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Pattern and severity of sleep apnea in a Saudi sleep center: The impact of obesity

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Abstract:

AIM: The aim of this study was to estimate the effect of obesity on the severity of obstructive sleep apnea (OSA) by assessing the relationship between OSA and body mass index (BMI).

MATERIALS AND METHODS: A cross-sectional study was conducted in 2017 among patients who had been referred to the sleep center at King Abdulaziz University Hospital (KAUH, Jeddah, Saudi Arabia) for polysomnography between January 2012 and September 2017. The data were abstracted from the medical records of these patients at KAUH. Initial data analysis included descriptive statistics; Chi-square test, *t*-test, and one-way ANOVA as appropriate were used to assess the associations between the variables.

RESULTS: The study included 803 patients; the average age of the patients was 45.9 years and 56.5% were male. About 70.4% were obese, 54% of whom were classified as having Class 3 obesity. Approximately, 75% patients had OSA. The prevalence of OSA was higher among obese patients (77.7%) compared to nonobese patients (22.3%). Moreover, the severity of OSA was higher in obese patients, with 85.3% of obese patients considered as having severe OSA.

CONCLUSION: Obesity is a considerable risk factor for developing OSA and could play a major role in increasing the severity of the disease. We encourage further studies on the impact of sedentary lifestyle and its association with OSA in Saudi Arabia, with an emphasis on the evaluation of the cost-effectiveness and burden of the disease.

Keywords:

Body mass index, obesity, sleep apnea

Introduction

Obstructive sleep apnea (OSA) is characterized by recurrent upper airway obstruction during sleep, resulting in decreased oxygenation inducing arousal from sleep.[1] The prevalence of OSA in the general population ranges from 9% to 38%, with obesity, aging, and male sex showing the highest incidence as related factors.[2] However, 82% of men and 93% of women remain undiagnosed. These cases, however, are clinically significant, indicating that the proportion of OSA may be much higher than expected.[3] In Saudi Arabia, the overall prevalence of obesity is 35.4%, and the country ranks 14th in the world with regard to the prevalence of obesity.[4]

Many studies have shown that obesity influences the severity of OSA. One such study conducted in Chile with 916 participants reported a significant association between general and central obesity and high OSA risk.[5] Another study done in Turkey on 499 patients who were diagnosed with OSA found that the risk of OSA was roughly proportional to the

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circumference of the upper neck.[6] This association is likely because high-fat accumulation in a patient's neck can weaken the pharyngeal muscle and decrease the pharyngeal cross-sectional area. Hence, obstruction of the upper airway, particularly while sleeping, is expected.[7] Furthermore, a study in Italy on 161 obese patients which examined the prevalence of sleep breathing disorders in these subjects demonstrated that >50% of the patients with a mean BMI higher than 40.0 kg/m^2 had OSA. Moreover, there appears to be an even higher percentage for women. In fact, the neck circumference of men and BMI of women seem to be the strongest and the most reliable predictive factors for the severity of OSA in obese patients.[8]

At present, the global burden of diseases and disabilities is dramatically affected by obesity, and the prevalence of obesity even in adolescents and children has significantly increased in the last few decades.[9,10] Hence, this study was to assess the relationship between OSA and BMI in patients attending the sleep center of King Abdulaziz University Hospital (KAUH) and estimate the effect of obesity on the severity of the disease.

Materials and Methods

This cross-sectional study was conducted among all patients referred to the sleep center of KAUH and had undergone polysomnography (PSG) between January 2012 and September 2017. The data were abstracted from the medical records of all these patients. KAUH is one of the biggest tertiary and referral centers in the western region of Saudi Arabia, with a capacity of 800 beds. The study was approved by the institutional review board, and written informed consent was obtained from all study participants.

A total of 878 patients referred to the sleep center of KAUH had undergone polysomnography (PSG) during the study period. Seventy five patients were excluded because of noninterpretable or missing data, such as BMI and apnea–hypopnea index (AHI) in the PSG report.

