NEW RESEARCH

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The Prevalence and Severity of Obstructive Sleep Apnea in Severe Obesity: The Impact of Ethnicity

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Study Objectives: The South Asian population is at increased risk of cardiovascular disease. We compared the prevalence and severity of obstructive sleep apnea (OSA) in South Asians and white Europeans with severe obesity.

Methods: Data from consecutive patients attending a specialist weight management service were analyzed. Self-reported age, gender, and ethnicity were recorded. Objective measurements of blood pressure, body mass index (BMI), and apnea-hypopnea index (AHI) were also acquired.

Results: A total of 308 patients (72.7% women; 13% South Asian) were included, with mean age and BMI of 46 ± 12 y and 49 ± 8 kg/m^2, respectively. South Asians had significantly increased prevalence of OSA compared to white Europeans (85% vs. 66% [p = 0.017]) and were more likely to have severe OSA (42.5% vs. 21.6% [p = 0.015]). South Asians had significantly higher median AHI (24 events/h: interquartile range [IQR] 9.3-57.6 vs. 9 events/h: IQR 3.4-26.6; p < 0.01), significantly lower minimum oxygen saturation (76%: IQR 64% to 84% vs. 83%: IQR 77% to 87%; p < 0.01), and spent a significantly greater amount of time < 90% oxygen saturation (8.4%: IQR 1.0% to 24.3% vs. 2.4%: IQR 0.2% to 16.0%; p = 0.03). South Asian ethnicity, independent of demographics, BMI, and comorbidities, was associated with β = 1.84 (95%CI: 1.27-2.65) increase in AHI+1 compared to white Europeans. Furthermore, we confirmed other independent OSA risk factors including increasing age, BMI, and male gender (all p < 0.001).

Conclusions: Severely obese South Asians had significantly greater prevalence and severity of OSA than white Europeans. OSA may contribute to increased cardiovascular risk in South Asians compared to white Europeans with severe obesity. Mechanisms mediating the observed associations between these ethnicities require further investigation.

Keywords: Ethnicity, sleep disordered breathing, diabetes mellitus, South Asian, obstructive sleep apnea

Commentary: A commentary on this article appears in this issue on page 859.

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SCIENTIFIC INVESTIGATIONS

Obstructive sleep apnea (OSA) is a common disorder characterized by frequent episodes of complete or partial upper airway obstruction during sleep, with subsequent oxygen desaturation, and sleep disturbance. OSA has been identified as an independent risk factor for cardiovascular disease (CVD)[1-3] and is also associated with increased all-cause mortality.[4] Chronic metabolic diseases including type 2 diabetes and metabolic syndrome are widespread in those with OSA.[5,6] Furthermore, obesity is a major risk factor and a common characteristic of OSA. For example, those who are extremely obesity (defined as body mass index [BMI] ≥ 40 kg/m^2) have a higher prevalence of OSA with additional clinical complications.[7,8]

Compared to white Europeans, South Asians (those of Indian/Pakistani/Bangladeshi/Sri Lankan origin) residing in western countries, have a higher prevalence of type 2 diabetes, dyslipidemia, and CVD.[9] Considering these conditions commonly accompany OSA, it is possible that South Asians are at greater risk of OSA than white Europeans. This potential ethnic difference has not, however, been comprehensively examined, particularly in the severely obese. Two population studies from India reported OSA prevalence of 9.3% to 19.5%,[10,11] considerably higher than some western countries,[12] suggesting ethnicity may play a key role in OSA prevalence. OSA severity may also be different among these ethnic groups, but this notion has not yet been explored. A recent United Kingdom (UK) study compared the prevalence of OSA in South Asians and white Europeans but found no difference between groups.[13] This study, however, characterized OSA based on subjective responses to the Berlin questionnaire, which has not previously demonstrated a high level of diagnostic

BRIEF SUMMARY

Current Knowledge/Study Rationale: Risk of cardiovascular disease (CVD) is heightened in South Asians compared to white Europeans. Since obstructive sleep apnea (OSA) has a close connection with obesity and CVD, our study was conducted to investigate potential ethnic differences between South Asians and white Europeans with severe obesity, in relation to the prevalence and severity of OSA.

Study Impact: Our study demonstrated that OSA was more prevalent in South Asians than white Europeans. We also showed that South Asians had significantly more severe OSA and an increased number of comorbidities, suggesting the need for better clinical management and educational programs to target this ethnic group.

specificity[14] and does not provide an objective measure of OSA. We therefore sought to assess potential differences in objectively determined OSA prevalence and severity between severely obese South Asians and white Europeans. We specifically assessed a severely obese patient population, as this group is at increased risk of CVD. Based on the evidence that South Asians have a higher prevalence of metabolic and cardiovascular morbidities, we hypothesized that severely obese South Asians would have a higher prevalence and more severe OSA than white Europeans.

