Electromechanical delay and 4-chamber longitudinal strain in patients with obstructive sleep apnea

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Original Article

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ABSTRACT

Objectives: The aim of this study was to evaluate atrial electromechanical delay (AEMD), apical 4-chamber longitudinal strain (4C-LS) and echocardiographic changes in patients with obstructive sleep apnea (OSA).

Design: Prospective cross-sectional study

Setting: Secondary care hospital

Subjects: Forty-six patients (32 male, 14 female) who were diagnosed as mild-to-severe OSA (AHI ≥5 events/h) and control group consisted of 35 healthy subjects (18 male, 17 female)

Intervention: Polysomnography, blood samples and transthoracic echocardiograhy (TTE) were evaluated

Main outcome measures: TTE was used to evaluate echocardiographic changes, AEMD and 4C-LS

Results: Left ventricle (LV) end-diastolic and end-systolic diameter, interventricular septum and posterior wall thickness were significantly higher; LVEF and Ea/Aa mitral ratio were lower; right ventricle basal, mid and vertical diameters, Emax, Amax, and Ea tricuspid, tricuspid regurgitan velocity, systolic pulmonary artery pressure, and systolic motion tricuspid were significantly higher in the OSA group. TAPSE was significantly lower and AEMD lateral/tricuspid, lateral/mitral and septal were significantly higher in the OSA group. Mid anterolateral, apicolateral, apex, apical septal strains and 4C-LS were decreased significantly in the OSA group

Conclusion: Right-left ventricular systolic-diastolic functions were impaired in patients with OSA. In these patients, apical 4C-LS was lower and AEMD was prolonged.

KEY WORDS: Atrial electromechanical delay, longitudinal strain, obstructive sleep apnea

INTRODUCTION

Obstructive sleep apnea (OSA) is a serious, life-threatening and common chronic disease affecting particularly middle-aged men in the populations [1, 2]. OSA is characterized by repetitive surcease episodes of breathing while sleeping, by the reason of complete or partial airway obstruction [1, 2]. Hypoxia and hypercapnia caused by OSA increases the arousal of the patient throughout the night [3]. These arousals associated with hypoxia and hypercapnia stimulate the sympathetic nervous system, increase catecholamine release and myocardial oxygen consumption [3]. Increased myocardial oxygen consumption causes cardiac ischemia, myocardial infarction, hypertrophy, hypertension, increase in left ventricle wall tension, congestive heart failure, arrhythmias such as atrial fibrillation and stroke [3,4]. Cardiovascular complications are the most common complications of OSA. Echocardiography is gold standard noninvasive imaging method for evaluating myocardial functions of these type of patients and assessing the effects of OSA on heart [5].

Atrial electromechanical delay (AEMD) is the time interval between the beginning of P wave on surface electrocardiography and beginning of the late diastolic wave (Am-wave) on Tissue Doppler Imaging

(TDI) [6]. The structural changes of atrial tissue cause delay between the electrical stimulation and mechanical contraction [6]. Atrial tissue changes can cause prolongation of P wave on surface electrocardiogram (ECG) [7]. Prolongation of P wave can be seen in patients undergoing coronary artery bypass surgery, patients with hypertrophic cardiomyopathy, right atrial dilatation, atrial septal defect hypertension and chronic obstructive pulmonary disease due to affected atrial tissue [7].

Two-dimensional speckle tracking echocardiography (2D-STE) is a strain measurement method to obtain the size of regional myocardial deformations [8,9]. Myocardial deformations can be obtained by this easily applied method in the longitudinal, circumferential and radial pointing [8,9].

The aim of this study was to evaluate AEMD, apical 4C-LS and echocardiographic changes in patients with OSA. We especially focused on AEMD, longitudinal strain, and diastolic-systolic functions of right-left heart in these patients.

