients without aortic diseases. In an early cross-sectional study in 76 patients with OSA, Meuleman et al. found that aortic root diameter ranged from 26.9 to 44.6 mm with a mean ($±$SD) of 35.3 $±$ 3.8 mm, and only 3 (3.9%) patients had aortic root dilation. The investigators of the study concluded that the prevalence of aortic root enlargement was not increased in OSA and reiterated in a published correspondence that they did not find any correlation of aortic root diameter with AHI or time spent with oxygen desaturation less than 90%.

In the latter study in 150 consecutive patients who were referred for confirmation of OSA, Cicek et al. found that patients with an AHI of 30 or higher had a...
significantly greater aortic root diameter than those with an AHI below 30 \( (n = 66) \). In a clinical experiment in 20 healthy subjects, Stöwhas et al. found that simulated hypopnea was associated with an increase in proximal aortic diameter. In patients with both hypertension and OSA, our present study extends the findings of these previous studies by showing a significant association between aortic root diameter and various severity parameters of OSA.

OSA is a known independent risk factor of cardiovascular diseases. Our current study showed that OSA might also be a risk factor for aortic root dilatation. Aortic root dilatation is a risk factor for aortic dissection or rupture and a predictor of aortic valve regurgitation or even progression to aortic dissection. Lam et al. showed that aortic root dilatation was associated with an increased risk of major adverse cardiovascular events and mortality. As a noninvasive examination, echocardiography is useful in the early diagnosis of aortic root dilatation. The long axis of the left ventricle beside the sternum can show the structure of the aortic root. It can show the changes of the aortic structure and hemodynamics in the subclinical stage. In 2096 American patients with hypertension but without overt cardiovascular disease, Palmieri et al. found a 4.6% of prevalence of echocardiographic aortic root dilation, slightly higher than 3.7% of prevalence in our current study.

The underlying mechanism for the association between severe OSA and increased aortic root diameter remains under investigation. It may involve intermittent hypoxia and reoxygenation, increased sympathetic nerve activity, and increased wall stress against intrathoracic organs induced by exaggerated negative intrathoracic pressure. Time spent with oxygen desaturation less than 90% is a measure of the duration of hypoxia, and has been shown to be a better predictor of cardiovascular disease mortality than AHI. Our study did show a slightly stronger association between this
measure and aortic root diameter, indicating a possible role of long lasting hypoxia and its consequences.

Why the aortic root diameter but not the measurements of cardiac structure and function differed between patients with severe, moderate, and mild OSA is not entirely understood. Our speculative explanation is that hypertension as a major cardiac risk factor must have played an important part. Our study patients had relatively severe hypertension. They had on average close to 10 years of hypertension history. Many of them were treated with combination antihypertensive therapy and still had inadequately controlled clinic and ambulatory blood pressure. Severe hypertension might to a large extent have accounted for the high proportion of left ventricular hypertrophy in our study participants. In fact, with regard to the association between OSA severity and cardiac structure and function, previous studies produced inconsistent results. Some, but not other studies showed significant differences across various severity groups of OSA. The differences between these studies might have been confounded by patients characteristics, such as the prevalence, severity, and management of hypertension.

Our study has to be interpreted within the context of its limitations. First, aortic root is a complex structure including aortic anulus, aortic leaflets junction, aortic sinuses, and sinutubular junction. Measurement of aortic root diameter at sinuses of Valsalva is most commonly used, but may not be sufficiently accurate in assessing the aortic root diameter. A more comprehensive evaluation including additional measurements at various levels of annulus, supraaortic ridge, or proximal ascending aorta could help. Second, our study was retrospective, and lack of measurement of early cardiac dysfunction. Third, our study is cross-sectional and hence does not allow to draw any causal inference. Finally, our study is cross-sectional and hence was retrospective, and lack of measurement of early cardiac dysfunction. Third, our study is cross-sectional and hence was retrospective, and lack of measurement of early cardiac dysfunction. Third, our study is cross-sectional and hence was retrospective, and lack of measurement of early cardiac dysfunction.

In conclusion, the severity of OSA was associated with the aortic root diameter. Patients with severe OSA had a greater aortic root diameter. Future studies should address whether OSA is a causal risk factor for aortic root dilatation, and whether treatment of OSA such as the continuous positive airway pressure would prevent aortic dilatation by correcting hypoxemia and negative intrathoracic pressures. If our finding is confirmed by these observational and interventional studies, aortic root diameter in obstructive sleep apnea as a risk marker in coronary artery disease. Chest 1996; 109:659–663.

