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Assessment of Myocardial Mechanics by Speckle Tracking Echocardiography in Patients with Obstructive Sleep Apnea

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a widespread health issue increasingly linked to cardiovascular complications, including dysfunction of both the left ventricle (LV) and right ventricle (RV), arterial hypertension, coronary artery disease, as well as heart failure, and the risk of arrhythmias.

Objectives: This study aimed to assess the impact of obstructive sleep apnea (OSA) on myocardial mechanics utilizing Two-dimensional speckle tracking echocardiography (2D-STE).

Subjects and methods: 45 patients diagnosed with OSA through polysomnography were set to be classified into three groups with respect to their apnea-hypopnea index (AHI): Mild, moderate and severe. Additionally, a control group comprising 45 healthy individuals carefully matched for sex and age was included. All participants underwent a thorough echocardiographic evaluation, which included both conventional 2D echocardiography and 2D-STE.

Results: While conventional 2D echocardiography did not reveal significant differences in LV dimensions and function among the study groups, 2D-STE analysis indicated statistically significant variations in LV and RV strain values. The most significant impairments were detected in the severe as well as the moderate OSA groups when compared to the other mild OSA and control groups. **Conclusion:** Our findings suggest that OSA adversely affects both LV and RV myocardial mechanics. The severity of these impairments correlates with the severity of OSA. By employing 2D-STE to assess LV and RV strain, clinicians can identify subtle cardiac alterations in OSA patients before traditional echocardiographic measures or the onset of heart failure. Early detection enables timely intervention and management strategies to address OSA-related cardiac complications.

Keywords: Obstructive sleep apnea, Strain, Speckle tracking echocardiography, Right ventricle, Left ventricle.

INTRODUCTION

Obstructive sleep apnea (OSA) is a rising health concern linked to various cardiovascular complications, including left and right ventricular dysfunction, hypertension, coronary artery disease, heart failure, and arrhythmias [1]. Recurring episodes of hypoxia associated with OSA can disrupt the delicate balance between myocardial oxygen demand and supply, potentially leading to subtle impairments in systolic function [2].

OSA diagnosis typically involves polysomnography or ambulatory cardiorespiratory monitoring. The severity of OSA is categorized based on the apnea-hypopnea index (AHI), with mild OSA defined as an AHI of 5-14, moderate OSA as 15-29, and severe OSA as 30 or more [3]. Speckle tracking echocardiography (STE) is a non-invasive imaging technique that enables the assessment of myocardial mechanics. STE can evaluate both global and regional myocardial strain without the limitations associated with Doppler echocardiography [4]. STE provides valuable insights into regional and global ventricular function, with reduced inter- and intra-observer variability in assessing regional left ventricular function. Key applications of STE include the evaluation of myocardial mechanics, ischemic heart disease, cardiomyopathies, and left ventricular diastolic dysfunction [5]. Global longitudinal strain (GLS) is a measure of myocardial shortening along the long axis of the heart. GLS is sensitive to subtle changes in myocardial function, particularly in the subendocardial layers, which are susceptible to hypoxic injury [6]. Circumferential strain measures systolic shortening in the short axis of the heart, while radial strain assesses myocardial thickening from the endocardium to the epicardium [7].

PATIENTS AND METHODS

Study population: Forty-five patients diagnosed with definitive obstructive sleep apnea (OSA), confirmed by polysomnography were enrolled in the study. These patients were split into 3 groups with accordance to their apnea-hypopnea index (AHI): Mild (AHI 5-14), moderate (AHI 15-29), and severe (AHI $\geq$ 30) [3]. A control group of 45 age- and sex-matched healthy volunteers was also included.

Exclusion criteria: Patients with congestive heart failure, a history of coronary artery disease, moderate or severe valvular heart disease, chronic obstructive pulmonary disease (COPD), and arrhythmias (frequent premature beats, atrial fibrillation, and atrial flutter). Also, myocardial & pericardial diseases, poor echogenic window, advanced hepatic or renal disease, decompensated or unstable cardiopulmonary diseases, cor-pulmonale and sarcoidosis or history of pulmonary embolism.

