Doppler Echocardiographic Evaluation in Obstructive Sleep Apnoea

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ABSTRACT

Obstructive sleep apnoea (OSA) is the commonest form of sleep disordered breathing. Cardiovascular morbidity and mortality are serious complications of OSA. This study was done to evaluate cardiac parameters by Doppler Echocardiography (DE) in patients with OSA in central India. Fifty patients with OSA, diagnosed on full night polysomnography, were recruited into the study. DE was done following standard guidelines. Eighty eight percent of the patients with OSA had normal DE. Thirty one patients were hypertensive. A mild increase in PAP was noted in 6 (12%) patients. Mild LV dysfunction was present in 4 patients. Six patients had diastolic dysfunction. Abnormal DE parameters were present in patients with severe OSA only. A binary logistic regression analysis showed that DE changes were seen in OSA patients with hypertension. Majority of the patients with OSA of all severity had normal DE. Severe OSA, in some cases, was associated with abnormal DE. Abnormal DE in these patients was related to hypertension than to OSA.

KEY WORDS: diastolic dysfunction (DD), doppler echocardiography (DE), left ventricle ejection fraction (LVEF), obstructive sleep apnoea (OSA), pulmonary artery pressure (PAP)

INTRODUCTION:

Obstructive Sleep Apnea (OSA) is the most common form of sleep disordered breathing.[1] OSA may cause many complications and morbidities.[2] Cardiovascular disturbances are one of the serious complications of OSA. These complications include hypertension, coronary artery disease, cardiac arrhythmias, corpulmonale and sudden nocturnal death. Pulmonary hypertension (PH) leading to corpulmonale is a potential complication of OSA. [2]

Doppler Echocardiography (DE) is a modern non-invasive technique to assess the cardiac status. Hence in the present study, we evaluated OSA through DE.

MATERIAL AND METHODS:

In a hospital based, prospective, non-randomized, cross sectional study done over a period of one and half years from January 2017 to June 2018 we evaluated 50 patients of OSA with DE. The study was approved by Research Advisory Committee and Institutional Ethics Committee of People's College of Medical Sciences & Research Centre, Bhopal and All India Institute of Medical Sciences (AIIMS), Bhopal.

Screening for OSA : Patients between the age of 18-80 years were enrolled in the study. Those with sleep complaints like loud snoring, choking, increased day time sleepiness and other risk factors were evaluated for OSA. These patients were subjected to complete physical examination, assessment of Mallampati score and Epworth Sleepiness Score (ESS). Neck circumference (NC) was measured. NC of $\geq$17 inches in men and $\geq$16 inches in women was considered high risk for OSA. Oral examination was done to rule out macroglossia and tonsilar hypertrophy.

Demographic variables including age, gender and occupation were recorded and other clinical variables including breathlessness, hypertension, diabetes mellitus, Body Mass Index (BMI), Electrocardiogram (ECG) changes, Chest X ray and Thyroid profile were also obtained.

Polysomnography: The screened patients were subjected to full night polysomnography (using Philips Alice-6 Diagnostic System) for confirmation of OSA.

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Table 1: Demographic data and Baseline characteristics of study population (n=50).

Baseline variables		Mild OSA (AHI = 5-15/hour)	Moderate OSA (AHI>15-30/hour)	Severe OSA (AHI>30/hour)	Total
Age (in years)	25 -35	0	0	2	2
	35-45	0	2	4	6
	45 -55	1	2	14	17
	55-65	1	2	18	21
	65-75	0	0	4	4
Gender	Male	1	4	29	34
	Female	1	3	12	16
BMI (in Kg/m^2)	<18.5	0	0	1	1
	18.5 -24.9	1	1	4	6
	25.29.9	0	3	8	11
	30-34.9	0	0	14	14
	35-39.9	1	1	7	9
	>40	0	1	8	9
WC (in cm)	Male (>90)	0	0	0	0
	Female (>80)	0	0	0	0
WHR	Male (=0.9)	1	4	28	33
	Female (=0.85)	1	2	12	15
NC (in cm)	Male (>17)	0	1	9	10
	Female (>16)	0	0	1	1
Mallampati score	1	0	0	5	5
	2	0	3	1	4
	3	1	0	11	12
	4	1	3	25	29
ESS	ESS <10	2	3	26	31
	ESS>=10	0	3	16	19
Hypertension	BP≥140/90mmHg	2	4	25	31

