ORIGINAL ARTICLE

Clinical and polysomnographic features of patients with OSAHS versus patients of stroke detected to have OSA

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Abstract

Objective: Obstructive sleep apnea (OSA) is commonly found in patients with stroke; however, most of these patients are undiagnosed because they do not seekhelp for the same. As part of a prevalence study, we conducted polysomnography (PSG) recordings in consecutive patients of stroke presenting to our department. We conducted this comparative study to assess for any clinical and PSG differences between these patients and those directly presenting to our sleep disorders clinicfor OSA.

Methods: We analyzed various clinical and PSG features of consecutive patients of stroke with sleep-disordered breathing admitted between September 2009 and February 2010 consecutively.PSG was carried out after 6 weeks or more of the stroke for all patients. An equal number of patients presenting with sleep apnea at our sleep disorders clinic, during the same period, formed the control population.

Results: We included 12 male patients of stroke with sleep-disordered breathing[apnea–hypopnea index (AHI) >5] on PSG and compared them with 12 male patients who had presented for OSA, confirmed on PSG (AHI>5).We found no significant difference in sleep architecture and respiratory parametersbetween these two groups. However, we found more patientswith body mass index of>24 kg/m^{2}among those seeking help for OSA (n=8) versus those with stroke and OSA (n=3). The former also had significantly higher Epworth sleepiness scale score(p =0.006).

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Conclusion: Patients of stroke found to have OSA are less sleepy in daytime as compared to those presenting for OSA and less likely to be as obese as the latter, despite the severity of sleep apnea and associated sleep disturbances being similar.

Keywords: Stroke, Obstructive sleep apnea.

Introduction

Obstructive sleep apnea–hypopnea syndrome (OSAHS) is among the most common chronic disorders of adults, with a prevalence in order of 24% for men and 9% for[1] women, and in patients of stroke, the prevalence is 50%–74%[2–5]. Obstructive sleep apnea (OSA)is characterized by snoring, recurrent episodes of partial or complete obstruction of the upper airway during sleep resulting in oxygen desaturation and arousal from sleep, excessive daytime sleepiness, and fatigue. Not only are there local obstructive phenomena in upper airwayamong these patients, there is now strong evidence that OSA is an independent risk factor for stroke[6–8] and it also contributestoward aggravating other stroke risk factors such as hypertension[9], diabetes mellitus[10], and hyperlipidemia[11]. Among patients with recent stroke, severe OSA increases the recurrence, mortality, and poor outcome from rehabilitation[12–14]. OSA may cause these effects through intermittent hypoxia,reduced cerebral perfusion, vascular inflammation, and fragmented sleep[15].

It has been reported that patients of stroke have less severe daytime clinical symptoms and these symptoms are not well correlated with severity of OSA diagnosed on polysomnography (PSG)[2]. This study aimed to compare PSGfeatures of patients of stroke with coexisting OSA with age- and sex-matchedpatients with OSA and to identify PSG findings that determine the daytime symptoms among patients of stroke detected with OSA.

Methods

This cross-sectional case–control study was carried out at the Department of Neurology, All India institute of Medical Sciences (AIIMS), New Delhi, India, from September 2009 to February 2010.Group 1 included consecutive stroke patients presenting to Neurology services at our center, confirmed clinically, by neuroimaging and diagnosed as OSA[apnea–hypopnea index (AHI)>5] on PSG (as a part of prevalence study) within 6 months of ictus. Group 2 (OSA alone) was formed by age-and sex-matched patients presenting to the Sleep Disorders Clinic, Department of Neurology, AIIMS, with symptoms of OSAS and confirmed to have OSA (AHI > 5) on PSG. Patients with serious medical or psychiatric illness, those with a known preexisting sleep disorder, those in coma or other neurological illness, and those on medication with primary effect on central nervous system during the time of study were excluded.

Clinical assessment

We assessed functional disability of patients with stroke on the day of PSG administering the modified Rankin Scale and Barthel Index and evaluating for risk factors for cardiovascular events such as body mass index (BMI), smoking, diabetesmellitus, and hypercholesterolemia. Berlin Questionnaire and Epworth Sleepiness Scale (ESS)[16] were administered to each patient for assessment of clinically evident OSA and for excessive daytime sleepiness, respectively.

Polysomnography

All patients of stroke weresubjected to PSG after 6 weeks of stroke. All patients were admitted to our sleep laboratory forovernight PSG on dedicated video PSG system (Nicolet 1;CareFusion, San Diego, CA). The recording included a 12-channel electroencephalography, electrooculography and for electromyogram (placed over submentalis muscles), with respiratory events recorded through both thermistor and nasal pressure transducer and chest and abdomen through peizo electric belts. A peizo electric sensor was placed over right lower limb to detect and record periodic limb movements. Sleep scoring was done in 30 sec epochs, according to standard AASM criteria (2007). Percentage of total sleep time (TST) for each sleep stage (N_1,N_2, N_3 and REM), apnea–hypopnea index (AHI), oxygen desaturation index (ODI) and arousal index (AI) were calculated for each participant. Apnea was defined as reduction in air flow (thermistor) more than 90% and hypopnea was defined as reduction in pressure transducer recording amplitude more than 30% from base linelasting at least 10 seconds and associated with arousal or desaturation. Number of apnea and hypopnea per hour were calculated to obtainthe AHI.