METHODS

Routine clinical data were prospectively collected from consecutive adult (≥ 18 years) patients attending the West Midlands regional specialist weight management service 2009-2011 at the Heart of England NHS Foundation Trust, Birmingham, UK. Over 10% of the West Midlands inhabitants (total 5.5 million) are South Asian.[15] All patients were referred to the service based on the following criteria: (1) BMI ≥ 35 kg/m^2 with at least one comorbidity, or (2) BMI ≥ 40 kg/m^2 without comorbidity. The aim of the specialist service is to provide assessment of obesity and its comorbidities, and development of a therapeutic plan that may include bariatric surgery. Patients and referring physicians are informed of the pathways and processes within the service and treatment options available.

In the specialist service, all patients are formally assessed for sleep disordered breathing (SDB). Anonymized data from patients attending the service with OSA assessment were included. Data available included age, gender, and ethnicity, objective anthropometry (height, weight, BMI), systolic and diastolic blood pressure (BP), and the presence of obesity-related comorbidities (type 2 diabetes [DM], hypertension and coronary artery disease [CAD]).

Respiratory Sleep Monitoring

Home sleep portable monitoring using the Embletta (Embla systems) was employed. Patients were given a demonstration of the equipment by a trained sleep physiologist on the afternoon of the test night. Measurement channels included airflow (nasal pressure device), chest and abdominal movements (inductance plethysmography), oxygen saturation (SpO_2), and heart rate. The sleep physiologist observed the patient undergoing self-application of the equipment before leaving the sleep center. Patients also completed a diary of sleep-wake times for the test night, which was completed the morning after the test night. This was used to determine sleep onset and wake as well as subjectively confirming that the patient achieved ≥ 4 h sleep duration. South Asian and white European patients did not differ in the interpretation of sleep diaries, which were in agreement with the respiratory data as seen by the movement artifacts. Equipment and diaries were returned the next day, and data were downloaded and reviewed. Studies with ≥ 4 hours of good quality respiratory signals were considered acceptable. A retest was offered if data were inadequate. All respiratory data were manually scored by blinded trained sleep-respiratory physiologists and rescored by a sleep physician for confirmation. All scorers were blinded to patient characteristics including name and ethnicity.

All parameters were scored based the American Academy of Sleep Medicine guidelines.[16] An apnea was defined as complete cessation of airflow ≥ 10 sec, while hypopnea was defined as reduction ≥ 30% of airflow with ≥ 4% reduction in oxygen saturation with the presence of chest and abdominal movement. The AHI was calculated as average number of episodes of apneas and hypopneas per hour of sleep. AHI was calculated for the whole night and did not differentiate supine and non-supine body position. Respiratory parameter data collected included: AHI, mean and minimum oxygen saturation during sleep (%), and percentage of time spent under 90% oxygen saturation while asleep. OSA was defined using the standard AHI cut-point ≥ 5 events per hour (events/h).[17] Mild, moderate, and severe OSA were defined as AHI of 5-15 events/h, 15-30 events/h, and ≥ 30 events/h, respectively.

The anonymized data collection for analysis was conducted as part of service evaluation and did not require formal ethics committee approval, as recommended by the UK National Research Ethics Service.[18]

Statistical Analysis

Statistical analysis was performed using SPSS version 19 (SPSS, Chicago, IL). Normality of continuous data was ascertained through visual inspection and the Kolmogorov-Smirnov test. Normally distributed data are reported as mean ± standard deviation (SD), while non-normally distributed variables are reported as median with interquartile range (IQR). Mann Whitney U-tests and independent *t*-tests were used for nonparametric and normally distributed data, respectively. Differences between categorical variables were analyzed using χ^2 test. We ran a series of Mann Whitney U-tests to assess the potential difference in AHI, mean O_2 saturation, minimum O_2 saturation, and percentage of time spent under 90% O_2 saturation according to ethnicity. Natural log transformation was performed for AHI+1 to ensure normal distribution prior to multivariate linear regression. Three models were explored for linear regression to examine if age, sex, ethnicity, and/or BMI were independent predictors of AHI. Models presented include Model 1, which was unadjusted; Model 2, which was adjusted for age, sex, ethnicity, and/or BMI, as appropriate; and Model 3, which was further adjusted for the presence of type 2 diabetes, hypertension, CAD, and insulin treatment. Clinically relevant results from log-transformed data were back-transformed, and results are reported as unstandardized beta coefficients (β) along with 95% confidence intervals (CI), for AHI+1. Results were considered statistically significant when $p < 0.05$.

