Discussion

This study revealed that OSA is associated with additional impact in the left ventricular dimension and LVM among Nigerian hypertensive subjects and that they may have additional increased CV risk. This is related to the associated increased frequency of LVH, a higher chamber wall dimension including PWT, interventricular septal thickness and even increased mean FBS. It is also related to significantly higher LVM among hypertensive subjects with increased risk of OSA than those with low risk for OSA. A similar finding was demonstrated in this study between hypertensive snorers and non-snorers.

This is in agreement with other similar studies that have demonstrated that OSA is associated with increased LVM and left ventricular chamber dimensions.[19-21] However, other studies have demonstrated that this change is particularly related to the pattern and contribution of obesity among these hypertensive subjects[22] while others have demonstrated no significant additional impact of OSA on the left ventricular structure and function among hypertensive and diabetic subjects.[23]

OSA is closely associated with many CV risk factors such as HTN, atherosclerosis, obesity, diabetes and dyslipidemia.[7,12,24,25] Repeated episodes of hypoxia, hypercapnia, microarousals and changes in intrathoracic pressure in OSA trigger pathophysiological mechanisms such as hyperactivity, oxidative stress, systemic inflammation, hypercoagulability and even endothelial dysfunction.[26-28] All these changes results in additional impact on the left ventricular remodeling pattern and may consequently produce increased LVM and chamber dimension. The apnea and hypopnea episode in OSA is also associated with increased inflammation, endothelial dysfunction and coagulation abnormalities.[20,24] This may be responsible for a higher BP profile among hypertensive subjects with high risk for OSA in this study and also among hypertensive snorers. The increased CV risk profile of hypertensive subjects with high risk for OSA and snoring may also be responsible for the elevated FBS compared with those with low risk of OSA. Increased FBS is associated with insulin resistance and endothelial dysfunction and can ultimately lead to frank diabetes.[7,12,15,27]

This study therefore revealed that OSA and/or snoring are associated with the double burden on the myocardium of hypertensive subjects with these conditions. It is however, possible that this burden may be alleviated by the use of antihypertensive therapy. This may be what is responsible for the finding in this study with respect to diastolic function. LVH is associated with increased prevalence of diastolic dysfunction.[29,30] However, we found out that the mean transmitral E/A velocity was lower among subjects with high risk for OSA and hypertensive snorers than those with low risk and hypertensive non-snorers respectively. This may be due to the fact that the study participants were already on treatment majority of who are on Angiotensin converting

Table 1: Demographic and clinical parameters between hypertensive with low and high risk of OSA

Variable	Low risk (49)	High risk (55)	*P*
Age (years)	58.8 (12.6)	58.6 (11.2)	0.82
Gender (females) (%)	29 (59.2)	34 (61.8)	0.73
WC (cm)	88.2 (11.4)	96.6 (11.9)	<0.001*
SBP (mmHg)	133.7 (15.2)	137.3 (21.1)	0.36
DBP (mmHg)	81.5 (11.8)	82.1 (14.0)	0.84
BMI (kg/m^2)	24.6 (5.4)	27.8 (5.1)	<0.01*
WHR	0.90 (0.006)	0.94 (0.007)	0.01*
PR (min^{-1})	80.5 (13.3)	84.7 (15.0)	0.21

*Statistically significant. WC: Waist circumference, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WHR: Waist hip ratio, PR: Pulse rate, OSA: Obstructive sleep apnea

Table 2: Echocardiographic parameters of hypertensive subjects with low and high risk of OSA

Variable	Low risk (49)	High risk (55)	*P*
LVIDd (mm)	49.1 (4.1)	50.9 (12.1)	0.71
LVIDs (mm)	32.6 (5.1)	37.3 (14.8)	0.42
PWTd (mm)	10.7 (2.1)	12.1 (2.7)	0.02*
IVSd (mm)	11.7 (2.4)	13.2 (3.1)	0.02*
EF (%)	65.2 (8.9)	60.6 (20.0)	0.60
FS (%)	34.0 (5.4)	30.0 (11.1)	0.37
E/A ratio	0.81 (0.21)	1.2 (0.44)	0.04*
LVM (g)	208.7 (47.0)	263.6 (112.8)	0.02*
RWT	0.44±0.07	0.50 (0.16)	0.03*
LVMI (g/m$^{2.7}$)	53.3 (11.3)	73.2 (30.9)	0.01*
LVH (n) (%)	28 (57.1)	39 (70.9)	<0.01*