Statistical analysis

Statistical analysis was done using the SPSS 11.5 for windows statistical package Descriptive statistics were determined for all demographic and sleep architecture characteristics. All categorical variables were analyzed by chi square test and all continuous variables were analyzed by tests for non-parametric data (Mann- Whitney test).

Results

A total of 15 stroke patients out of 25 underwent overnight sleep study and 12 were diagnosed as OSA, fitting inclusion criteria for group 1. Out of 12 stroke patients, 7 patients had major vessel infarcts and all were ambulatory except one who had significant disability due to stroke (Table 1). Twelve age and sex matched consequent patients who were diagnosed in the sleep disorders clinic and laboratory as OSAHS during the same periodwere included in group 2. The mean age of group 1 vs group 2 was (51.67±12.80 vs 51.58±9.59, *p*= 0.70). All were men and their clinical characteristics were statistically similar except snoring, fatigue and subjective sleepiness in terms of ESS (Table 2A).The patients with stroke presented with less subjective sleepiness than the OSAHS patients as evident through ESS scores which were significantly lower in group 1 compared to group 2 (*p* = 0.006).Screeningthrough Berlin Questionnaire showed fewer patients from group 1 to be in high risk category (group1 6(50%) vs. group2 10(83.33%), *p* =0.02) (Table 2B).

It was interesting that the risk factors for stroke were similar among both groups (Table 3) and PSG parameters were also statistically similar among both groups but mean duration of apneas was much higher in OSA patients versus stroke patients (46.3±31.29 sec versus 26.27±13.84 sec respectively; (median 42.05, IQR= 47.65 versus median 25.85, IQR =18.35) (*p*=0.07; Table 4).

Table 1: Clinical characteristics of patients with stroke

Patients	Topography of lesion	Barthel Index	MRS	TIA
1	Right MCA territory infarct	100	1	No
2	Right MCA territory infarct	100	1	Yes
3	Left MCA territory infarct	85	1	No
4	Right MCA territory infarct	100	1	No
5	Right MCA territory infarct	100	0	No
6	Left MCA territory infarct	100	1	No
7	Right basal ganglia bleed	100	1	No
8	Left basal ganglia bleed	100	1	No
9	Left Parietal hematoma	100	1	No
10	Right basal ganglia bleed	100	1	No
11	Left basal ganglia bleed	5	5	No
12	Right MCA territory infarct	100	1	No

Table 2A: Demographic details and clinical feature among patients of stroke with coexisting OSA(Group1)vs. patients with OSA (Group2)

Variable	Group 1 (stroke patient with SDB)				Group 2 (patient of OSA)				P Value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR	
Age	51.67	12.8	49	20.5	51.58	9.59	53	10.5	0.707
Weight	66.25	7.04	66	9.25	77.33	22.3	70	36.25	0.258
Hight	164.3	5.47	164.5	6.25	165.5	5.9	165.5	8.25	0.622
BMI	24.59	2.68	24.08	4.12	27.95	6.33	26.51	10.84	0.204
ESS	6	6.26	6	8	15.09	5.77	16	10	.006

Table 2B: BerlinQuestionnaire: comparisonamong patients of stroke with coexisting OSA(Group1) vs. patients presenting with OSA (Group2)

Berlin Questionnaires	Category 1		Category2		Category3		High Risk	P value
	+ve	-ve	+ve	-ve	+ve	-ve		.021
Group 1	9	3	2	10	8	4	6 (50%)	
Group 2	12	0	10	2	4	8	10(83.33%)	

Table 3: Assessment of risk factorsfor stroke among patients of group 1versus group 2

	Group 1 n (%)	Group 2 n(%)	P Value
Hypertension	8 (66.66)	4(33.33)	0.22
Diabetes mellitus	2(16.66)	2(16.66)	1.00
Hyperlipidemia	3(25)	2(16.66)	0.47
Smoking	5(41.66)	3(25)	1.55
Alcohol	3(25)	2(16.66)	0.47
Sedentary Life Style	9(75)	11(91.66)	0.59

Table 4: Comparison of polysomnographic parameters among patients in group 1 versus group 2

