Sleep Medicine 58 (2019) 42–47

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Prevalence and risk factors of people at-risk of obstructive sleep apnea in a rural community of Odisha, India: a community based cross-sectional study

Anurag Choudhury [a], Dipanweeta Routray [b, *], Swagatika Swain [a], Aswin Kumar Das [a]

[a] S.C.B. Medical College, Cuttack, Odisha, India
[b] Department of Community Medicine, S.C.B. Medical College, Cuttack, Odisha, India

ARTICLE INFO

Article history:
Received 2 August 2018
Received in revised form
23 December 2018
Accepted 25 March 2019
Available online 28 March 2019

Keywords:
Berlin questionnaire (BQ)
Multi-staged sampling
Obesity
Obstructive sleep apnea (OSA)
Prevalence
Rural Odisha

ABSTRACT

Background: Obstructive sleep apnea (OSA) is a progressive sleep disordered breathing condition characterized by repeated episodes of upper airway collapse during sleep. Despite being the most common sleep apnea, it often remains undiagnosed and untreated, especially in rural communities. There is a paucity of literature estimating the prevalence of the disease and associated risk factors from the rural population of Odisha.

Methods: It is a cross-sectional study that was conducted in a rural community of Odisha, India to find out the prevalence of people living with the risk of OSA. Multi-staged sampling was done. Participants were evaluated using the Berlin questionnaire (BQ) after obtaining informed consent. Data were analyzed using SPSS with the significance level set at 95%. Univariate and multivariate analysis was done to evaluate the risk factors associated with the condition.

Results: Out of 200 community dwellers surveyed using the BQ, 25% had a high likelihood of OSA. Among these high risk subjects, snoring was reported by 70%, excessive daytime sleepiness by 50%. Out of those who were at risk of OSA, 28% were smokers and 48% reported alcohol use. The associated risk factors were age >35 years (OR = 4.5, 95% CI = 1.4–13.8, $p < 0.05$), BMI $\geq$ 25 kg/m^2 (aOR = 3.5, 95% CI = 1.2–10.5, $p < 0.05$), alcoholism (aOR = 4.5, 95% CI = 1.8–11.1, $p = 0.001$), and hypertension (aOR = 11.5, 95% CI = 4.7–28, $p < 0.001$). The prevalence was not affected by the type of diet (vegetarian vs non-vegetarian), use of tobacco or tea consumption.

Conclusion: OSA is highly prevalent in the rural community of Odisha. Independent risk factors associated with such subjects were obesity, regular alcohol consumption, and hypertension. Further prevalence studies are recommended.

1. Introduction

Obstructive sleep apnea (OSA) is a highly prevalent sleep-disordered breathing condition with potentially life-threatening complications affecting mostly the middle and old aged population globally. It is caused by repetitive obstruction of the upper airway during sleep resulting in partial or complete cessation of airflow [1]. Apnea is defined as complete cessation of airflow for at least 10 s whereas reduction of airflow by $\geq$ 30% for $\geq$10 s with either $\geq$3% decrease in oxygen saturation or arousal from sleep is termed as hypopnea [2]. Patients with obstructive sleep apnea experience symptoms like loud snoring, frequent arousals, and disruption of sleep [1,3]. Disturbed sleep further results in excessive daytime sleepiness, impaired concentration and increased risk of road traffic accidents [1,4,5]. Various health-related complications associated with OSA are hypertension, diabetes mellitus, cardiorespiratory complications, metabolic complications, cognitive impairment which leads to unanticipated admissions to ICU [1,3–7]. The etiology of OSA is multifactorial where old age, male gender, and craniofacial abnormalities constitute the non-modifiable risk factors and obesity, smoking and alcohol are the major modifiable risk factors [3,5,8,9]. Out of these, obesity is the most important preventable risk factor [8]. Studies report one standard deviation increase in body mass index (BMI) increases the prevalence of the disease by four folds [5,8]. Prevalence of OSA in India varies from 4.6% to 27.3% using questionnaire method and from 1.7% to 12.2% using two staged

* Corresponding author.
E-mail address: drdipanweeta@gmail.com (D. Routray).

https://doi.org/10.1016/j.sleep.2019.03.014
1389-9457/

questionnaire-cum-polysomnography method respectively [10]. Since performing polysomnography is not only expensive but also requires a sleep laboratory with skilled technicians, various validated questionnaires are used in resource limited setting to screen out OSA cases in a community [1,5,11]. Among the various questionnaires available, the Berlin questionnaire (BQ) has the best negative predictive value (80–90%) along with high sensitivity (73.1–93.8%) and specificity (89.1–95.7%) for the screening of at-risk OSA cases [1,4,10,12–14]. Despite the high prevalence and deadly complications associated with the disease, 85% cases remain undiagnosed partly because of lack of information to primary care physicians and partly due to expensive diagnostic tests [3,10]. Since there is a paucity of studies from the rural population of Odisha in India, the present study is designed to estimate the prevalence of people at risk of OSA using the BQ, to study the socio-demographic parameters and assess their associated risk factors in a rural community of Odisha.