REFERENCES
1. Mooe T, Rabben T, Wiklund U, Franklin KA, Eriksson P. Sleep-disordered breathing in men with coronary artery disease. Chest 1996; 109:659–663.
2. Schärer H, Koehler U, Ewig S, Hasper E, Tasci S, Lüderitz B. Obstructive sleep apnea as a risk marker in coronary artery disease. Cardiology 1999; 92:79–84.
3. Monahan K, Redline S. Role of obstructive sleep apnea in cardiovascular disease. Curr Opin Cardiol 2011; 26:541–547.
4. Cormican LJ, Williams A. Sleep disordered breathing and its treatment in congestive heart failure. Heart 2005; 91:1265–1270.
5. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. N Engl J Med 2005; 353:2034–2041.
6. Kohler M, Pitcher A, Blair E, Risby P, Senn O, Forfar C, Wordsworth P, Stradling JR. The impact of obstructive sleep apnea on aortic disease in Marfan’s syndrome. Respira- tion 2013; 86:39–44.
7. Mason RH, Ruegg G, Perkins J, Hardinge M, Amann-Vesti B, Senn O, Stradling JR, Kohler M. Obstructive sleep apnea in patients with abdominal aortic aneurysms: highly prevalent and associated with atherosclerotic expansion. Am J Respir Crit Care Med 2011; 183:668–674.
8. Zhou X, Liu F, Zhang W, Wang G, Guo D, Fu W, Wang L. Obstructive sleep apnea and risk of aortic dissection: a meta-analysis of observational studies. Vascular 2018; 26:515–523.
9. Masugata H, Senda S, Murao K, Okuyama H, Inukai M, Hosomi N, Iwado Y, Noma T, Kohno M, Himoto T, Goda F. Aortic root dilatation as a marker of subclinical left ventricular diastolic dysfunction in patients with cardiovascular risk factors. J Int Med Res 2011; 39:64–70.
10. Iarussi D, Caruso A, Galderisi M, Covino FE, Dialetto G, Bossone E, de Divitiis O, Cotrufo M. Association of left ventricular hypertrophy and aortic dilatation in patients with acute thoracic aortic dissection. Angiology 2001; 52:447–455.
11. Lin CH, Lurie RC, Lyons OD. Sleep apnea and chronic kidney disease: a state-of-the-art review. Chest 2005; 127:673–685.
12. Vasan RS, Larson MG, Levy D. Determinants of echocardiographic aortic root size. The Framingham Heart Study. Circulation 1995; 91:734–740.
13. Roman MJ, Devereux RB, Kramer-Fox R, O’Loughlin J. Two-dimensional echocardiographic aortic root dimensions in normal children and adults. Am J Cardiol 1989; 64:507–512.
14. Fukaya AOJ, Otohinoyi DA, Omoné AE, Oladele C, Kalejaiye A, Ouwegbui A, Nwali E, Talikdar D, Eriñikota O. Correlating possible predisposing demographics and systemic conditions with the aortic root. Ann Afr Med 2018; 17:133–139.
15. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, Horton K, Oguyenkin KO, Palma RA, Velazquez EJ. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr 2019; 32:1–64.
16. Mül G, Nardi E, Morreale M, Castiglia A, Geraci G, Altieri D, Cacciatori V, Schillaci M, Vaccaro F, Cottone S. The relationship between aortic root size and hypertension: an unsolved conundrum. Adv Exp Med Biol 2017; 956:427–445.