SUBJECTS AND METHODS

Study Population

This prospective study was conducted with the approval of a university hospital Caucasian University Medical Faculty ethics committee between April and August 2018. The patient group consisted of 46 patients (32 male, 14 female) who were referred to the Sleep Disorders Center of Caucasian University Hospital and were diagnosed as mild-to-severe OSA (AHI ≥5 events/h) on polysomnographic evaluation. The control group consisted of 35 healthy subjects (18 male, 17 female) who were found not to have OSA (AHI<5 events/h) on polysomnographic evaluation.

The exclusion criteria were as follows: chronic obstructive pulmonary disease on pulmonary function tests, patients with valvular and structural heart disease, wall-motion abnormality, coronary artery disease, acute coronary syndrome, heart failure, atrioventricular conduction abnormalities, previous history of atrial fibrillation, ejection fraction <50%, use of drugs that affect atrioventricular conduction system, uncontrolled hypertension, pulmonary embolism, pneumonia, insulin dependent diabetes mellitus, history of cerebrovascular disease, history of continuous positive airway pressure, renal impairment, hypo-hyperthyroidism, anemia, electrolyte disorders, acid-base disorders, malignancy, patients using two or more oral antidiabetic drugs, systemic inflammatory response syndrome, poor echocardiographic view.

The following parameters of all patients were evaluated: age, gender, body mass index (BMI), comorbidity, blood glucose, electrolytes, liver function tests, renal function tests, complete blood count, transthoracic echocardiography and polysomnography. All patients were informed about the study. An informed consent was obtained for all procedures and then the patient's signature was accepted.

Blood samples

All blood samples were drawn from the vein in the forearm and collected into 2 mL Lavender (EDTA) top tube and 5 mL Yellow top tube were analyzed with Pentra DF Nexus, Horiba Medical, Japan with Automated Cell Counter Methodology and Cobas C 501, Roche. The complete blood samples were

stabilized optimally when run within in 4 hours of collection, stable for 24 hours at room temperature, and stable for 36 hours at 2 – 8 degrees C. The biochemical samples were stabilized optimally when run within 2 hours of collection, stable for 24 hours at +4-degree C.

Polysomnography

Overnight PSG was performed in all patients using conventional and analysis according to the American Academy of Sleep Medicine [10]. All patients spent one entire night in the sleep laboratory with the aim of capturing a typical night's sleep. The wakefulness, sleep stages, respiration, cardiopulmonary functions and body movements of patients were evaluated. Electroencephalography, electro-oculography, and chin muscle electromyography channels were used to sleep stages. Airflow and respiratory effort channels were used to assess sleep-disordered breathing. Arterial oxygen saturation was measured with finger pulse oximetry channel. Movement changes of the chest and abdomen during breathing were recorded by using respiratory inductive plethysmography. Limb EMG channels were placed on the legs (tibialis anterior muscle) and were evaluated periodical limb movements. An oronasal flow cannula attached to pneumotachograph and apneas-hypopneas. Apneas were defined as the cut-off of airflow for more than ≥10 s. Hypopnea were defined: peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure, duration of the ≥30% drop in signal excursion is ≥10 seconds and ≥3% oxygen desaturation from pre-event baseline and/or the event is associated with an arousal [11]. The Apnea/Hypopnea Index (AHI) was obtained by dividing the total number of apneas and hypopneas during the entire sleeping time. OSA classification was made according to American Academy of Sleep Medicine: mild OSA was defined AHI of 5-15, moderate OSA was defined AHI of 15-30 and severe OSA was defined AHI of more than 30 [10].

