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Echocardiographic Profile in Newly Diagnosed Patients with Obstructive Sleep Apnoea (OSA) and Normal LV Ejection Fraction: A Prospective Study

Anender Kaur Dhariwal [α], Prakash Sanzgiri [σ], Charan Reddy KV [ρ], Vidya Suratkal [ω] & Suresh Vijan[¥]

Abstract- OSA is considered as an independent risk factor for cardiovascular morbidity and mortality. Hypertension, atrial fibrillation, heart failure with reduced ejection fraction, stroke and metabolic syndrome are also known to be associated with OSA. Each of these conditions are associated with 2D-ECHO abnormalities and often present with increased hospitalization rates or morbidity. However, echocardiographic parameters in newly detected OSA, without any other associated illness, is poorly defined. The aim of this study is to evaluate systolic and diastolic dysfunction using 2D speckle tracking in patients with newly diagnosed OSA and normal left ventricle ejection fraction. The association between diastolic dysfunction, Global Longitudinal Strain (GLS), LV hypertrophy, LV mass, estimated pulmonary artery pressures and severity of OSA was also studied. The results indicated that most of the patients with OSA, without any other cardiovascular diseases, exhibited normal left ventricular (LV) ejection fraction (EF), but had clinical signs and symptoms of LV systolic dysfunction.

Keywords: obstructive sleep apnoea, left ventricular ejection fraction, echocardiography, systolic dysfunction.

I. Introduction

Obstructive sleep apnoea (OSA) is a common condition affecting nearly 5-15% of adult population in both developing and developed countries. Prevalence increases with age, obesity, and other chronic diseases.OSA is considered as an independent risk factor for cardiovascular morbidity and mortality. Left ventricular systolic function is generated by radial and longitudinal fibre shortening. Radial shortening is predominantly dependent on the contraction of circumferential myocardial fibres, which are more resistant to ischemia. However, longitudinal shortening is generated by both longitudinal sub-endocardial and sub-epicardial fibres, where the sub-endocardium is more vulnerable to myocardial ischaemia. Hence, assessment of LV longitudinal function is considered as a sensitive marker for early detection of the left ventricular systolic dysfunction.

A recent meta-analysis that compared left ventricular ejection fraction (LVEF) and Global longitudinal strain (GLS) in predicting major adverse cardiac events in patients with different cardiovascular diseases reported that GLS had superior prognostic value to EF for predicting all-cause mortality, cardiac death, malignant arrhythmia, hospitalization due to heart failure, urgent valve surgery or heart transplantation and acute coronary ischemic events[1]. The potential of Continuous positive airway pressure (CPAP) therapy to reverse functional and structural remodeling of the heart has been confirmed in several studies.

The present study was undertaken to evaluate early systolic and diastolic dysfunction using 2D speckle tracking in patients with newly diagnosed OSA with normal left ventricle ejection fraction. The association between diastolic dysfunction, LV hypertrophy, LV mass, estimated pulmonary artery pressure with severity of OSA, studied using Apnoea-hypopnoea Index (AHI).

II. Material and Methods

The current work is based on single centre, observational, non-randomised prospective study, which was undertaken to assess and highlight the Echocardiographic parameters in patients having Hypertension, Atrial Fibrillation, Heart failure with reduced ejection fraction, Stroke and Metabolic syndrome, which are known to be associated with OSA. Each of these medical conditions cause 2D-Echo abnormalities with high morbidity and increased hospitalization rates. However, 2D-echocardiographic parameters in newly detected OSA, without any other associated illness, is poorly defined. In this study we had selected such patients and sub-grouped them based on the severity of sleep apnoea.

a) Patients selection

Inclusion criteria: This was an observational study of 50 patients (included both inpatients and outpatients-males & females above 18 years), recently diagnosed to have Obstructive sleep apnoea by polysomnography (PSG), with rigid exclusion criteria. Patients with OSA often have co-existing disorders that are prone to diastolic dysfunction such as aging, obesity, hypertension and diabetes. Obstructive sleep apnoea (Apnoea hypopnoea index >5 episodes/hr), satisfying the inclusion and exclusion criteria were selected in study after written informed consent. Patients were graded as mild (AHI ≥5), moderate (AHI ≥15) and

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severe (AHI ≥ 30) obstructive sleep apnoea as per American academy of sleep medicine[2].

