



## Echocardiographic Assessment of Left Ventricular Functions in Females with Severe Obstructive Sleep Apnea Syndrome

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### ABSTRACT

**Background:** Because obstructive sleep apnea (OSA) was once thought to be a condition exclusively affecting men, women with OSA were frequently underdiagnosed and undertreated in comparison to men. Consequently, this study aimed to study, the deleterious effect of severe obstructive sleep apnea on left ventricular functions (Systolic & Diastolic) in female patients. **Methods:** This case-control study was carried out at both Cardiology Department and chest department (sleep disorder & breathing unit), Faculty of Medicine, Zagazig University in the duration from August 2023 to February 2024. Included 32 patients were subdivided into 2 groups, Group I: Included 16 females with Severe Obstructive Sleep Apnea Syndrome as cases group. Group II: Included 16 healthy females similar gender and age as control group. **Results:** There are no statistical significant differences in demographic characters of studied groups P value ( $\geq 0.05$ ). E/e' ratio was statistically significantly higher in Group I compared to Group II. PASP was significant higher in Group I compared to Group II. There was no significant difference in EF by SIMPSON between Group I and Group II. GLS was significant less negative in Group I in compared to Group II. **Conclusion:** We found that the sever cases of OSA were associated with subtle systolic and diastolic LV dysfunction.

**Keywords:** Echocardiographic, Obstructive sleep apnea, Left ventricular, AHI.

### INTRODUCTION

The prevalent and chronic disorder known as obstructive sleep apnea (OSA) is defined by periods of complete or partial

obstruction of the upper airway, which results in frequent arousals, intermittent hypoxia and daytime sleepiness. Known risk factors for OSA include male gender, age, obesity, upper

airway structural anomalies and hormonal impacts [1]. In the past, obstructive sleep apnea (OSA) was thought to be a condition exclusive to men. Because of this, compared to males with OSA, female patients were frequently underdiagnosed and undertreated. Nonetheless, current research indicates that a number of the detrimental cardiovascular effects of OSA are more severe in women [2]. The significance of OSA in women became more acknowledgeable, as certain noteworthy gender-based variations in the condition's symptoms, diagnosis, outcomes and management; they have shorter apneas and hypopneas and a lower apnea-hypopnea index (AHI) and women typically suffer less severe OSA than men. Women are more likely to experience episodes of upper airway resistance that do not fit the criteria for apneas [3].

The Apnea-Hypopnea Index (AHI), which measures the frequency of episodes of breathing cessation (apneas) and hypopneas (events associated with diminished breathing and a drop in oxygen saturation) per hour, is the primary indicator of the severity of sleep apnea [4]. The cause, severity, and natural history of the elevated upper airway resistance determine the best course of treatment [5].

For a variety of disorders, left ventricular (LV) systolic and diastolic functions are well-established prognostic indicators of poor cardiovascular fate. Studies evaluating LV function in "pure" OSA patients (i.e., without other confounding comorbidities) are few. Small studies have reported LV dysfunction of unknown etiology in the OSA population

due to the commonly accompanying comorbidities with OSA, such as obesity, coronary artery disease and hypertension, which can independently affect LV function [6]. Individuals of open-heart surgery at risk of having heart failure. Assessing OSA as a heart failure risk factor and implementing therapeutic measures are crucial in stage A patients with chronic heart failure [7].

The most widely used and verified echocardiographic marker of systolic performance is left ventricular ejection fraction or LVEF. Due to its use as selection criterion for therapeutic trials, which form the basis of current treatment recommendations' evidence, LVEF holds a special place in the field of cardiology [8]. Speckle tracking strain imaging in two and three dimensions shows potential for measuring left ventricular function, especially in individuals with marginal LV function [9].

The prevalence of OSA in cardiovascular disorders is very high. The characteristic feature of OSA, intermittent hypoxia, results in endothelial dysfunction, oxidative stress, inflammation and sympathetic hyperactivity, all of which contribute to cardiovascular comorbidities [10]. The most dangerous side effects of obstructive sleep apnea (OSA) condition are cardiovascular disruptions. Systemic hypertension, abrupt myocardial infarction, nocturnal arrhythmias, cor pulmonale and unexpected nighttime mortality are some of these consequences [11]. This study aimed to study, the deleterious effect of severe obstructive sleep apnea on left ventricular functions (Systolic & Diastolic) in female patients.

