ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

The Relationship Between Obstructive Sleep Apnea Severity and Right Heart Cavities Echocardiographic Features

Obstrüktif Uyku Apne Şiddeti ile Sağ Kalp Kavitelerinin Ekokardiyografik Özellikleri Arasındaki İlişki

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ABSTRACT

Aim: *Obstructive sleep apnea (OSA) has been attributed to an increased risk of cardiovascular disease and death from all causes. It has been demonstrated that OSA affects the right ventricle's (RV) diastolic and systolic functioning, with diastolic dysfunction usually appearing before systolic failure. Delaying the progression of right ventricular failure may be clinically beneficial if RV diastolic insufficiency is accurately assessed and treated early. This study aimed to investigate right ventricular diastolic function in patients with OSA and its relationship with disease severity.*

Material and Method: *The study comprised 88 participants with an OSA diagnosis who had transthoracic echocardiography. Based on their apnea and hypopnea index, the individuals were split into two groups as mild-moderate OSA (mild; apnea and hypopnea index 5–14 events/hour, moderate; apnea and hypopnea index 15–29 events/hour) and severe OSA (apnea and hypopnea index ≥30 events/hour).*

Results: *The severe group consisted of forty-three people, whereas the mild-to-moderate group had forty-five. The right atrial volume index (RAVI) (13.26±4.81 mL/m² vs. 24.24±10.75 mL/m2; p <0.001) and E/Em tricuspid ratio (5.70±2.32 vs. 7.21±3.83; p=0.046) of the severe group were substantially higher than those of the mild-moderate group.*

Conclusion: *The severity of OSA can be accurately predicted using the echocardiographic measures RAVI and tricuspid E/Em, which are practical, affordable, and easily available.*

Key words: *obstructive sleep apnea; right ventricular diastolic dysfunction; right atrial volume index*

ÖZET

Amaç: *Obstrüktif uyku apne sendromu (OUAS) kardiyovasküler riskte artış ve tüm ölüm nedenli ölümler ile ilişkilidir. Obstrüktif uyku apne sendromu sağ ventrikülün (SğV) hem sistolik hem de diyastolik fonksiyonlarını bozar. SğV'nin diyastolik fonksiyonları sistolik fonksiyonlardan önce bozulur. SğV diyastolik fonksiyon bozukluğunun doğru değerlendirilmesi ve erken müdahale sağ kalp yetmezliğinin ilerlemesini geciktirmede klinik olarak faydalı olabilir. Bu çalışmada OUAS hastalarında SğV diyastolik fonksiyon bozukluğunu tespit etmeyi amaçladık.*

Materyal ve Metot: *88 OUAS tanısı alan hastanın transtorasik ekokardiyografi kayıtları incelendi. Çalışmaya alınan tüm hastalar apne-hipopne endeksi'ne göre hafif-orta (hafif; apne-hipopne endeksi 5–14/saat, orta; apne-hipopne endeksi 15–29/saat) ile şiddetli (apne-hipopne endeksi ≥30/saat) olmak üzere iki gruba ayrıldı.*

Bulgular: *Hafif-orta grupta 45, ağır grupta 43 OUAS hastası vardı. Triküspit E/Em oranı (5,70±2,32 vs. 7,21±3,83 p=0,046) ve sağ atriyal volüm endeksi (13,26±4,81 mL/m2 vs. 24,24±10,75 mL/m2; p <0,001) değeri şiddetli grupta, hafif-orta gruba göre anlamlı derecede daha yüksek bulundu.*

Sonuç: *Ekokardiyografik parametrelerden sağ atriyal volüm endeksi ve triküspit E/Em, obstrüktif uyku apne şiddetini predikte edebilen, kolay ulaşılabilir ve uygulanabilir ölçümlerdir.*

Anahtar kelimeler: *obstrüktif uyku apne sendromu; sağ ventrikül diyastolik disfonksiyonu; sağ atriyal volüm indeksi*

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Introduction

Obstructive sleep apnea (OSA) is a common disorder characterized by intermittent cessation of breathing during sleep, and it represents a significant public health concern[1]. Obstructive sleep apnea has been associated with an increased risk of cardiovascular events, stroke, all-cause mortality, and the development of diabetes mellitus[2].