2. Methodology

2.1. Study setting and design

It is a community based cross-sectional study conducted in a duration of six months (September 2016 to February 2017) by the Department of Community Medicine of a tertiary care teaching hospital of the central revenue zone of Odisha. After getting approval from the Institutional Ethics Committee (IEC), the survey was carried out by paying visits from house to house in the selected community.

2.2. Subjects

The study subjects comprised adults of both sexes residing in the rural community of central revenue zone for at least six months. Persons with diagnosed insomnia, major depression (or other psychiatric illnesses), congestive heart failure, chronic kidney diseases, severe anemia, or cerebrovascular diseases (stroke) were excluded. Moribund or severely ill subjects were also excluded from the study.

Sample size was calculated to be 190 by taking the prevalence of 13.7% with an absolute precision of 5% [11,15,16]. Taking a non-response rate of 10%, the estimated sample size was 210. The power of the study was calculated to be 83.8% at this sample size.

2.3. Sampling

In this study, we performed multi-staged sampling. The sampling unit was a household with an eligible participant. In each stage, simple random sampling was done using lottery method except in the final stage of choosing the households, where the random number table was used. In the first stage, out of the 10 districts under the central revenue zone of Odisha, one district (Kendrapara) was selected randomly. Kendrapara district covers 12 blocks and 1592 villages with 1,440,361 populations; 94.2% population of Kendrapara district lives in rural areas of villages [17]. In the second and third stages, one out of 12 blocks (ie, Rajnagar), and five out of 12 villages were selected respectively, by lottery. Important sources of livelihood in Rajnagar block are farming, working as daily wage laborers, and cow rearing. The list of households in those villages was obtained from the Accredited Social Health Activist (ASHA) workers. In each village, 42 households were visited randomly using random number table. From each household, one eligible person was interviewed in presence of their bed partner after taking written informed consent.

2.4. Questionnaire

A predesigned, pretested and validated questionnaire was used for data collection. It contained questions on socio-demographic profile (age, sex, and socioeconomic status), lifestyle factors (dietary habits, alcohol consumption, and tobacco use), BQ criteria and other possible risk factors. Body mass index (BMI) was classified according to the new Asian criteria [18]. In our study, the socioeconomic status of the participants was assessed using Uday Pareek's scale [19]. Blood pressure was measured using mercury sphygmomanometer in right arm sitting posture in the home environment. Two readings were taken in a gap of 15 min and an average was noted. Persons who had already been diagnosed with hypertension and those with BP $\geq$ 140/90 mmHg at the time of the study were included under hypertensives. The questionnaire was validated by translating into local language (Odia) and was reviewed by a group of experts.

2.5. Statistical analysis

Data were entered using Microsoft Excel 2010 and analyzed using Statistical Package for Social Sciences version 18 (PASW statistics for Windows, Chicago: SPSS Inc.).

Descriptive statistics were used and the results were expressed as mean ± standard deviation or frequency and percentage. Normally distributed quantitative and categorical variables were compared using Students t-test and Pearson Chi–Square test respectively. The risk factors associated with lifestyle variables were dichotomized into two categories; predominant diet type as vegetarian or non-vegetarian, socioeconomic status (lower/middle class), tea and smoking (either daily or occasionally), alcohol consumption (regularly/occasionally), and predominant tobacco chewers (current users/non-users). Odds ratios (OR) and 95% confidence intervals (CI) were determined. Risk factors showing association with an α value of <0.25 in univariate analysis were considered for use in multivariate analysis. Adjusted odds ratio (aOR) was calculated using logistic regression to identify significant independent risk factors for people at risk of OSA. Hosmer and Lemeshow tests were used to examine the fitness of the model. Asymptotic 2-tailed p value of <0.05 was considered statistically significant.

3. Results

Out of the 223 households visited, 200 community dwellers could be surveyed using the BQ, and 25% (50/200) had a high likelihood of OSA. The average age of the participants was 50 years (±16.3 years). People in the age group 36–60 years comprised of 43.5% (n = 87) and those in the age group >60 years comprised of 35.5% (n = 71) of the participants. Mean BMI of the participants was 20.4 kg/m^2 (±4.08).