(AHI= Apnea Hypopnea Index; BMI=Body Mass Index, WC=Waist Circumference, WHR=Waist Hip Ratio, NC= Neck circumference, ESS=Epworth Sleepiness Score)

Doppler Echocardiography : DE (using Siemens Acuson X300) was performed in patients with confirmed diagnosis of OSA. More than 3 cardiac cycles were used to measure DE parameters. Left ventricular posterior wall thickness (LVPW) was measured using M mode in parasternal long axis view, Left Ventricle Ejection Fraction (LVEF) was calculated by the modified biplane Simpson method. Mean Pulmonary Artery Pressure (PAP) was estimated using Tricuspid Regurgitation (TR) jet velocity.

Statistical analysis : All the data analysis was done using SPSS (Statistical Package for the Social Sciences) ver. 20. Quantitative data is expressed as mean ± SD whereas categorical data is expressed as number and percentage. Student-t test and one way ANOVA was used for quantitative data whereas chi Square test was used for categorical data. Level of significance was assessed at 5%.

RESULTS:

Fifty patients with polysomnography confirmed diagnosis of OSA were selected for DE evaluation. There were 2 patients with mild OSA, 6 with moderate OSA and 42 with severe OSA. General characteristics of patients and DE variables are depicted here in (Table 1 & Table 2).

Majority of the patients (n=46) with OSA had normal LVEF of >50%. Four patients had mildly deranged LVEF of 40-49%. All these 4 patients had severe OSA.

Maximum number of patients ie, 44 (88%) had no Diastolic Dysfunction (DD). Grade 2 DD was

Table 2: DE characteristics of study population (n=50).

	DE variables	Mild OSA	Moderate OSA	Severe OSA	Total	p value
LVEF (%)	<29% (Severe dysfunction)	0	0	0	0	NA
	≥30- 39% (Moderate dysfunction)	0	0	0	0	NA
	40-49% (Mild dysfunction)	0	0	4	4	0.046
	>50% (Normal)	2	6	38	46	0.031
DD (Grades)	0	2	6	36	44	-
	1	0	0	0	0	NA
	2	0	0	5	5	0.432
	3	0	0	1	1	0.876
TR	Present	0	0	0	14	0.021
	Absent	2	6	28	36	-
PAP (mm Hg)	15-22 (Normal)	2	6	36	44	0.041
	>22-40 (Mild PAH)	0	0	6	6	0.048
	41-55 (Moderate PAH)	0	0	0	0	NA
	>55 (Severe PAH)	0	0	0	0	NA

(LVEF= Left Ventricular Ejection Fraction, DD= Diastolic dysfunction, TR= Tricuspid Regurgitation, PAP= Pulmonary Artery Pressure)

present in 5 (10%) patients. Grade 3 DD was present in only one patient. No patient had grade 1 DD. All patients with Grade 2 and 3 DD had severe OSA. Tricuspid regurgitation (TR) was present in 14 (28%) patients (Table 2). TR was present only in patients with severe OSA. Almost all OSA patients (n=44) had normal Pulmonary Artery Pressure (PAP). Mild rise in PAP (>22-40 mmHg) was present in 6 patients. All of them had severe OSA.

Binary logistic regression analysis showed a positive correlation between cardiac parameters like TR, DD, LVEF and PAP and presence of hypertension in patients with OSA. There was a higher OR for the presence of TR, high RVSP, DD, and reduced LVEF in hypertensives with OSA than in non-hypertensives with OSA (Table 3).

Table 3: Binary logistic regression analysis of cardiac parameters with hypertension and OSA (adjusted OR).

DE Variables	OSA patients with Hypertension	OSA patients without Hypertension
TR (present)	OR=1.21, p=0.032	OR=0.11, p=0.342
DD (grade 2 and 3)	OR =1.57, p=0.022	OR =0.52, p=0.522
LVEF (<50%)	OR =1.65, p=0.010	OR =0.35, p=0.611
PAP (<22mm)	OR 0.510, p=0.322	OR =0.41, p=0.427

DISCUSSION:

In our study 2 (4%), 6 (12%) and 42 (84%) patients had mild, moderate and severe OSA respectively. The higher prevalence of severe disease in the present study is probably due to hospital based referral bias model of the study as patients seek advice only in late stages.