Obstructive sleep apnea can contribute to the development of multiorgan dysfunction and cause both systolic and diastolic dysfunction of the right ventricle[3]. Right ventricle (RV) diastolic dysfunction usually occurs before RV systolic dysfunction and dilation[4]. Prompt intervention and early detection of RV diastolic dysfunction may be essential in preventing the progression to right heart failure[5,6]. There is little data on how OSA affects RV diastolic function, despite its clinical significance.

Echocardiography is a widely used non-invasive diagnostic technique for assessing heart function. The purpose of this research is to identify early indicators of RV diastolic dysfunction in OSA patients.

Material and Method

Population Study

This study comprised 88 individuals over the age of 18, 58 of whom were male (64.9%) and had been diagnosed with OSA. Patients who underwent both polysomnography and transthoracic echocardiography (TTE) between January 2023 and May 2024 were scanned from hospital records. Those diagnosed with OSA on polysomnography were included in the study. The TTE of the patients included in the study was evaluated in June 2024 by a cardiologist blinded to the apnea and hypopnea index. Apnea and hypopnea index (AHI) index scores of patients were recorded from the hospital records. The classification was based on the AHI, which is the total number of apnea and hypopnea events per hour of sleep. Obstructive sleep apnea was diagnosed using the criteria defined by the American Academy of Sleep Medicine. Patients were divided into two groups according to AHI values: mild-moderate OSA (mild; AHI 5–14 events/hour, moderate; AHI 15–29 events/hour) and severe OSA (AHI ≥30 events/hour)[7].

The study excluded patients with obstructive lung disease, a history of smoking, or pre-existing cardiovascular problems (such as congenital heart disease, atrial fibrillation, or coronary artery disease (CAD)). Furthermore, those with a left ventricular ejection fraction (LVEF) below 50% or poor-quality echocardiograms were not eligible to participate. The local ethics committee approved this study.

Imaging Procedure

The Philips Epic 7c echocardiography device was used to perform the TTE evaluation. Every measurement was carried out in compliance with the American Society of Echocardiography's recommendations[8].

Using the biplane method of discs, the LVEF was computed to evaluate left ventricular function. The biplane approach was used to measure the maximal left atrial volume from apical two- and four-chamber views. The left atrial volume index (LAVI) was subsequently computed by indexing these volumes to body surface area. In order to assess diastolic function, transmitral inflow parameters, such as peak early (E) and late (A) diastolic velocities, were measured using pulsed-wave Doppler echocardiography. The E/A ratio was then calculated. Tissue Doppler imaging was used to measure the diastolic velocities (Em and Am) at the septal and lateral mitral annular regions. To gain a better knowledge of diastolic function, E/Em ratios for the lateral and then septal walls have been established.

The tricuspid annular plane systolic excursion (TAPSE) and RV end-diastolic diameter (RVDD), two essential echocardiographic measures for assessing right ventricular (RV) function, were acquired using 2D mode. Pulsed-wave Doppler imaging was used to quantify the early (E) and late (A) diastolic peak flow velocities from the apical four-chamber view. E′, A′, and S′ cardiac velocities were measured using tissue Doppler imaging at the tricuspid annular plane, which was also acquired from the same point of view. The right atrial volume index (RAVI) has been determined using the adjusted Simpson's method and then normalized to the body surface area. Additional data, including tricuspid E/A and E/Em ratios, right ventricular fractional area change (RVFAC), TAPSE / tricuspid regurgitation velocity (TRV), and TAPSE / pulmonary artery systolic pressure (PASP) ratios, were obtained from recorded measurements.