PSG is the standard test for the diagnosis of OSA in adult patients[11] and is used to diagnose and monitor sleep disorders related to breathing and movement or neurological causes. Respiratory parameters were collected, including AHI and minimum O_2 saturation during sleep (%). The AHI was determined as the number of apnea and hypopnea events per hour recorded during sleep. Apnea was defined as complete cessation of breathing during an interval of 10 s or more, while hypopnea is defined as a decrease of at least 30% in airflow, accompanied by a decrease in oxygen saturation of 4% or greater, with chest and abdominal movement. Patients who had an AHI of five episodes per hour or more were defined as having OSA.[2] Regarding the severity of OSA, an AHI of 5–14.9 events/h was considered mild, an AHI between 15 and 29.9 events/h was moderate, and an AHI of 30 events/h or more was considered severe.

The coordinator of sleep medicine and research center in the hospital gave the details of the PSG procedure, as applied in KAUH. Initially, a pretest questionnaire is given to the patient, and the procedure will be explained in detail. The patient is then admitted to the sleep center and tested with continuous monitoring of the vital signs. Observations made by a technician during the PSG are recorded in the technician's notes. The recordings are paused if the study is interrupted at any time. According to the KAUH protocol, the test is terminated when the patient wakes up and is unable to fall asleep again; otherwise, the test ends at 6:00 AM. At the end of the procedure, the patient is disconnected from the machine and discharged after the completion of a postsleep study questionnaire. The patient is given an appointment in the sleep disorder clinics, 2 weeks after the test, for a follow-up on the results.

For the study, body measurements including the height, measured to the nearest centimeter, and the weight, measured without shoes, were extracted from the PSG reports of the patients. Height and weight are measured routinely on hospital admissions and in the clinics. These measurements are taken by an experienced nurse staff who follows a stringent hospital protocol of taking accurate weight measurement to the nearest kilogram, using a balance beam scale. BMI was calculated by dividing the individual's weight, in kilograms (kg), by the square of the body height, in meters (m). Subjects were grouped into four BMI categories: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obese (>30.0 kg/m^2). Patients in the obese category were grouped into three classes: BMI between 30 and 34.99 was defined as Class 1, BMI between 35 and 39.99 was defined as Class 2, and BMI higher than 39.99 was defined as Class 3.[12] Laboratory results for the patients, including glycosylated hemoglobin, triglyceride, high-density lipoprotein, and low-density lipoprotein, as well as demographic data such as age and sex were included in the analyses to look for possible associations between these variables and different classes of BMI.

The data were analyzed using Statistical Package for the Social Sciences (SPSS) version 23 (IBM Corp., Armonk, NY, USA). Descriptive analyses included frequency distribution and percentages for categorical variables; mean and standard deviations computed for continuous variables. Chi-square test, *t*-test, and one-way ANOVA,

as appropriate, were used to assess the associations between the variables. All tests were performed at 5% level of significance.

Results

A total of 803 patients were included in the study. The demographic/clinical and Laboratory results for study participants are presented in Table 1. The mean age of the patients was 45.9 years, and 56.5% were male. About 70.0% patients were obese, 54% of whom were classifed as having Class 3 obesity.

PSG data showed that 74.8% patients had OSA, with a mean AHI of 19.93 (±21.58) and mean minimum O_2 saturation of 82.42 (±10.95). Males had a significantly higher percentage of OSA and mean AHI than the female patients [Table 1].

The prevalence of OSA was 77.7% among obese patients and 22.3% among non-obese patients; this was statistically significant, *P*-value <0.0001 [Table 2] with a *P*<0.001. The mean AHI was also significantly higher in obese (24.20 ± 22.86) than that in nonobese patients (12.11 ± 15.35). Furthermore, 50.1% of the obese patients who had OSA were classified as Class 3 obese.

Regarding the severity of OSA, AHI was positively correlated with BMI ($P < 0.0001$). About 85% of the patients with severe OSA were obese, compared to 14.7% who were non-obese, $P < 0.0001$ [Table 2]. The patients in Obese category 3 had significantly higher body weight, BMI, and sleep AHI [Table 3]. Moreover, of the obese patients with severe OSA, 54% were classified as having Class 3 obesity [Table 4].