Patients with OSA were selected from Sleep Laboratory of Chest Department through the period from October 2022 to December 2023. The control group comprised healthy volunteers who met the study's inclusion criteria for normal cardiac structure and

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function and reported no symptoms of night snoring or daytime sleepiness.

Study protocol: Patients who met the specified criteria were chosen, and their baseline demographic information was recorded. A detailed clinical history was obtained from all participants, along with a physical examination. Information collected included cardiovascular risk factors, symptoms of obstructive sleep apnea and heart failure. Measurements such as body weight, height and neck circumference were taken. All patients were investigated by twelve-lead surface ECG, plain chest X-ray, routine laboratory investigations in the form of complete blood picture, kidney function tests, liver function tests and blood glucose test. Additionally, all patients underwent detailed 2D transthoracic echocardiographic examination with the patient supine or in left lateral position, connected to a single lead ECG.

Polysomnography (PSG): Patients underwent full-night polysomnography using Philips Respironics Alice6 LDXS (Philips, Cambridge, Massachusetts, USA). Overnight PSG was carried out in the sleep laboratory of Menoufia University Hospital. The PSG involved recording of various physiological measurements such as brain activity, muscle movement, heart activity, and breathing patterns throughout the entire sleep duration. Special sensors were used to continuously monitor airflow and oxygen levels. Sleep parameters were measured according to specific criteria set by the American Academy of Sleep Medicine [8]. For example, apnea was defined as a complete pause in breathing for at least ten seconds, while hypopnea was characterized by shallow breathing accompanied by reduced oxygen levels or patient arousal. The diagnosis of OSA was determined according to the number of apnea and/otherwise hypopnea occurrences per hour of sleep, as mild (AHI from 5 to14), moderate (AHI 15 to 29) and severe (AHI $\geq$ 30) [3].

Conventional transthoracic echocardiography: Echocardiographic data were collected using a Vivid 9 ultrasound system (GE Vingmed, Norway) outfitted with a harmonic M5S variable-frequency phased-array transducer (1.7-4 MHz). Standard echocardiographic views were obtained from apical and parasternal windows using 2D, M-mode, color Doppler, and pulsed-wave Doppler techniques. Cardiac chamber dimensions and functionalities were assessed following the guidelines established by the American Society of Echocardiography.

M-mode from the parasternal long-axis image was used to compute the left ventricular ejection fraction (LVEF). In the apical four-chamber view, pulsed-wave Doppler facilitated the measurement of early (E) and late (A) diastolic filling velocities at the mitral valve annulus, with the E/A ratio computed as a conventional indicator of left ventricular filling. Systolic (S'), early diastolic (E'), and late diastolic (A') velocities at the septal mitral annulus were assessed using tissue Doppler imaging, once more in the apical four-chamber view. These observations were used to calculate the mitral E/E' ratio.

2D Speckle tracking echocardiography: For the examination of myocardial strain, one cardiac cycle was chosen from both the apical and short-axis views. Global longitudinal, radial, circumferential, and right ventricular strain were measured using two-dimensional speckle tracking imaging. The endocardial border was traced manually, frame-by-frame, and software divided the region of interest encompassing the myocardium into segments for further speckle tracking analysis. Image analysis was conducted offline with the help of custom analysis software (Echopac PC, Version 1.8.1.X, GE Healthcare).

Ethical approval: Written informed consent was obtained from each participant, and the study was authorized by The Ethics Committee of Menoufia University. The study was registered with international review board (IRB) (approval number: 6/2022 CARD 45). The study adhered to the Helsinki Declaration throughout its execution.

Statistical Analysis

Statistical evaluations were carried out using the Statistical Package for the Social Sciences (SPSS) version 25 (SPSS Inc., Chicago, IL, USA) as well as Microsoft Excel 2019. Quantitative data were presented as means ± standard deviation, while qualitative data were expressed as frequencies and percentages. Categorical variables underwent analysis via Chi-square tests (including Fisher's exact test or Monte Carlo simulation). The Kruskall-Wallis H test, serving as a non-parametric alternative to ANOVA, compared multiple groups for non-normally distributed quantitative variables. Appropriate statistical methods, including Student's t-test and regression analysis, were also utilized. The threshold for statistical significance was established at a p-value of ≤ 0.05.