Table 1. *Demographic, clinical, and laboratory characteristics of all patients with mild to moderate and severe OSA, with p value*

Variable	Mild to moderate OSA (n=45)	Severe OSA (n=43)	Total patients (n=88)	p-value
Age (years)	48±14	56±12	52±14	0.009
Female gender, n (%)	17 (37.8)	13 (30.2)	30 (34.1)	0.458
BMI (kg/cm²)	30.27±6.19	35.13±5.75	32.64±6.42	**<0.001**
HT, n (%)	13 (28.9)	30 (69.8)	43 (48.9)	**<0.001**
DM, n (%)	6 (13.3)	7 (16.3)	13 (14.8)	0.699
CAD, n (%)	5 (11.1)	11 (25.6)	16 (18.2)	0.080
WBC (10^3/uL)	7.55±1.29	7.95±2.21	7.75±1.80	0.960
Neutrophil (10^3/uL)	4.34±1.20	4.59±1.42	4.47±1.31	0.542
Hgb (g/dL)	14.10±1.67	14.81±2.29	14.45±2.02	0.093
Platelet (10^3/uL)	238±57	227±62	233±59	0.318
ALT (mg/dL)	19 (14–33)	24 (16–35)	21 (15–35)	0.197
CRP, mg/dL	3.2 (1.8–6.0)	4.8 (2.1–9.0)	3.6 (2.1–8.0)	0.222
Creatinine (mg/dL)	0.94±0.43	0.94±0.15	0.94±0.32	0.133
MAX apnea time	23.0 (17.0–33.7)	51.6±18.6	37.6 (21.3–60.0)	**<0.001**
AHI (events/h)	13.8±6.1	69.8±28.6	41.2±34.7	**<0.001**
OSA severity (%)				
1	26 (57.8)	0 (0.0)	26 (29.5)	
2	19 (42.2)	0 (0.0)	19 (21.6)	**<0.001**
3	0 (0.0)	43 (100.0)	43 (48.9)	
HR (bpm)	72±11	74±15	73±13	0.792

OSA: obstructive sleep apnea, BMI: body mass index, HT: hypertension, DM: diabetes mellitus, CAD: coronary artery disease, WBC: white blood cell, Hgb: hemoglobin, ALT: alanine aminotransferase, CPR: C-reactive protein, AHI: Apnea-Hypopnea index, HR: heart rate.

Statistical Analysis

Statistical Package for Social Sciences (SPSS) program version 22.0 (IBM Inc., Chicago, IL) was used to analyze the research data. The descriptive continuous variables which have the normal distribution were described as mean and standard deviation, as median and interquartile ranges for continuous variables which do not have the normal distribution, and as percentage values for categorical variables. Kolmogorov-Smirnov test was used to analyze the normal distribution characteristics of continuous variables. To determine the differences between the groups for continuous variables, the Mann-Whitney U test or the two-sample student's *t*-test was used. Variables that were significant in univariate analysis were included in multivariate logistic regression to identify independent predictors of severe OSA. The optimal cut-off values of the significant variables in the multivariate analysis in predicting the development of severe OSA were determined by calculating ROC (receiver operating characteristic curves) and their AUC (area under the curve) values. The p-value <0.05 was taken for statistical significance.

Results

A total of 88 patients diagnosed with obstructive sleep apnea syndrome and evaluated at the cardiology echocardiography laboratory of Kafkas University Hospital between January 2023 and May 2024 were included in the study. Of them, forty-three patients were allocated to the severe OSA group and forty-five patients to the mild-to-moderate OSA group.

The body mass index (BMI) of the severe OSA group was higher than that of the mild-to-moderate group. The severe group was older and had a higher prevalence of hypertension (HT). No significant differences were observed between the two groups regarding sex, diabetes mellitus (DM), heart rate (HR), coronary artery disease (CAD), or laboratory parameters. The classification of baseline demographic information and lab results by group is shown in Table 1.