Discussion

The main purpose of this study was to explore the relationship between BMI and OSA and to develop an understanding of BMI as a risk factor for OSA incidence and severity. Majority (74.8%) of patients included in this study were diagnosed with OSA. Patients are sent for PSG testing based on a combination of symptoms and risk factors indicative of OSA, including daytime snoring, sleepiness, and obesity. Therefore, the proportion of OSA cases among the patients referred to sleep center are not expected to reflect the prevalence of the disease in the general population. One study found that snoring has a positive predictive value for OSA in 84.7% of the patients.[13] Another study reported that snoring was a symptom in all the patients who experienced apnea while sleeping.[8] Besides, obesity has been described by various studies as a major risk factor for OSA.[14] Therefore, keeping in mind the commonality of snoring and obesity in our sample, a high prevalence of OSA was expected.

Table 1: Demographic and clinical characteristics of study participants by gender (*n*=803)

Variables	Male (*n*=454) *N* (%)	Female (*n*=349) *N* (%)	*P*-Value	Total
Age (years) Mean±SD	44.9±15.9	47.2±15.6	0.04	45.9±15.9
Weight (Kg) Mean±SD	99.8±30.9	96.6±27.5	0.13	98.4±29.5
Height (cm) Mean±SD	167.0±12.8	156.5±10.2	<0.0001	162.5±12.9
BMI				
Underweight	15 (60.0)	10 (40.0)	0.88	25 (31.0)
Normal	36 (65.5)	19 (34.5)	0.21	55 (6.8)
Overweight	110 (69.2)	49 (30.8)	<0.0001	159 (19.8)
Obese class 1	113 (72.9)	42 (27.1)	<0.0001	155 (19.3)
Obese class 2	62 (48.8)	65 (51.2)	0.07	127 (15.8)
Obese class 3	118 (41.8)	164 (58.2)	<0.0001	282 (35.1)
HbA1c Mean±SD	6.55±1.6	6.5±1.6	0.54	6.5±1.6
TG Mean±SD	1.53±0.9	1.5±1.5	0.48	1.5±0.9
HDL Mean±SD	1.1±0.4	2.3±10.3	0.21	1.6±6.9
LDL Mean±SD	2.9±0.9	3.1±0.9	0.12	3.1±0.9
Sleep AHI Mean±SD	23.5±23.1	16.9±18.9	<0.0001	19.9±21.6
Overall prevalence of OSA	359 (59.7)	242 (40.3)	0.002	601 (74.8)
OSA severity				
No OSA	95 (47.0)	107 (53.0)	0.002	202 (25.2)
Mild OSA	137 (56.4)	106 (43.6)	0.95	243 (30.3)
Moderate OSA	92 (55.1)	75 (44.9)	0.73	167 (20.8)
Severe OSA	130 (68.1)	61 (31.9)	<0.0001	191 (23.8)
Minimum O_2 saturation Mean±SD	82.6±10.9	82.5±10.9	0.95	82.42±10.95

Values are presented as mean±SD or *n* (%). BMI=Body mass index, TG=Triglyceride, HbA1c=Glycosylated hemoglobin, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, AHI=Apnea–hypopnea index, OSA=Obstructive sleep apnea, SD=Standard deviation

Another factor that may have contributed to the severity of OSA in our sample was the mean age (45.90 ± 15.86 years) of the patients. The sleep Heart Study conducted in 2002, reported that the prevalence and severity of OSA increased steadily with the age until 60 years of age when it reached a plateau.[15] Furthermore, the study showed that males had a significantly higher prevalence of OSA and mean AHI than females. These findings are in agreement with previous reports that in general, males had a higher prevalence of OSA than females.[16] In the present study, almost 30% of diagnosed males were found to have a severe form of OSA, which is in accordance with earlier studies.[17] This can be associated with the higher prevalence of both obesity[18] and snoring[19] in males. Moreover, studies have shown that BMI is the best predictor for OSA in females, while neck circumference is a better risk predictor for males.[8] However, the overall prevalence of OSA in morbidly obese women is still lower than that of males.[17] This observation leads to the conclusion that regardless of age and weight, the female population, in general, has lower prevalence of OSA compared to the male population.[20] The reasons for the differences between males and females are only partially understood; one of these reasons may be the difference in the structure and function of the upper airway in the sexes.[20] Although the present study could not distinguish between the effects of BMI and neck circumference on sleep apnea in both sexes, it does underline the crucial effect of BMI on the risk of having OSA in both men and women.

