Obstructive Sleep Apnea and Associated Factors Among Hypertensive Patients Attending a Tertiary Cardiac Center in Tanzania: a Comparative Cross-sectional Study

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Abstract

Background: There is mounting evidence for a reciprocal yet bidirectional association between sleep-disordered breathing and hypertension. Obstructive sleep apnea (OSA), a common cause of systemic hypertension is an independent risk factor for hypertension-related cardiovascular morbidity and mortality. In this comparative hospital-based cross-sectional study, we sought to explore the burden of obstructive sleep apnea and its associated risk factors among hypertensive patients attending Jakaya Kikwete Cardiac Institute.

Methodology: A total of 1974 individuals (i.e. 1289 hypertensive and 685 normotensives) were consecutively enrolled in this study. The Berlin questionnaire and Epworth Sleepiness Scale were utilized in the assessment of OSA and excessive daytime sleepiness (EDS) respectively. Logistic regression analyses were employed in the determination of associated factors for OSA.

Results: The mean age was 53.4 years and females constituted the large majority (60.4%) of participants. About three quarters (74.1%) of participants had excess body weight, 11.6% had diabetes, 8.0% had asthma and 18.6% had history of recurrent nasal congestion. Positive family history of snoring was reported by 43.1% of participants and 36.9% had a personal history of snoring. Persons with hypertension displayed a higher frequency (42.1%) of OSA compared to their normotensive counterparts (11.8%), p<0.001. Multivariate logistic regression analyses revealed hypertension (OR 5.1, 95% CI 3.2-8.2, p<0.001), diabetes mellitus (OR 2.2, 95% CI 1.3-3.5, p<0.01), chronic nasal congestion (OR 1.6, 95% CI 1.1-2.5, p = 0.01), obesity (OR 2.4, 95% CI 1.8-3.3, p<0.001), increased neck circumference (OR 2.7, 95% CI 1.2-6.4, p = 0.02), family history of snoring (OR 5.5, 95% CI 4.0-7.5, p<0.001), and working >8hrs/24hr (OR 0.6, 95% CI 0.4-1.0, p = 0.03) to have an independent association for OSA. Furthermore, participants with hypertension displayed superior odds for OSA compared to their normotensive counterparts across all subgroup analyses.

Conclusion: OSA is considerably common among hypertensives in a tertiary health care setting in Tanzania. Positive family history of snoring was the strongest associated factor; however, excess body weight proved to be the strongest modifiable risk factor. In view of its pervasiveness, OSA should be an integral part of the medical evaluation in hypertensive individuals.

Background.

There is mounting evidence for a reciprocal yet bidirectional, and a complex however intriguing association between sleep-disordered breathing and hypertension.[1–19] Obstructive sleep apnea (OSA), a common cause of systemic hypertension is associated with poor blood pressure control and resistant hypertension.[1–19] The mechanisms underlying the association between OSA and hypertension have not been well elucidated. Nevertheless, sympathetic nervous system overactivity is considered to be the primary mechanism linking OSA to the development of hypertension. It has been postulated that the intermittent hypoxia (i.e. hypoxemic - apneic episodes) induced by OSA leads to chemoreceptor activation

and increased sympathetic outflow causing inflammation, neurohormonal dysregulation and endothelial dysfunction that predisposes to increased carotid intima-media thickness and arterial stiffness inevitably leading to the development of hypertension.[20–26]

Although less than one-tenth of the general adult population is estimated to be affected,[27] OSA is known to distress between 30% – 80% of hypertensives and 70% – 90% of the resistant hypertension subgroup.[27–30] Conversely, between 35% and 80% of individuals with OSA have elevated blood pressure.[29, 31–33] Evidence from epidemiological studies strongly implicate OSA as an independent risk factor for hypertension-related cardiovascular morbidity (i.e. arrhythmias, cerebrovascular disease, ischemic heart disease, large vessel disease and heart failure)[30, 34–43] and mortality.[37, 44–46] Additionally, OSA is associated with increased incidence of numerous nocturnal cardiovascular events including angina pectoris, myocardial infarction and sudden cardiac death.[47, 48] Owing to the paucity of data on the association between OSA and hypertension in sub- Saharan Africa (SSA) in general and Tanzania in particular, we conducted this comparative hospital-based cross-sectional study to explore the burden of OSA and its associated factors among hypertensive patients attending Jakaya Kikwete Cardiac Institute (JKCI).