Table 2. *Echocardiographic parameters of all patients with mild to moderate and severe OSA, with p value*

Variable	Mild to moderate OSA (n=45)	Severe OSA (n=43)	Total patients (n=88)	p-value
E wave, (cm/s)	68±17	68±18	68±17	0.967
A wave (cm/s)	76±16	78±20	77±18	0.767
E/A, mitral	0.93±0.31	0.98±0.67	0.95±0.52	0.323
Em lateral, mitral (cm/s)	10.1±4.1	10±3.3	10.1±3.7	0.822
Em septal, mitral (cm/s)	7.4±2.1	7.2±1.9	7.3±2.0	0.333
Em mean, mitral	8.75±2.64	8.58±2.22	8.67±2.43	0.723
E/Em septal, mitral	10.09±4.1	10.22±4.36	10.16±4.20	0.844
E/Em lateral, mitral	7.61(5.13–9.67)	6.45(5.39–8.63)	6.93(5.32–8.90)	0.679
E/Em mean, mitral	8.48±3.28	8.50±3.96	8.49±3.61	0.686
LVEF, (%)	58±5	57±7	58±6	0.314
E, tricuspid (cm/s)	53.4±13.9	59.3±25.1	56.3±20.2	0.304
A, tricuspid (cm/s)	60.1±18.3	59.0±20.6	59.6±19.3	0.793
E/A, tricuspid	0.95±0.32	1.04±0.37	0.99±0.35	0.155
RV Em (cm/s)	10.4±3.6	9.1±2.9	9.8±3.3	0.123
RV Am (cm/s)	13.5±4.1	14.1±4.8	13.8±4.4	0.628
RV S'(cm/s)	12.5±2.8	12.7±3.2	12.6±3	0.825
LAVI (mL/m²)	17.05±8.82	24.45±13.35	20.67±11.8	**0.001**
E/Em, tricuspid	5.70±2.32	7.21±3.83	6.44±3.22	**0.046**
TRV, m/s	1.5±0.6	1.9±0.8	1.7±0.7	0.050
TAPSE, mm	2.4±0.5	2.3±0.5	2.4±0.5	0.703
IVC, mm	1.4±0.4	1.4±0.5	1.4±0.4	0.927
sPAP, mmHg	14.86±8.03	20.62±12.14	17.68±10.59	0.044
TAPSE/TRV, mmx $(m/s)^{-1}$	1.87±1.09	1.58±0.93	1.73±1.02	0.071
TAPSE/sPAP	0.17 (0.13–0.27)	0.13(0.07–0.27)	0.16(0.09–0.27)	0.072
RVDD (mm)	3.8±0.7	4.2±0.6	4.0±0.7	0.008
MPAd, mm	2.1±0.4	2.2±0.3	2.1±0.4	0.016
RAVI, mL/m²	13.26±4.81	24.24±10.75	18.63±9.90	**< 0.001**
RVFAC (%)	0.48±0.15	0.50±0.16	0.49±0.15	0.861

E: peak early inflow velocity, A: peak late inflow velocity, Em: early diastolic tissue Doppler velocity, LAVI: left atrial volume index, LVEF: left ventricle ejection fraction, RV Em, RV Am, and RV S'; right ventricle peak early (Em), Late (Am) and systolic (S') tissue Doppler velocities, LAVI: left atrial volume index, TRV: tricuspid regurgitation velocity, TAPSE: tricuspid annular plane systolic excursion, IVC: inferior vena cava, sPAP: systolic pulmonary artery pressure, RVDD: right ventricular end-diastolic diameter; MPAd: main pulmonary artery diameter at diastole, RAVI: right atrial volume index, RVFAC: right ventricle fractional area change.

A summary of the echocardiographic results, broken down by group, is shown in Table 2. The severe group's LAVI was substantially greater than that of the mild-to-moderate group (17.05±8.82 mL/m² vs. 24.45±13.35 mL/m²; p=0.001) in terms of left-sided characteristics. The groups' LVEFs did not differ significantly. The severe group had considerably higher right-sided measures, including the right atrial volume index (RAVI) (13.26±4.81 mL/m² vs. 24.24±10.75 mL/m²; p <0.001) and the E/Em tricuspid ratio (5.70±2.32 vs. 7.21±3.83; p=0.046).

According to Table 3's multivariate analysis, severe OSA was independently predicted by HT, BMI, and RAVI (HR: 1.231, 95% CI: 1.120–1.353; p <0.001). A RAVI cutoff value of 17.3 predicts severe OSA with 72.1% sensitivity and 86.7% specificity, according to the ROC curve analysis shown in Fig. 1 (AUC=0.822, 95% CI: 0.726–0.895; p <0.001).