## METHODS

After protocol approval by our Local Ethics Committee (IRB#10884-13-6-2023), this study was performed at both Cardiology Department and Chest department (sleep disorder & breathing unit), Faculty of Medicine, Zagazig University in the duration from August 2023 to February 2024. We 32 recruited all female cases with sever OSAS from sleep laboratory and have no item of exclusion criteria & recruited similar number of age matched healthy individual. All patients provided written informed consent to participate in the study. Study protocol conformed to the ethical guidelines of the Declaration of Helsinki (1975) for studies involving humans.

The study included all female patients age range 20-70 years and were diagnosed as OSA with apnea hypopnea index  $>30/h$ , which neither diabetic nor hypertensive. Group II Female Patients age range 20-70 years with apnea hypopnea index  $< 5/h$ , which neither diabetic nor hypertensive. Reported enrollment of outpatients or patients undergoing elective admission. The exclusion criteria were having elevated fasting glucose of venous whole blood  $\geq 120$  mg/dl and having the characteristic signs of diabetes. The only requirement set by the WHO for sustained blood glucose rise in an oral glucose tolerance test (OGTT) sample obtained between the 75-g glucose dose administration and two hours later is that the 2-hour venous whole blood sample must have a blood glucose level of at least 180 mg/dl [12].

Every patient underwent a complete medical history review, a comprehensive physical

examination and a 12-lead electrocardiogram (ECG) both upon admission and following symptom resolution. ECG was conducted at a paper speed of 25 mm/s and an amplification of 10 mm/mv using the ECG Comen CM 100 and the ECG Schiller AT 102. ECG was analyzed regarding heart rate, rhythm, PR interval, QRS duration and the Bazett formula-corrected QT interval. Examining Polysomnography Sleep Study (PSG) data with an emphasis on the Apnea-Hypopnea Index (AHI), which indicates the quantity of apneas and hypopneas in an hour of sleep, and evaluating OSA AHI $<5$  indicates normal severity, AHI 5–14 indicates mild, AHI 15–29 indicates moderate and AHI $\geq 30$  indicates severe.

The quantity of major oxygen desaturations per hour of sleep (a substantial oxygen desaturation was defined as one in which the SaO<sub>2</sub> dropped by more than 4% from the baseline that was immediately before it). Conventional Transthoracic Echo Doppler study VIVID E95 ultrasound system (GE Healthcare), the patient was examined in the left semi-lateral position, using the chamber views of the left parasternal long axis, short axis, apical 4, apical 5 and apical 2. The Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging was followed in the recording and calculation of various parameters. The American Society of Echocardiography's journal.

Left ventricular ejection fraction (LVEF) was measured in apical four-chamber and two-chamber views using Biplane disk assumption method. Diastolic trans-mitral flow velocities (E, A) was obtained with a pulsed-wave Doppler in an apical four-chamber view. Using tissue Doppler imaging (TDI) and the sample volume positioned at the septal and lateral annuli, the diastolic mitral annular velocities (e', a') were also assessed in the same image in order to determine the average e' [13].

#### **Left ventricular systolic function:**

Ejection fraction (EF%); Simpson's approach was used to compute the ejection % from apical 4- and 2-chamber images. End-diastolic and end-systolic endocardial borders were manually sketched on frozen two- and four-chamber sections of the apical heart to determine the end-diastolic volume (EDV) and end-systolic volume (ESV). Using the formula, the LV EF was determined [14].

It is calculated also from the formula:

$$EF = \frac{EDV - ESV}{EDV} \times 100$$

EDV

Normally it is 50- 70 %. global longitudinal strain (GLS).

#### **Tissue Doppler Echocardiography (TDE):**

To evaluate global and regional myocardial systolic as well as diastolic function. Pulsed-wave (PW) Doppler is used to estimate E velocity; this is often done in the apical 4-chamber view. For the best position of the Doppler beam's sample volume, color flow imaging is useful. During diastole, a 1-3 mm sample volume is positioned in between the tips of the mitral leaflets. Following spectral gain and wall filter configuration

improvement, mitral inflow wave patterns may be clearly recorded. Mitral inflow velocities ought to be assessed with a greater sweep speed (100mm/sec) at the end of expiration (Fig. 1). Incorrect usage of location, sample volume, or respiratory condition can result in errors [15].