Males comprised of 62.5% (n = 125) and 37.5% (n = 75) were females as more number of males consented to participate. According to Uday Pareek's scale for socioeconomic status [19], 50.5% (n = 101) of participants were from lower socioeconomic status. Middle class participants constituted 49.5% (n = 99/200) which included upper middle class (n = 20/99; 20.2%), middle class (n = 30/99; 30.3%), and lower middle class (n = 49/99; 49.4%). Asian criteria for the classification of BMI [18] reported 9% of the participants to be overweight (BMI: 23–24.99 kg/m^2) and 13% as obese (BMI $\geq$25 kg/m^2). Vegetarians comprised of 68% (n = 136) and the rest 32% were non-vegetarians (n = 64). Tea was consumed daily by 75% of the participants. Current users of tobacco, predominantly by chewing and by smoking consisted of 32% (n = 64) and 18.5% (n = 37) respectively. Regular users of alcohol (or *Handia,*

a local alcoholic drink) consisted of 21.5% of participants (n = 43). Hypertension was present in 40.5% participants (n = 81). [Table 1]

Table 2 demonstrates the distribution of symptoms suggestive of OSA as experienced by both males and females. In our study, the majority of participants reported tiredness or fatigue after waking up (n = 145) followed by continually feeling tired during their wake time (n = 114). Snoring as reported by bed partners was present in 32.5% of the participants (n = 65/200). Nodding off during driving or daily work was present in 58.8% of males and 41.2% of females. Most of the symptoms of OSA commonly occurred in males except spells of apnea (ie, breathing cessation) which was more commonly associated with females.

Table 3 reports the complete symptom profile of the study population based on BQ. Among the at-risk OSA positive cases, 70% of participants (n = 35/50) complained of snoring with approximately 23% of participants snoring every day. While 30% of the participants (n = 15/50) experienced tiredness or fatigued after waking from sleep, 26% of participants (n = 13/50) felt tiredness during their wake time. Half of the participants (n = 25/50) accepted to have nodded off or fallen asleep while driving or during work. Hypertensives comprised of 82% of the participants at risk of OSA (n = 41/50). Only 6% of participants had BMI >30 kg/m^2 among the at-risk OSA subjects.

Risk factors assessment from Table 4 shows increasing age (>35 years), BMI (>23.0), hypertension, predominant diet type, chewing tobacco currently, as well as daily smoking and regular consumption of alcohol (or *Handia*) are independently associated with increased risk of OSA on univariate analysis ($p < 0.25$). On multivariate analysis and adjusting the odds ratio, it was seen that BMI $\geq$25 kg/m^2, hypertension and regular alcohol consumption (including *Handia*) are significantly associated with increased risk of OSA ($p < 0.001$). Participants in the age group 36–60 years have four fold increased risk of developing OSA than the participants in the age group 18–35 years. BMI cut-offs at 23.0 kg/m^2 (overweight according to Asian criteria) posed an increased risk for developing OSA with an adjusted odds ratio of 2.4 ($p = 0.058$). Hypertension is independently associated with increased risk of OSA with an odds ratio of 11.5 ($p < 0.001$). As revealed by the dietary assessment, being a predominantly vegetarian or non-vegetarian and drinking tea on a daily basis did not pose any significant risk for developing OSA ($p > 0.05$).

Table 1
Clinico-social and demographic profiles of study subjects (N = 200).

Parameters	Mean ± SD; n (%)
Age (years)	50.6 ± 16.3
18–35 y	42 (21)
36–60 y	87 (43.5)
> 60 y	71 (35.5)
Sex	
Male	125 (62.5)
Female	75 (37.5)
Socioeconomic status	
Lower class	101 (50.5)
Middle class	99 (49.5)
Height (cm)	
Male	164.2 ± 8.01
Female	152.6 ± 6.6
Weight (kg)	
Male	55.2 ± 11.6
Female	47.7 ± 10.8
BMI (kg/m^2)	20.45 ± 4.08
< 18.5	65 (32.5)
18.5–22.99	91 (45.5)
23–24.99	18 (9)
≥ 25	26 (13)
Diet type	
Vegetarian	136 (68)
Non-vegetarian	64 (32)
Tea	
Daily	150 (75)
No/occasionally	50 (25)
Tobacco chewing	
Current users	64 (32)
Non-users	136 (68)
Smoking	
Daily	37 (18.5)
Non-users/occasionally	163 (81.5)
Alcohol	
Regular users	43 (21.5)
Non-users/occasionally	157 (78.5)
Hypertension	
Yes	81 (40.5)
No	119 (59.5)
At risk OSA outcome	
OSA Present	50 (25)
OSA Absent	150 (75)

Table 2
Gender wise distribution of symptoms suggestive of OSA among the participants (N = 200).