Methods.

Study design, Recruitment process, and Definition of terms.

This comparative cross-sectional hospitalized-based study was conducted at JKCI, a tertiary care public teaching hospital in Dar es Salaam, Tanzania between July 2020 and March 2021. A consecutive sampling method was utilized to recruit consented hypertensive outpatients (cases) and normotensive patient escorts (controls) during their scheduled clinic visit. A structured questionnaire bearing questions pertaining to sociodemographic and clinical characteristics, measurement of key vitals (blood pressure, blood sugar, height, weight, and waist and neck circumference) was used during participants' interviews. Moreover, standard tools (i.e. the Berlin questionnaire[49] and Epworth sleepiness scale[50]) were utilized in the assessment of OSA and EDS respectively. We defined underweight as BMI < 18.5 kg/m^2, normal: BMI 18.5–24.9 kg/m^2, overweight: BMI 25.0-29.9 kg/m^2 and obese: BMI ≥ 30.0 kg/m^2.[51] Individuals who smoked at least 1 cigarette in the past 6 months were regarded as current smokers, those who last smoked over 6 months or self-reported quitting smoking were considered past smokers and those who never smoked were regarded as non-smokers. Alcohol drinking was defined as at least a once consumption every week. Hypertension was defined as SBP ≥ 140 mmHg or DBP ≥ 90 mmHg, or use of blood pressure lowering agents.[52] Diabetes was diagnosed using a random blood glucose (RBG) ≥ 11.1 mmol/L and/or fasting blood glucose (FBG) ≥ 7 mmol/L or use of glucose-lowering agents.[53]

Statistical analysis.

All statistical analyses utilized STATA v11.0 software. Summaries of continuous variables are presented as means (± SD) and categorical variables are presented as frequencies (percentages). Pearson Chi

square and Student's T-test were used in comparison of categorical and continuous variables respectively. Logistic regression analyses were used to assess for factors associated with OSA. Stepwise and forward selection procedure was used to add and assess the statistically significant variables in the multivariate regression model. The multivariate model was fitted with baseline covariates associated with OSA by bivariate analysis at the < 0.05 significance level. Furthermore, subgroup analyses to determine the odds for OSA by hypertension status was performed and the findings are presented graphically in a forest plot. Odd ratios with 95% confidence intervals and p-values are reported. All tests were 2-sided and $p < 0.05$ was used to indicate a statistical significance.

Results.

Study population characteristics.

A total of 1974 individuals (i.e. 1289 hypertensive and 685 normotensives) were enrolled in this study. Table 1 displays the sociodemographic and clinical characteristics of study participants. The mean age was 53.4 years and 63.8% of participants were aged 60 years or less. Females constituted the large majority (60.4%) of participants. 54.0% of participants had attained primary school as their highest level of education and 67.9% were in marriage. Over 80% of participants dwelled in urban areas, 63.3% had a regular income generating activity and 26.4% worked at least 8 hours a day. Over one-sixth (15.9%) had a positive smoking history and 10% were current alcohol consumers. Nearly three quarters (74.1%) of participants had excess body weight, 11.6% had diabetes, 8.0% had asthma and 18.6% had history of recurrent nasal congestion. Positive family history of snoring was reported by 43.1% of participants and 36.9% had a personal history of snoring. Surprisingly, none of the participants in this study was aware of OSA and its potential in causing or worsening CVDs and other chronic conditions.