Wave with an annular pulse Using a 1- to 2-mm size sample volume, Doppler tissue imaging may also be obtained from the apical 4-chamber view (Fig. 2). It is best to minimize the angle (<20°) between the ultrasound beam and the plane of heart action. It is preferable to average the e' velocity from the lateral side of the mitral annulus and the septum. Excessive Doppler gain can lead to spectrum widening, which can lead to errors. If this happens, a modal velocity measurement should be taken (see figure 2).

#### **Global Longitudinal Strain (GLS):**

The definition of longitudinal strain is the change in an object's length in a particular direction with respect to its baseline length:  $(L1-L0)/L0 = \text{Strain\%}$  [16]. The published GLS values range widely, from -15.9% to -22.1% [17]. Each of the 17 longitudinal LV segments had its peak negative systolic longitudinal strain measured; the segmental values were then averaged to produce the global longitudinal strain (GLS).

**Statistical analysis:** Using IBM SPSS Statistics for Windows, Version 23.0 (Armonk, NY: IBM Corp. 2015), all data were gathered, tabulated, and statistically evaluated. Qualitative data were presented as percentages and figures, while quantitative data were given as the mean  $\pm$  SD & median (range). Two groups of normally distributed variables were compared using the t test. Two

group variables that were not normally distributed were compared using the Mann-Whitney U test. The Pearson correlation coefficient was computed to evaluate the association among the different research variables. A statistically significant P-value was defined as one less than 0.05. Using IBM SPSS Statistics for Windows, Version 23.0 (Armonk, NY: IBM Corp. 2015), all data were gathered, tabulated, and statistically evaluated. Qualitative data were presented as percentages and figures, while quantitative data were given as the mean  $\pm$  SD & median (range).

### Results

As regard age in-group I was (ranged from 28–65 years), versus (ranged from 27–68 years) group II. This difference was statistically non-significant ( $t=1.83$ ,  $p>0.05$ ). Group I, BMI was (ranged from 30.3–51.2), while in Group II, it was (ranged from 28.2–50.2). This difference was statistically non-significant ( $t=1.98$ ,  $p>0.05$ ). In both groups; all studied females were not smokers and did not have special habit (Table 1). As regard clinical data group I, Heart rate was (ranged from 65–86 beat/minute), while in group II, it was (ranged from 68–80 beat/minute), the difference was statistically non-significant ( $t=1.03$ ,  $p>0.05$ ). Group I, systolic blood pressure was (ranged from 100–136 mmHg), while in group II, it was (ranged from 110–128 mmHg), the difference was statistically non-significant ( $t=1.42$ ,  $p>0.05$ ). Group I, diastolic blood pressure was (ranged from 65–85 Hg/mm), while in group II, it was (ranged from 72–82 mmHg), the difference was statistically non-significant ( $t=1.44$ ,  $p>0.05$ ). Logically all parameters (AHI, AI, HI, Base SPO<sub>2</sub>, Minimum SPO<sub>2</sub> and Summation of Desaturation) of obstructive sleep apnea syndrome patients were more significant