RESULTS

This case control study was conducted on 45 cases of confirmed OSA patients fulfilling our inclusion criteria and 45 age- and gender-matched control case. The study groups were carefully matched for age and sex. Analysis revealed significant differences in body mass index (BMI) and neck circumference, both of which were elevated in the OSA group relative to the control group.

Furthermore, the OSA group demonstrated a higher smoking prevalence compared to the control group ($p < 0.001$). Only 9 cases of OSA group had DM and or hypertension, on the other side, none of the control group cases had DM or HTN (Table 1).

Table (1): Demographic data with the risk factors between all groups

	OSA n=45 (Mean ± S.D)	Control n=45 (Mean ± S.D)	T	P value
Demographic data				
Age(years)	36.99 ± 3.63	34.98±3.74	0.954	0.079
Gender			X2	P value
Male n=58 No. (%)	33(73.3%)	25 (55.6%)	3.103	0.123
Female n=32 No. (%)	12 (26.7%)	20 (44.4%)		
Neck circumference (cm)	39.55 ± 4.65	35.11±2.38	4.780	0.007*
BMI (Kg/m2)	33.88 ± 4.65	28.89±3.77	6.160	0.001**
Risk Factors				
Diabetes No. (%)	9 (20%)	0 (0%)	-	-
Hypertension No. (%)	9 (20%)	0 (0%)	-	-
Smoking No. (%)	13 (28.9%)	2 (4.4%)	FE= 26.879	<0.001**

FE: Fisher-Freeman-Halton Exact test Pearson's Chi-square test ($\chi 2$) **T**: Student t test *: Significant **: highly significant.

Regarding 2D transthoracic echocardiographic data, there was insignificant differences between OSA group and control group regarding EF, LV dimensions measured by M-mode in parasternal long axis view and represented as LVIDd, LVIDs, IVSd and LVPWd. Also, there was no significant difference in aortic and left atrial diameter, Avg MAPSE and TAPSE. Nevertheless, there was a substantial difference between the 2 studied groups regarding ESPAP with higher pulmonary artery pressure in OSA group compared to control group. As regards LV filling parameters obtained from conventional and tissue Doppler and represented as E/A and E/e. OSA group had higher LV filling compared to control group with lower " E/A" and higher "E/e". Additionally, patients with obstructive sleep apnea had significantly lower strain values compared to control group. This included various components of LV strain, such as longitudinal, circumferential and radial strains, also RV longitudinal strain (Table 2).

Table (2): Comparison between OSA group and control group regarding 2D conventional echocardiography data and strain values

	OSA group=45 (Mean ± S.D)	**Control group=45 (Mean ± S.D)**	**Student t test**	**P value**
LVIDd (mm)	48.09 ± 12.7	50.80 ± 2.27	1.344	0.079
LVIDs(mm)	29.96± 3.73	30.67 ±3.29	0.587	0.558
IVSd(mm)	9.13 ± 0.87	8.78 ± 0.59	2.960	0.056
LVPWd (mm)	9.44 ± 3.47	8.96 ± 0.71	1.707	0.080
EF(%)	65.26 ± 10.03	67.47±2.12	1.520	0.082
Aorta(mm)	31.68 ± 3.72	33.17 ± 2.22	1.160	0.067
LA diameter (mm)	37.89± 7.59	37.73 ± 1.41	0.804	0.424
ESPAP (mmHg)	30.53± 2.33	25.42±2.12	7.415	0.001**
TAPSE (cm)	2.42 ± 0.24	2.424± 0.215	0.049	0.961
Avg MAPSE (cm)	1.5± 0.1	1.704 ±0.099	1.320	0.570
E/A	1.01 ± 0.21	1.345 ± 0.131	9.162	0.001**
E/e	10.41± 0.74	7.78±0.12	6.876	0.009*
LV strain				
LV-GLS %	-19.1± 2.46	-21.08 ± 1.09	2.555	0.041*
LV circumferential strain %	-17.43± 0.73	-22.04± 1.02	21.858	<0.001**
LV radial Strain %	37.17± 5.0	43.26±3.57	6.053	0.001**
RV strain				
RV-GLS %	-19.34± 1.96	-21.16± 0.89	6.448	0.001**