Table 1
Sociodemographic characteristics of study participants by hypertension status.

Characteristic	ALL N = 1974	Hypertensive n = 1289	Normotensive n = 685	p-value
Age (Mean, SD)	53.4 (15.0)	59.8 (11.8)	41.3 (13.0)	**< 0.001**
Range	18–98	18–98	18–83	
Age group				
>60	714 (36.2%)	653 (50.7%)	61 (08.9%)	
≤60	1260 (63.8%)	636 (49.3%)	624 (91.1%)	**< 0.001**
Sex				
Male	782 (39.6%)	483 (37.5%)	299 (43.7%)	
Female	1192 (60.4%)	806 (62.5%)	386 (56.4%)	**< 0.01**
Education				
No Formal	117 (05.9%)	100 (07.8%)	17 (02.5%)	**< 0.001**
Primary	950 (48.1%)	643 (49.9%)	307 (44.8%)	**0.03**
Secondary	513 (26.0%)	318 (24.6%)	195 (28.5%)	0.06
University	394 (20.0%)	228 (17.7%)	166 (24.2%)	**< 0.001**
Marital status				
Single	192 (09.7%)	48 (03.7%)	144 (21.0%)	**< 0.001**
Married	1341 (67.9%)	873 (67.7%)	468 (68.3%)	0.79
Divorced	128 (06.5%)	82 (06.4%)	46 (06.7%)	0.80
Widowed	313 (15.9%)	286 (22.2%)	27 (04.0%)	**< 0.001**
Occupation				
Jobless	371 (18.8%)	253 (19.6%)	118 (17.2%)	0.19
Self-employed	888 (45.0%)	487 (37.8%)	401 (58.5%)	**< 0.001**
Employed	362 (18.3%)	219 (17.0%)	143 (20.9%)	**0.03**
Retired	353 (17.9%)	330 (25.6%)	23 (03.4%)	**< 0.001**
Work duration (hours)				
Mean (SD)	6.2 (3.8)	5.4 (3.5)	7.7 (3.9)	**< 0.001**
>8 hours/day	521 (26.4%)	256 (19.9%)	265 (38.7%)	**< 0.001**

Characteristic	ALL N = 1974	Hypertensive n = 1289	Normotensive n = 685	p-value
Residence				
Urban	1618 (82.0%)	1030 (79.9%)	588 (85.8%)	
Rural	356 (18.0%)	259 (20.1%)	97 (14.2%)	**< 0.01**
Personal Disease History (% Yes)				
Diabetes	228 (11.6%)	211 (16.4%)	17 (02.5%)	**< 0.001**
Asthma	158 (08.0%)	107 (08.3%)	51 (07.5%)	0.53
Stroke	113 (05.7%)	103 (08.0%)	10 (01.5%)	**< 0.001**
Recurrent nocturnal nasal congestion	367 (18.6%)	270 (21.0%)	97 (14.2%)	**< 0.001**
Smoking status				
Ever smoker	313 (15.9%)	284 (14.3%)	129 (18.8%)	
Never	1661 (84.1%)	1105 (85.7%)	556 (81.2%)	**< 0.01**
Alcohol intake (current)				
Yes	197 (10.0%)	70 (05.4%)	127 (18.5%)	
No	1777 (90.0%)	1219 (94.6%)	558 (81.5%)	**< 0.001**
Body Mass Index (mean, SD)	29.1 (6.2)	30.3 (6.3)	26.9 (5.5)	**< 0.001**
BMI categories				
Underweight	34 (01.7%)	11 (0.9%)	23 (03.4%)	**< 0.001**
Normal	478 (24.2%)	225 (17.4%)	253 (36.9%)	**< 0.001**
Overweight	680 (34.5%)	447 (34.7%)	233 (34.0%)	0.76
Obese	782 (39.6%)	606 (47.0%)	176 (25.7%)	**< 0.001**
Waist circumference (mean[cm], SD)	98.1 (14.0)	101.0 (13.4)	92.8 (13.4)	**< 0.001**
Men > 90cm	486 (62.1%)	357 (73.9%)	129 (43.1%)	**< 0.001**
Women > 80cm	1089 (91.4%)	766 (95.0%)	323 (83.7%)	**< 0.001**
Neck circumference (mean[cm], SD)	36.8 (3.5)	37.2 (3.4)	35.9 (3.6)	**< 0.001**
Men > 37cm	434 (55.5%)	314 (65.0%)	120 (40.1%)	**< 0.001**
Women > 31cm	1095 (91.9%)	767 (95.2%)	328 (85.0%)	**< 0.001**