### Echocardiogram to assess left ventricular function of studied groups:

In Group I, E/e' ratio was  $11.06\pm 0.73$  (ranged from 9.5–11.7), while in Group II, it was  $7.21\pm 0.76$  (ranged from 6.1–8), E/e' ratio was statistically more in group I compared to group II, ( $t=14.68$ ,  $p<0.01$ ). In group I, E/A ratio was  $0.78\pm 0.14$  (ranged from 0.6–1), while in group II, it was  $1.22\pm 0.074$  (ranged from 1.1–1.3), E/A ratio was statistically lower in group I compared to group II ( $t=11.23$ ,  $p<0.01$ ). In group I, DT was  $203.63\pm 16.07$  (ranged from 184–222), while in group II, it was  $156.06\pm 3.89$  (ranged from 149–162), DT was statistically more in cases group compared to control group, ( $t=11.509$ ,  $p=0.0001$ ). In group I, IVRT was  $108.81\pm 3.54$  ms (ranged from 103–113), while in group II, it was  $83\pm 11.09$  ms (ranged from 70–100), IVRT was statistically more in group I compared to group II, ( $t=8.87$ ,  $p<0.01$ ). In group I, PASP was  $32.16\pm 3.53$  mmHg (ranged from 27–36.5), while in group II, it was  $15.34\pm 3.97$  mmHg (ranged from 10.5–23), PASP was statistically more in group I compared to group II, ( $t=12.66$ ,  $p<0.01$ ). In group I, EF by M-mode was  $63.69\pm 2.53$  % (ranged from 62–70), while in group II, it was  $65.12\pm 2.47$  % (ranged from 62–70), there was no statistical difference in EF by M-mode between cases and control, ( $p>0.05$ ). In group I, EF by SIMPSON was  $62.75\pm 1.47$  % (ranged from 61–65), while in group II, it was  $63.69\pm 1.7$  % (ranged from 62–69), there was no statistical difference in EF by SIMPSON between cases and control, ( $p>0.05$ ). In group I, GLS was  $-15.49\pm 1.76$  % (ranged from -17.2–-13.7), while in group II, it was  $-20\pm 1.13$  % (ranged from -22–-18), GLS was statistically less negative in group I compared to Group II, ( $t=6.64$ ,  $p<0.01$ ) (table 2).

**Table 3;** showed that there was significant and direct relation between, EF by M-mode and Base SPO2, Minimum SPO2. While there is significant and inverse relation between, EF by M-mode and Apnea Hypopnea Index, Apnea Index, Sum of Desaturation. There was significant and direct relation between, EF by SIMPSON and Base SPO2, Minimum SPO2. While there is significant and inverse relation between, EF by SIMPSON and Apnea Hypopnea Index, Apnea Index, Hypopnea Index and Sum of Desaturation.

Table 4; showed that there was significant and direct relation between, left ventricle GLS and Base SPO2, minimum SPO2. While there is significant and inverse relation between, left ventricle GLS and Apnea Hypopnea Index,

Apnea Index, Hypopnea Index and Sum of Desaturation.

Table 5; showed that there is significant and inverse relation between, E/e` and Base SPO2, minimum SPO2. While there is significant and direct relation between, E/e` and Apnea Hypopnea Index, Apnea Index, Hypopnea Index and Sum of Desaturation.

Table 6 show that left ventricle GLS at cut off level  $\leq -17.25$  show sensitivity 68.8%, specificity 100% and accuracy 84.4% to discriminate severe OSA (AHI) in females.

Table 7 show that E/e` value at cut off level  $\geq 8.8$  show sensitivity 100%, specificity 100% and accuracy 100% to discriminate severe OSA (AHI) in females.

**Table (1):** Demographic characters of studied groups:

Parameters	Group I n.16	Group II n.16	t-test	p-value
Age per years Mean $\pm$ SD (range)	54.44 $\pm$ 15.07 28-68	45.44 $\pm$ 12.69 27-65	1.83	>0.05
BMI(k/m <sup>2</sup> ) Mean $\pm$ SD (range)	43.04 $\pm$ 7.8 30.3-51.2	37.89 $\pm$ 6.92 28.2-50.2	1.98	>0.05
<b>Clinical examination</b>				
Heart rate(beat/minute) Mean $\pm$ SD (range)	77.06 $\pm$ 7.67 65-86	74.81 $\pm$ 4.1 68-80	1.03	>0.05
Systolic blood pressure(mmHg) Mean $\pm$ SD (range)	125.5 $\pm$ 11.23 100-136	121.06 $\pm$ 5.48 110-128	1.42	>0.05
Diastolic blood pressure (mmHg) Mean $\pm$ SD (range)	77.94 $\pm$ 5.87 65-85	75.56 $\pm$ 3.05 72-82	1.44	>0.05

t :student't (t)