Echocardiography

Transthoracic echocardiography (Epiq 7; Philips) was evaluated by a practitioner in a standard protocol in all patients. A 2.5 MHz probe was used for the Doppler measurements and 2.5-3.5 MHz probe for tissue Doppler measurements. Patients were monitored using electrocardiographic leads and were placed in the left lateral decubitus position. Echocardiographic images were obtained from the parasternal views (long axis, short axis), the apical four-chamber view and, the subcostal view. Echocardiographic measurements were performed at the end of expiration according to the recommendations of the American Society of Echocardiography/ European Association of Echocardiography [12]. 1) Diameters of right ventricle (RV), measured in apical view. 2) Left ventricle (LV) diameter and wall thickness were measured in the parasternal view. 3) Left atrial diameter, measured in the parasternal view. 4) Aortic root diameters, measured at the sinus of Valsalva. 5) LV ejection fraction, measured in apical 4-chamber view by modified Simpson method. 6) Right and left ventricle functions were evaluated as follows: a) maximal peak velocity of early diastolic flow (Emax), maximal peak velocity of atrial contraction (Amax), and ratio of these (Emax/Amax), measured over the mitral and tricuspid valves; b) Tissue Doppler imaging, measured in the mitral and tricuspid lateral

annulus at early diastole (Ea), atrium systole (Aa) and ratio of these (Ea/Aa); c) The ratio of Emax/Ea. 7) Aortic, tricuspid, mitral and pulmonary valvular evaluation. 8) Tricuspid regurgitant velocity (TRV) recorded by continuous wave Doppler.

AEMD was calculated from colored-TDI recordings. AEMD was determined as the time interval between the beginning of echocardiographic P wave to the initial of Am-wave (late diastolic wave) in TDI recordings. AEMD was measured from lateral/tricuspid, lateral/mitral and septal anulus from apical 4-chamber views.

The echocardiographic examinations, and standard 2D measurements for strain were acquired according to American Society of Echocardiography recommendations [12]. The images were digitally stored, and measurements were performed by same practitioner. Images were obtained at a frame rate of 50 to 70 per second, and digital loops were saved onto optical disc for off-line analysis. The cardiac cycle with the best image quality and without any artefacts was selected for reporting results. Two and three-chambers images were not used due to intense artefacts. Longitudinal strain images were obtained apical 4-chamber views. The practitioner identified three points on each view (two borders of the mitral annulus and the apex). Speckles were tracked frame by-frame throughout the LV wall during the cardiac cycle and basal, mid, and apical regions of interest were created.

Statistical Analysis

All statistical calculations were performed with SPSS 23.0 (SPSS for Windows, Chicago, IL, SA). All continuous variables were expressed as mean±standard deviation; categoric variables were defined as percentages (%). The categorical parameters were compared with Chi Square test and Fischer's exact test. The normal distribution was determined by histogram and Kolmogorov-Smirnov test. Mean values of continuous variables were compared between the groups using Mann-Whitney U test. All tests were applied as two tailed; the statistical significance level was $p<0.05$.

RESULTS

Baseline clinical characteristics of the study population are presented in Table 1. BMI and number of patients with hypertension were higher in the OSA group ($p<0.001$).

Biochemical blood parameters of the study population are presented in Table 2. Glucose levels, urea, creatinine, uric acid, AST and ALT were higher significantly in the OSA group.

Complete blood counts of the study population are presented in Table 3. Hemoglobin, hematocrit, eosinophil count, and percent were significantly higher in the OSA group.

Conventional and tissue Doppler echocardiographic parameters of left heart and septum for two groups are shown in Table 4. LV end-diastolic diameter, end-systolic diameter, interventricular septum and posterior wall thickness were significantly higher in the OSA group ($p<0.05$). LV ejection fraction and Ea/Aa mitral ratio were lower in the OSA group.

Conventional and tissue Doppler echocardiographic parameters of right heart for two groups are shown in Table 5. Right ventricle basal, mid and vertical diameters, Emax, Amax, and Ea tricuspid, TRV,

systolic pulmonary artery pressure, and systolic motion tricuspid were significantly higher in the OSA group. Tricuspid annular plane systolic excursion (TAPSE) was significantly lower in the OSA group compared to healthy subjects (p<0.001).

