ORIGINAL ARTICLE

# Prevalence of left ventricular diastolic dysfunction in OSA patients: a retrospective study

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**Abstract**

**Introduction:** OSA has been increasingly implicated in the initiation and progression of cardiovascular diseases. We attempted to find the association of LVDD with OSA in terms of its frequency as well as in terms of several important parameters.

**Materials and methods:** It was a retrospective study where 32 OSA patients diagnosed by polysomnography who also underwent echocardiography were looked for the presence of LVDD. The patients were divided into two groups based on the presence or absence of LVDD and compared them according to different parameters.

**Results:** The overall prevalence of LVDD in our OSA patients has been found to be 46.8%. There has been found to be no significant differences as per the different parameters considered. On the contrary, severity of sleep apnea was found to be higher in the non LVDD group tallying to their higher BMI, higher neck and waist circumference.

**Conclusion:** It has not been possible to associate severity of OSA with risk or prevalence of LVDD as there has been found to be no difference between the two groups, with and without LVDD. This area needs further probing so as to look into the origin of LVDD in certain OSA sufferers.

**Keywords:** OSA (Obstructive Sleep Apnea), LVDD (Left Ventricular Diastolic Dysfunction), CPAP (Continuous Positive Airway Pressure), COPD (Chronic Obstructive Pulmonary Disease), ESS (Epworth Sleepiness Score)

## Introduction

Obstructive sleep apnea (OSA), being a fairly common, but neglected disorder has been associated with several cardio vascular morbidities as hypertension, acute myocardial infarction, cardiac arrhythmias, coronary artery disease, heart failure and stroke.[1-3] Some studies have recorded that apparently unexplained left ventricular failure in some patients is actually contributed by OSA. Incidentally, diastolic heart failure has been found to be more common than systolic heart failure.[4] Such diastolic dysfunction may have important association with OSA severity and it is observed that treatment of OSA with nasal CPAP may actually improve the diastolic dysfunction in OSA.[5]

We looked for association of LVDD with OSA in terms of its frequency and tried to see the difference, if any, between patients with and without LVDD, especially in relation to several different important parameters taken into consideration in evaluation of OSA.

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## Materials and Methods

It has been a retrospective study. We looked at our records for the presence of left ventricular diastolic dysfunction in our patients of OSA since the time we started keeping a note of positive findings of echocardiography (LVDD, LVSD, RMWA, PAH), if done in our patients. For all these patients, we had detailed record of sleep study results done at our lab by Embla S 4000 machine between June 2009 and July 2010.

Based on the presence or absence of LVDD, we have divided the patients into two groups and compared them according to the different parameters as-

a. Morphometric measurements (age, BMI, neck circumference, waist circumference)
b. Resting general condition (PR, $SaO_{2,}$ BP)
c. Co morbidities (Hypertension, COPD, diabetes, IHD )
d. Addiction habits (smoking and alcoholism)
e. Sleepiness (ESS score) and different sleep related parameters
f. Oxygenation status

All data were presented as mean ± SD unless otherwise stated. The statistical analysis was performed by unpaired *t* test.

## Results

We could collect the data of 32 patients with significant OSA who were predominantly men (27 of 32 patients). Mean age of the patients were 50.53 ± 9.63 years and the mean BMI was 33.32 ± 6.11 kg/$m^2$ respectively and the mean ESS score was 11.93 ± 6.68. Polysomnography revealed severe OSA in all, with a mean AHI of 51.84 ± 30.25. Out of 32 patients screened, LVDD was present in 15 patients and in 17 patients LVDD was found to be absent. The details of these two groups of patients were charted on different parameters as shown in table 1.

The results reflect that there have been no significant differences as per demographic data, presence of co morbidities many of which are known to pre dispose LVDD and different clinical measurements. On comparing the different sleep related parameters as ESS, total sleep duration, sleep efficiency, apnoea or hypopnea per hour, the difference between the two groups were again not found to be significant. Except for duration of snoring, all the other parameters were more severe in patients without LVDD. Tallying to the higher BMI, higher neck and waist circumference, severity of sleep apnea was higher in the non LVDD group.

## Discussion

OSA is a common disorder characterized by periodic reduction or cessation of airflow due to narrowing of the upper airways during sleep.[6] Prevalence surveys estimate that 2% of women and 4-5% of men of middle age are affected by this syndrome.[7] It has significant deleterious effects on patients' health, as there is considerable evidence suggesting an increased risk for cardiovascular diseases.[8] However, many risk factors for OSA, including male gender, advanced age, and obesity, are the same for cardiovascular disease, which makes it difficult to recognize the role of OSA as an independent risk factor. Some studies have demonstrated an association of OSA with certain cases of left ventricular (LV) failure of otherwise unknown etiology, especially diastolic dysfunction. [10-11]

Left ventricular diastolic dysfunction is a condition with increased resistance to filling of the left ventricle, leading to an inappropriate rise in the diastolic pressure-volume relationship and causing symptoms of pulmonary congestion during exercise.[11] Of late, LVDD has attracted attention of the concerned people. The overall prevalence of LVDD in our OSA patients has been found to be 46.8%, such a figure of prevalence being observed by others too.[12] The first direct evidence that OSA might play a role in causing left ventricular dysfunction came from Hedner and colleagues[13]. They reported that OSA patients had thicker LV walls and LV mass, their mass index to body surface area, was approximately 15% higher among normotensive OSA patients than in normotensive control subjects. Noda et al[14] reported LV hypertrophy (LVH) in 41% of 51 OSA patients. Alchanatis et al[5] also reported that LV diastolic function was impaired in 15 OSA patients with neither history of nor present systemic hypertension, compared to 11 subjects matched for age and BMI.