P value  $\geq$  0.05: no significant

**Table (2):** left ventricular functions of studied groups:

Parameters	Group I n.16	Group II n.16	t-test	p-value
E/e` Mean ±SD (range)	11.06±0.73 9.5-11.7	7.21±0.76 6.1-8	14.684	<0.001 *
E/A Mean ±SD (range)	0.78±0.14 0.6-1	1.22±0.074 1.1-1.3	11.26	<0.001 *
DT Mean ±SD (range)	203.63±16.0 7 184-222	156.06±3.89 149-162	11.509	<0.001 *
IVRT Mean ±SD (range)	108.81±3.54 103-113	83±11.09 70-100	8.87	<0.001 *
PASP Mean ±SD (range)	32.16±3.53 27-36.5	15.34±3.97 10.5-23	12.66	<0.001 *
EF by M-mode Mean ±SD (range)	63.69±1.53 62-66	65.12±2.47 62-70	1.97	>0.05
EF by SIMPSON Mean ±SD (range)	62.75±1.47 61-65	63.69±1.7 62-69	1.67	>0.05
GLS Mean ±SD (range)	-15.49±1.76 -17.2— -13.7	-20±1.13 -22--18	8.81	<0.001 *

t :student't (t)      P value < 0.05: Significant.      P value < 0.01: Very Significant (\*)

**Table (3):** Correlation matrix of EF by M-mode, SIMPSON and Apnea Hypopnea Index, Apnea Index, Hypopnea Index, Base SPO2, Minimum SPO2, Sum of Desaturation, (n.32)

variables	r	p
EF by M-mode		
Apnea Hypopnea Index	-.45**	<0. 001
Apnea Index	-.51**	<0. 001
Hypopnea Index	-0.37	<0. 001
Base SPO2	.51**	<0. 001
Minimum SPO2	.55**	<0. 001
Sum Of Desaturation	-.45**	<0. 001
EF by SIMPSON		
Apnea Hypopnea Index	-.46**	<0. 001
Apnea Index	-.57**	<0. 001
Hypopnea Index	-0.41	<0. 001
Base SPO2	.46**	<0. 001
Minimum SPO2	.55**	<0. 001
Sum Of Desaturation	-.46**	<0. 001

Pearson' correlation coefficient (r) \*\* Correlation is significant at the 0.01 level (2-tailed). \* Correlation is significant at the 0.05 level (2-tailed).p>.05 no significant

**Table (4):** Correlation matrix of LV by GLS and Apnea Hypopnea Index, Apnea Index, Hypopnea Index, Base SPO2, Minimum SPO2, Sum of Desaturation, (n.32)

variables	GLS	
	r	p
Apnea Hypopnea Index	.854**	<0.001
Apnea Index	.73**	<0.001
Hypopnea Index	.55**	<0.001
Base SPO2	-.61**	<0.001
Minimum SPO2	-.90**	<0.001
Sum Of Desaturation	.84**	<0.001

**Table (5):** Correlation matrix of E/e` and Apnea Hypopnea Index, Apnea Index, Hypopnea Index, Base SPO2, Minimum SPO2, Sum of Desaturation, (n.32)

Variables	E/e`	
	r	p
Apnea Hypopnea Index	.91**	<0.001
Apnea Index	.71**	<0.001
Hypopnea Index	.66**	<0.001
Base SPO2	-.36*	<0.05
Minimum SPO2	-.79**	<0.001
Sum Of Desaturation	.98**	<0.001

Pearson' correlation coefficient (r) \*\* Correlation is significant at the 0.01 level (2-tailed).

\* Correlation is significant at the 0.05 level (2-tailed), p>.05 no significant

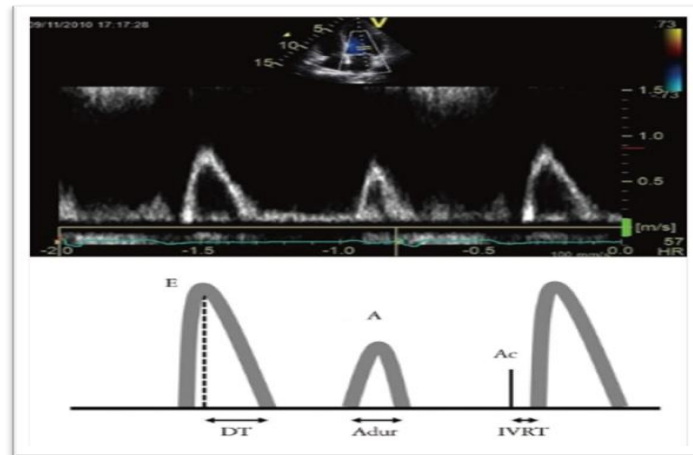
**Table (6):** Performance of LV by GLS to diagnose severe OSA (AHI) in recruited females