IVSd: interventricular septum during diastole**, LVIDd**: left ventricle internal diameter in diastole, **LVIDs**: left ventricle internal diameter in systole**, LVPWd**: left ventricular posterior wall thickness during diastole ,**EF %**: ejection fraction ,**LA diameter**: left atrium diameter ,**ESPAP**: the estimated systolic pulmonary artery pressure, **TAPSE**: tricuspid annular plane systolic excursion, **Avg MAPSE**: average of mitral annular plane systolic excursion. **LV-GLS**: left ventricular global longitudinal strain, **RV-GLS**: right ventricular global longitudinal strain.

The 45 patients in OSA group were further subdivided into three groups according to apnea-hypopnea index and polysomnography results. Of those 45 patients, 12 patients (representing about 26.6% of patients) had mild degree of OSA, 9 patients had moderate degree of OSA representing about 20% of patients however, 24 patients had severe degree of OSA representing about 53.4% of patients. Demographic data and risk factor analysis revealed that the study groups were well-matched for age and sex. Patients with severe OSA exhibited a higher prevalence of diabetes mellitus, hypertension, and smoking compared to those with mild or moderate OSA. Additionally, a statistically significant difference in BMI was observed across the groups (P=0.003), with the severe OSA group having the highest BMI and the mild group the lowest. There was no discernible variation in BMI between the groups with mild and moderate OSA. Regarding neck circumference, both moderate and severe OSA groups had larger neck circumferences compared to the mild group, with no significant difference between the moderate and severe groups (Table 3).

Table (3): Comparison between OSA subgroups regarding demographic data and risk factors

	OSA n=45			**H**	**P value**	**Post hoc**
	Mild=12	**Mod=9**	**Sever=24**			
Demographic data						
Age (years) Mean ± SD	35.58±3.68	38.56±3.47	36.83±3.74	2.630	0.75	
Gender **Male (n=33)** No (%) **Female (n=12)** No (%)	8(66.7%) 4(33.3%)	7(77.8%) 2(22.2%)	18(75%) 6(25%)	FE= 2.251	0.960	
Neck circumference (cm) Mean± SD	36.58±5.21	40.44±4.98	41.63±3.77	41.855	≤0.001**	*P1 = 0.015** *P2= 0.002*** *P3 = 0.395*
BMI (kg/m2) Mean± SD	32.01±3.79	32.39±5.18	37.25±4.98	12.500	0.003**	P1 = 0.842 *P2 ≤0.001*** *P3 = 0.003***
Risk factors						
Diabetes No (%)	2 (11%)	2 (22.2%)	6 (25%)	8.670	0.017**	*P1 = 0.882* *P2 = 0.012** *P3 = 0.013**
Hypertension No (%)	1 (8.3%)	1 (11.1%)	7 (29.2%)	14.808	0.003**	*P1 = 0.960* *P2 = 0.023** *P3 = 0.021**
Smoking No (%)	1 (8.3%)	3 (33.3%)	9 (37.5%)	1.39	0.002**	*P1 = 0.537* *P2 = 0.007** *P3 = 0.013**

P1 = mild group vs moderate group P2 = mild group vs severe group P3 = moderate group vs severe group
H: Kruskall-Wallis H *: Significant **: highly Significant.

Regarding conventional echocardiographic data in OSA subgroups, the findings revealed statistically non-significant variances among the studied groups regarding LV dimensions and function. Also, there was neither significant increase nor decrease in aortic and left atrium diameter, ESPAP, TAPSE and avg MAPSE. Additionally, as regards LV filling pressure parameters, there was statically significant difference in" E/A" across all groups with the lowest value in severe and moderate groups. "E/e" had the highest value in severe group followed by moderate group. Severe group had the highest LV filling pressure compared to other groups. Regarding LV strain, there were significant differences in the values of (apical 4-chamber, apical 3-chamber, apical 2-chamber longitudinal strains, LV global longitudinal and circumferential and radial strain). Severe in addition to moderate groups had lower values as compared to mild group. This also applies to RV longitudinal strain with the lowest strain value in severe group (Table 4).