Table 2: Demographic and clinical characteristics of study participants by obesity (*n*=803)

Variables	Non-obese (*n*=239) *N* (%)	Obese (*n*=564) *N* (%)	*P*-Value*
Age (years) Mean±SD	44.34±15.63	46.55±15.92	0.073
Sex			
Male	161 (35.5)	293 (64.5)	<0.0001
Female	78 (22.3)	271 (77.7)	
Weight (Kg) Mean±SD	69.69±13.07	110.53±25.95	<0.0001
Height (cm) Mean±SD	164.75±10.97	161.74±13.51	0.001
BMI Mean±SD	24.78±5.59	43.72±20.24	<0.0001
HbA1c Mean±SD	6.52±1.63	6.50±1.60	0.935
TG Mean±SD	1.52±0.87	1.50±0.82	0.876
HDL Mean±SD	1.23±0.59	1.7975±8.05	0.545
LDL Mean±SD	2.94±0.92	3.09±0.96	0.194
Sleep AHI Mean±SD	12.11±15.35	24.20±22.86	<0.0001
OSA	134 (22.3)	467 (77.7)	<0.0001
OSA severity			
No OSA	105 (52.0)	97 (48.0)	<0.0001
Mild OSA	75 (30.9)	168 (69.1)	0.71
Moderate OSA	31 (18.6)	136 (81.4)	0.001
Severe OSA	28 (14.7)	163 (85.3)	<0.0001
Minimum O_2 saturation Mean±SD	83.43±10.80	82.20±10.96	0.166

Values are presented as mean±SD or *n* (%). **P* values were derived via independent *t*-test for categorical and continues variables, and Chi-square test for categorical variables. BMI=Body mass index, TG=Triglyceride, HbA1c=Glycosylated hemoglobin, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, AHI=Apnea–hypopnea index, OSA=Obstructive sleep apnea, SD=Standard deviation

The most relevant finding of this study was that the prevalence of OSA in obese patients was significantly higher than in nonobese patients. Moreover, AHI, a predictor for the severity of OSA, was found to be significantly higher in the obese population than that in the nonobese population. These findings suggest a positive correlation between obesity and the severity of OSA, as subjects with higher BMI scored higher AHI values on the PSG test. This result was expected as obesity is widely considered a risk factor for OSA and is thought to increase the severity of the syndrome. This effect of obesity on the severity of OSA may be caused by alterations in the normal airway functioning owing to excessive fat accumulation around the pharynx, leading to a reduction of the size of the airway passage.

Table 3: Comparison of demographic and clinical characteristics of study participants by different classes of obesity (*n*=566)

Variables	Obese Class 1 (*n*=155) Mean±SD	Obese Class 2 (*n*=127) Mean±SD	Obese Class 3 (*n*=281) Mean±SD	*P*-Value*
Age	48.66±16.74	48.45±14.52	44.53±15.85	0.011
Weight	88.97±13.15	99.44±12.71	127.39±24.18	<0.0001
Height	164.50±11.52	162.85±9.91	159.18±15.42	<0.0001
BMI	32.32±1.42	37.37±1.43	52.85±25.41	<0.0001
HbA1c	6.45±1.69	6.76±1.68	6.41±1.52	0.273
TG	0.79±0.08	0.80±0.10	0.85±0.07	0.679
HDL	0.37±0.04	16.35±2.29	0.66±0.07	0.211
LDL	2.99±0.85	3.26±1.10	3.06±0.95	0.220
Sleep AHI	21.48±19.24	21.12±21.47	27.08±24.92	0.011
OSA	127 (27.2)	106 (22.7)	234 (50.1)	0.938
Minimum O_2 saturation	82.79±9.56	81.44±11.91	82.22±11.267	0.611