Characteristic	ALL N = 1974	Hypertensive n = 1289	Normotensive n = 685	p-value
Family history of snoring				
Positive	851 (43.1%)	566 (43.9%)	285 (41.6%)	0.33
Negative	982 (49.8%)	617 (47.9%)	365 (53.3%)	**0.02**
Don't Know	141 (07.1%)	106 (08.2%)	35 (05.1%)	**0.01**
Personal history of snoring				
Positive	729 (36.9%)	564 (43.8%)	165 (24.0%)	**< 0.001**
Negative	1156 (58.6%)	659 (51.1%)	497 (72.6%)	**< 0.001**
Don't Know	89 (04.5%)	66 (05.1%)	23 (03.4%)	0.08
OSA awareness				
Yes	0 (0%)	0 (0%)	0 (0%)	1.0
No	1974 (100%)	1289 (100%)	685 (100%)	1.0

Prevalence and correlates of OSA.

Overall, 624 (31.6%) of all participants were categorized as having high-risk for OSA. Persons with hypertension displayed a higher frequency (42.1%) of OSA compared to their normotensive counterparts (11.8%), $p < 0.001$. Furthermore, the prevalence of OSA was similar among hypertensives regardless of their BP range i.e. 41.1% in SBP < 140, 43.2% in SBP140-159 and 42.4% in SBP ≥ 160. Moreover, individuals with hypertension had a 6.8% prevalence of EDS compared to 5.6% among controls, $p = 0.3$, Table 2. During bivariate analyses, participants with high-risk OSA were older (57.9 vs 51.3, $p < 0.001$) and a positive linear correlation between age and OSA until the age group 60–70 years was observed i.e. <40 years, 13.4%; 41–50 years, 29.0%; 51–60 years, 36.7%; 61–70 years, 42.9%; and > 70 years, 36.3%. Moreover, participants with high-risk for OSA had a higher comorbidity history [hypertension (87.0% vs 55.3%, $p < 0.001$), diabetes (18.3% vs 8.4%, $p < 0.001$), stroke (7.2% vs 5.0%, $p = 0.05$) and recurrent nasal congestion (25.8% vs 15.3%, $p < 0.001$)], and a higher frequency of known risk factors i.e. positive smoking history (18.3% vs 14.7%, $p = 0.04$), increased body weight (88.9% vs 67.2%, $p < 0.001$), increased neck circumference (88.6% vs 72.3%, $p < 0.001$), post-menopause (83.5% vs 59.8%, $p < 0.001$), positive family (70.2% vs 30.6%, $p < 0.001$) and personal history of snoring (99.7% vs 7.9%, $p < 0.001$), and a higher ESS score (42.8% vs 27.6%, $p < 0.001$); Table 3. Males and females displayed similar odds of OSA (i.e. 31.7% vs 31.5%, $p = 0.9$).

Table 2
Prevalence and pattern of EDS by hypertension status.

EDS	*Frequency (%)*		*p - value*
	Hypertensive	**Normotensive**	
Unlikely	635 (49.3%)	373 (54.5%)	0.03
Average	210 (16.3%)	116 (16.9%)	0.7
Excessive situational	356 (27.6%)	158 (23.1%)	0.03
Excessively sleepy	88 (06.8%)	38 (05.6%)	0.3

Table 3

Sociodemographic and Clinical characteristics comparison by OSA status.