Table (4): Comparison between OSA subgroups according to 2D conventional echocardiographic data and strain values

	OSA n=45 (Mean± SD)			H	P value	Post hoc
	Mild=12	Mod=9	Sever=24			
LVIDd (mm)	47.33±2.9	47.78±5.6	49.17 ± 4.2	4.031	0.298	
LVIDs(mm)	28.42 ± 3.9	30.44 ±3.4	31.04 ±3.9	5.558	0.135	
IVSd(mm)	9 ± 0.95	8.78 ± 0.67	9.63 ± 1.01	3.044	0.729	
LVPWd (mm)	9 ± 1.07	9.11 ± 1.1	10.21 ± 1.3	1.769	0.955	
EF (%)	65.03±3.33	66.87±2.6	63.88±4.1	6.213	0.061	
Aortic diameter(mm)	31.08± 4.188	31.89 ± 4.1	32.08 ± 2.9	8.820	0. 320	
LA diameter (mm)	36.75± 2.63	38.11 ± 4.1	38.83 ± 2.6	4.577	0.205	
EPASP (mmHg)	29.67±0.0	31.89 ± 2.6	30.04 ± 4.4	3.347	0.670	
TAPSE (cm)	2.4±0.23	2.46 ± 0.3	2.4 ± 0.2	0.556	0.906	
MAPSE (Avg) (cm)	1.60 ± 0.1	1.55 ± 0.1	1.50 ± 0.1	0.929	0.914	
E/A	1.15± 0.22	0.99±0.18	0.90 ± 0.23	30.872	≤0.001**	P1 <0.001* P2= 0.002* P3 = 0.188
E/e	7.91±0.42	9.60±1.19	13.74±0.62	26.109	≤0.001**	*P1 = 0.554* *P2= 0.017** *P3= 0.250*
LV strain						
LV-GLS %	-21.4±1.9	-18.2 ± 2.9	-17.9 ± 2.6	27.057	≤0.001**	*P1 < 0.001** *P2< 0.001** *P3 = 0.691*
Radial strain%	41.8 ± 4.4	34.94 ± 6.5	34.78 ± 4.1	38.194	< 0.001*	*P1 = 0.001** *P2 < 0.001** *P3 = 0.930*
Circumferential Strain%	-20.2 ± 0.6	-16.1 ± 0.6	-16 ± 1	73.102	< 0.001*	*P1 < 0.001** *P2 < 0.001** *P3 = 0.829*
RV strain						
RV-GLS%	-20.22 ± 1.8	-19.6 ± 2.4	-18.2 ± 1.7	36.911	< 0.001*	*P1 = 0.306* *P2 < 0.001** *P3= 0.018**

IVSd: interventricular septum during diastole**, LVIDd**: left ventricle internal diameter in diastole, **LVIDs**: left ventricle internal diameter in systole**, LVPWd**: left ventricular posterior wall thickness during diastole ,**EF %**: ejection fraction ,**LA diameter**: left atrium diameter ,**ESPAP**: the estimated systolic pulmonary artery pressure, **TAPSE**: tricuspid annular plane systolic excursion, **Avg MAPSE**: average of mitral annular plane systolic excursion. **LV-GLS**: left ventricular global longitudinal strain, **RV-GLS**: right ventricular global longitudinal strain.

In univariate as well as multivariate logistic regression analyses parameters that impact LV and RV strain values. Regarding LV-GLS, in the univariate analysis, BMI showed a p-value of 0.013, with odds ratios greater than 1, indicating a statistically significant association with impaired LV-GLS. Also, apnea-hypopnea index (AHI), reflecting OSA severity, revealed a p-value of 0.032 for moderate and 0.002 for severe OSA groups, and with odds ratio greater than 1. Conversely in the multivariate analysis, apnea-hypopnea index (AHI) for both moderate and severe OSA continued to show statistical significance, suggesting that AHI is an independent risk factor for impaired LV-GLS in patients with OSA. Regarding RV-GLS, in the univariate analysis, BMI showed a p-value of 0.050, with odds ratios greater than 1, indicating a statistically significant association with impaired RV-GLS. Also, the apnea-hypopnea index (AHI), revealed a p-value of 0.001 for the severe OSA group, also with odds ratios greater than 1. In the multivariate analysis, factors such as BMI and AHI continued to be significant suggesting they are strong independent risk factors for impaired RV-GLS in OSA patients (Table 5).