Symptoms	Males [n (%)]	Females [n (%)]	Total [n (%)]
Snoring	41 (63.1)	24 (36.9)	65 (32.5)
Snores disturbing others	13 (61.9)	8 (38.1)	21 (10.5)
Quit breathing	18 (38.3)	29 (61.7)	47 (23.5)
Tired/feeling fatigued after sleep	79 (54.5)	66 (45.5)	145 (72.5)
Tired during wake time	62 (54.4)	52 (45.6)	114 (57)
Nodded off during driving/work	40 (58.8)	28 (41.2)	68 (34)

4. Discussion

Despite deliberate efforts made by researchers in India in the last decade, OSA still remains largely unrecognized and undiagnosed, especially in the resource-limited settings [10]. Compounding the lack of awareness on the part of the patient or their bed partners, few health professionals have the knowledge necessary to make the diagnosis [3,10]. OSA is a chronic and progressive disease of growing importance because of its associated neurocognitive, cardiorespiratory and metabolic sequelae [7]. An accurate diagnosis of OSA can be made using Polysomnography or Home sleep apnea testing methods. Medical therapy with continuous positive airway pressure (CPAP)/biphasic positive airway pressure (BiPAP) is the mainstay treatment modality. However, both the diagnosis and management of OSA using these modalities is expensive in a resource-limited setting like India [3,10]. Evaluating people at risk of OSA using simpler techniques and promoting lifestyle changes can help prevent this condition in rural communities. Various studies have validated certain questionnaires to screen out OSA cases in communities. The BQ has the best negative predictive value (80–90%) along with high sensitivity (73.1–93.8%) and specificity (89.1–95.7%) [4,10,12–14].

There are a limited count number of studies conducted in rural communities to estimate the prevalence of OSA. The present study is first of its kind to estimate the prevalence of people at risk of OSA along with the assessment of risk factors, in a rural community of Odisha in India.

Most studies have reported snoring as the major presenting symptom [8,10], however, our study reported feeling tired or fatigued after waking up from sleep as the most common symptom (72.5%). Only one-third of participants in our study had the habit of snoring (n = 65/200), although the prevalence of snoring was as high as 70% among the OSA positive cases. Out of every 10 people screened, seven reported feeling tired or fatigued after waking up as the most common symptom. The manifestation of weakness or fatigue could be due to various medical conditions like

Table 3
Symptom profile of study subjects at risk of OSA as per Berlin Questionnaire (N = 200).

Berlin Questionnaire	OSA Present (n = 50)	OSA Absent (n = 150)
Category 1	n (%)	n (%)
Do you snore?		
Yes	35 (70)	30 (20)
No	9 (18)	108 (72)
Don't know	6 (12)	12 (8)
Total	50	150
Your snoring is?		
Loud as breathing	15 (42.8)	14 (46.6)
Loud as talking	8 (22.8)	6 (20)
Louder than talking	8 (22.8)	9 (30)
Very loud	4 (11.4)	1 (3.3)
Total	35	30
How often do you snore?		
Almost everyday	8 (22.8)	8 (26.6)
3–4 times/week	23 (65.7)	9 (30)
1–2 times/week	4 (11.4)	11 (36.6)
1–2 times/month	0 (0)	2 (6.6)
Never/almost never	0 (0)	0 (0)
Total	35	30
Has your snoring ever bothered others?		
Yes	15 (42.8)	6 (20)
No	20 (57.1)	24 (80)
Total	35	30
Has anyone noticed you quit breathing during sleep?		
Almost everyday	0 (0)	0 (0)
3–4 times/week	2 (5.7)	4 (11.4)
1–2 times/week	5 (14.2)	1 (2.8)
1–2 times/month	4 (11.4)	5 (14.2)
Never/almost never	24 (68.5)	20 (57.1)
Total	35	30
Category 2		
How often do you feel tired/fatigue after sleep?		
Almost everyday	15 (30)	22 (14.6)
3–4 times/week	18 (36)	24 (16)
1–2 times/week	10 (20)	41 (27.3)
1–2 times/month	0 (0)	15 (10)
Never/almost never	7 (14)	48 (32)
Total	50	150
During your wake time, do you feel tired/fatigued/not up to par?		
Almost everyday	13 (26)	9 (6)
3–4 times/week	9 (18)	12 (8)
1–2 times/week	17 (34)	37 (24.6)
1–2 times/month	2 (4)	15 (10)
Never/almost never	9 (18)	77 (51.3)
Total	50	150
Have you ever nodded off/fallen asleep while driving vehicle?		
Yes	25 (50)	43 (28.6)
No	25 (50)	107 (71.3)
Total	50	150
How often you nodded off/fallen asleep while driving vehicle		
Almost everyday	10 (20)	9 (6)
3–4 times/week	8 (16)	14 (9.3)
1–2 times/week	7 (14)	15 (10)
1–2 times/month	0 (0)	5 (3.3)
Never/almost never	25 (50)	107 (71.3)
Total	50	150
Category 3		
Do you have high BP?		
Yes	41 (82)	40 (26.6)
No	9 (18)	110 (73.3)
Don't know	0 (0)	0 (0)
Total	50	150
BMI > 30 kg/m^2		
Yes	3 (6)	1 (0.6)
No	47 (94)	149 (99.3)
Total	50	150

