



ARAŞTIRMA / RESEARCH

Evaluation of right heart functions by echocardiography and tissue Doppler imaging echocardiography in obese and non-obese patients with obstructive sleep apnea

Obez ve nonobez obstrüktif uyku apne sendromlu hastalarda sağ kalp fonksiyonlarının ekokardiyografi ve Doppler ekokardiyografi ile değerlendirilmesi

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Abstract

Purpose: The aim of our study was to compare the right ventricular systolic and diastolic functions and pulmonary artery pressure (PAP) in obese and non-obese patients with obstructive sleep apnea syndrome (OSAS) by tissue Doppler imaging (TDI) echocardiography.

Materials and Methods: This study was conducted with 69 patients, 34 obese and 35 non-obese, diagnosed moderate or severe OSAS by an overnight polysomnographic sleep study. In all patients, LV (left ventricle) and RV (right ventricle) size, left atrial (LA) and RA (right atrial) dimensions, LV and RV systolic and diastolic functions and systolic PAPs were measured by M-mode, two-dimensional analysis, color flow Doppler and TDI.

Results: RV diastolic dysfunction was detected in both groups; this impairment was significantly higher in the obese group (lateral tricuspid late diastolic myocardial annular zone velocity A'a: 0.13 ± 0.03 in non-obese patients and 0.11 ± 0.04 in obese patients). The mean systolic PAP was significantly higher in obese patients (31.2 ± 5.6 , 27.1 ± 5.8 , respectively)

Conclusion: Obstructive Sleep Apnea Syndrome increases cardiovascular morbidity and mortality. In our study, left ventricular and right ventricular diastolic dysfunction was determined by tissue Doppler imaging in patients with moderate and severe Obstructive Sleep Apnea Syndrome. Obesity contributes to this impairment regardless of Obstructive Sleep Apnea Syndrome.

Keywords: Obesity, obstructive sleep apnea syndrome, tissue doppler echocardiography

Öz

Amaç: Çalışmamızın amacı, obez ve nonobez obstrüktif uyku apne sendromu (OUAS) hastalarında; doku doppler (DD) ekokardiyografi ile sağ ventrikül sistolik ve diastolik fonksiyonlarının ve pulmoner arter basınçlarını (PAB) karşılaştırmaktır.

Gereç ve Yöntem: Çalışmamıza Uyku Laboratuvarında polisomnografik inceleme yapılmış ve orta ya da ağır OUAS tanısı konmuş 35 nonobez ve 34 obez olmak üzere 69 hasta alındı. Tüm hastalarda M-mode, iki boyutlu inceleme, renkli akım Doppler ve Doku Doppler yardımıyla sol ventrikül ve sağ ventrikül boyutları, sol atrium (LA) ve sağ atrium (RA) boyutları, sol ventrikül ve sağ ventrikül sistolik ve diastolik fonksiyonları ve sistolik PAB' ları ölçüldü.

Bulgular: Her iki grupta da RV diastolik fonksiyonlarında bozulma saptanmış olup, obez grupta anlamlı olarak daha fazla bozulma saptanmıştır (lateral triküspit annuler bölge geç diastolik miyokardial hız A'a: nonobez hastalarda 0.13 ± 0.03 ve obez hastalarda 0.11 ± 0.04). Obez grupta ortalama sistolik PAB anlamlı olarak daha yüksek bulunmuştur (Sırasıyla 31.2 ± 5.6 ve 27.1 ± 5.8).

Sonuç: Obstrüktif Uyku Apne Sendromu kardiyovasküler morbidite ve mortaliteyi artırır. Çalışmamızda orta ve ağır OUAS hastalarında doku doppler incelemesi ile SV ve RV diastolik fonksiyonlarında bozulma saptanmıştır. Obezite OUAS'tan bağımsız olarak bu bozukluğa katkıda bulunmaktadır.

Anahtar kelimeler: Obezite, obstrüktif uyku apne sendromu, doku doppler ekokardiyografi

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INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a disease characterized by repetitive partial or complete airway obstruction that develops during sleep. Cardiovascular complications are commonly seen in the patients with OSAS, causing increases in morbidity and mortality^{1,2}. OSAS is closely associated with obesity; However it may also exist in the non-obese patients. The relation of obesity with OSAS has not completely been clarified with the explanations of causes and effects; however it is well-known that obesity shows parallelism with the severity of OSAS³.