Variable	Group 1				Group2				P value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR	
TST	334.3	93.1	333	108	282	81.8	310	125	0.166
Sleep efficiency	65.72	17.5	67.9	34.15	72.41	11.2	71.8	17.3	.386
Sleep latency(Sec)	1660	835	1670	2303	683.6	549	630	825	0.204
REM latency	123.02	98.7	115.5	200.3	147.6	95.3	164.5	172.8	0.525
AHI	26.53	29.1	18.26	27.13	34.73	28.7	26.92	58.56	0.686
Arousal Index	27.4	13.2	24	25.15	39.26	29.8	33.9	51	0.498
Mean Duration of Sleep apnea	26.27	13.84	25.85	18.35	46.3+/-	31.29	42.05	47.65	0.07
Desaturation index	21.45	30.6	12	20.7	17.74	15.2	14.7	17.68	0.833
Total stage shift	163.7	67.4	150	85.75	152.2	98.3	164	108.5	0885
WASO	45.25	19.2	45.5	36	50	38.4	37.5	65.25	0.773
Arousal with A/H	9.01	7.9	6.1	8	18.97	20.01	12.5	25.53	0.291
Spontaneous arousals	14.25	10.3	12.55	12.73	12.21	10.3	8.35	15	0.51
Arousal with desaturation	21.45	30.6	12	20.7	17.74	15.2	14.7	17.68	0.833

Discussion

OSA is an independent risk factor for stroke[6–8] and there are suggestions in previous studies that PSG should be considered in all stroke and TIA patients because clinical history of OSA may not be identified in all these patients[17]. Though the currentstudy was not sufficiently powered for evaluation of various parameters, nevertheless, it showed that cardinal features of OSA, viz. snoring,excessive daytime sleepinessand fatigueare less prevalent in patients with stroke. We found that stroke patients are less obese than OSA patients and the previously observed association between BMI and severity of OSA in community samples was also not found in stroke patients with OSA. On Berlin Questionnaire OSA screening, fewer stroke patients paradoxically fall in the high risk category.

One case controlled study has shown that stroke patients,despite severe OSA have less EDS and are often not obese[18] as compared with community samples. This observation is similar to that in the present study. Other observational studies also report stroke patients to have low prevalence of these cardinal features of OSA[2,19,20]. These observations were reported by Bassetti et al[2] who foundthat 26 of 152 patients with stroke had severe OSA with an AHI > 30 but their mean ESS score was low (6.8) and mean BMI was27.9 kg/m^2 indicating that they were generally neither subjectively sleepy nor obese.Similarly, Wessendrof and colleagues[21] observed that in 105 patients with stroke with moderate to severe OSA (AHI > 15), the mean ESS was 7.2 and mean BMI was 28.7 kg/m^2.

The reported prevalence of OSA among stroke patients, in previous studies is 50 – 74%and the reason of variability may be the use of different scoring rules for apnea and hypopnea. In the present study sleep stage and respiratory events were scored according to identical criteria in patients with stroke and OSA, so we have validcomparison of their PSG features and we were also able to analyze mean differencesin EDS and BMI after controlling for potential confounding factors (age and sex, lifestyle, substance abuse). Accordingly, we demonstrate that lower ESS scores and lower BMI in patients with stroke compared to patients seeking help for OSA cannot be explained by differences in the severity of OSA, age,sex and BMI. In addition, the OSAHS group had more daytime somnolence and higher BMI, after the controlling for known confounding factors. This suggests that, in patients with stroke, subjective

ESS and obesity are not sensitive predictors of presence of OSA.

There have been no studies in patients with stroke examining relationship between ESS and objective measures of sleepiness, while there are studies correlating ESS,with the AHI as in our populations and objective measures of sleepiness and sleep resistance such as multiple sleep latency test[22] and oxford sleep resistance[23] test.

Interestinglythese findings in patients with stroke and OSA are similar to patients with heart failure who also have lower ESS scores at given AHI compared to sleep clinic OSA controls.

Another finding of our study isthe lack of an association between BMI and severity of OSA in our stroke patientscontrary to thewell established relationship between high BMI and increasing severity of OSA[24,25].

No differences in PSG parameters of sleep architecture and quality as well as of respiratory events, were observed between the two groups. The length of obstructive events was found to be longer among the patients with OSA than stroke patients, though it does not assume statistical significance in our study. In a recently published study it was shown that the cerebral flow velocity is increasedfrom 22% to 42% for amplitude and 22% to 33% for area respectively[26] as the apnea duration increases above> 30 sec. It is possible that longer duration of apnea may be protective for ischemic stroke as damage of vessels may be compensated by increased cerebral blood flow. Another study shows that termination of apnea was not determined by reduced cerebral oxygen delivery. The oxygen delivery was maintained by commensurately increased CBF[27]. These findings warrant future research in this direction.

Conclusion

Despite no PSG differences between both groups,patients of OSA with stroke are clinically different from the OSAHS patients who come to sleep clinics for seeking help for the same. Since stroke patients with OSA are less likely to be obese and sleepy during the daytime, and in view of the potential benefits on prognosis and functional outcome of patients with stroke when coexisting OSA is treated, indications of PSG to diagnose OSA in stroke population must be different.

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