Table (5): Logistic regression analysis for the variables affecting strain

	Logistic regression analysis for the variables affecting LV-GLS.				**Logistic regression analysis for the variables affecting RV-GLS.**			
	Univariate		**#Multivariate**		**Univariate**		**#Multivariate**	
	p	**OR (LL – UL 95%C.I)**	**P**	**OR (LL – UL 95%C.I)**	**p**	**OR (LL – UL 95%C.I)**	**p**	**OR (LL – UL 95%C.I)**
Age (years)	**0.543**	0.066(0.135-1.533)			**0.336**	0.027(0.150-0.420)		
gender	0.117	0.152(1.16-2.88)			0.870	0.084(0.006-0.100)		
Neck circumference	**0.414**	0.252 (0.152-0.732)			**0.697**	0.031 (0.310-0.734)		
DM	**0.979**	0.069(0.123-1.240)			**0.357**	0.625(0.124-0.253)		
HTN	**0.673**	0.147(0.112-0.880)			**0.195**	0.916(0.115-0.234)		
BMI (kg/m2)	**0.013***	2.306(1.67-2.95)	0.158	0.261 (0.020-0.154)	**0.050***	1.901(0.543-1.276)	0.043*	1.197 (0.113-0.845)
Smoking	**0.845**	0.312(0.154-0.760)			**0.288**	0.438(0.143-0.541)		
Degree of OSA (AHI)								
Mild	**0.810**	0.015(0.232-0.411)			**0.91**	0.030(0.238-0.962)		
Moderate	**0.032***	1.49(0.852-1.99)	0.041*	1.080 (0.365-1.744)	**0.160**	0.560(0.169-1.353)		
Severe	**0.002***	1.88(0.112-0.83)	0.019*	0.954 (1.126-1.799)	**0.001***	3.670(0.025-1.982)	0.025*	1.46 (0.932-1.142)

OR: Odd`s ratio **C.I:** Confidence interval, **LL:** Lower limit , **UL:** Upper Limit, **#:** All variables with p<0.05 was included in the multivariate , Statistically significant at $p \leq 0.05$.