Repetitive hypopnea and apnea episodes in OSAS result in hypoxemia, increased sympathetic activity, increases in pulmonary and systemic arterial pressures and changes in heart rate that are reasons for high cardiac risk. Episodes of repetitive transient nocturnal hypoxemia may cause increase in pulmonary artery pressure, right ventricular hypertrophy, cardiac arrhythmias, polycythemia, and even sudden deaths during sleep^{1,4}. Another important fact is that, the main risk factors (obesity, hypertension, etc.) for cardiovascular disease frequently exist simultaneously in the OSAS patients.

Significant relations have been determined between OSAS, and systemic and pulmonary hypertension, cardiac failure, coronary heart disease, arrhythmias, sudden death and cerebrovascular diseases^{1,5}. There are various studies indicating left ventricular function disorders in OSAS. However, the relation between OSAS and right ventricular function disorder is a matter of debate, since ratios changing from 0% to 70% have been reported in various studies⁶.

Various echocardiographic methods are used in the determination of cardiac and hemodynamic changes that develop in OSAS. Cardiac complications developing secondary to the disease in early phases are difficult to determine by the conventional methods. In the early period; transthoracic echocardiography and tissue Doppler echocardiography, a technique with high sensitivity, are applied for the evaluation of the effects of disease on cardiac parameters; these methods can serve as useful guides for the diagnosis, follow-up and treatment of the disease. Thus early diagnosis and treatment of the patients that carry cardiac risks, can affect cardiovascular morbidity and mortality in a positive manner in the long term⁶.

The aim of the present study is to investigate and compare the right ventricular systolic and diastolic functions, right atrial dimensions and pulmonary artery pressures by means of conventional echocardiography and tissue Doppler echocardiography, in obese and non-obese patients with moderate and severe sleep apnea syndrome.

MATERIALS AND METHODS

This study included patients who were hospitalized for polysomnographic evaluation at the Mustafa Kemal University Sleep Clinic. The polysomnographic data of all patients were evaluated simultaneously by two physicians with a sleep certificate at the same time. Patients with moderate and severe OSAS in the test results were invited to the study, 126 patients were invited to the study. 57 patients with exclusion criteria were excluded from the study. Patients (n = 69) investigated by polysomnographic study, and diagnosed moderate (AHI = 15-30/h) or severe (AHI >30/h) OSAS in the Sleep Laboratory, were included in the study.

Our study included patients over 18 years of age who had moderate to severe Osas in their polysomnography, who did not have additional respiratory, metabolic and cardiac disease, who approved the use of the data. These patients were not included in the study because the distinction between the underlying societal and cardiac diseases and whether the pathology was related to OSAS or to the underlying disease would not be clear. For this reason, Patients with severe chronic obstructive lung disease (COLD), hypertension (HT), cardiac failure, renal failure, cardiac valve disease, lung disease causing respiratory failure, atrial fibrillation (AF), cardiomyopathy (CMP), cerebrovascular disease, and hypothyroidism were excluded from the study.

Procedure

Confirmation of the Local Ethics Committee was obtained. Our study was approved by the Ethics Committee of Mustafa Kemal University Faculty of Medicine with the date and number of 2011/04. The purpose of our study was explained to the participants in detail, and informed by Helsinki rules, and an informed consent form was obtained.

The patients were informed about what procedures would be applied, and they completed informed consent forms. Each patient was interviewed after the

polysomnography test, demographic data of the patients, height and weight measurements were looked at. Similar numbers of patients were included in both groups. Patients were questioned with regards to age, gender, associated additional diseases, existing symptoms and risk factors, smoking habits and alcohol use. Age, gender, height and weight values of the patients were noted, and body mass indices were calculated ($BMI = \text{Weight} / \text{Height}^2$) (kg/m^2). Patients were separated to obese ($BMI \geq 30$) and non-obese ($BMI < 30$) groups, considering the BMI.

Mallampati scores, total cholesterol, triglyceride, LDL (low-density lipoprotein) and HDL (high-density lipoprotein) levels, thyroid function tests and respiratory function tests were noted. Mallampati scores were evaluated by the same physician. Patients were divided into 4 groups according to their oropharyngeal appearance by performing oral examination, and looking at uvula and tonsil size. Class I: Uvula, soft palate, tonsil bed, front and back pillars are easily seen; Class II: Uvula and soft palate are seen; Class III: Soft palate and uvula base are visible; Class IV: Uvula was completely closed by the root of the tongue and the pharyngeal wall was not seen and the Mallampati scores of the patients were recorded. Radiographic examination of the lung was performed to exclude a possible lung disease. Electrocardiograms (ECG) were evaluated for an additional possible cardiac disease.