Characteristic		High-risk OSA n = 624	Low-risk OSA n = 1350	p - value
Age (Mean, SD)		57.9 (12.3)	51.3 (15.7)	**<0.001**
	n			
<40 years[β]	442	59 (13.4%)		-
41-50	338	98 (29.0%)		**<0.001**
51-60	480	176 (36.7%)		**<0.001**
61-70	480	206 (42.9%)		**<0.001**
>70 years	234	85 (36.3%)		**<0.001**
Female sex		376 (60.3%)	816 (60.4%)	1.0
≤ Primary education		346 (55.5%)	721 (53.4%)	0.38
Married		439 (70.4%)	902 (66.8%)	0.11
Work duration >8hrs/day		135 (21.6%)	386 (28.6%)	**0.001**
Urban residency		505 (80.9%)	1113 (82.4%)	0.42
Comorbidities history				
Hypertension		543 (87.0%)	746 (55.3%)	**<0.001**
Diabetes		114 (18.3%)	114 (08.4%)	**<0.001**
Asthma		48 (07.7%)	110 (08.2%)	0.70
Stroke		45 (07.2%)	68 (05.0%)	**0.05**
Recurrent nocturnal nasal congestion		161 (25.8%)	206 (15.3%)	**<0.001**
Menopause[#]		314 (83.5%)	488 (59.8%)	**<0.001**
Ever smoker		114 (18.3%)	199 (14.7%)	**0.04**
Current drinker		43 (06.9%)	154 (11.4%)	**0.002**
Measures of obesity				
BMI≥25		555 (88.9%)	907 (67.2%)	**<0.001**
Neck>37cm M/31cm F		553 (88.6%)	976 (72.3%)	**<0.001**
Waist>90cm M/80cm F		572 (91.7%)	1003 (74.3%)	**<0.001**
Snoring history				

Positive family		438 (70.2%)	413 (30.6%)	**<0.001**
Positive personal		622 (99.7%)	107 (07.9%)	**<0.001**
SBP≥140 and/or DBP≥90*		365 (67.2%)	482 (64.6%)	0.26
SBP<140*β	504	207 (41.1%)		-
SBP 140-159*	424	183 (43.2%)		0.51
SBP≥160*	361	153 (42.4%)		0.70
ESS score ≥10		267 (42.8%)	373 (27.6%)	**<0.001**
#: assessed in women, n = 1192 ; *: assessed in hypertensives, n = 1289. ; β: reference group				

In a logistic regression model of 16 characteristics, 7 factors i.e. hypertension (OR 5.1, 95% CI 3.2–8.2, p < 0.001), diabetes mellitus (OR 2.2, 95% CI 1.3–3.5, p < 0.01), chronic nasal congestion (OR 1.6, 95% CI 1.1–2.5, p = 0.01), obesity (OR 2.4, 95% CI 1.8–3.3, p < 0.001), increased neck circumference (OR 2.7, 95% CI 1.2–6.4, p = 0.02), family history of snoring (OR 5.5, 95% CI 4.0-7.5, p < 0.001), and working > 8hrs/24hr (OR 0.6, 95% CI 0.4-1.0, p = 0.03) were found to have an independent association for OSA, Table 4. Furthermore, as displayed in Fig. 1, participants with hypertension had superior odds for OSA compared to their normotensive counterparts across all subgroup analyses. For instance, among individuals with BMI ≥ 25 those with hypertension displayed a 4-fold chance of having OSA compared to normotensive persons, OR 4.0, 95% CI 3.0-5.5, p < 0.001. Similarly, among participants with a positive family history of snoring, those with hypertension had a 7-fold likelihood of having OSA compared to their counterparts with negative history of hypertension, OR 7.1, 95%CI 5.1–10.1, p < 0.001. Nevertheless, participants with diabetes displayed similar odds of OSA regardless of their hypertension status, OR 1.9, 95%CI 0.6–6.6, p = 0.21.