Cut level	off	Sensitivity	Specificity	PPV	NPV	Accuracy
GLS ≤ 17.25	-	68.8%	100.0%	100.0%	76.2%	84.4%

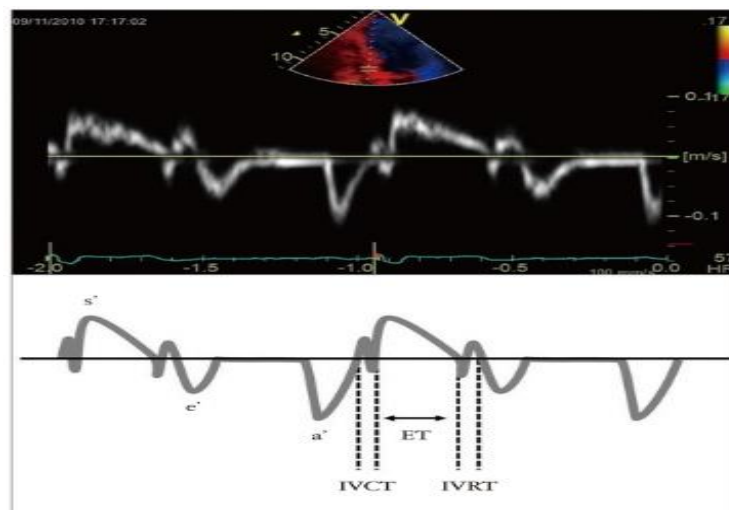
**Table (7):** Performance of E/e` to diagnose severe OSA (AHI) in recruited females

Cut off level	Sensitivity	Specificity	PPV	NPV	Accuracy
E/e` ≥ 8.8	100.0%	100.0%	100.0%	100.0%	100.0%





**Figure (1):** Mitral inflow velocities obtained by pulsed wave Doppler technique and their schematic diagram. Peak mitral inflow velocity during early diastole (E wave), peak mitral inflow velocity at atrial contraction (A wave), mitral deceleration time (DT), duration of A wave (Adur), and interval between aortic valve closure (Ac) and start of mitral inflow (IVRT) are labelled. IVRT: isovolumic relaxation t



**Figure (2):** Mitral annular velocities obtained by tissue Doppler echocardiography. s' velocity: systolic velocity, e' velocity: early diastolic velocity, a' velocity: late diastolic velocity, IVCT: isovolumic contraction time, ET: ejection time, IVRT: isovolumic relaxation time.

**DISCUSSION**

The current investigation found that the two groups under consideration did not differ statistically significantly in terms of age or BMI. This study supports the findings of D'Andrea et al. [18], who evaluated diastolic reserve and left ventricular (LV) dynamic myocardial deformation in patients with obstructive sleep apnea (OSA). They found that although there were statistically significant differences in BMI between the two groups under study, there were no statistically significant differences in age between the OSA group and the control. Comparably, our findings are in line with

Zhou et al.'s [19] assessment of dyssynchrony and right ventricular (RV) regional systolic performance in individuals who have just been diagnosed with obstructive sleep apnea using real-time three-dimensional (3D) echocardiography. According to their findings, there were statistically significant variations in BMI between the two groups under study, but not in age between the OSA group and the control group.

Varghese et al. [20], who used cutting-edge speckle tracking echocardiographic (STE) techniques to examine the subclinical systolic ventricular failure of patients with OSA, further supports our findings. They

demonstrated that the age and BMI of the OSA group and the control group did not differ statistically significantly.

Regarding the clinical examination, the current study found that heart rate, systolic and diastolic blood pressure did not differ statistically significantly between the control group and the severe OSA group. Furthermore, our data support the findings of Zhou et al. [19], who found no statistically significant variations in heart rate, systolic and diastolic blood pressure between the severe OSA group and the control group.