Epworth Sleepiness Scale (ESS) was completed for each patient. The Epworth sleepiness scale is a test used to demonstrate daytime sleepiness. It consists of 8 questions in total. Each question is filled by the patient himself/herself to give 0-3 points. In this survey, the possibility of falling asleep in certain situations is questioned on an ordinary day when the patient is not overly tired. Total scores of the patients are recorded. If the total score is 10 or more, it indicates the presence of excessive daytime sleepiness. Validity and reliability of the daytime sleepiness has been demonstrated in domestic and international studies.

Polysomnography

PSG was performed using Compumedics E Series 44 channel polysomnograph, Profusion PSG3 Software (Abbotsford, VIC, Australia). EEG, EOG, submental EMG, ECG, finger pulse oximetry, thoracic and abdominal movements, body position, and bilateral anterior tibial EMG were recorded. An

oronasal thermistor and nasal cannulas were used to detect apnea and hypopnea.

All signals were digitized and stored on a personal computer. Scoring was performed according to the recommendations of Rechtschaffen and Kales and the AASM rules^{7,8}.

By investigating polysomnographic analyses of the patients; AHI assessments, OSAS levels, sleep efficiency, percent values of sleep stages, the lowest saturation, the mean saturation, the mean desaturation, obstructive, mixed or central types of apnea (via AHI), arousal index, and the lowest, the highest and the mean pulses were recorded.

Echocardiographic evaluation

Echocardiographic evaluations were evaluated by a single cardiologist after patients rest for at least 30 minutes. For echocardiographic examination 2.5 MHz, Vivid 7 Digital ultrasound device was used (Horten, Norway, GE); 2-dimensional, M-mode, pulsed-wave Doppler and pulsed wave tissue Doppler echocardiographic parameters were obtained. Echocardiographic evaluation was made via parasternal and apical images, by telling patients to lie down at the left lateral position. Cases with technical insufficiency were not included in the study. Measurements and records were made at the end of normal inspiration and expiration. Parameters like left atrial dimensions, regional wall motion abnormalities and left ventricular hypertrophy (LVH) were evaluated via 2-dimensional B-mode images. With the ECG records, the LV dimensions, interventricular septum and LV posterior wall measurements were obtained by cutting perpendicularly the long axis at the tip of the mitral leaflets, in the M-mode. Right ventricle dimensions were measured in parasternal long axis view in M-mode, cutting the right ventricle perpendicularly at the tip of the tricuspid valve. Right ventricular free wall thickness $\leq 5\text{mm}$ was considered as right ventricular hypertrophy (RVH). The right atrium size was analyzed from apical 4-chamber view.

Left ventricular and right ventricular wall motions were analyzed at the left semi lateral position, from the standard left parasternal and apical views. Transmitral and tricuspid valve flow velocities were recorded using pulse-wave Doppler technique, from the apical window by using 1 to 3mm sampling volume between the open leaflet tips that lie passively throughout the diastole. By using pulse-wave

Doppler and via the mitral and tricuspid valves, peak flow velocity in early diastole, the E wave, and the late end-diastolic A wave produced by atrial contraction, were measured.

Table 1: Summary of reference limits for recommended measures of right heart structure and function⁹

Variable	Unit	Abnormal
Chamber dimensions		
RV basal diameter	cm	>4.2
RV subcostal wall thickness	cm	>0.5
RVOT PSAX distal diameter	cm	>2.7
RVOT PLAX proximal diameter	cm	>3.3
RA major dimension	cm	>5.3
RA minor dimension	cm	>4.4
RA end-systolic area	cm ²	>18
Systolic function		
TAPSE	cm	<1.6
Pulsed Doppler peak velocity at the annulus	cm/s	<10
Pulsed Doppler MPI	-	>0.55
Tissue Doppler MPI FAC	%	<35
Diastolic function		
E/A ratio	-	<0.8 or >2.1
E/E' ratio	-	>6
Deceleration time	ms	<120

FAC, Fractional area change; MPI, myocardial performance index; PLAX, parasternal long-axis; PSAX, parasternal short-axis; RA, right atrium; RV, right ventricle; RVD, right ventricular diameter; RVOT, right ventricular outflow tract; TAPSE, tricuspid annular plane systolic excursion.