RESULTS

Patient Characteristics

A total of 343 consecutive patients referred to the Specialist Weight Management Clinic underwent SDB assessment during the study period. Data from 4 patients were not analyzable; 7 patients had no BMI data available; and 24 patients were from another ethnic group and were excluded, leaving complete data on 308 patients for subsequent analyses.

The study sample characteristics are shown in **Table 1**. The majority of the sample was white European (87.3%), reflecting the ethnic composition of the local population.[15] The majority of patients were female (71.1%), in agreement with attendance at services catering for severely obese patients.[19] No significant

Table 1—Patient characteristics in 308 obese patients according to ethnic group

	White European (n = 268)	South Asians (n = 40)	p value
Age, years	46.6 ± 12.2	43.8 ± 10.9	0.17
Sex, n (%)			0.10
Male	74 (27.6)	16 (40.0)	
Female	194 (72.4)	24 (60.0)	
Weight, kg	133.2 ± 24.9	134.9 ± 30.9	0.71
BMI, kg/m^2	48.6 ± 8.0	49.8 ± 10.9	0.42
Systolic BP, mm Hg*	142 ± 18	142 ± 15	0.99
Diastolic BP, mm Hg*	87 ± 12	82 ± 14	0.09
Overall prevalence of OSA, AHI ≥ 5, n (%)	177 (66.0)	34 (85.0)	0.017
OSA severity, n (%)			0.015
No OSA, AHI < 5	91 (34.0)	6 (15.0)	
Mild OSA, AHI 5-15	77 (28.7)	10 (25.0)	
Moderate OSA, AHI 15-30	42 (15.7)	7 (17.5)	
Severe OSA, AHI > 30	58 (21.6)	17 (42.5)	
AHI	9.0 (3.4-26.6)	24.0 (9.3-57.6)	< 0.01
Comorbidities			
DM, %	33.1	60.0	< 0.01
HTN, %	36.6	42.5	0.48
CAD, %	7.3	7.5	0.96
Presence of comorbidities (DM, HTN, or CAD)			0.02
None, %	50.9	27.5	
One, %	28.7	37.5	
Two, %	16.4	32.5	
Three, %	4.0	2.5	
HbA1c, %	6.3 (5.8-7.3)	7.4 (6.3-8.6)	< 0.01
HbA1c, mmol/mol	45 (40-56)	57 (45-71)	< 0.01
% on diabetes medication	25.1	50.0	< 0.01
Number of DM medications			< 0.01
None, %	74.9	50.0	
One, %	10.5	10.0	
Two, %	10.2	30.0	
Three or more, %	4.4	10.0	

Data are reported as mean ± standard deviation or median (IQR), unless otherwise stated. OR, odds ratio; CI, confidence interval; BMI, body mass index; BP, blood pressure; AHI, apnea-hypopnea index (events/h); DM, diabetes mellitus; HTN, hypertension; CAD, coronary artery disease. *Blood pressure data presented were available in 212 patients. p values were calculated using either independent t-test, Mann Whitney U-test or χ^2, as appropriate.

differences were found between ethnic groups for age, sex, BMI, body weight, or the prevalence of hypertension or CAD. The percentage of South Asians with type 2 diabetes mellitus was, however, almost double that observed in white Europeans (60.0% vs. 33.1%, p < 0.01). A greater proportion of the South Asian group took diabetes medication (50.0% vs. 25.1%, p < 0.01) and had significantly worse glycemic control than white Europeans. South Asians also had a significantly greater prevalence of comorbidities than white Europeans (p = 0.02). In those with severe OSA (AHI > 30 events/h), South Asians were significantly younger (45 ± 13 years) than white Europeans (52 ± 11 years), p = 0.04.

Prevalence and Severity of OSA

The prevalence of OSA (AHI ≥ 5 events/h) in our study population was 69%. The South Asian group had significantly higher AHI than white Europeans (24 events/h [IQR 9.3-57.6] vs. 9 events/h [IQR 3.4-26.6]), p < 0.01). South Asians had significantly greater prevalence of OSA (85% vs. 66%, p = 0.017) and more severe OSA (p = 0.015) than white Europeans.

Table 2 highlights a number of statistically significant differences according to ethnic group in relation to 3 of the 4 SDB measures. South Asians had a greater AHI (p < 0.01), spent more time under 90% oxygen saturation (p = 0.03), and had significantly lower minimum oxygen saturation (p < 0.01) than white Europeans.