anemia, diabetes, hypothyroidism and, insomnia [20–25]. Certain occupations that involve working at night such as bus/truck drivers, security guards etc., are also potential confounders which can present with symptoms similar to that of OSA [26,27]. However, anemic and insomnia patients were already restricted from our study, and none of the participants under our survey worked night shifts. Nodding off during day-to-day work or driving is an independent risk for road traffic accidents, which was present in every six out of 10 male participants. Al-Jewair et al., reported that individuals with OSA have 2–10 times increased risk of road traffic accidents than those without OSA [3].

Among the sociodemographic factors, people aged 35 years or older have a greater risk of developing OSA which is similar to other studies [1,3,5,28–30]. The odds of developing OSA are 4.5 times higher in the age group 36–60 years and 3.2 times higher for participants more than 60 years when compared to their younger counterparts (18–60 years). The reason for the declining risk in the elderly group is still controversial and is explained by Punjabi et al., and Young et al., where they reported the prevalence of OSA plateaus after age 60 years possibly due to relative increase in the mortality from OSA after 60 years [5,30]. Male gender is known to be significantly associated with OSA [3,5]. Yet, the present study does not show the gender preponderance as supported by the findings of other studies carried out in Lucknow (Uttar Pradesh, India) and Brazil [10,12]. Punjabi et al., mentioned that males are at increased risk for OSA, and the symptoms experienced by both genders are different [5].

Obesity is an independent risk factor for developing OSA. Most of the earlier studies had taken cut-off of BMI >30 kg/m^2 to assess the risk for OSA [3,8,10,31]. We took cut-off levels for BMI according to Asian criteria [18] to demonstrate that being obese (BMI $\geq$ 25 kg/m^2) can pose an increased risk of developing OSA. The risk of developing OSA significantly increases as the BMI crosses 25 kg/m^2 (aOR = 3.553). We inferred from our study that being overweight (BMI $\geq$ 23 kg/m^2) increases the odds of OSA [OR: 3.065(1.5–6.261), aOR: 2.399 (0.97–5.931)], although statistical significance is not established. The Kuprio sleep apnea group reported that obesity and OSA are bi-directionally associated; obesity leads to increased risk of upper airway collapse resulting in sleep fragmentation, which further increases appetite leading to weight gain and obesity [31]. Leptin, "the hormone of energy expenditure" inhibits appetite in the hypothalamus by counteracting the effects of orexigenic hormone neuro-peptide Y and ghrelin [32,33]. It also enhances the synthesis of appetite suppressant alpha-melanocyte stimulating hormone (αMSH) [32]. Sleep fragmentation attenuates leptin-signaling pathways in hypothalamus leading to an increase in ghrelin to leptin ratio [33]. This leads to uncontrolled appetite and food intake resulting in weight gain and obesity [32,33]. Sleep fragmentation disrupts energy homeostasis by inducing endoplasmic reticulum stress and activating unfolded protein response in the hypothalamus, which ultimately results in weight gain [32]. The present study also reveals that the prevalence of at-risk OSA considerably increases from 21.8% in the study population to 46.1% in the population having BMI $\geq$25 kg/m^2, which means every other obese person is at risk of having OSA, which is in accordance with other studies [10].

Hypertension and metabolic abnormalities are the root causes of cardiovascular diseases. There is significant overlap between OSA and systemic hypertension [34]. Upper airway collapse in OSA leads to intermittent hypoxia which results in overactivation of sympathetic output and endothelin system [34]. Thus, a cascade of changes occurs which further leads to elevated adrenergic tone resulting in hypertension [34]. In our study, hypertension was associated with 82% of OSA positive subjects and 50.6% of hypertensives are estimated to have OSA. Further analysis reported hypertensives are at 12 times increased risk for developing OSA than normotensives. Similar findings have been reported by Desalu et al., and others [5,8,10,28,35,36]. The risk of OSA remains unaffected by

Table 4
Risk factors assessment of subjects at risk of OSA.