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DISCLOSURE
The authors declared no conflict of interest.
17. Echocardiography Working Group of the Chinese Society of Medical Ultrasonography. Guidelines for echocardiographic examinations in adult Chinese. Chin J Med Ultrasonogr 2016; 25:645–666.
18. Cistulli PA, Wilcox I, Jeremy R, Sullivan CE. Aortic root dilatation in Marfan’s syndrome: a contribution from obstructive sleep apnea? Chest 1997; 111:1763–1766.
19. Liu W, Zhang W, Wang T, Wu J, Zhong X, Gao K, Liu Y, He X, Zhou Y, Wang H, Zeng H. Obstructive sleep apnea syndrome promotes the progression of aortic dissection via a ROS-HIF-1α-MMPs associated pathway. Int J Biol Sci 2019; 15:2774–2782.
20. Meuleman C, Boccarda F, Nguyen XL, Di Angelantonio E, Ederhy S, Janower S, Dufaitre G, Haddour N, Boyer-Chatenet L, Rakotonahary D, Fleury B, Cohen A. Is the aortic root dilated in obstructive sleep apnea syndrome? Arch Cardiovasc Dis 2008; 101:391–397.
21. Cicek D, Lakadamyali H, Yaşbasan BD, Sapmaz I, Müderrisoglu H. Obstructive sleep apnea and its association with left ventricular function and aortic root parameters in newly diagnosed, untreated patients: a prospective study. J Int Med Res 2011; 39:2228–2238.
22. Serizawa N, Yumino D, Takagi A, Gomiya K, Kajimoto K, Tsurumi Y, Hagiwara N. Obstructive sleep apnea is associated with greater thoracic aortic size. J Am Coll Cardiol 2008; 52:885–886.
23. Meuleman C, Boccarda F, Ederhy S, Dufaitre G, Fleury B, Cohen A. Is obstructive sleep apnea associated with greater thoracic aortic size? J Am Coll Cardiol 2009; 53:815–816.
24. Cicek D, Balcioglu AS, Lakadamyali H, Müderrisoglu H. Effects of three month nasal continuous positive airway pressure treatment on electrocardiographic, echocardiographic and overnight polysomnographic parameters in newly diagnosed moderate/severe obstructive sleep apnea patients. Int Heart J 2015; 56:94–99.
25. Stőwhas AC, Namdar M, Biaggi P, Russi EW, Bloch KE, Stradling JR, Kohler M. The effect of simulated obstructive sleep apnea and hypopnea on aortic diameter and BP. Chest 2011; 140:675–680.
26. Lam CS, Gona P, Larson MG, Aragam J, Lee DS, Mitchell GE, Levy D, Cheng S, Benjamin EJ, Vasan RS. Aortic root remodeling and risk of heart failure in the Framingham Heart study. JACC Heart Fail 2013; 1:79–83.
27. Brown OR, DeMots H, Kloster FE, Roberts A, Menashe BD, Beals RK. Aortic root dilatation and mitral valve prolapse in Marfan’s syndrome: an ECHOCARDIOgraphic study. Circulation 1975; 52:651–657.
28. Palmieri V, Bella JN, Arnett DK, Roman MJ, Oberman A, Kitzman DW, Hopkins PN, Paranasis M, Rao DC, Devereux RB. Aortic root dilatation at sinuses of valsalva and aortic regurgitation in hypertensive and normotensive subjects: the Hypertension Genetic Epidemiology Network Study. Hypertension 2001; 37:1229–1235.
29. Narkiewicz K, Somers VK. Sympathetic nerve activity in obstructive sleep apnoea. Acta Physiol Scand 2003; 177:383–390.
30. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. Lancet 2009; 373:82–93.
31. Sadr N, Bin YS, Sutherland K, Cook K, Dissnayake H, Cistulli P, Chazal P. Is cumulative time of oxygen desaturation a better predictor of cardiovascular mortality than apnoea hypopnoea index? Ann Int Conf IEEE Eng Med Biol Soc 2020; 2020:2788–2791.
32. Holtsrand Hjalm H, Fu M, Hansson PO, Zhong Y, Caidahl K, Mandalenakis Z, Morales D, Ergatoudes C, Rosengren A, Grote L, Thunström E. Association between left atrial enlargement and obstructive sleep apnea in a general population of 71-year-old men. J Sleep Res 2018; 27:252–258.
33. Cicek D, Lakadamyali H, Yaşbasan BD, Sapmaz I, Müderrisoglu H. Obstructive sleep apnea and its association with left ventricular function and aortic root parameters in newly diagnosed, untreated patients: a prospective study. J Int Med Res 2011; 39:2228–2238.
34. Dursunoglu D, Dursunoglu N, Evrençül H, Özkürt S, Kuru O, Kılıç M, Fisekci F. Impact of obstructive sleep apnoea on left ventricular mass and global function. Eur Respir J 2005; 26:283–288.