Contrary to available information, in our study, there is no difference between the two groups, with and without LVDD. The prevalence of LVDD has been found to be higher in patients of less severe OSA (mean AHI being 42.78 ± 27.55 per hour) which needs explanation.

Table: 1

| *Morphometric Measurements* | | | |
|---|---|---|---|
| Parameters | LVDD (n=15) | No LVDD (n=17) | p value |
| Age (years) | 52.9±7.83 | 48.41±10.77 | 0.19 |
| BMI (kg/m$^2$) | 31.10±3.83 | 35.27±7.13 | 0.052 |
| Neck ccf (cms) | 42.10±2.92(n=14) | 42.2±2.82 | 0.92 |
| Waist ccf (cms) | 109.28±10(n=14) | 118.02±15.30 | 0.07 |
| *Resting Clinical State* | | | |
| Resting PR (/min) | 80.93±17.09 | 80.87±14.90(n=16) | 0.99 |
| Resting SaO2(%) | 96.92±1.59 (n=14) | 96.18±2.71(n=16) | 0.37 |
| Systolic BP (mmHg) | 135.66±17.81(n=16) | 132.18±19.74 | 0.6 |
| Diastolic BP (mm/Hg) | 83.2±7.65 (n=16) | 85.31±15.96 | 0.64 |
| *Co Morbidities* | | | |
| Hypertension | 11 (73.33%) | 11 (64.7%) | 0.88 |
| Diabetes mellitus | 4 (26.66%) | 5 (29.41%) | 0.82 |
| IHD | 1 (6.66%) | 2 (11.76%) | 0.90 |
| Hypothyroidism | 1 (6.66%) | 3 (17.64%) | 0.68 |
| COPD | 1(6.66%) | 1 (5.88%) | 0.52 |
| Dyslepidemia | 0 | 1(5.88%) | 0.94 |
| *Addiction Habits* | | | |
| Smoker (past & present) | 9 (60%)11 | (64.70%) | 0.92 |
| Alcoholic (past & present) | 9 (60%) | 6 (35.29%) | 0.29 |
| *Sleep Related Parameters* | | | |
| AHI (/hr) | 42.78±27.55 | 59.83±30.12 | 0.10 |
| ESS score | 11.93±6.8 | 15.41±5.13 | 0.11 |
| Total Sleep Time (mins) | 211.06±128.51 | 202.17±71.83 | 0.81 |
| Sleep Efficiency(%) | 84.03±20.98 | 91.27±5.41 | 0.18 |
| *Sleep Abnormalities* | | | |
| Obstructive Apnea (/hr) | 18.88±21.35 | 32.68±28.88 | 0.13 |
| Central Apnea (/hr) | 1.393±4.63 | 0.35±0.70 | 0.35 |
| Mixed Apnoea (/hr) 3.5±4.77 | 4.65±7.70 | 0.60 | |
| Hypopnoea (/hr) | 19.01±14.28 | 22.12±15.93 | 0.56 |
| Snore Time (mins) | 44.09±60.53 | 19.45±14.29 | 0.11 |
| *Oxygenation* | | | |
| Avg SAO2 (%) | 92.77±4.49 | 91.25±5.31(n=16) | 0.39 |
| Lowest SAO2 (%) | 75.26±11.02 | 67.62±12.64(n=16) | 0.08 |
| Avg Desat (%) | 8.51±3.18 | 9.73±3.59 (n=16) | 0.32 |
| Sat<90% (%) | 22.44±27.77 | 29.44±29.05 | 0.49 |
| Sat <80% (%) | 5.82±11.15 | 8.04±11.98 | 0.59 |
| Sat <70% (%) | 0.94±1.97 | 1.78±12.51 | 0.79 |

However, OSA patients with LVDD were found to be slightly older than their counterparts. Duration of snoring has also been found to be more in favor of patients with LVDD, though none of the relations on either way was statistically significant. It is not possible, therefore, from the results, to associate severity of OSA with risk or prevalence of LVDD. From this point of view, the origin of LVDD in certain OSA sufferers becomes a more interesting area of research. The data and the analysis do not point to any variable in consideration being associated with the presence of diastolic dysfunction of left ventricle. Perhaps, some other factors, apparently not clear, are playing a crucial role in our patients. COPD has recently been implicated as an association of LVDD.[15] We have not performed spirometry to any of these patients that could have been

helpful to identify COPD.

There are several limitations in our study. This was a retrospective study with the total number of patients being very small. There was no control group as well. Another limitation is the echocardiography was done at different places by different persons using different machines. The actual prevalence of diastolic dysfunction could be much higher if the echocardiography could have been done following a stringent protocol. We are not aware of the values of different parameters for LVDD in echocardiography. However, in general, left ventricular ejection fraction (LVEF), IV septal thickness, LV internal diameter, E/A ratio have been usually looked for according to those reports.

Although it is difficult to derive any inference with such a small number of patients, the higher association of LVDD in snorers needs further evaluation. The study signifies the need for a well planned prospective investigation in this area with determination of the right incidence, markers and the significance of LVDD in OSA patients.

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