Exclusion criteria: Patients excluded in this study had central sleep apnoea, coronary artery disease or electrocardiographic changes suggestive of myocardial infarction, global LV systolic dysfunction (LVEF<50%) or a history of congestive heart failure, diabetes mellitus, moderate to severe valvular heart diseases, hypertrophic cardiomyopathy, history and clinical features of restrictive or chronic obstructive pulmonary disease or asthma, arrhythmias like atrial fibrillation, previous diagnosis of OSA and/or the previous use of continuous positive airway pressure therapy (CPAP), chronic renal impairment (serum creatinine > 112μmol/L), individuals with systemic and metabolic diseases which could adversely affect the cardiac function and cigarette smoking were excluded. Also excluded were patients <18 years, pregnant females, and those with prior surgical treatment for Obstructive sleep apnoea, and those who are unwilling or uncooperative patients.

b) Methods

Overnight fully attended PSG monitoring was performed with the Alice 4 Sleep System (Respironics Inc., Murrysville, PA, USA) using standard recording technique. All Echocardiographic examinations were performed by an experienced cardiologist who was blinded for the results of polysomnography. All measurements were performed with the subjects in the left lateral decubitus position by M-mode, two dimensional, and Doppler ultrasound echocardiography. The equipment used was Vivid I (GE healthcare, Horten, Norway). Basic measurements of left ventricular dimensions in diastole and systole, thickness of inter-ventricular septum (IVS), left ventricular posterior wall (LVPW) and LV Mass (LVM) were measured by the M-mode technique and LVM was divided with body surface area to obtain LVM index (LVMI). LVH was said to be present when the LVMI crossed the reference upper limits of 95g/m2 in females and 115g/m2 in males (2015 chamber quantification)[10].

LVEF was measured using biplane Simpson's method according to the recommendation of European Association for Echocardiography. LV Global longitudinal strain was measured using commercially available 2D strain software (EchoPAC PC, version 6.0, GE Healthcare, Horten, Norway). Those with GLS of less than -20% were labeled as low GLS and those with GLS≥-20% were labeled as normal GLS (2015 chamber quantification guidelines)[3]. LV diastolic dysfunction was evaluated according to the guidelines of the American society of Echocardiography. Right ventricle dimension (RVD) and right ventricular fractional area change (RVFAC) were also measured.

III. Discussion

Repeated episodes of hypoxia, hypercapnia, microarousals, and changes in intra-thoracic pressure, trigger pathophysiological mechanisms such as sympathetic hyperactivity, oxidative stress, systemic inflammation, hypercoagulability and endothelial dysfunction which can lead to the development of vascular disease. Hypertension, commonly seen in OSA, is the most common risk factor for LVH. However, *Hedner et al (1990)*[4] reported that OSA patients had a thicker LV wall and LV mass, and their mass index to body surface area, was approximately 15% higher among normotensive OSA patients than in normotensive control subjects. In the present study, the percentage of subjects having mild, moderate and severe OSA were 38%, 30% & 32% respectively *(Table-1)*. Amongst the subjects with mild, moderate and severe OSA, the percentage of LVH was 10.5%, 60% and 93.8% respectively. A statistically significant association between OSA and LVH was observed *(Table-2).* Recurrent episodes of hypoxaemia and increased sympathetic activity observed during OSA would also contribute to development of LVH in patients with OSA, and would correlate with severity, duration of OSA and degree of hypoxemia.