Pulsed-wave tissue Doppler images were obtained at transducer frequency of 3.5-4.0 MHz, color Doppler screening ratio of 100-140 Hz, and Nyquist limit of 15-20 cm/s. These measurements were obtained from apical 4-chamber view, and by using 5 mm sampling volumes in basal septal and basal lateral tricuspid annular regions. Systolic (Sm: basal septal region, Sa: lateral tricuspid annulus longitudinal systolic velocity), early diastolic (Em: basal septal region, Ea: lateral tricuspid annular region), and late diastolic (Am: basal septal region, Aa: lateral tricuspid annular region) myocardial velocities, and velocities from tricuspid valve annular region, and isovolumetric contraction time (IVCT), contraction time (CT) and isovolumetric relaxation time (IVRT) were measured. Additionally, tricuspid annular plane systolic excursion (TAPSE), which indicates the RV systolic function, was also evaluated. This parameter was analyzed from the right ventricle free wall, by

placing M-mode on the lateral tricuspid annulus (Table 1).

Statistical analysis

Data analysis was performed using 'Statistical Package for Social Sciences (SPSS) 13 for Windows' (SPSS Inc., Chicago, Illinois) statistical program. Groups and descriptive data were compared by Chi-square test. Pearson correlation analysis was used in comparison of two continuous variables. When parametric test assumptions were provided, the means of two groups were compared by using the independent-samples-t-test. A p value <0.05 was accepted to be significant. Average - standard deviation from central spreading measures was used for continuous variables. Other distributions were expressed in rates and analyzed by chi-square tests. It was decided to perform parametric tests using the Shapiro Wilk test, one of the normality tests for continuous variables. One Way ANOVA and Student-t tests were used from the parametric tests. Of the nonparametric tests, Mann Whitney U and Kruskal Wallis tests were used. Descriptive statistics were used for the demographic features specified in the study.

RESULTS

A total of 69 patients, 15 of whom were females, were included in the study. Ages of the cases varied from 28 to 67. Patients were separated to obese and non-obese groups, by considering the body mass indices (BMI) (kg/m²). These groups involved 35 non-obese (6 females, 29 males) and 34 obese (9 females, 25 males) cases. The mean ages of the non-obese and obese patients were found to be 48.8±10.7 years and 46.6±8.9 years, respectively. Tobacco and alcohol use did not differ between the groups (p<0.05) (Table 2).

OSAS symptoms like snoring, witnessed apnea and excessive daytime sleepiness questioned before the polysomnographic evaluation, were similar in both groups (Table 2). When the patients were separated to moderate and severe OSAS, demographic data were found to be similar, whereas systolic blood pressure levels were significantly higher in the severe OSAS group (p<0.001).

AHI was determined to be significantly higher in the obese patients; a total of 53 (76%) patients were in the severe OSAS group, and 30 cases (88.2%) from the obese group and 23 cases (35%) from the non-

obese group contributed to this total number of severe OSAS patients. In the obese group; the lowest saturation during sleep and the mean saturation

during sleep were significantly lower; the lowest and the mean saturations during sleep were found to decrease more as the AHI increased.

Table 2. Properties of the obese and non-obese patients

Variable	Non-obese (n=35)	Obese (n=34)	P value
Gender (n,%) Female	6 (%17.1)	9 (%26.5)	0.394
Male	29 (% 82.9)	25 (%73.5)	
Age (mean±SD)	48.8 (±10.7)	46.6 (±8.9)	0.357
Smoking (n,%)			0.440
Still smoking	22 (% 62.9)	25 (% 73.5)	
Never smoked/gave up smoking	13 (% 37.1)	9 (% 26.5)	
Alcohol (n, %)			0.513
User	28 (% 80)	30 (% 88.2)	
Non-user	7 (%20)	4 (% 118)	
Witnessed apnea (n,%)	25 (% 71.4)	30 (% 88.2)	0.133
Excessive daytime sleepiness (n,%)	30 (% 85.7)	32 (% 94.1)	0.428
Snoring (n,%)	34 (%97.1)	34 (% 100)	1

However, the mean desaturation did not differ significantly between the groups ($p=0.234$). Minimum oxygen saturation decreased as AHI increased; these two parameters were inversely and significantly correlated with each other ($r=-0.450$, $p=0.013$). When patients were separated to moderate and severe OSAS cases; the lowest saturation (86 ± 7 and 74 ± 12 respectively, $p=0.020$) and the mean saturation (94 ± 1 and 89 ± 6 , respectively; $p=0.003$) were indicated to be significantly lower in the severe OSAS group, but the mean desaturation values were significantly higher (3 ± 1 and 9 ± 9 respectively, $p<0.001$). When arousal indexes were evaluated in two patient groups; arousal index correlated negatively and significantly with the lowest saturation and the mean saturation ($p<0.001$); as AHI increased, arousal index also increased significantly, and positively.