Colored-TDI measurements of AEMD and 2D-STE measurements of apical 4C-LS for two groups are presented in Table 6. AEMD lateral/tricuspid, lateral/mitral and septal were significantly higher in the OSA group (p<0.001). Mid anterolateral, apicolateral, apex, apical septal strains and 4C-LS were decreased significantly in the OSA group (p<0.001).

Polysomnographic findings of OSA patients according to classifications are presented in Table 7. AHI and hypopnea were significantly differed between three groups (p<0.001).

DISCUSSION

Obstructive Sleep Apnea is a well-known disease with numerous systemic complications, which can cause cardiovascular diseases in multifold phases and pathological pathways [13]. In OSA patients, apnea-hypopnea periods revealed by partial and complete airway obstruction cause sleep fragmentations [14-16]. In patients with OSA, hypoxia and hypercapnia-induced sustained sympathetic nervous system activation, low-grade chronic inflammation, oxidative stress, and vascular inflammation are the main mechanisms that explain cardiac diseases [2,17]. In this study, cardiac changes of caused by OSA were evaluated by echocardiography.

Our results suggest that LV end-diastolic and end-systolic diameters, interventricular septum and posterior wall thickness were significantly higher in patients with OSA. LV ejection fraction and Ea/Aa mitral ratio known as early diastole/atrium systole were lower in these patients. There was an increase in both LV hypertrophy and LV diameters in patients with OSA. The increase in LV end-diastolic diameter was associated with lower EF. Ea/Aa, which is the indicator of diastolic function, was lower in patients with OSA. Hypoxia and hypercapnia periods may influence the proportion in myocardial oxygen requirement and supplement [17]. Myocardial ischemia, oxidative stress and activation of sympathetic nervous system increase left ventricular afterload and decrease in left ventricular preload [17,18]. OSA may impair LV function and additively LV hypertrophy is associated with elevated blood pressure levels during sleep [18].

In this study, right ventricle basal, mid and vertical diameters, Emax, Amax and Ea tricuspid, tricuspid regurgitant velocity, and systolic pulmonary artery pressure were significantly higher in patients with OSA. TAPSE was significantly lower in the OSA group. The diameters and TAPSE were used to evaluate right ventricular systolic functions. Increased basal diameter and decreased TAPSE were indicative of right ventricular systolic dysfunction. Although Emax/Amax, which is a diastolic function indicator, insignificantly decreased in patients with OSA. A larger scale working group is needed. Increased TRV and SPAP showed increased pulmonary artery pressure. In OSA patients, pulmonary vasoconstriction secondary to hypoxia period may contribute to pulmonary hypertension, and this hypertension leads to right ventricular dysfunction and hypertrophy [19,20]. TAPSE is a measurement for right ventricle function and it gives information about RV ejection fraction [21].

In the present study, we found that lateral/tricuspid, lateral/mitral and septal AEMD were significantly higher in patients with OSA. The prolongation of AEMD was an expected result for patients with OSA. AEMD is known as the time intervals between the intervals between the atrial depolarization and the beginning of atrial mechanical contraction [22]. Prolonged AEMD is predisposed to atrial fibrillation [22]. In OSA patients, repetitive forced inspiration against an obstructed airway may cause a negative intrathoracic pressure that may lead to elevated cardiac afterload, increased atrial dimension, wall tension and resulting atrial remodeling [14]. Autonomic nervous system irregularity with elevated-depressed heart rates triggered by hypoxemia, hypercapnia and acidosis may stimulate electrical changes in atrium [14]. Structural remodeling of the heart with increased left atrial dilatation and increased fibrosis of tissue caused by intrathoracic pressure shift is another mechanism for prolonged AEMD [23,24]. The nightlong renin-angiotensin system fluctuations and increased aldosterone in OSA patients also may precipitate resistant hypertension and atrial fibrillation [25,26]. As in similar study samples, AEMD prolongs significantly in patients with mild-to-severe OSA [27,28].