Table 4
Logistic regression analyses for factors associated with OSA.

Characteristic	Comparative	OR	95% CI	p-value	Adj.OR	95% CI	p-value
Age > 60	Age ≤ 60	1.9	1.6–2.3	**< 0.001**	1.2	0.8–1.7	0.32
Male	Female	1.0	0.8–1.2	0.94	-	-	**-**
Diabetes	Diabetes-free	2.4	1.8–3.2	**< 0.001**	2.2	1.3–3.5	**< 0.01**
Asthmatic	Asthma-free	0.9	0.7–1.3	0.73	-	-	**-**
History of stroke	Stroke-free	1.5	1.0-2.2	**0.05**	0.8	0.4–1.6	0.57
Chronic nasal congestion	No congestion	1.9	1.5–2.4	**< 0.001**	1.6	1.1–2.5	**0.01**
Ever smoker	Never smoker	1.3	1.0-1.7	**0.05**	0.8	0.4–1.8	0.63
BMI ≥ 30	BMI < 30	3.7	3.0-4.5	**< 0.001**	2.4	1.8–3.3	**< 0.001**
Neck > 37cm M/31cm F	Normal Neck	3.0	2.3–3.9	**< 0.001**	2.7	1.2–6.4	**0.02**
Family history of snoring	Negative history	6.1	4.9–7.6	**< 0.001**	5.5	4.0-7.5	**< 0.001**
Uncontrolled BP	BP < 140/90	0.9	0.7–1.1	0.33	-	-	**-**
ESS score ≥ 10	ESS score < 10	2.0	1.6–2.4	**< 0.001**	1.3	0.9–1.8	0.12
Hypertensive	Negative history	5.4	4.2-7.0	**< 0.001**	5.1	3.2–8.2	**< 0.001**
Alcohol intake	Non drinker	0.6	0.4–0.8	**0.002**	1.4	0.7–3.1	0.34
Post-menopausal	Pre-menopausal	3.4	2.5–4.6	**< 0.001**	1.2	0.8–1.9	0.36
Works > 8hrs/day	works ≤ 8hrs/day	0.7	0.6–0.9	**0.001**	0.6	0.4-1.0	**0.03**

Discussion.

Over the past five decades, OSA, a potentially treatable disorder that is characterized by cyclic intermittent hypoxia and the disruption of sleep architecture has been increasingly recognized as a widespread syndrome causing significant population health burden. Despite its ubiquity and potential to either cause or modify the course of other chronic disorders, OSA continues to be underdiagnosed and undertreated worldwide.[54, 55] With over two-fifth of hypertensives having OSA, this present study echoes findings from previous studies[1–30] that OSA is highly prevalent among hypertensive individuals. In unison with the literature,[56–59] this study has revealed critically low awareness regarding OSA and its associated health-related consequences. Amid the rapid rising obesity, hypertension and diabetes rates, it is pivotal to intensify the ongoing health education campaigns to increase awareness so that they coincide with the temporal trends in public knowledge of OSA amongst others.

Moreover, a positive linear correlation between age and OSA until the age group 60–70 years was observed in this present study. Such findings are literally identical to the Sleep Heart Health Study which demonstrated a simple, positive linear correlation between age and OSA until the age of about 65 years.[60] Furthermore, this linear trend between OSA and age until about the age 65 then levelling off or even decreasing has been reported by a couple of other recent studies.[61–63] We observed similar EDS rates among hypertensives and normotensives. Previous studies have revealed conflicting results with some[64,65] showing independent association between EDS and hypertension while others did not.[19,66,67] As witnessed in this study, such controversy can largely be explained by a high prevalence of overweight and obesity which independently increases the risk of EDS.[68–70]