Similarly, our findings in line with Altekin et al, [21] who demonstrated there were no statistically significant differences between control group and severe OSA group regarding systolic and diastolic blood pressure. As well, our results in consistent with Chen et al. [22] who revealed that Heart rate did not alter statistically significantly between the OSAS group and the control systolic and diastolic blood pressure.

In our study E/e` ratio was significantly higher in group I in comparison with group II which is agreed with Altekin et al. [21] who revealed that the mean E/e' ratio in severe OSA group was significantly higher in OSA group when compared to control. E/A ratio was significantly lower in group I in comparison with group II which is agreed with, Chen et al. [22] and disagree with Varghese et al. [20] which found no significant difference regarding mitral E/A ratio, their study includes 31 patients who were newly diagnosed with sever OSA with AHI >40 with an equal matched age, sex and BMI and hypertension of both genders, while we recruited only female patients. Surprisingly Altekin et al., [21] they found lower E/A ratio in mild cases of OSA and did not found that in moderate and severe cases,

which does not agree with our study. Where the participants in this observational cross-sectional study were twenty-one healthy persons and fifty-eight OSA patients of both genders. E wave DT was significantly higher in group I compared to group II which agreed with Al-Sadawi et al. [23].

Our results showed that the mean IVRT in cases was highly significantly higher group I when compared to group II. Similarly, our results in agreement with Altekin et al. [21] who revealed that the mean isovolumic relaxation time (IVRT) in severe OSA group was highly substantially worse OSA group in comparison to control group.

Concerning PAP it was significantly higher in group I in comparison with group II in our study which it agreed with Bady et al [24] .Also, our results in consistent with D'Andrea et al. [17] who demonstrated that the mean systolic pulmonary artery pressure (SPAP) was much higher in the OSA group as compared to the control group. Also, our study in consistent with Zhou et al, [19] who illustrated that the mean SPAP in severe OSA group was notably greater in groups with severe OSA than in the control group. In contrary our study disagreed with Altekin et al [21] who found no significant differences between PAP of different degree of OSA severity which include 21 healthy individuals and 58 OSA patients of both genders because we only recruited severe cases of OSA patients.

Concerning left ventricular function, literature reports were controversial, some found that OSA severity is insignificantly correlated with a reduction in LVEF as in our work there was no significant difference between group I and group II in EF by m mode and modified Simpson which disagreed with Hammerstingl et al [25], wherein 82 patients with OSA who

required CPAP therapy were prospectively enrolled, received TTE at study inclusion, and were followed up for six months. Our study in agreement with Zhou et al [19], there were no significant difference in EF by M mode and modified Simpson. Similarly, our study can be supported by D'Andrea et al, [18] they found that the left ventricular ejection fraction did not differ statistically significantly between the OSA group and the control.

The current study revealed that the mean GLS in group I was significantly less negative in group I compared to group II. Also, our results in agreement with D'Andrea et al. [18] who showed that the GLS was considerably less negative in the OSA group than the control group, with a mean of  $-13.4 \pm 3.8\%$  in the OSA group and  $-18.4 \pm 3.3\%$  in the control group.

Similarly, our findings in line with Varghese et al. [20] who reported that the mean GLS in OSA group was  $-15 (1.8)$  GLS, in control group was  $-19 (1.6)$  and in individuals with OSA, the global longitudinal LV strain (GLS) was considerably lower. Also agreed with Zhou et al [19] Sleep apnea severity as measured by AHI was the only significant predictor of abnormal longitudinal strain in these patients.

**The limitations of the study:** Due to our limited sample size, we were probably underpowered for various assessments, especially in the subgroup analysis. We also acknowledge that there are too many confounders present for us to be able to discern between the effects of hypoxemia and severe OSA. The real length of the study individuals' severe OSA was unknown, and the study included a very limited number of patients.

## CONCLUSION

We found that the severe cases of OSA were associated with subtle systolic and diastolic LV dysfunction measured mainly by GLS and E/e'; these parameters are easy to use, broadly relevant and reproducible for estimating the global function of the left ventricle in patients with (OSA).

### Declaration of interest

The authors report no conflicts of interest. The authors along are responsible for the content and writing of the paper.

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None declared

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