The basal longitudinal strain measurements such as mid anterolateral, apicolateral, apex, apical septal and 4C-LS we used to evaluate left ventricular myocardial function were significantly low in patients with OSA compared to healthy subjects. Ventricular strain and strain rates, which are deformation indicator of myocardium, are used for to measure ventricular dysfunction [29]. The LV consists of three non-homogenous fiber layers. Reverse positioning of subendocardial and sub epicardial layer fibers is important for redistribution of the strain in the heart. Heterogenous deterioration of basal, middle and apical ventricular segments provide coordinated ventricular contraction. This LV contraction, which shows strain of the heart, can be impaired after decreased arterial oxygen saturation and increased negative intrathoracic pressure [30,31]. Nocturnal pulse oximetry, which is a method for monitoring the arterial blood oxygen saturation, can demonstrate significant changes in patients with OSA due to recurrent apnea periods [32]. Hypoxia with apnea episodes may lead to decreased myocardial oxygenation, decreased LV contraction and decreased strain. Longitudinal strain reduction is expected in patients with OSA and there are studies supporting this result [5,8,33].

The small number of patients and the presence of longitudinal strain images in only 4-chamber due to artefacts are the main limitations of the study. The patients were enrolled from only one sleep center hence limiting the variegation of the patients.

CONCLUSION

This study revealed that, OSA is a disease that can impair the left-right ventricular systolic-diastolic functions of the heart and it may cause structural changes in the heart. Moreover, OSA may cause prolongation of atrial conduction times and it may decrease the contraction forces of the heart.

This prospective clinical study is important because of the combined evaluation right-left ventricular functions, AEMD and longitudinal strain.

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Conflict of interest: None declared.

Authors Contribution

FK did data collection and designed of manuscript. SA & GP did analysis, manuscript writing.

FK & SA did review and final version of manuscript.

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None

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Table 1: Clinical characteristics of the study population

Clinical Profile		OSA (n=46)	Controls (n=35)	p value
		Mean±sd / n (%)		
Age		49.3±10.5	48.3±5.1	NS
Gender				
	Male	32 (69.6%)	18 (51.4%)	NS
	Female	14 (30.4%)	17 (48.6%)	NS
BMI (kg/m^2)		35.67±5.77	28.41±4.1	**<0.001**
Comorbidities				
	Hypertension	28 (60.9%)	3 (8.6%)	**<0.001**
	Diabetes Mellitus	-	1 (2.9%)	NS
	Hyperlipidemia	6 (13%)	1 (2.9%)	NS

OSA: obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS: non-significant; BMI: body mass index.

Table 2: Biochemical parameters of the study population

Biochemical parameters	OSA	Controls	p value
	(Mean±sd)		
Glucose	108.1±15.5	98.5±11.8	**0.007**
Urea	35.6±9.2	30±7.4	**0.001**
Creatinine	0.87±0.16	0.72±0.19	**0.001**
Uric acid	6.22±1.3	4.89±1.57	**<0.001**
HDL	42.6±8.2	48.9±12.6	**0.048**
Triglyceride	176±61	170.6±80.7	NS
C-reactive protein	0.59±0.87	0.37±0.33	NS
Albumin	4.31±0.33	4.59±0.24	**<0.001**
AST	24.5±8.2	20.1±8.6	**0.018**
ALT	33.6±15.2	21.6±14.3	**<0.001**
LDH	188±36.6	198.9±40.1	NS
Calcium	9.5±0.3	9±0.4	NS
Sodium	140.7±3.2	140.5±1.9	NS
Potassium	4.49±0.35	4.42±0.34	NS
ALP	82.4±24.8	100.5±13.4	NS
Total Bilirubin	0.43±0.21	0.46±0.24	NS
Protein	7.07±1.68	7.34±0.45	NS

OSA: obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS: non-significant; HDL: high density lipoprotein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase; ALP: alkaline phosphatase.