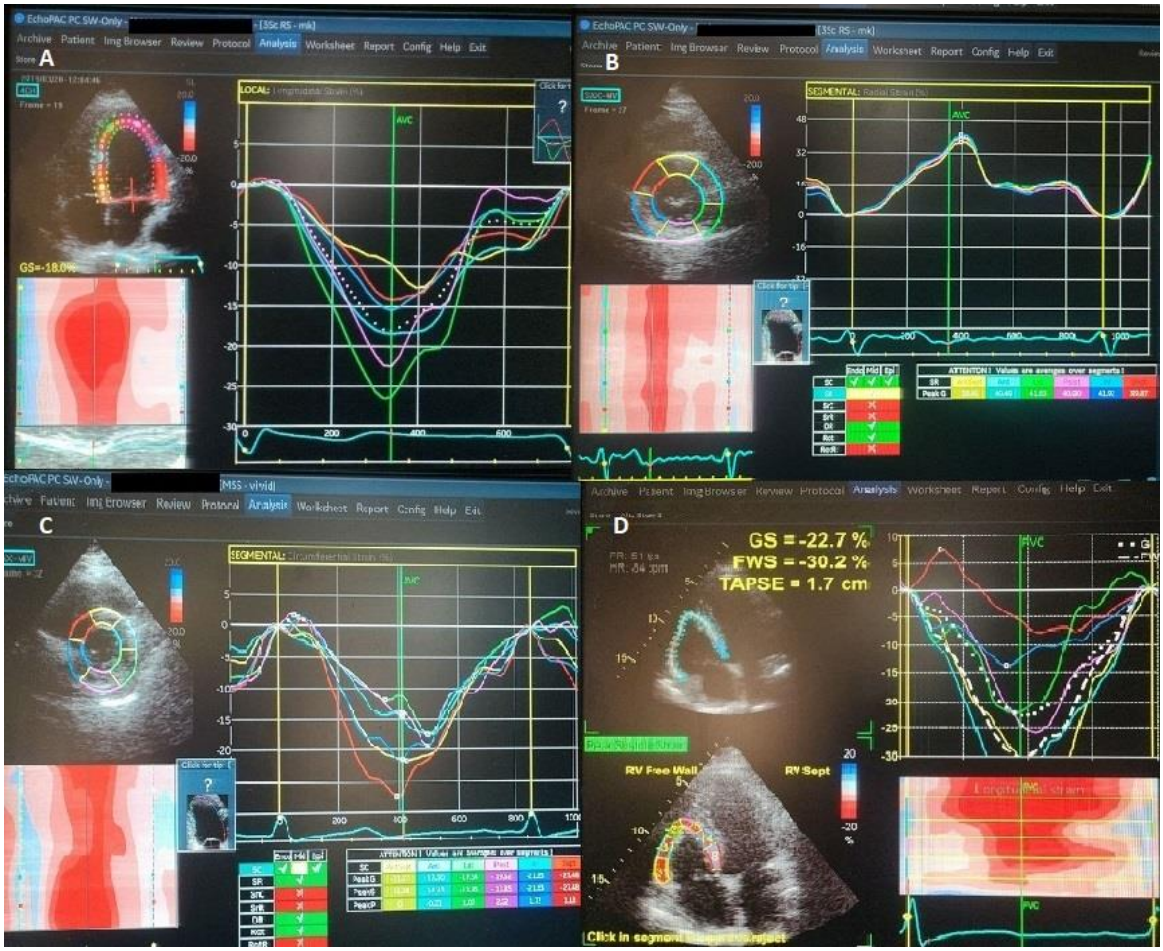

Figure (1): Demonstrating strain measurement by 2D speckle tracking echocardiography. (a) LV longitudinal strain (4-chamber view), (b) radial strain, (c) circumferential strain, (d) RV-GLS. LV: Left ventricle, RV-GS: Right ventricle global longitudinal stain.

DISCUSSION

Obstructive sleep apnea (OSA) has emerged as a significant factor contributing to cardiovascular morbidity globally. While, the link between OSA and cardiovascular issues is well-documented, the depth of its effects remains an active area of research [11].

OSA impacts cardiac mechanics through multiple pathways. The recurrent obstruction of the upper respiratory tract during sleep results in hypoxia, hypercapnia, and fluctuations in intrathoracic pressure, which can activate pathophysiological processes such as sympathetic nervous system overactivity, oxidative stress, systemic inflammation, increased blood coagulability, and endothelial dysfunction. These alterations may adversely affect myocardial structure and function. Timely diagnosis and treatment of OSA are crucial for mitigating cardiovascular risks and enhancing overall health outcomes [12].

The primary objective of our study was to assess left ventricular (LV) and right ventricular (RV) functions in patients with OSA through two-dimensional speckle tracking echocardiography (2D-STE). The research involved 45 individuals with a confirmed diagnosis of OSA and 45 age- and gender-matched controls.

We analyzed several risk factors, including diabetes mellitus (DM), hypertension (HTN), body mass index (BMI), neck circumference, and smoking habits. Our findings indicated a higher prevalence of these risk factors among OSA patients compared to controls. This aligns with research by **Loredo *et al.*** [13] who found that a significant percentage of OSA patients experienced nocturnal hypertension. Additionally, **Walia *et al.*** [10] demonstrated that severe untreated OSA was linked to elevated blood pressure, even among patients receiving intensive antihypertensive treatment, suggesting that untreated OSA worsens blood pressure management despite aggressive medication regimens.

Our study confirmed a notable prevalence of DM within the OSA group, which is consistent with the work of **Vale *et al.*** [14] who observed a high incidence of OSA among both type 1 and type 2 diabetes patients. Their research indicated that many of the diabetes patients involved had OSA.