When the lowest, the highest, and the mean heart rates determined during sleep were compared between the two groups, the highest and the mean heart rate values were found to be higher in the obese cases, but this difference between the groups was not statistically significant. When transthoracic echocardiography was evaluated; right atrial (RA) size, right ventricular end-systolic diameter (RVESD), right ventricular end-diastolic diameter (RVEDD), ejection fraction (EF), and left atrial (LA) and left ventricular end-diastolic diameters were found to be similar in both groups (Table 3).

PAP in the obese patient group was significantly higher when compared to the non-obese cases

(31.2 ± 5.6 mmHg and 27.1 ± 5.8 mmHg, respectively; $p=0.04$) (Table 3).

In both patient groups, right and left heart functions were primarily evaluated by conventional Doppler; E/A ratio was determined, which was superior to other parameters especially in indicating diastolic dysfunction. In a total of 22 patients, E/A was determined to be <0.8 for the right heart (normal range 0.8-2.1); 10 of these patients (28.6%) were from the non-obese, and 12 (35.3%) were from the obese group. In 32 patients, E/A was found to be <1 for the left heart (normal value >1); 18 of these patients (51.4%) were from non-obese group, and 14 were (41.2%) from the obese patient group (Table 4). However the mean E/A values did not differ significantly between the two groups (E/A for the right heart: in obese group 1.11 ± 0.28 , in non-obese group 1.25 ± 0.55 , $p=0.225$; E/A for the left heart: in obese group 1.08 ± 0.33 , in non-obese group 1 ± 0.29 , $p=0.257$).

When the right ventricular tissue Doppler parameters were examined; isovolumetric relaxation time (IVRT), one of the parameters that shows diastolic dysfunction (normal value 43-53, mean 48) was determined to be below the mean level in 6 (17.6%) of the obese patients, and in three cases (8.6%) of the non-obese group; however no significant difference was found between the groups ($p=0.306$). A'a (lateral tricuspid annular region late diastolic myocardial velocity normal value:12-14 cm/s) was found to be significantly lower in the obese cases (Table 5).

Table 3. Evaluation of transthoracic echocardiography results in the obese and non-obese OSAS patients.

	Non-obese (mean±SD)	Obese (mean±SD)	P value
Aortic root	3.3 (±0,33)	3.2 (±0,51)	0.174
Ascending aorta	3.2(±0,35)	3.2 (±0,34)	0.822
LA	3.4 (±0,25)	3.4 (±0,23)	0.686
LVESD	3.15 (±0,6)	3.27 (±0,64)	<0.001
LVEDD	4.53 (0,51)	4.59 (±0,53)	0.637
İVST	0.99 (±0,12)	1.01 (±0,12)	0.441
LVPWT	0.83 (±0,05)	0.87 (±0,05)	0.006
LV-EF	61.9 (±2,8)	60.7(±2,7)	0.062
Pulmonary velocity	0.89 (±0,08)	0.92 (±0,11)	0.083
Aortic velocity	1.43 (±0,2)	1.48 (±0,22)	0.412
PAP	27.1(±5,8)	31.2 (±5,6)	0.04
TAPSE	2.6 (±0,26)	2.56(±0,26)	0.498
RA	2.9 (±0,53)	2.8 (±0,40)	0.420
RVEDD	2.5 (±0,48)	2.5 (±0,28)	0.734
RVESD	1.79 (±0,29)	1.79 (±0,28)	0.074

LA; Left atrium, LVESD; Left ventricular end-systolic diameter, LVEDD; Left ventricular end-diastolic diameter, İVST; interventricular septum thickness, LVPWT; Left ventricular posterior wall thickness, LV-EF; Left ventricle ejection fraction PAP; pulmonary artery pressure TAPSE; tricuspid annular plane systolic excursion, RA; Right atrium size, RVEDD; Right ventricular end-diastolic diameter, RVESD; Right ventricular end-systolic diameter.