Table 3: Complete blood counts of the study population

Complete Blood Count Parameters	OSA	Controls	p value
	(Mean±sd)		
Hemoglobin (mg/dL)	15.9±1.44	15.02±1.26	**0.011**
Hematocrit	48.05±4.4	45.15±3.5	**0.002**
WBC	7.86±1.87	7.38±2.18	NS
MPV	8.44±0.58	8.91±0.79	**0.006**
Platelet count	262.4±58.8	259.4±57	NS
Lymphocyte count	2.36±0.73	2.34±0.63	NS
Lymphocyte percent	31.03±8.95	32.47±7.12	NS
PLR	122±47.32	118.83±41.94	NS
Neutrophil count	4.61±1.36	4.26±1.7	NS
Neutrophil percent	58.91±7.81	56.82±8.13	NS
NLR	2.17±1.08	1.91±0.85	NS
Eosinophil count	0.26±0.15	0.21±0.29	**0.002**
Eosinophil percent	3.16±1.42	2.77±2.99	**0.009**
RDW	15.4±1.98	15.12±1.23	NS
PCT	0.22±0.06	0.23±0.04	NS

OSA: obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS: non-significant; WBC: white blood cell; MPV: mean platelet volume; PLR: platelet-to-lymphocyte ratio; NLR: neutrophil-to-lymphocyte ratio; RDW: red blood cell distribution width; PCT: plateletcrit.

Table 4: Echocardiographic findings of the left heart and septum in both groups

Left Heart and Septum	OSA	Controls	p
	(Mean±sd)		
Left Heart			
Dimensions			
Left atrium (parasternal long axis)			
Diameter (mm)	38±0.38	35.2±3.1	0.007
Left ventricle (parasternal long axis)			
End-diastolic diameter (mm)	49.4±0.42	46.2±3.9	**0.004**
End-systolic diameter (mm)	31±0.36	26.6±0.31	**<0.001**
Left ventricle wall thickness			
Interventricular septum (mm)	11.8±1.4	10.7±1.3	**<0.001**
Posterior wall (mm)	10.5±1.4	9.6±1.2	**0.003**
Ventricular function			
Systolic function			
LV ejection fraction (%)	58.27±5.54	61.73±3.31	**0.008**
Diastolic function			
Emax mitral (cm/s)	74.59±16.57	70.18±13.62	NS
Amax mitral (cm/s)	75.96±12.33	70.43±19.13	NS
Emax/Amax mitral	1.01±0.27	1.33±1.94	NS
Ea (tissue doppler lateral mitral) (cm/s)	10.93±3.48	11.25±3.06	NS
Aa (tissue doppler lateral mitral) (cm/s)	12.34±3.28	11.38±2.46	NS
Ea/Aa mitral	0.65±0.24	0.78±0.29	**<0.001**
Emax/Aa	0.97±0.45	1.05±0.42	NS
Emax/Ea	7.32±2.13	6.69±2.48	NS
Mitral E wave decelaration time (ms)	153.9±21.6	145.1±24.7	NS
Aortic root diameter (cm)	3.52±0.25	3.41±0.33	NS
Septum			
Ea (tissue doppler septal) (cm/s)	7.41±1.63	7.7±2.7	NS
Aa (tissue doppler septal) (cm/s)	11±2.12	10.77±2.6	NS
Ea/Aa septal	0.71±0.27	0.76±0.35	NS
Emax/Ea	1.08±0.54	1.01±0.71	NS
Systolic motion mitral	9.91±3.11	8.99±1.98	NS
Systolic motion septal	8.42±1.47	7.78±1.35	NS
Heart rate, beats/min	76.6±8.8	73.6±10.1	NS

OSA, obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS, non-significant; LV, left ventricle.