Wachter group (2013)[5] reported that moderate-to-severe OSA is independently associated with diastolic dysfunction. The prevalence of diastolic dysfunction in their study increased with the severity of sleep apnoea from 44.8% (none) to 56.8% (mild) to 69.7% (moderate-to-severe sleep apnoea) (p-0.002). The degree of diastolic dysfunction also increased with sleep apnoea severity. e' was significantly reduced in OSA and E/e' was significantly increased with increasing severity of OSA5. Similar pattern was noted in our study. We observed 42.1% of cases with mild OSA, 73.3% with moderate OSA & 75.1% (grade I diastolic dysfunction-43.8%, grade II- 31.3%)of cases with severe OSA had diastolic dysfunction. There was a significant association (p-0.0034) between grades of OSA and diastolic dysfunction. AHI was the only significant predictor of diastolic dysfunction in our study group; more the AHI, more likely is the patient to have diastolic dysfunction (*Table-5).*

The percentage of subjects with mild, moderate and severe OSA having PAH were 5.3%, 20% and 43.8% respectively. There was a statistically significant association (p-0.035) between grades of OSA and PAH. Patients with severe OSA had greater predisposition to having pulmonary hypertension on first detection of OSA. However all of them had normal RV function *(Table-4).* In our study, the estimated LVEF was no different in all subgroups of OSA. LV hypertrophy, LV mass and left ventricular mass index (LVMI) was increased in moderate and severe OSA groups. GLS was statistically abnormal in moderate and severe OSA

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groups. Mean Pulmonary artery pressure (mPAP) was significantly increased in moderate and severe OSA sub-groups *(Table-3).* Systolic LV function is commonly estimated by assessing LV ejection fraction. But fall of LVEF is a rather late echocardiographic finding. This is due to the fact that normal value of LVEF does not always imply normal LV systolic function. On the other hand, diastolic function is often impaired in OSA. Myocardial ischemia and oxidative stress are the pathophysiological explanations of these disturbances.

The recent development of the 2D-STE (speckle-tracking echocardiography) enables accurate and reliable measurements of both the global and regional myocardial strain and strain rates. The limitations of EF in assessing systolic function and predicting prognosis in the context of LV hypertrophy (or increased LV wall thickness) are well recognized. STE has gained increasing clinical popularity in this setting as a means of identifying early, subtle systolic dysfunction in the context of normal LVEF, aiding diagnosis of rarer causes of LV hypertrophy, such as hypertrophic cardiomyopathy (HCM) or cardiac amyloidosis (CA).It can also be used to predict and assess short and long term prognosis with the study of global longitudinal strain (GLS), which is an accurate echocardiographic method of early LV dysfunction[6]. 50% of OSA cases had low GLS (GLS< -20%) and 50% had normal GLS (GLS≥ -20%).

Estimated LVEF was no different in all subgroups of OSA. LV hypertrophy, LV mass and left ventricular mass index (LVMI) was increased in moderate and severe OSA groups. GLS was statistically abnormal in moderate and severe OSA groups. Mean mPAP was significantly increased in moderate to severe OSA subgroups (*Table-3*). None of the subjects with mild OSA had low GLS, while 60% of subjects with moderate OSA and all 100% of subjects with severe OSA had low GLS. There was a significant association (p-1.86E-08) between grades of OSA and low GLS *(Table-6). Altekin et al (2012)*[7] used 2D-speckle tracking echocardiography (2D-STE) to evaluate subclinical LV systolic dysfunction in patients with OSA patients with preserved LVEF and without any confounding diseases that may result myocardial dysfunction. In their study, the mean GLS values for mild, moderate & severe OSA patients were -25.3±-1.67, -20.22±-2.4 & -16.62±-2.48 respectively, almost similar to our findings[7,8]. In moderate OSA patients, the GLS values decreased with the severity of the disease.