Values are presented as mean±SD or *n* (%). **P* values were derived via one-way ANOVA for categorical and continues variables, and Chi-square test for categorical variables. BMI=Body mass index, TG=Triglyceride, HbA1c=Glycosylated hemoglobin, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, AHI=Apnea–hypopnea index, OSA=Obstructive sleep apnea, SD=Standard deviation

Table 4: Comparison between severity of obstructive sleep apnea and different classes of obesity (*n*=566)

OSA severity	Obese Class 1 (*n*=155) *N* (%)	*P*-Value*	Obese Class 2 (*n*=127) *N* (%)	*P*-Value*	Obese Class 3 (*n*=281) *N* (%)	*P*-Value*
No OSA	28 (28.9)	0.03	21 (21.6)	0.02	48 (49.5)	<0.0001
Mild OSA	46 (27.4)	0.93	48 (28.6)	0.05	74 (44.0)	0.08
Moderate OSA	40 (29.4)	0.1	24 (17.6)	0.64	72 (52.9)	0.01
Severe OSA	41 (25.2)	0.44	34 (20.9)	0.45	88 (54)	<0.0001

Values are presented as mean±SD or *n* (%). **P* values were derived via Chi-square test. OSA=Obstructive sleep apnea, SD=Standard deviation

Previous studies reported that there was a 32% increase in AHI score following an increase of 10% of body weight, leading to a 6-fold increase in the risk of developing severe OSA.[16] The results of the present study are consistent with the findings of multiple past studies, including a Chinese study done in 2002, which reported a higher AHI score in subjects with higher body mass compared to subjects with lower body mass, with values of AHI smaller than five reported for patients with a mean BMI of 24.4 and AHI values of >30 reported for patients with a mean BMI of 29.5.[21,22]

Similarly, a Turkish retrospective study conducted in 2011, which divided subjects into a control group (AHI <5) and an OSA group (AHI >5), found that the OSA group had a higher BMI (mean value of 32.15 ± 5.48) than the control group (mean value of 26.94 ± 5.1).[5] Based on these results, we can infer a linear relationship between increasing body weight and AHI; the latter being an indicator of the severity of OSA and progression from mild to severe OSA.

Of our obese study subjects who were diagnosed with OSA, 50.1% were classified as having Class 3 obesity, with a BMI of >39.99 kg/m^2. This finding correlates well with the other results of our study which show an important linear relationship between obesity and OSA since OSA is more prevalent in subjects with greater BMI.[5,8,17,19-23] However, this apparent linear relationship between BMI and OSA could result from the fact that our sample (n = 803) contained 564 subjects classified as obese (70.2%), 282 of which (~50% of the obese patients) were classified as Class 3 obese. Nevertheless, the results of the present study are in accordance with those of earlier studies which underscored the fact that higher BMI increased the risk of OSA.

A study conducted in the UK in 2012 among 50 years or older patients diagnosed with OSA, reported that of all of the obese subjects diagnosed with OSA during the period of their study, only 21.1% were classified with Class 3 obesity.[24] The difference in the results of UK study and our study may reflect disparities in the lifestyle and the obesity prevalence in Saudi Arabia (35.4%)[4] and England (25%).[25] In general, we can infer that OSA prevalence and its severity are higher in morbidly obese patients, which would represent a greater fraction of samples taken from countries with a prevalence of obesity.

Our study had a few limitations with regard to incomplete documentation. Many of the patients our hospital regularly admits do not return for follow-up, so there are always issues with missing data. Other limitations include inaccurate data from records and lack of information about ethnicity.

Conclusion

There was significantly greater prevalence and severity of OSA in obese patients compared to nonobese patients, particularly in males. Furthermore, the majority of patients with severe OSA were classified with Class 3 obesity. Therefore, obesity is a considerable risk factor for the development of OSA and could play a major role in increasing the severity of the disease. We encourage further studies on the impact of sedentary lifestyle and its association with OSA in Saudi Arabia, with an emphasis on the evaluation of the burden of the disease.

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Conflicts of interest

There are no conflicts of interest.

References

1. Senaratna CV, Perret JL, Lodge CJ, Lowe AJ, Campbell BE, Matheson MC, *et al.* Prevalence of obstructive sleep apnea in the general population: A systematic review. Sleep Med Rev 2017;34:70-81.
2. Sleep-related breathing disorders in adults: Recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep 1999;22:667-89.

3. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. Sleep 1997;20:705-6.
4. Dillinger J. The Most Obese Countries in the World. WorldAtlas; 2018. Available from: https://www.worldatlas.com/articles/29-most-obese-countries-in-the-world.html. [Last accessed on 2018 Aug 16].
5. Wosu AC, Vélez JC, Barbosa C, Andrade A, Frye M, Chen X, *et al.* The relationship between high risk for obstructive sleep apnea and general and central obesity: Findings from a sample of Chilean college students. ISRN Obes 2014;2014:871681.
6. Soylu AC, Levent E, Sarıman N, Yurtlu S, Alparslan S, Saygı A. Obstructive sleep apnea syndrome and anthropometric obesity indexes. Sleep Breath 2012;16:1151-8.
7. Brennick MJ, Pack AI, Ko K, Kim E, Pickup S, Maislin G, *et al.* Altered upper airway and soft tissue structures in the New Zealand obese mouse. Am J Respir Crit Care Med 2009;179:158-69.
8. Resta O, Foschino-Barbaro MP, Legari G, Talamo S, Bonfitto P, Palumbo A, *et al.* Sleep-related breathing disorders, loud snoring and excessive daytime sleepiness in obese subjects. Int J Obes Relat Metab Disord 2001;25:669-75.
9. Peeters A, Backholer K. Is the health burden associated with obesity changing? Am J Epidemiol 2012;176:840-5.
10. World Health Organization. Obesity. World Health Organization; 2008. Available from: http://www.who.int/topics/obesity/en/. [Last accessed on 2014 Apr 07].
11. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, *et al.* Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: An American academy of sleep medicine clinical practice guideline. J Clin Sleep Med 2017;13:479-504.
12. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. Arch Intern Med 1998;158:1855-67.
13. Romero E, Krakow B, Haynes P, Ulibarri V. Nocturia and snoring: Predictive symptoms for obstructive sleep apnea. Sleep Breath 2010;14:337-43.
14. Flemons WW. Clinical practice. Obstructive sleep apnea. N Engl J Med 2002;347:498-504.
15. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ, *et al.* Predictors of sleep-disordered breathing in community-dwelling adults: The sleep heart health study. Arch Intern Med 2002;162:893-900.
16. Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc 2008;5:136-43.
17. Daltro C, Gregorio PB, Alves E, Abreu M, Bomfim D, Chicourel MH, *et al.* Prevalence and severity of sleep apnea in a group of morbidly obese patients. Obes Surg 2007;17:809-14.
18. Al-Nozha MM, Al-Mazrou YY, Al-Maatouq MA, Arafah MR, Khalil MZ, Khan NB, *et al.* Obesity in Saudi Arabia. Saudi Med J 2005;26:824-9.
19. Wali SO, Abaalkhail BA. Prevalence and predictors of habitual snoring in a sample of Saudi middle-aged adults. Saudi Med J 2015;36:920-7.
20. O'Connor C, Thornley KS, Hanly PJ. Gender differences in the polysomnographic features of obstructive sleep apnea. Am J Respir Crit Care Med 2000;161:1465-72.
21. Ip MS, Lam B, Ng MM, Lam WK, Tsang KW, Lam KS. Obstructive sleep apnea is independently associated with insulin resistance. Am J Respir Crit Care Med 2002;165:670-6.
22. Malhotra A, White DP. Obstructive sleep apnoea. Lancet 2002;360:237-45.
23. Koeck ES, Barefoot LC, Hamrick M, Owens JA, Qureshi FG, Nadler EP. Predicting sleep apnea in morbidly obese adolescents undergoing bariatric surgery. Surg Endosc 2014;28:1146-52.
24. Wall H, Smith C, Hubbard R. Body mass index and obstructive sleep apnoea in the UK: A cross-sectional study of the over-50s. Prim Care Respir J 2012;21:371-6.
25. Public Health England; 2018. Available from: https://www.gov.uk/government/organisations/public-health-england. [Last accessed on 2018 Aug 16].