Discussion

This study searched the subclinical effects of severe OSA on cardiac structure and function using two-dimensional echocardiography. The main findings revealed that patients with severe OSA had significantly higher values of LAVI, tricuspid E/Em ratio,

Table 3. *Univariate and multivariate logistic regression analysis of demographic, biochemical, and echocardiographic parameters for diagnosis of severely OSA*

	Univariate			Multivariate		
Variable	Odds ratio	95% CI	p-value	Odds ratio	95% CI	p-value
Age	1.049	1.013–1.085	0.007			
HT	5.680	2.273–14.197	<0.001	4.928	1.383–17.558	**0.014**
BMI	1.153	1.059–1.255	0.001	1.128	1.009–1.261	**0.034**
LAVI	1.073	1.019–1.131	0.008			
E/Em Tricuspid	1.181	1.010–1.380	0.036			
TRV, m/s	2.011	1.067–3.792	0.031			
sPAP	1.056	1.012–1.103	0.013			
RV basal diameter	2.323	1.155–4.674	0.018			
MPAd	4.475	1.222–16.383	0.024			
RAVI	1.231	1.120–1.353	<0.001	1.256	1.120–1.410	**<0.001**

HT: hypertension, BMI: body mass index, LAVI: left atrial volume index, E: peak early inflow velocity, Em: early diastolic tissue Doppler velocity, TRV: tricuspid regurgitation velocity, sPAP: systolic pulmonary artery pressure, RV: right ventricle, MPAd: main pulmonary artery diameter at diastole, RAVI: right atrial volume index.

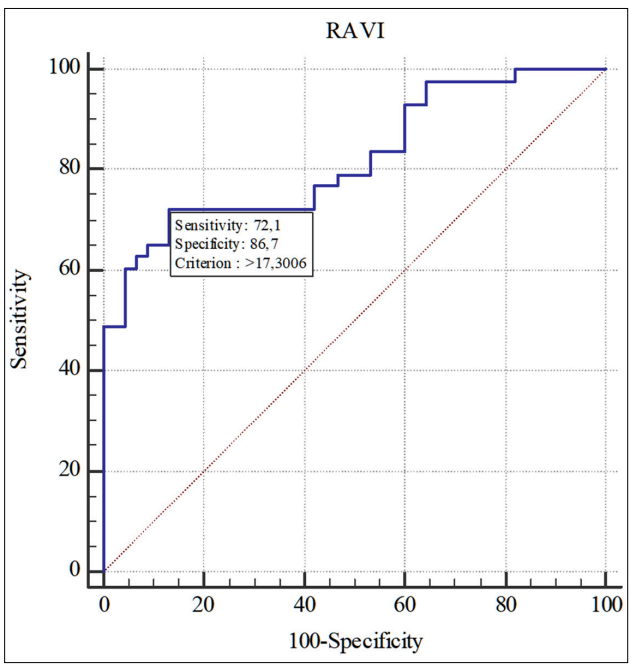

Figure 1. *Receiver operating characteristic (ROC) curve analysis for RAVI for severe OSA group.*

and RAVI compared to those with mild-to-moderate OSA. Furthermore, severe OSA was independently linked to RAVI.

The literature currently in publication emphasizes the difficulties in precisely evaluating RV morphology and function in clinical settings, with studies frequently presenting contradictory results. Patients with OSA exhibit functional and structural dysfunction in their RV, and the severity of the illness is connected with these changes, according to studies employing both conventional and tissue Doppler imaging techniques[9]. Accurate measurement of RV function in OSA is crucial for prompt management, monitoring therapy effects, predicting prognosis, and reducing deleterious cardiovascular events. Myocardial performance index, RVFAC, TAPSE, and other 2D echocardiographic parameters can be used to evaluate global right ventricular function. Another important and sensitive early sign for assessing RV impairment is RV diastolic insufficiency[10]. In conventional echocardiography, the metrics used to assess the right ventricle's diastolic function are similar to those used to assess the left. RAVI, tissue Doppler imaging parameters like Em, Am, and the E/Em ratio, and pulse Doppler imaging parameters like E, A, and the E/A ratio are among them[11]. For the detection of subclinical RV dysfunction, tissue Doppler imaging is more sensitive than 2D echocardiography[10]. The TAPSE and RVFAC groups did not differ in the current investigation; however, in line with earlier research[10,12], we demonstrated that patients with severe OSA had greater RAVI and E/Em ratios than patients in the mild-to-moderate OSA group. Furthermore, RAVI was discovered to be an independent predictor of severe OSA in the current investigation. There aren't many studies assessing E/A and E/Em among those assessing right ventricular diastolic dysfunction in the literature. We discovered that patients with severe OSA had higher E/Em values, which is in line with the work of Li et al.[12]. Among other techniques, including strain calculation, this is the most useful and appropriate way to assess the RV's diastolic dysfunction. In patients with OSA, right ventricular dysfunction may result from a number of processes.