Our study showed that decreased longitudinal systolic deformation occurs early in OSA patients despite normal LVEF and that Longitudinal systolic deformation is strongly associated with the severity of OSA, with AHI being a independent predictor of GLS. The possible explanation for this is that the apnoea-hypopnea periods affect the sub-endocardially located longitudinal fibres thereby, increasing LV wall tension and preload caused by the OSA leading to LV longitudinal systolic dysfunction. In order to decrease LV wall tension and protect myocardial function, LV hypertrophy and remodeling develops as compensatory mechanisms. As the LV hypertrophy and concentric remodeling progresses, the sub-endocardial myocardial layer responsible for the longitudinal shortening becomes more susceptible to ischemic apoptosis and fibrous transformation, resulting in reduced LV longitudinal shortening in the early stages of the OSA. This conclusion also supports the theory that the longitudinal fibres are affected in the early stages of OSA, as they are sub-endocardially located & are more susceptible to myocardial ischemia caused by the recurrent apnoea-hypopnea episodes of the OSA. Haruki et al have shown that after effective CPAP therapy for a period of 3 months, AHI and minimal oxygen saturation were significantly improved, with an elimination of sleep-induced GLS abnormality in OSA patients[9].

Pulmonary hypertension (PH) in OSA is often overlooked.PH secondary to OSA is usually mild to moderate. 27%–30% of patients with OSA without left ventricular dysfunction or hypoxemic lung disease have PH. It was previously considered that PH is a manifestation of long standing OSA. However, our study has demonstrated that PH can be present at the first detection of OSA, and is directly related to severity of OSA. OSA is associated with a higher mortality among patients with PH than without PH. In presence of PH, treatment modality specific to OSA associated with PH should be planned. The possible coexistence of other conditions (pulmonary parenchymal disease, Mitral regurgitation, auto-immune disease, obesity hypoventilation syndrome, chronic pulmonary thromboembolism), which worsen PH, should be considered as it requires a different management strategy. The majority of patients with OSA experience cyclical oxygen desaturation during sleep. These episodes can last from few seconds occurring several times an hour followed by arousals with complete or partial recovery of oxygen saturation. This cumulative effect of intermittent hypoxia can lead to PH[10].The findings in our study can be summarized as follows:

1). Left ventricular hypertrophy (LVH) was present in 52% (26 out of 50) patients. There was a significant association between various grades of OSA and LVMI. The LVMI was higher in subjects with moderate and severe OSA as compared to mild OSA, albeit the difference was not significant. 2)62% of newly diagnosed OSA patients in our study had diastolic dysfunction (grade I diastolic dysfunction -52% and grade II diastolic dysfunction- 10%), of which 67.74% had LVH and 54.84% had history of hypertension. 3).The prevalence of diastolic dysfunction increased with the severity of OSA. There was a statistically significant

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association (p- 0.0034) between severity of OSA and diastolic dysfunction. AHI was the only significant predictor of diastolic dysfunction; more the AHI, more likely is the patient to have diastolic dysfunction. 4).Apnoea hypopnoea index was found to be a significant predictor of GLS. None of the subjects with mild OSA had low GLS, while 60% of subjects with moderate OSA and all 100% of subjects with severe OSA had low GLS. There was a statistically significant association (p-1.86E-08) between grades of OSA and low GLS. Thus, a decreased longitudinal systolic deformation (measured as GLS) occurs early in OSA patients despite normal LVEF and that longitudinal systolic deformation is significantly associated with the severity of OSA, with AHI being a significant predictor of GLS. 5) The percentage of subjects with mild, moderate and severe OSA having PAH were 5.3%, 20% and 43.8% respectively. There was a statistically significant association (p-0.035) between grades of OSA and PAH. Patients with severe OSA had greater predisposition to having pulmonary hypertension on first detection of OSA. However all of them had normal RV function.