Risk factors	OSA Present (n = 50)	OSA Absent (n = 150)	Unadjusted OR (95% CI); p-value	Adjusted OR (95% CI); p-value
Age groups				
18–35 y	4	38	–	–
36–60 y	28	59	4.508 (1.465–13.876); p = 0.009	
>60 y	18	53	3.226 (1.011–10.3); p = 0.048	
Sex				
Male	29	96	0.777 (0.404–1.493); p = 0.448	–
Female	21	54		
Socio-economic status				
Lower	24	77	1.143 (0.602–2.168); p = 0.683	–
Middle	26	73		
Height (cm)				
Male	165.03 ± 7.6	164.03 ± 8.1	1.016 (0.964–1.071); p = 0.557	–
Female	151.5 ± 6.4	153.07 ± 6.7	0.965 (0.892–1.043); p = 0.37	
Weight (kg)				
Male	60.2 ± 14.5	53.7 ± 10.2	1.049 (1.011–1.089); p = 0.008	–
Female	51.1 ± 10.6	46.4 ± 10.6	1.042 (0.993–1.093); p = 0.088	
BMI ≥ 25 kg/m^2				
Yes	12	14	3.068 (1.31–7.18); p = 0.008	3.553 (1.2–10.46); p = 0.021
No	38	136		
BMI ≥ 23 kg/m^2				
Yes	19	25	3.065 (1.5–6.261); p = 0.002	2.399 (0.97–5.931); p = 0.058
No	31	125		
Hypertension				
Yes	41	40	12.528 (5.59–28.080); p < 0.001	11.45 (4.68–27.99); p < 0.001
No	9	110		
Predominant diet type				
Non-Vegetarian	20	44	1.606 (0.825–3.126); p = 0.161	1.57 (0.673–3.646); p = 0.297
Vegetarian	30	106		
Tea				
Daily	40	110	1.45 (0.66–3.18); p = 0.346	–
No/occasionally	10	40		
Tobacco chewing				
Current users	22	42	2.02 (1.042–3.92); p = 0.036	1.855 (0.778–4.426); p = 0.163
Non-users	28	108		
Smoking				
Daily	14	23	2.15 (1.004–4.594); p = 0.046	2.033 (0.717–5.766); p = 0.182
Non-users/occasionally	36	127		
Alcohol				
Regular users	24	19	6.364 (3.054–13.265); p < 0.001	4.501 (1.822–11.123); p = 0.001
Non-users/occasionally	26	131		

the type of diet (predominantly vegetarian vs non-vegetarian diet) consumed by participants in this community (p > 0.05). Regular intake of tea did not increase the risk of OSA (95% CI: 0.66–3.18). We did not come across any study directly linking OSA with the type of diet and consumption of tea. Results of a meta-analysis reported there was insufficient published data to evaluate whether OSA is associated with caffeine intake [37].

Regular tobacco chewing or smoking are considered potential risk factors for OSA [5,9]. Smoking induces upper airway inflammation leading to swelling of uvula, resulting in narrowing of the air passage and its collapse [38,39]. Compton postulated that it is actually nicotine withdrawal which increases the risk for OSA rather than nicotine itself as nicotine being a potent stimulant of the parasympathetic system prevents the airway from collapsing [38]. The “rebound effect” of nicotine withdrawal is regarded as the main mechanism for the impact of smoking on sleep apnea [39–41]. During the first few hours of sleep, nicotine acts as a stimulant, thus reducing the number of apneas and hypopneas [37,39–41]. However, as nicotine withdrawal continues throughout the night, sleep apnea increases in the later part of the night thus explaining the “rebound effect” of nicotine withdrawal [37,39–41]. Although our study provides epidemiological evidence for a potential association between OSA and tobacco intake, an independent risk could not be established. Singh et al., reported similar findings while comparing the risk of OSA among smokers and non-smokers [10]. Sleep heart health study found an inverse association between smoking and OSA after adjusting for age and BMI [10]. Further study is needed to clarify this issue, as there is not enough evidence to confirm the association with tobacco [37].

Alcohol is another potential risk factor and is independently associated with OSA [5,10,42,43]. A body of evidence shows that alcohol causes relaxation of oropharyngeal muscles by acute brain depression and reduction of ventilatory responses to asphyxia, thus increasing the susceptibility for upper airway collapse [5,43]. The crucial event terminating obstructive apneas is the arousal induced by asphyxia; arousal from sleep causes a return of tone in the muscles of the tongue, soft palate and pharynx, and so opens the oropharyngeal airway [43]. Moreover, it depresses the arousal mechanism leading to increased duration of OSA symptoms [5,43]. The present study reports alcoholics or participants consuming alcohol regularly (including *Handia drink*) have a five times increased risk for OSA (95% CI: 1.8–11.1).