During apnea episodes, inspiratory efforts against the constricted pharynx cause the right ventricle to distend, increase venous return, and cause volume overload[13]. Additionally, the increased myocardial oxygen demand brought on by right ventricular (RV) structural remodeling, in conjunction with decreased coronary artery perfusion from vasoconstriction and vascular endothelial remodeling, may exacerbate RV dysfunction[14] and cause RV ischemia. This syndrome may also be caused by a number of other conditions, including CAD, obesity, and systemic HT.

Left atrial volume index was considerably higher in the patients with the severe OSA group than in the patients with the mild-to-moderate OSA group, which is consistent with evidence from the literature[15]. Left atrial volume index may have been higher in the severe OSA group due to increased rates of HT, DM, and CAD, which are predictors of LV dysfunction, albeit this effect was not statistically significant. Furthermore, blood pressure might rise due to sympathetic hyperactivity and nocturnal hypoxia, and transmural pressure can rise as a result of significant negative intrathoracic pressure brought on by respiratory effort during apnea. Over time, these events cause the left and right ventricles to experience increasing afterload, which causes ventricular hypertrophy and cardiac malfunction in both the diastolic and systolic phases, ultimately leading to heart failure (HF)[16]. Similar to HF, the development of HT as a result of OSA is a normal occurrence. Heart failure is a significant predictor of the severity of OSA, and our study demonstrated that the prevalence of HT was elevated in individuals with severe OSA, aligning with existing literature.

Numerous studies have shown a correlation between severe OSA and BMI. According to Young T. et al., a 5.3 BMI gain increases the likelihood of getting OSA by four times[17]. Indeed, BMI severity and apnea severity were both numerically examined and associated in a recent study by Fattal D. et al.[18]. In line with these, our study found that individuals with severe OSA had higher BMI values than those with mild to moderate OSA. Additionally, our research revealed that BMI is a reliable indicator of the existence of severe OSA. This condition can be explained by a variety of variables. Because fat deposits, especially around the upper airways, can result in a smaller airway lumen and higher collapsibility, weight gain is believed to exacerbate apnea. Moreover, the accumulation of fat around the chest decreases resting lung volume and chest compliance. Such decreases in lung volume promote pharyngeal collapsibility by decreasing tracheal traction[19].

Knowing that OSA exhibits structural and functional alterations of right heart chambers, detecting the changes using imaging methods seems to be reasonable. Right atrial volume index and E/Em are a part of the 2D echocardiography imaging method and are easily measurable. Because of being user-friendly and accessible, echocardiography may be used in the first stage, although there are more advanced imaging methods.

Conclusion

Given the high prevalence of OSA and its association with cardiovascular complications, routine assessment of disease severity is crucial. Our findings suggest that echocardiographic parameters such as RAVI and tricuspid E/Em ratio are effective, affordable, and practical markers for predicting OSA severity, and may aid in early identification of patients at risk for cardiac involvement.

Limitations

This study has several limitations. First, the lack of a non-OSA control group limits our ability to evaluate the absolute contribution of OSA to echocardiographic changes. Second, the sample size was relatively small, which may affect statistical power. Third, although comorbid conditions such as CAD, hypertension, and diabetes were included due to their frequent association with OSA, they may have confounded the relationship between OSA severity and echocardiographic parameters.

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