Table 5: Echocardiographic findings of the right heart in both groups

Right Heart	OSA	Controls	p
	(Mean±sd)		
Right Heart			
Dimensions			
Right ventricle (mm)			
Basal	35.8±4.5	32.4±4	**0.001**
Mid	23.7±3.4	21.6±4.4	**0.007**
Vertical	58.1±5.9	54.2±4.6	**0.002**
TAPSE (mm)	23.3±0.27	27.6±2.8	**<0.001**
Ventricular function			
Diastolic function			
Emax tricuspid (cm/s)	57.39±11.09	52.59±8.11	**0.009**
Amax tricuspid (cm/s)	55.87±14.97	47.32±8.71	**0.007**
Emax/Amax tricuspid	1.11±0.39	1.14±0.23	NS
Ea (tissue doppler tricuspid) (cm/s)	10.35±2.55	9.38±2.52	**0.048**
Aa (tissue doppler tricuspid) (cm/s)	13.75±3.52	13.84±3.1	NS
Ea/Aa tricuspid	0.82±0.34	0.72±0.29	NS
Emax/Ea	5.8±1.51	5.91±1.49	NS
Assessment of pulmonary hypertension			
TRV (m/s)	2.412±0.429	2.217±0.306	**0.030**
Systolic pulmonary arterial pressure (mmHg)	24.1±8.3	20.1±5.5	**0.034**

COPD: chronic obstructive pulmonary disease; continuous variables are expressed as mean ± standard deviation; NS: non-significant; TAPSE: tricuspid annular plane systolic excursion; TRV: tricuspid regurgitant velocity

Table 6: Atrial conduction times and apical 4-chamber longitudinal strains are both groups

Atrial conduction times and strains	OSA	Controls	p
	(Mean±sd)		
Atrial Electromechanical Delay			
Lateral/tricuspid (msec)	26.4±9	16.9±8.9	**<0.001**
Lateral/mitral (msec)	62.7±12	38.6±14.6	**<0.001**
Septal (msec)	41.5±8.8	22.4±10.1	**<0.001**
Longitudinal Strain			
Basal Anterolateral	-20±3.3	-20.9±3.3	NS
Mid Anterolateral	-17.3±3.6	-20.1±3	**<0.001**
Apicolateral	-18.8±3.3	-21.8±2.3	**<0.001**
Apex	-21.7±3.3	-25.5±2.5	**<0.001**
Apical Septal	-24.7±4.2	-28.8±4.1	**<0.001**
Mid Inferoseptal	-14.7±4.9	-16.4±4.2	NS
Basal Inferoseptal	-15.2±2.4	-15±2.5	NS
4C-LS	-18.69±2.16	-21.07±1.93	**<0.001**

OSA: obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS: non-significant; 4C-LS: 4-chamber longitudinal strain.

Table 7: Polysomnographic findings of the OSA group according to classification

Polysomnographic findings	OSA Classification			p		
	Mild OSA (n=8)	Moderate OSA (n=6)	Severe OSA (n=32)	Mild vs. Moderate	Mild vs. Severe	Moderate vs. Severe
	Mean±sd / n (%)					
Sleep latency (minute)	27.75±14.51	49.67±21.75	18.38±18.39	**0.037**	NS	**0.002**
Sleep efficiency (%)	78.3±9.66	73.47±17.57	81.83±13.97	NS	NS	NS
AHI	9.6±2.77	23.33±3.25	52.13±21.81	**0.002**	**<0.001**	**<0.001**
Obstructive apnea	0.93±0.46	2.53±3.08	11.34±15.13	NS	**0.004**	**0.045**
Central apnea	0.43±0.46	0.5±0.63	2.42±2.89	NS	**0.010**	**0.016**
Mix apnea	0±0	0±0	2.13±5.45	NS	**0.026**	NS
Mean oxygen saturation	88.08±3.44	88.1±4.13	89.22±4.23	NS	NS	NS
Minimum oxygen saturation	70.25±12.41	73.67±14.46	71.25±12.07	NS	NS	NS
Hypoapnea	7.98±1.88	20.3±0.78	36.18±12.93	**0.002**	**<0.001**	**<0.001**

OSA: obstructive sleep apnea; continuous variables are expressed as mean ± standard deviation; NS: non-significant; AHI: apnea/hypopnea index.