The major advantage of the present study is that it is first to estimate the prevalence of people living at risk of OSA in a rural community of Odisha. Screening followed by confirmatory tests like PSG will increase the accuracy of the study. Secondly, the adequate sample size, robust sampling method, and the use of a standardized questionnaire as a screening tool have added to the external validity of our study. Alternately, other potential risk factors like presence of co-morbidities such as diabetes mellitus, bronchial asthma, chronic obstructive pulmonary disease, and hypothyroidism may be explored in future studies to overcome the limitation of the current study. Further, quantification of tea intake, alcohol consumption, tobacco chewing, and smoking may be done

to enhance the reliability of the odds of association of OSA with the risk factors in this community.

5. Conclusion

This is a population-based survey from a rural community of Odisha in India estimating the prevalence of people at risk of OSA to be 25% (males and females being 14.5% and 10.5% respectively). Statistically significant and independent risk factors associated with such subjects were obesity (BMI $\geq$ 25 kg/m^2), regular alcohol consumption, and hypertension. Further studies on estimation of prevalence of OSA are warranted from other regions of Odisha by the BQ followed by polysomnography or with all feasibility considerations.

Conflict of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2019.03.014.

References

[1] Qaseem A, Dallas P, Owens DK, et al. Diagnosis of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2014 Aug 5;161(3):210–20.

[2] Malhotra RK, Kirsch DB, Kristo DA, et al. Polysomnography for obstructive sleep apnea should include arousal-based scoring: an American academy of sleep medicine position statement. J Clin Sleep Med 2018 Jul 15;14(07):1245–7.

[3] Al-Jewair TS, Nazir MA, Al-Masoud NN, et al. Prevalence and risks of habitual snoring and obstructive sleep apnea symptoms in adult dental patients. Saudi Med J 2016 Feb;37(2):183.

[4] Vasu TS, Grewal R, Doghramji K. Obstructive sleep apnea syndrome and perioperative complications: a systematic review of the literature. J Clin Sleep Med 2012 Apr 15;8(2):199.

[5] Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc 2008 Feb 15;5(2):136–43.

[6] Liao P, Yegneswaran B, Vairavanathan S, et al. Postoperative complications in patients with obstructive sleep apnea: a retrospective matched cohort study. Can J Anesth 2009 Nov 1;56(11):819.

[7] Silva KV, Rosa ML, Jorge AJ, et al. Prevalence of risk for obstructive sleep apnea syndrome and association with risk factors in primary care. Arq Bras Cardiol 2016 Jun;106(6):474–80.

[8] Barnes M, Goldsworthy UR, Cary BA, et al. A diet and exercise program to improve clinical outcomes in patients with obstructive sleep apnea–a feasibility study. J Clin Sleep Med 2009 Oct 15;5(5):409.

[9] Kashyap R, Hock LM, Bowman TJ. Higher prevalence of smoking in patients diagnosed as having obstructive sleep apnea. Sleep Breath 2001 Oct 1;5(4):167–72.

[10] Singh A, Prasad R, Garg R, et al. A study to estimate prevalence and risk factors of Obstructive Sleep Apnoea Syndrome in a semi-urban Indian population. Monaldi Arch Chest Dis 2017 May 18;87(1).

[11] Sharma SK, Ahluwalia G. Epidemiology of adult obstructive sleep apnoea syndrome in India. Indian J Med Res 2010 Feb 1;131(2):171.

[12] Modena DA, Cazzo E, Cândido EC, et al. Obstructive sleep apnea syndrome among obese individuals: a cross-sectional study. Revista da Associação Médica Brasileira 2017 Oct;63(10):862–8.

[13] Amra B, Javani M, Soltaninejad F, et al. Comparison of Berlin questionnaire, STOP-bang, and epworth sleepiness scale for diagnosing obstructive sleep apnea in Persian patients. Int J Prev Med 2018;9.

[14] Tan A, Yin JD, Tan LW, et al. Using the Berlin questionnaire to predict obstructive sleep apnea in the general population. J Clin Sleep Med 2017 Mar 15;13(3):427.

[15] Charan J, Biswas T. How to calculate sample size for different study designs in medical research? Indian J Psychol Med 2013 Apr;35(2):121.

[16] Naing L, Winn T, Rusli BN. Practical issues in calculating the sample size for prevalence studies. Arch Orofac Sci 2006;1:9–14.

[17] Office of the Registrar General & Census Commissioner, India, Ministry of Home Affairs, Government of India. Census of India. 2011. http://www.censusindia.gov.in/pca/SearchDetails.aspx?Id=442680.

[18] Misra A, Chowbey P, Makkar BM, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. JAPI 2009 Feb;57(2):163–70.

[19] Singh T, Sharma S, Nagesh S. Socio-economic status scales updated for 2017. Int J Res Med Sci 2017 Jun 24;5(7):3264–7.