Normal LVEF of ≥50% was present in majority (n=46) of the patients. Mild dysfunction with LVEF of 40-49% was present in 4 patients and all of them had severe OSA. In a study by Niromound et al, LVEF <60% was present in 45 out of 883 patients. They concluded that OSA in the absence of hypertension and obesity was not associated with decreased LVEF. Other studies have also confirmed that reduced LVEF was absent in mild to moderate form of the disease. [3],[4]

The cause of decreased LVEF is multifacto-rial and it may be due to hypertension, obesity or OSA. A binary logistic regression analysis of our study indicated that LVEF reduction was more related to hypertension than to OSA. DD was not common in patients with OSA. Only 6 patients had DD and all of them had severe OSA. Binary logistic regression analysis of DE parameters revealed that DD was related to hypertension than to OSA. Several pathophysiological mechanisms have been postulated for the development of DD in patients with OSA like Left Ventricular hypertrophy, activation of sympathetic nervous system and hypertension. [5]

Danica et al [5] in their study concluded that the prevalence of DD was higher in patients with OSA than in the general population. In the study done by Baguat et al, DD was present in 32 (22.8%) out of 150 patients with OSA and 81% of those with DD were hypertensive.[6] Echocar-diographic studies have shown both systolic and diastolic dysfunction with increasing AHI. [7]

Kanwar et al reported that the severity of DD was associated with increasing AHI. Diastolic dysfunction was present only in severe OSA with AHI

>30/hour. [8]

TR was absent in majority (36 out of 50) of our OSA patients. Patients with mild and moderate OSA did not have TR. Only 14 out of 42 patients with severe OSA had TR. This indicates that TR is not common in OSA. Because of small numbers of patients with mild and moderate OSA, we could not conclude whether progressively increasing OSA severity was associated with progressively increasing presence of TR. Moro et al reported TR in 4% of patients with OSA.[2]

OSA was not associated with PAH in most of our patients. Only six out of fifty patients had PAH. PAH was seen only in patients with severe OSA. All these patients had mild PAH. This indicates that OSA is not usually associated with PAH and if PAH occurs, it is of mild degree.

Studies done by Sanner et al and Bady et al also did not find a high prevalence of PAH in patients with OSA. They reported a DE determined mild rise in PAP (20-26 mm Hg) in 18/92 (20%) and 12/44 (27%) patients respectively. [11],[13] Sajkov et al found PAP >22 mm Hg in 11/27 (41%) patients. However, they did not find any correlation with the severity of OSA. [12] Fisher et al in their study compared PAP diagnosed by DE with right heart catheterization (RHC). They reported that the magnitude of pressure underestimation (33%) was greater than overestima-tion (13%) by DE. [10]

Patients with OSA have higher prevalence of hypertension. [3],[7] In our study, 31 (62%) OSA patients were hypertensive. Both OSA and hypertension are known to cause DE changes. DE changes in heart were not common in patients with OSA without hypertension. DE parameters such as DD, decreased LVEF were more related to hypertension, which is not uncommon in patients with OSA, than to OSA. Verbraecken et al also did not find DE changes in patients with OSA without hypertension[3].

In our study patients with OSA had mostly normal DE. It is possible that with a longer duration of untreated OSA, cardiac changes would occur. Most of our patients had symptoms of OSA ranging from a few months to a few years. Patients should, therefore, be followed up longitudinally to assess the effect of OSA on heart.

Most of our patients had severe OSA. This could be because of a hospital based referral bias of the study. A better comparison of cardiac parameters would have been achieved with adequate numbers of patients with mild and moderate OSA.

Only 6 patients in our study had PAH and all these patients had mild PAH. The Gold standard test for the diagnosis of PAH is RHC. Fisher et al in their study compared PAH measured by DE with that measured by RHC. They reported inaccuracy of DE in diagnosing PAH in 48% of their patients. This could be because of suboptimal visualization of Doppler signal across the tricuspid valve.

CONCLUSION:

Normal DE parameters were present in majority of the patients with OSA of all severity. Severe OSA, in some cases, was associated with abnormal DE. The DE changes that were present in these patients were more related to hypertension than to OSA per se.

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