Table 4. Comparison of conventional echocardiography results in the obese and non-obese OSAS patients.

	Non-obese (mean±SD)	Obese (mean±SD)	P value
Right V.PWE	0.52 (±0.13)	0.51 (±0.11)	0.774
Right V.PWA	0.45 (±0.14)	0.47 (±0.11)	0.623
Right E/A	1.25 (±0.55)	1.11 (±0.28)	0.225
Left V.PWE	0.70 (±0.15)	0.69 (±0.18)	0.024
Left V.PWA	0.68(±0.15)	0.66 (±0.14)	0.569
Left E/A	1 (±0.29)	1.08(±0.33)	0.257

Table 5. Comparison of tissue Doppler echocardiographic results of right heart functions in obese and non-obese OSAS patients.

	Non-obese (mean±SD)	Obese (mean±SD)	P value
Sa	0.13 (±0.02)	0.12 (±0.02)	0.282
E'a	0.11(±0.03)	0.10(±0.03)	0.148
A'a	0.13 (±0.03)	0.11 (±0.04)	0.020
İVRT	69.7(±17.1)	64.5 (±14.4)	0.175
İVCT	71.4(±18.1)	71.8 (±15.2)	0.933
CT	265.5(±29.5)	260.9(±34.4)	0.551
E/E'	4.9 (±2.19)	5.5 (±2.2)	0.092

Sa: lateral tricuspid annulus longitudinal systolic velocity E'a: lateral tricuspid annular region early diastolic myocardial velocity A'a: lateral tricuspid annular region late diastolic myocardial velocity, İVCT: isovolumetric contraction time, CT: Contraction time, İVRT: isovolumetric relaxation time

When the left ventricular tissue Doppler parameters were examined, late diastolic myocardial velocity (A), one of the parameters indicating left ventricular diastolic function impairment, was found to be

significantly lower in the obese patient group when compared to non-obese cases. Other parameters were found to be similar in both groups (Table 6).

Table 6. Comparison of tissue Doppler echocardiographic results of left heart functions in obese and non-obese OSAS patients

	Non-obese (mean±SD)	Obese (mean±SD)	P value
Sm	0.08 (±0.11)	0.09(±0.14)	0.100
E'm	0.09(±0.27)	0.08(±0.17)	0.062
A'm	0.1 (±0.02)	0.08 (±0.02)	0.02
E/E'	7.7 (±2.1)	8.3 (±1.7)	0.206

Sm: basal septal region systolic peak flow velocity, E'm: basal septal region early diastolic and A'm: basal septal region late diastolic myocardial velocity

DISCUSSION

Obstructive sleep apnea (OSA) is a syndrome that affects 1% to 5% of the population, and it increases cardiovascular morbidity and mortality^{1,2}. OSAS frequency generally increases with age. In a study, prevalence of the patients with AHI value ≥ 15 increased by a direct proportion until 60 years of age, and the prevalence remained constant at about 20% above 60 years⁶. In our study, no significant statistical difference between age groups, moderate and severe OSAS.

It is known that there is a strong relation between BMI and OSAS¹⁰. It was indicated in a study that increases in weight by 10%, increases AHI by 32%, and causes a six-fold increase in the risk of moderate to severe OSAS development¹¹. Being similar with the literature, AHI increased significantly in our study, by the increase of BMI.

The primary cardiovascular diseases associated with OSAS are HT, ischemic heart disease, cardiac arrhythmias, left heart failure, PHT, right heart failure, polycythemia, stroke and sudden death. The frequency of this syndrome has been determined to be 50% in HT patients, 25% in the patients with chronic heart failure, 30% in those with acute coronary syndrome, and 60% in the cases that developed stroke¹².

Negative effects of OSAS on the LV functions have been demonstrated in various studies. Impairment of LV functions caused by OSAS is considered due to increase in the intrathoracic negative pressure, intermittent hypoxia and short awakening periods^{13,14}. In a study Hanlon and his colleagues conducted on 61 children who were obese and had hypertension, Children with OSAS were found to be more likely to have left ventricular hypertrophy (LVH) (85.7% vs. 59.4%, $p = 0.047$). (OSAS was associated with 4.11 times greater odds of LVH (95%

CI 1.15, 14.65; $P = 0.030$), remaining significant after adjustment for age, sex, race, and BMI z-score (after adjustment for hypertension, $P = 0.051$). The higher the intensity of OSAS, the greater the risk of LVH was determined¹⁵. Acute pulmonary hemodynamic changes during apneas have also been well-defined¹⁶; however, it is not well-known yet whether OSAS is an independent risk factor for the PHT¹⁷.