IV. Conclusion

All newly diagnosed patients with OSA should undergo detailed echocardiographic evaluation to see diastolic function and strain imaging should also be performed in them even if have a normal LV ejection fraction. New cases of OSA patients without clinically diagnosed cardiovascular diseases usually present with early signs of cardiac hypertrophy, LV diastolic or LV systolic dysfunction as seen by abnormal GLS and pulmonary artery pressures. The severity of the OSA also appears to play a major role in LV re-modeling. Hence, early therapeutic interventions can be undertaken to reverse many of abnormalities like LVH, diastolic and systolic dysfunction[11].

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Table 1: Distribution of OSA grades among study subjects. The percentage of subjects having mild, moderate & severe OSA were 38%, 30% and 32% respectively.

Sleep Study: OSA grade	No patients	%
Mild (AHI ≥ 5)	19	38.0%
Moderate (AHI ≥ 15)	15	30.0%
Severe (AHI ≥ 30)	16	32.0%
Total	50	100.0%

Table 2: Association among the cases between- OSA grade and echocardiography LVH

Sleep Study: OSA grade		Echocardiography: LVH		Total
		Present	Absent	
Mild	No.	2	17	19
	%	10.5%	89.5%	
Moderate	No.	9	6	15
	%	60.0%	40.0%	
Severe	No.	15	1	16
	%	93.8%	6.3%	
Total	No.	26	24	50
	%	52.0%	48.0%	

Table 3: Comparison of various echocardiography variables between OSA grades

Echo Variables	Sleep Study: OSA grade	Mean	SD	Median	IQR	F-value	p-value
LVEF (Simpson's)	Mild	60.79	2.30	60.00	3.00	2.601	0.085
	Moderate	59.27	3.11	59.00	5.00	Difference is not significant	
	Severe	59.06	1.91	59.50	3.00		
	Severe	39.94	2.96	40.00	5.00		
mPAP	Mild	18.21	2.68	18.00	3.00	14.371	0.00076
	Moderate	21.60	5.15	20.00	2.00	Difference is significant	
	Severe	25.06	6.77	22.00	14.00		
IVS (mm)	Mild	10.51	0.91	10.50	1.10	39.421	8.88E-11
	Moderate	12.19	0.72	12.00	1.30	Difference is significant	
	Severe	13.07	0.94	13.20	1.60		
PW (mm)	Mild	10.28	0.92	10.00	1.50	38.659	1.18E-10
	Moderate	11.86	0.74	11.90	1.10	Difference is significant	
	Severe	12.77	0.86	12.95	1.30		
LV mass (g)	Mild	143.68	38.48	135.00	65.00	27.299	1.36E-08
	Moderate	209.67	44.98	199.00	70.00	Difference is significant	
	Severe	248.38	44.68	254.00	72.00		
LVMI (g/m2)	Mild	75.21	21.62	70.00	39.00	20.174	4.73E-07
	Moderate	102.07	18.18	105.00	28.00	Difference is significant	
	Severe	119.69	22.35	119.50	41.00		
GLS	Mild	23.86	1.53	24.00	2.20	122.779	2.18E-19
	Moderate	19.67	1.33	19.40	2.20	Difference is significant	
	Severe	16.17	1.46	15.90	2.40		

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Table 4: Association among the cases between- OSA grade and echocardiography: PAH

Sleep Study: OSA grade		Echocardiography: PAH		Total
		Mild PH	No PH	
Mild	No.	1	18	19
	%	5.3%	94.7%	
Moderate	No.	3	12	15
	%	20.0%	80.0%	
Severe	No.	7	9	16
	%	43.8%	56.3%	
Total	No.	11	39	50
	%	22.0%	78.0%	

Table 5: Association among the cases between-OSA grade and grade of diastolic dysfunction

Sleep Study: OSA grade		Grade of Diastolic dysfunction			Total
		No diastolic dysfunction	Grade I	Grade II	
Mild	No.	11	8	0	19
	%	57.9%	42.1%	0.0%	
Moderate	No.	4	11	0	15
	%	26.7%	73.3%	0.0%	
Severe	No.	4	7	5	16
	%	25.0%	43.8%	31.3%	
Total	No.	19	26	5	50
	%	38.0%	52.0%	10.0%	