Table 3 demonstrates a significant positive association between South Asian ethnicity and AHI+1 after adjustment for a range of potential confounders with β = 1.84 (95%CI: 1.27-2.65), compared to white Europeans. Thus, South Asian ethnicity was significantly associated with an 84% increase in AHI+1, after adjustment. As expected, male gender was also an independent predictor of increasing AHI+1 (p < 0.001). Similarly, positive linear relationships were observed for age and BMI with AHI+1, where p < 0.001.

DISCUSSION

OSA is increasingly appreciated as an important contributor for CVD risk[1-3] and mortality.[4] The prevalence and severity of

Table 2—Comparison of AHI, mean oxygen saturation, time under 90% oxygen saturation, and minimum oxygen saturation between white Europeans and South Asians

	White European (n = 268)	South Asian (n = 40)	p value
AHI, events/h	9 (3.4-26.6)	24 (9.3-57.6)	< 0.01
Mean O_2 saturation, %	93 (92-95)	93 (91-95)	0.41
Percentage of sleep time spent under 90% O_2 saturation	2.4 (0.2-16.0)	8.4 (1.0-24.3)	0.03
Minimum O_2 saturation, %	83 (77-87)	76 (64-84)	< 0.01

Data are presented as median (IQR). IQR, interquartile range; AHI, apnea-hypopnea index; O_2, oxygen. p values were calculated using Mann Whitney U-test.

Table 3—Independent predictors of AHI+1 in 308 obese patients

	Model 1	Model 2	Model 3
South Asian	1.95 (1.29-2.95)**	1.83 (1.28-2.63)**	1.84 (1.27-2.65)**
Age, years	1.03 (1.02-1.04)***	1.03 (1.02-1.04)***	1.03 (1.02-1.04)***
Males	2.31 (1.72-3.11)***	2.38 (1.81-3.12)***	2.42 (1.84-3.20)***
BMI, kg/m^2	1.03 (1.02-1.05)***	1.04 (1.03-1.06)***	1.05 (1.03-1.06)***

Data are presented as unstandardized beta coefficients (95% CI) from multivariate linear regression analyses. Model 1: unadjusted. Model 2: adjusted for age, sex, BMI, ethnicity, as appropriate. Model 3: further adjusted for diabetes mellitus, hypertension, coronary artery disease, and insulin. *p < 0.05, **p < 0.01, ***p < 0.001.

OSA in the South Asian population, a group at increased risk of CVD, have not been comprehensively examined. Our study is the first to objectively investigate the prevalence and severity of OSA in severely obese South Asians residing in the west. Our results demonstrate South Asians have a significantly greater prevalence of OSA with more severe OSA compared to white Europeans. We also show, after adjustment, that South Asian ethnicity was significantly associated with an 84% increase in AHI+1.

To date, only one study has investigated ethnic differences in OSA, which reported no significant ethnic differences.[13] The UK study recruited participants from a community setting, and obesity was not part of the inclusion criteria. Furthermore, OSA was determined through the Berlin questionnaire - a screening, rather than a diagnostic tool. Given that our patient group was obese, a greater prevalence of OSA was anticipated compared to the other study (69% vs. 28%).[13] The OSA prevalence in our study is higher than two population studies from India,[10,11] possibly due to differences in obesity levels between these population studies and our sample, which only included severely obese patients.

In extreme obese populations (BMI $\geq$ 40 kg/m^2), prevalence rates of OSA have been reported as 64% to 98%.[7,8] In our patients attending the regional specialist weight management service, the prevalence of OSA is comparable to these reports. The prevalence of OSA in obese populations is elevated compared to the general population,[11,12] which is not surprising, given the close association obesity has with OSA.[7,12] While there were no significant differences in BMI between the South Asian and white European groups in our sample, South Asians have greater adiposity for an equivalent BMI. This has resulted in adoption of lower BMI cut-points for overweight/obesity for South Asians.[20] This greater propensity to visceral adiposity may explain the greater prevalence and severity of OSA. In our previous analysis, we observed that in those with moderate to severe OSA, 58% of OSA was attributable to excess body weight.[21] While this percentage is likely to be much higher in those with obesity, other factors including craniofacial anatomy and airway structure[22] and tone[23] may also be important. In the South Asian group, it is plausible that airway tone could have been altered through greater severity of diabetes and potential diabetic neuropathy. A few studies have reported ethnic differences in craniofacial morphology,[22] which may predispose to OSA, especially in obese individuals. Future studies could incorporate assessment of visceral obesity and craniofacial and airway anatomy. Whether there is also a genetic basis for the observed ethnic differences in OSA also requires exploration.