There are not a large number of studies investigating the relation between OSAS and RV morphology, and the results of studies are variable. In studies evaluating RV diameters and hypertrophy; it was reported that OSAS does not lead to a change in RV end-diastolic and end-systolic diameters, but causes an increase in RV free wall thickness. In a study by Berman et al, RV free wall thickness was observed to increase in 71% of the cases diagnosed as OSAS⁶. In a study by Hanly et al., RV and LV diameters did not differ significantly between the cases of snoring without apnea or the patients with OSAS¹⁸. Guidry et al. have compared two groups, one possessing a mean RDI value of 42, and the other 5. These groups did not differ significantly with regard to RA and RV diameters, and RV systolic function; however, RV anterior free wall thickness was found to increase significantly in the group with high RDI value¹⁹. In a meta-analysis published in recent years that evaluated the results of 25 studies; RV dilation, increased wall thickening and changes in RV function were observed in OSA patients²⁰. In our study, in accordance with the previous studies, RV diastolic and systolic diameters and right atrial size were found to be in normal ranges in both groups, and no significant differences were determined between the obese and non-obese groups ($p > 0.05$).

Tavil and the colleagues determined the impairment of right ventricular systolic and diastolic functions in the OSAS patients with/without hypertension²¹. In the study, they claimed that TAPSE (tricuspid annular plane systolic excursion) is an important

parameter in evaluating right ventricular functions, and tricuspid annular systolic velocity (Sa) obtained by tissue Doppler is important in the evaluation of right ventricular systolic functions. In the study of Karaçaglar and the colleagues in 34 OSAS and 28 healthy people, TAPSE values were found to be significantly lower in OSAS patients, PAP values were found to be significantly higher in the OSAS patient group²². In our study, the values of TAPSE and tricuspid annular systolic velocity were within normal ranges in all patients, and did not differ between the obese and non-obese OSAS groups. However, in our study, PAP values were significantly higher in obese patients group, similar to the literature.

Willens and the colleagues determined the effects of obesity on the right ventricular and left ventricular functions, by the tissue Doppler imaging; in the study mitral annular systolic velocity, ratio of early-to-late-diastolic tricuspid annular velocity, early-diastolic tricuspid annular velocity, early-diastolic mitral annular velocity, and ratio of early-to-late diastolic mitral annular velocity were found to decrease markedly²³. In our study, late-diastolic tricuspid annular velocity (A^{λ}), a tissue Doppler parameter of the right heart, was found to decrease in both groups, and additionally the decrease in the obese OSAS group was significantly more marked, when compared to the non-obese group.

In some studies, it was found out that the impairment of right ventricular function might be seen in overweight and obese individuals without any clinical sign of a marked heart disease. In a study on this subject performed with tissue Doppler imaging, right ventricular dysfunction independent of OSAS was reported to develop in the overweight and obese patients²⁴.

In the study of Li and his colleagues, right ventricular anterior wall thickness (RAW) values gradually increased with OSAS severity and in severe groups, the control was higher than the light groups. TAPSE values in the severe and middle groups were lower than the control group²⁵.

In several studies performed with conventional Doppler technique, left ventricular diastolic functions were determined to be affected in the patients with OSAS. Conventional Doppler technique was used in most of the limited number of studies investigating the effects of OSAS on the left ventricular diastolic functions²⁶. In a study by Kim et

al., it was aimed to investigate a tissue Doppler parameter that can be used to evaluate the left ventricle in the patients with OSAS, and early diastolic wave related with left ventricle was indicated to be markedly lowered in the severe OSAS group. It was additionally observed that this finding was not affected by diabetes mellitus, hypertension and body mass index; they consequently reported that left ventricular early diastolic wave is the most reliable parameter²⁷. In our study, left ventricular end-systolic diameter and left ventricle posterior wall thickness, which are the variables of 2-dimensional echocardiography, were determined to be significantly increased in the obese patients. As also determined in the literature, magnitude of left ventricular early diastolic wave (E_m), which is a parameter of Doppler and tissue Doppler, significantly decreased more in the obese patient group when compared with the non-obese group.