Neck circumference measurements were higher in both severe and moderate OSA patients compared to those with mild disease and control subjects. This observation supports findings by **Katz *et al.*** [15], who suggested that neck circumference and obesity serve as significant predictors of sleep apnea.

In terms of two-dimensional echocardiography results, we haven't spot statistically significant variations in LV dimensions and performance across the studied groups. **Wang *et al.*** [16] reported similar findings, indicating no notable differences in LV global systolic function among those with varying severity of OSA. **Dursunoglu's** [17] research noted normal LV ejection fraction among OSA patients but highlighted an increased left atrial diameter in individuals with severe OSA compared to those with milder forms.

Our results designated that the severe OSA group exhibited higher LV filling pressures than the mild and control groups. **Chen *et al.*** [18] corroborated this by demonstrating a reduced E/A ratio among patients with moderate to severe OSA who maintained preserved LV systolic function. Further, **Varghese *et al.*** [19] exposed that those patients who reported extreme OSA had reduced e' velocities relative to the controls. Furthermore, E/e was considerably higher in the OSA group than in the control group, according to **Kim *et al***.[20].

A significant change regarding the estimated pulmonary artery systolic pressure (ESPAP) was identified between OSA patients and controls, which aligns with **Arias *et al***. [21] who indicated higher pulmonary artery pressures in individuals with OSA.

Research using tissue Doppler imaging (TDI) showed that OSA can manifest as subclinical LV dysfunction [22]. However, TDI values are susceptible to the angle of the ultrasound beam, making comprehensive assessments challenging. The development of 2D-STE has enabled more accurate and reliable measurements of both global and regional myocardial strain, free from angular dependencies [23].

Our study highlighted significant differences in LV and RV strain values between groups, with the extent of dysfunction correlating with the severity of OSA. Longitudinal, circumferential, and radial strains were diminished in those with severe and moderate OSA when compared to mild cases and controls. **Zhou *et al.*** [24] also noted a significant decline in longitudinal strain in the severe OSA group, affirming a correlation between the degree of dysfunction and OSA severity. Similarly, **Altekin *et al.*** [25] reported changes to both systolic and diastolic functions that aligned with disease severity.

Although, global longitudinal strain (GLS) values decreased starting from moderate OSA, radial and circumferential strain reductions were observed only in the severe group. This suggests that the early stages of OSA may preserve ejection fraction due to increased radial systolic function. **Haruki *et al.*** [2] verified that continuous positive airway pressure (CPAP) therapy not only reduced OSA severity but also improved sleep-related longitudinal LV dysfunction. **Vitarelli *et al.*** [26] remarked that while myocardial longitudinal functions were diminished in OSA patients compared to healthy peers, LVEF remained unchanged. The combined findings from **Haruki *et al.*** [22] and **Vitarelli *et al.*** [26] suggest that apnea-hypopnea episodes during sleep particularly impact longitudinal myocardial fibers, supporting the idea of their vulnerability in the early stages of OSA. **Varghese *et al.*** [19] speculated that decreased LV longitudinal strain could result from the susceptibility of subendocardial fibers to hypoxia in OSA.

Due to the RV's complicated form and retrosternal location in the mediastinum, which make echocardiographic evaluation of the RV problematic, RV mechanics estimation remains tough. In comparison with the mild group and control patients, our investigations revealed that the RV-GLS strain values were lower in the severe and moderate OSA groups. A research by **Buonauro *et al.*** [27] demonstrated that speckle tracking in OSA can identify asymptomatic RV impairment. Even though TAPSE was normal, RV-GLS was impacted. Although RV structural characteristics and RV systolic and diastolic function parameters by 2D-echocardiography did not change between OSA and control groups **D'Andrea *et al.*** [28] found that RV global and free wall longitudinal stresses were considerably lower in OSA patients.

CONCLUSION

From our results it seems that in spite of normal LV systolic function measured by 2D conventional TTE and represented as LVEF, OSA cause subtle myocardial affection when LV function was assessed by 2D STE in terms of LV-GLS, circumferential and radial strains, also the degree of affection is proportionate to disease severity.

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