*Statistical significant. LVIDd: Left ventricular internal dimension in diastole, LVIDs: Left ventricular internal dimension in systole, PWTd: Posterior wall thickness in diastole, IVSd: Interventricular septal thickness in diastole, EF: Ejection fraction, FS: Fractional shortening, LVM: Left ventricular mass, RWT: Relative wall thickness, LVMI: Left ventricular mass index, LVH: Left ventricular hypertrophy, OSA: Obstructive sleep apnea, E/A: transmitral early (E) to late atrial (A) flow velocity

Table 3: The clinical and echocardiographic parameters between hypertensive snorers and non-snorers

Variable	Hypertensive non-snorers (52)	Hypertensive snorers (52)	*P*
Age (years)	58.6 (12.02)	58.1 (11.7)	0.81
Waist circumference (cm)	88.1 (10.7)	97.2 (12.2)	<0.001*
SBP (mmHg)	132.5 (13.8)	138.6 (22.12)	0.12
DBP (mmHg)	81.0 (12.4)	82.7 (13.6)	0.54
FBS (mmol/l)	5.1 (1.7)	5.7 (0.82)	0.04*
LVIDd (mm)	49.3 (3.6)	51.0 (12.7)	0.70
PWTd (mm)	10.6 (1.8)	12.0 (2.7)	0.01*
IVSd (mm)	11.8 (2.0)	13.4 (3.2)	0.02*
E/A ratio	0.98 (0.37)	1.2 (0.46)	0.32
LVM (g)	208.3 (40.7)	268.8 (11.7)	0.01*
BMI (kg/m^2)	24.4 (4.73)	28.3 (5.5)	<0.001*

*Statistically significant. SBP: Systolic blood pressure, DBP: Diastolic blood pressure, FBS: Fasting blood glucose, LVIDd: Left ventricular internal dimension in diastole, PWTd: Posterior wall thickness in diastole, IVSd: Interventricular septal thickness in diastole, LVM: Left ventricular mass, BMI: Body mass index, E/A: Transmitral early (E) to late atrial (A) flow velocity

enzyme inhibitors, Angiotensin receptor blockers, which have been reported to improve diastolic function and reverse CV remodeling.[7,9,14]

An important association of high risk for OSA and snoring in this study was obesity. Hypertensive snorers and those with high risk for OSA had a significantly higher mean WC and BMI than those with low risk for OSA and hypertensive non-snorers. While obesity may be associated with increased LVM, Sukhija *et al*.[19] showed that OSA was an independent predictor of LVH after controlling for other factors including BMI and WC.

Some other relationships between OSA and HTN have been reported by other authors from other part of the world. Myslinski *et al*. reported that left ventricular end diastolic dimension was increased in hypertensive subjects with ineffectively treated HTN and also was positively correlated with apnea and hypopnea index.[31]

This study revealed no significant association between left ventricular dimension and OSA. This may be because the mean WC and BMI in were significantly higher in that study by Myslinski *et al*. than this present study. Another possible reason may be because our patients are treated hypertensives and the use of antihypertensives might have altered the remodeling pattern. However, Wachter *et al*. showed that OSA is not associated with increased LVM and/or impaired LVDF independently of obesity, HTN or advancing age.[32]

The clinical significance of this study is that Nigeria hypertensive subjects with snoring or increased risk for OSA may have a higher CV risk due to the increased LVM, chamber wall dimension and a higher chance of LVH and may therefore require further attention in order to reduce the CV risk. Continuous positive airway pressure has been shown to ameliorate the increased CV burden in these subjects including lifestyle modification aimed at reducing weight among obese subjects.[13,21,31] One limitation of this study is that it is a cross-sectional study and do not have the power to determine the causality of statistically related variables. Further research is therefore necessary including prospective study designs as well as randomized trials in order to determine the relationship of LVM and OSA among hypertensive subjects.

In conclusion, this study revealed that OSA and snoring are possibly associated with increased CV risk due to the significant increased LVM, chamber wall dimension, FBS, obesity and frequency of LVH in Nigerian hypertensive subjects. Further attention may therefore be needed among them to further reduce their CV risk.

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