Another important parameter in evaluating diastolic dysfunctions is the ratio of early diastolic- to late diastolic-mitral annulus velocities (E/A); a value of $E/A < 1$ indicates diastolic dysfunction. In the study conducted by Papanikolaou and the colleagues, 30 severe OSAS ($AHI > 30$) and 25 control ($AHI < 5$) patients included in the study. In the study, the risk of left ventricular diastolic dysfunction increased in OSAS patients. As AHO increased, there was an increase in the severity of dysfunction. In addition, in the OSAS group, E wave velocity decreased, E/A rates decreased, E_m/A_m rates increased, and this increase was associated with right ventricular diastolic dysfunction²⁸. In our study, E/A was determined to be < 1 in 32 (46%) cases; of these, 18 (51.4%) were non-obese, and 14 (41.2%) were obese patients. However, the difference between these groups was not found to be significant. This finding indicates that OSAS causes impairment of the left ventricular diastolic functions, independent from obesity.

In a study evaluating the relationship between OSAS and hypertension, a linear relation was determined between AHI and blood pressure in the cases taking no antihypertensive treatment²⁹. It is considered that this relation may partly be explained by BMI. Even after corrections for BMI and demographic properties were performed, systolic and diastolic blood pressures were found to be increased with the increase of AHI, and this relation was determined to be significant statistically. We also determined positive and significant correlations between AHI and systolic and diastolic blood pressures.

In a study evaluating whether respiratory disorders during sleep are independent risk factors for hypertension, a high RDI caused statistically significant increases in systolic and diastolic blood pressures in the normotensive cases, and in patients newly diagnosed as hypertensive³⁰. In a study in which Wnag and colleagues investigated the detection rate and factors affecting obstructive sleep apnea syndrome in middle-aged and elderly patients with hypertension, They administered the Berlin survey to 440 hypertension patients and performed polysomnography (PSG) on 235 patients at high risk of OSAS. As a result, they found higher incidence of OSAS in middle-aged and elderly patients with hypertension. They detected high BMI, systolic and diastolic blood pressure as independent risk factors of OSAS³¹. In our study, systolic and diastolic blood pressure values were determined to be significantly higher in the patients with severe OSAS. In another study, systolic and diastolic blood pressures were found to be higher in the group of obese patients³². In our study also, systolic and diastolic blood pressure values were found to be significantly higher in the obese patients with OSAS, when compared with the non-obese OSAS group.

Alveolar hypoxia developing due to repetitive apneas during sleep may cause pulmonary vasoconstriction and increase of pulmonary artery pressure. Daylong persisting pulmonary hypertension and cor pulmonale have been reported in about 20% of the patients. The reason of this fact may be the persistent intimal hyperplasia and media hypertrophy³³.

It has been reported that sudden increases in pulmonary artery pressure are frequent in the patients with OSAS; however, proven pulmonary hypertension exists in about 20% of these cases³⁴. In the OSAS patients that pulmonary functions are also impaired, prevalence of pulmonary hypertension has been reported to increase by 41%³⁵. In our study, the mean systolic pulmonary artery pressure in the obese patient group was found to be significantly higher when compared with the non-obese cases. When the patients were evaluated in two separate groups as cases with moderate or severe OSAS, pulmonary artery pressure values did not differ significantly between the groups.

Our study has several limitations. First of all, in addition to the obese and non-obese OSAS group, we think that the absence of our control group without OSAS creates a significant limitation. Thus, we think that OSAS will be able to see more clearly

the effect on right heart functions. Secondly, we think that there may be an improvement in cardiac functions after OSAS treatment. For this, we think that more studies are needed to show the possible benefits of OSAS treatment.

The results of our study have led to the following conclusions: right ventricle systolic and diastolic functions are impaired in the patients with OSAS. Systolic functions of the left ventricle are conserved, whereas LV diastolic dysfunction is indicated by the tissue Doppler echocardiography. These impairments in cardiac functions are determined significantly in higher rates in obese OSAS cases when compared with the non-obese patients. Because of Cardiovascular complications are considerably frequent in the patients with obstructive sleep apnea, we consider that tissue Doppler echocardiography will be helpful in early determination of the impairment in diastolic functions of both ventricles, especially in the obese OSAS patients. OSAS patients with diastolic function disorders have to be more carefully investigated for the diastolic heart failure symptoms, have to be treated properly for the heart failure and sleep apnea, and also have to be followed-up more frequently.

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