Studies in predominantly white populations have found that OSA is associated with premature mortality,[4] and obese individuals have a lower life expectancy compared normal BMI counterparts.[24] This study found that South Asians not only had an elevated risk of severe OSA, but that those with severe OSA were seven years younger than white Europeans, in line with our study. This suggests that earlier exposure to intermittent hypoxia may contribute to more severe CVD and premature mortality, particularly in obese South Asian populations. The mechanisms involved investigating the link between OSA and ethnicity are still unknown and require detailed investigation.

Several studies have suggested that metabolic syndrome is associated with OSA.[5,6] Although our study did not assess metabolic syndrome per se, we demonstrated that South Asians had a significantly higher prevalence of type 2 diabetes, a major risk factor for CVD, compared to white Europeans. A study from India found that Indians with coexisting OSA have significantly higher C-reactive protein,[25] associated with increased CVD risk. Brady and colleagues reported South Asians had greater body fat percentage, lower HDL levels, and were younger upon initial presentation/investigations for OSA.[13] While OSA has been linked with increased risk of mortality[4] and stroke,[3] the majority of the studies have been performed in white Europeans, although not exclusively. Our observations are suggestive

of greater risks in South Asians, which may be an important consideration in clinical practice.

A large Norwegian primary care study has shown that South Asians living in Norway with diabetes mellitus were younger and had the greatest risk of being on either oral hypoglycemic medications or combination treatment with insulin. The study also found that risk of poor glycemic control was 3-fold higher in South Asians.[26] Allsworth and colleagues examined diabetes care in nursing home residents and found that diabetes medications was more prominent in Asians.[27] Our study is consistent with these findings, where diabetes mellitus was significantly higher in South Asians compared to white Europeans. Glycemia was also less well controlled and more anti-diabetes medications were required for South Asians, suggesting that this population has significantly more severe and more challenging diabetes mellitus.

Whilst our study is the first to report on the ethnic differences of OSA prevalence and severity, it is important to acknowledge the limitations. Our population was recruited from the specialist weight management service and may be subject to selection bias with findings only being representative of severely obese individuals. Furthermore, ethnic differences in those who initially present with obesity and those who are referred by physicians are unknown, and may have produced further selection bias in our sample. While all UK residents have access to the National Health Service, we cannot rule out the possibility of ethnic group differences for the presentation of obesity. However, as previously noted, the West Midlands South Asian population is approximately 10% of the total population. Our patient population of South Asian's referred to our Specialist Weight Management Clinic was 13% and thus may be representative of this minority group in the West Midlands. Referral bias may explain the greater prevalence of comorbidities and a younger age of presentation of South Asians. While we acknowledge that referral bias may play a role, a recent assessment that utilized the National Bariatric Surgery Registry and census data from the UK and Ireland, demonstrated that ethnic minority groups have equal access to this type of service/procedure.[28] One further limitation is that sleep was not assessed using full polysomnography, which is impractical and not cost-effective in large clinical populations. Validated instruments for assessment of sleep apnea were however used. We did not assess or collect data for OSA symptoms as most of our patients are referred to us for considerations of bariatric surgery. Since OSA increases the risks of perioperative complications and prolonged apnea periods with risks of respiratory arrest,[29] we routinely assess potential SDB prior to consideration for bariatric surgery. Moreover, Kapur et al. found that subjective sleepiness is not present in more than half of the individuals with moderate to severe OSA.[30] Carneiro et al.[7] also found the Epworth Sleepiness Score was not a useful predictor of OSA in obese populations. While our study benefitted from adjustment for a range of potential confounders, we did not consider smoking and alcohol consumption as this data were unavailable. Cigarette smoking has a positive correlation to OSA, although no causal association has been found.[31]

In summary, OSA prevalence and comorbidities was greater in severely obese South Asians compared to obese white Europeans. South Asians also had more severe OSA compared to BMI-matched white Europeans. Our data also support previously confirmed risk factors for AHI severity including higher BMI, older age, and male gender. The precise mechanisms involved in these ethnic differences are still to be explored and understood although we hypothesize that visceral adiposity plays a role. Other potential contributory factors may be genetically mediated and include craniofacial structure and differences in pharyngeal muscular tone. Our study demonstrates ethnic differences in OSA prevalence and severity. Further exploration of mechanisms underlying ethnic differences in OSA severity is likely to extend the current understanding of OSA.

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DISCLOSURE STATEMENT

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