[20] Bager P. Fatigue and acute/chronic anaemia. Dan Med J 2014 Apr 1;61(4):B4824.

[21] Singh R, Kluding PM. Fatigue and related factors in people with type 2 diabetes. Diabetes Educat 2013 May;39(3):320–6.

[22] Park H, Park C, Quinn L, et al. Glucose control and fatigue in type 2 diabetes: the mediating roles of diabetes symptoms and distress. J Adv Nurs 2015 Jul;71(7):1650–60.

[23] Kaltsas G, Vgontzas A, Chrousos G. Fatigue, endocrinopathies, and metabolic disorders. PM&R. 2010 May 1;2(5):393–8.

[24] Wang J, Wei Q, Liang W. Relationship of daytime fatigue and hyperarousal in patients with primary insomnia. Zhonghua Yixue Zazhi 2015 Aug;95(29):2355–8.

[25] Lee MH, Lee SA, Lee GH, et al. Gender differences in the effect of comorbid insomnia symptom on depression, anxiety, fatigue, and daytime sleepiness in patients with obstructive sleep apnea. Sleep Breath 2014 Mar 1;18(1):111–7.

[26] Meng F, Li S, Cao L, et al. Driving fatigue in professional drivers: a survey of truck and taxi drivers. Traffic Inj Prev 2015 Jul 4;16(5):474–83.

[27] Yumang-Ross DJ, Burns C. Shift work and employee fatigue: implications for occupational health nursing. Workplace Health & Saf 2014 Jun;62(6):256–61.

[28] Desalu OO, Onyedum CC, Adeoti AO, et al. Identifying patients at high risk for obstructive sleep apnoea syndrome in Nigeria: a multicentre observational study. Malawi Med J 2017;29(2):183–8.

[29] Cadelis G, Fayad OY. Prevalence of symptoms and risk of obstructive sleep apnea syndrome assessed by the Berlin Questionnaire among professionals of a health facility. Revue d'epidemiologieet de santepublique 2016 Dec;64(6):405–14.

[30] Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. Jama 2004 Apr 28;291(16):2013–6.

[31] Kuopio Sleep Apnea Group. Sustained improvement in mild obstructive sleep apnea after a diet- and physical activity–based lifestyle intervention: post-interventional follow-up. Am J Clin Nutr 1 October 2010;92(4):688–96. https://doi.org/10.3945/ajcn.2010.29485.

[32] Hakim F, Wang Y, Carreras A, et al. Chronic sleep fragmentation during the sleep period induces hypothalamic endoplasmic reticulum stress and PTP1b-mediated leptin resistance in male mice. Sleep 2015 Jan 1;38(1):31–40.

[33] Spiegel K, Tasali E, Penev P, et al. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med 2004 Dec 7;141(11):846–50.

[34] Hla KM, Young TB, Bidwell T, et al. Sleep apnea and hypertension: a population-based study. Ann Intern Med 1994 Mar 1;120(5):382–8.

[35] Sahlin C, Franklin KA, Stenlund H, et al. Sleep in women: normal values for sleep stages and position and the effect of age, obesity, sleep apnea, smoking, alcohol and hypertension. Sleep Med 2009 Oct 1;10(9):1025–30.

[36] Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Jama 2000 Apr 12;283(14):1829–36.

[37] Taveira KV, Kuntze MM, Berretta F, et al. Association between obstructive sleep apnea and alcohol, caffeine and tobacco: a meta analysis. J Oral Rehabil 2018 Nov;45(11):890–902.

[38] Compton J. Nicotine, smoking and sleep apnea. https://www.sleepresolutions.com/blog/smoking-and-sleep-apnea. [Accessed 2018 Jun 11].

[39] Kim KS, Kim JH, Park SY, et al. Smoking induces oropharyngeal narrowing and increases the severity of obstructive sleep apnea syndrome. J Clin Sleep Med 2012 Aug 15;8(04):367–74.

[40] Li Q, Zhou L, Lin Y. Smoking and OSA: a vicious cycle and synergistic effects. Austin J Sleep Disord 2015;3(2):1016.

[41] Lin YN, Li QY, Zhang XJ. Interaction between smoking and obstructive sleep apnea: not just participants. Chin Med J 2012 Sep;125(17):3150–6.

[42] Scrima L, Broudy M, Nay KN, et al. Increased severity of obstructive sleep apnea after bedtime alcohol ingestion: diagnostic potential and proposed mechanism of action. Sleep 1982 Sep 1;5(4):318–28.

[43] Issa FG, Sullivan CE. Alcohol, snoring and sleep apnea. J Neurol Neurosurg Psychiatr 1982 Apr 1;45(4):353–9.