Nonetheless, with a 2-fold likelihood for OSA among obese individuals, our findings are in consonance with numerous other studies which have revealed excessive body weight as a potential factor for the development and progression of OSA.[60, 71–79] Furthermore, as it was vivid in this study, other anthropometric measurements including neck and waist circumference have revealed a monotonic relationship with OSA similar to previous studies.[60, 80–83] Moreover, it is postulated that each unit increase in BMI is associated with a 14% increased risk of developing OSA and the odds of moderate-severe OSA increases 6-fold for every 10% weight gain.[74] Conversely, a 10–15% reduction in body weight has been shown to reduce OSA severity by 50%.[84] Our findings similar to majority of previous studies imply that excessive body weight is the strongest modifiable risk factor for OSA.

Our association analysis furthermore disclosed a 2-fold increase in OSA likelihood among diabetes type 2 patients compared to their diabetes-free counterparts. Several cross-sectional and longitudinal studies have demonstrated an independent association between OSA and incident type 2 diabetes[85–91] as well as with insulin resistance but diabetes-free persons.[92–95] Furthermore, a positive family history of snoring instigated an over 5-fold increase in OSA likelihood and was found to be the strongest predictor of OSA in this study. Similarly, a Cleveland Family Study, the largest familial study investigating OSA revealed a near 2-fold risk among relatives of OSA compared to their control neighbors.[96] Furthermore, studies involving monozygotic twins and siblings/children of persons with OSA have shown an up-to 50% increased odds of developing OSA compared to the general population.[97–100] Additionally, numerous

genetic studies[101–109] have identified several novel genetic loci associated with a sleep apnea phenotype suggesting around two-fifth[110–112] of the OSA variance is attributable to genetic factors.

Strengths and Limitations.

The strengths of this study include (i) a sufficiently large sample to estimate the prevalence of OSA and conduct analyses stratified according to potential effect modifiers, (ii) the use of rigorous and standardized tools for data collection and utilization of qualified and competent personnel in all measurements, and (iii) presence of a comparative group in this study made the demonstration of outcome measure risk more robust. Nonetheless, this study is not short of limitations. The cross-sectional nature and convenience sampling method technique utilized in this study cannot preclude bias and limits both causality exploration and generalizability of findings. Prospective studies on the longitudinal association between OSA and hypertension incidence will help elucidate the true nature and magnitude of this intriguing association. Lastly, the gold standard for the diagnosis of OSA is an overnight polysomnography; however, because of its limited availability, complex technical support, and high cost, this study relied on a self-reported but validated assessment modality.

Conclusion.

In conclusion; OSA, a largely modifiable CVD risk factor is considerably common among hypertensives in a tertiary health care setting in Tanzania. Positive family history of OSA was found to be the strongest associated factor; however, excess body weight proved to be the strongest modifiable risk factor. In view of its pervasiveness, OSA ought to be an integral part of the medical evaluation in hypertensive individuals. Furthermore, a multipronged approach to curb the escalating obesity epidemic is paramount in the battle against sleep-related breathing disorders.

List Of Abbreviations.

95% CI, 95% Confidence Interval; BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; DBP, diastolic blood pressure; EDS, excessive daytime sleepiness; FBG, fasting blood glucose; JKCI, Jakaya Kikwete Cardiac Institute; OR, Odd Ratio; OSA, Obstructive sleep apnea; RBG, random blood glucose; SSA, sub Saharan Africa; SBP, systolic blood pressure.

Declarations.

Ethics approval and consent to participate.

The study protocol was submitted to, and approved by the Ethical Committee, of the Jakaya Kikwete Cardiac Institute. Written informed consent was obtained from all study participants. This research was conducted in accordance with the Declaration of Helsinki.

Availability of data and materials.

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Consent to publish.

Not applicable

Competing interest.

The authors have no conflict of interest to declare.