Table 6: Association among the cases between sleep Study: OSA grade diastolic dysfunction and echocardiography GLS

Sleep Study: OSA grade		Echocardiography: GLS		Total
		Low	Normal	
Mild	No.	0	19	19
	%	0.0%	100.0%	
Moderate	No.	9	6	15
	%	60.0%	40.0%	
Severe	No.	16	0	16
	%	100.0%	0.0%	
Total	No.	25	25	50
	%	50.0%	50.0%	

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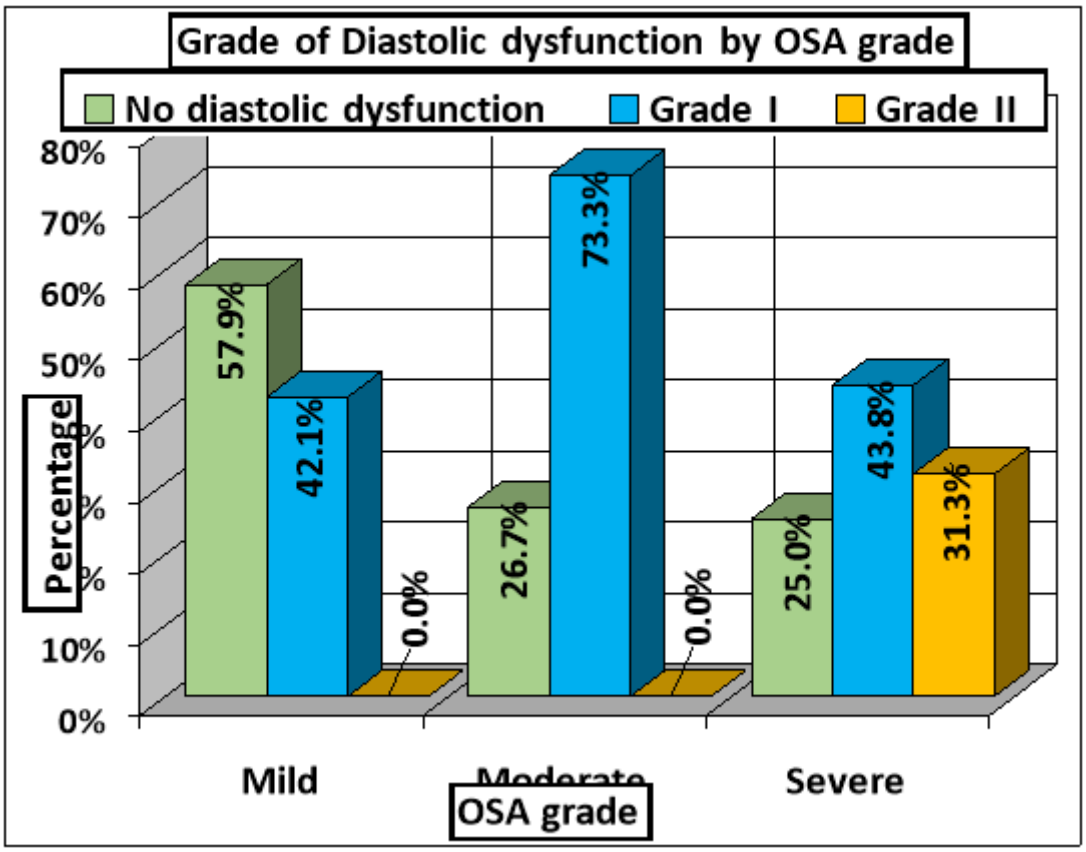


Figure 1: The mean age of study of study subjects (n=50) with newly diagnosed OSA was 47.16 ± 2.902 years, 42% were females and 58% were males

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