Author contributions.

PP and LRM conceived the study. MK, JM, NM, and HJS conducted all the interviews, as well as anthropometric and blood pressure measurements. ZM entered all the data. HM, SB, SW, and MJ participated in patient management including counseling. PP performed all the data cleaning and analysis. The corresponding author (PP) wrote the first draft of the manuscript, and other authors contributed to and approved it. All authors made the decision to submit the manuscript for publication. All authors undertake responsibility for the accuracy and integrity of the analysis.

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Figures

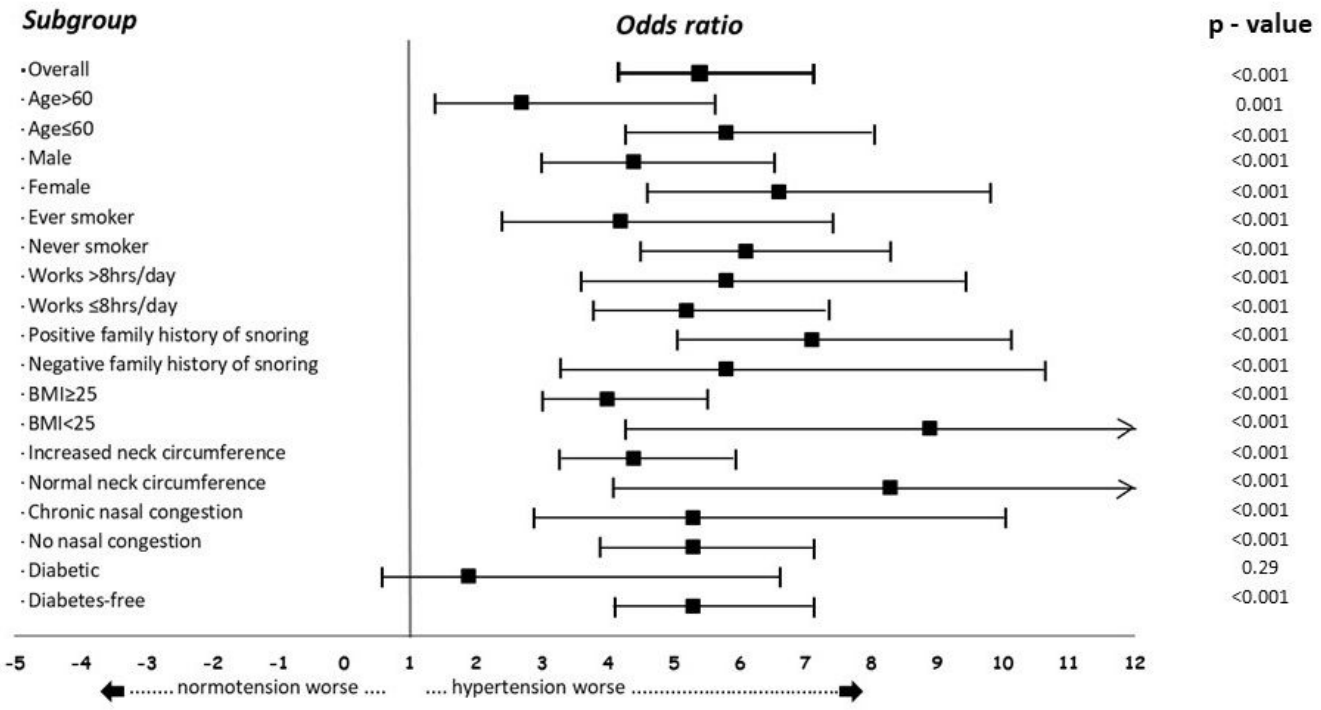

Figure 1

Odd ratios for OSA by hypertension status. This forest plot shows the odd ratios (black squares), 95% CIs (horizontal lines), and p-values for the interaction between OSA and any subgroup variable by

hypertension status.

Supplementary Files

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