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FULL PAPER

The role of upper airway morphology in apnea versus hypopnea predominant obstructive sleep apnea patients: an exploratory study

¹TÜLAY ÖZER, MD, ²ADIN SELÇUK, MD, ³ZAHİDE YILMAZ, MD, ¹NURAY VOYVODA, MD, ¹İSA ÇAM, ²HALİL ERDEM ÖZEL, MD, ²FATİH ÖZDOĞAN, MD, ²ERKAN ESEN, MD, GÜLDEN GENÇ, MD and ²SELAHATTIN GENÇ, MD

¹Department of Radiology, Health Sciences University, Derince Education and Research Hospital, Kocaeli, Turkey

²Department of ENT, Health Sciences University, Derince Education and Research Hospital, Kocaeli, Turkey

³Department of Neurology, Health Sciences University, Derince Education and Research Hospital, Kocaeli, Turkey

Address correspondence to: Dr Tülay Özer

E-mail: ozertulay@yahoo.com

Objective: The objective of this study was to investigate the association between upper airway morphology and apnea vs hypopnea predominance in patients with obstructive sleep apnea (OSA) syndrome.

Methods: A retrospective study on consecutively CT scans obtained from 54 OSA patients and 53 non-snoring controls. CT scans were measured to evaluate upper airway and surrounding structures' morphologic characteristics. OSA patients (matched for age, gender and body mass index) compared as two groups; apneic group: apnea ratio >50% and hypopneic group: hypopnea ratio >50%. Morphologic characteristics were analyzed using Kruskal-Wallis test and Pearson's correlation test.

Results: Apneic group (17 patients) showed 76.56% apnea rate and hypopneic group (37 patients) showed 78.46% hypopnea rate. Minimal lateral and

anteroposterior dimensions of velopharynx in the apneic group (0.86 ± 0.73 and 0.21 ± 0.13 cm, respectively) was statistically lesser from that of the hypopneic group (1.2 ± 0.42 and 0.54 ± 0.22 cm, respectively). Minimum cross-sectional area of the velopharynx was also lesser in apneic group (0.21 ± 0.16 cm²) than that in hypopneic group (0.65 ± 0.38 cm²). Almost all upper airway parameters in both apneic and hypopneic groups were tended to be smaller than in controls.

Conclusion: Decrease in airway volume does not signify the type of respiratory event, but significant narrowing of velopharynx in both dimensions; thus having the narrowest value below a certain level causes more apnea.

Advances in knowledge: We did not find a similar study when we did a literature search, showing the relationship of apnea vs hypopnea predominance and upper airway parameters in CT in patients with OSA.

INTRODUCTION

Overnight polysomnography (PSG) provides quantitative data regarding the frequency and duration of two distinct obstructive sleep apnea (OSA) event types; hypopneas and apneas. Hypopnea refers to partial airway obstruction and apnea refers to complete airway obstruction.¹ Despite the respiratory events in OSA patients are well-defined; little is known about the reason of hypopnea vs apnea predominance. As a result of polysomnographic findings, some patient exhibit sleep disordered breathing characterized with extreme hypopnea ratio, while some shows more apnea predominance. The apnea is observed as static obstruction of the upper airway during sleep, whereas the hypopneas occur with dynamic obstruction of upper airway.² Different upper airway morphological characteristics may play a role in the pathophysiology of apneas and hypopneas. As a

consequence of polysomnographic observations and based on the above, we investigated whether certain morphological differences in upper airway and its surrounding tissues define type of respiratory event in OSA patients.

This is the first study in the literature showing the relationship of apnea vs hypopnea predominance and upper airway parameters in CT in patients with OSA.

METHODS AND MATERIALS

Study sample

A group of patients with diagnosis of OSA syndrome and having complaints of nasal obstruction or partial nasal blockage and thus have been obtained paranasal sinus CT for evaluation of presence of nasal septal deviation or turbinate pathology were studied as patient group. PSG

was performed in all patients at the sleep laboratory. Patients were divided into two groups according to having more hypo-pnea or apnea in their PSG reports. Subjects having similar nasal obstruction complaints or paranasal sinus pathology and thus have been obtained paranasal sinus CT but no history of snoring or witnessed apnea were evaluated as control group. PSG was not performed to control group. The control subjects also did not have complete questionnaires regarding sleepiness. We excluded patients using endocrine medications, pregnant, or who had formerly been subjected to upper airway or neck operation or trauma. Oral and written information was given to the patients and their approvals were taken for participation in the work.

The body mass indexes (BMIs) and neck circumference (NC) were recorded. Epworth sleepiness scale (ESS) was obtained from all patients which shows daytime sleepiness.³

The local ethics committee approval was retrieved (KU GOKAEK 2017/6) and Helsinki declaration regulations were followed.

Polysomnography data

A full-night PSG was performed in Kocaeli Derince Educational and Research Hospital Sleep Laboratory, with Somnologica or REMLogic (Embla, Broomfield, CO). Signals comprised electroencephalograms (leads C3-A2, C4-A1, and O1-A2), tibial electromyogram, left and right electrooculograms, submental electromyogram, electrocardiogram, and oxyhemoglobin saturation. End-tidal CO₂ was measured in all participants. Airflow was acquired with a nasal cannula connected to a differential pressure transducer. Respiratory effort was evaluated with abdominal and thoracic inductive plethysmography. Body position was tracked by using infrared video camera. One of the junior authors (ZY) who is neurology specialist interpreted all results.

Polysomnography analysis

PSG was scored according to the American Academy of Sleep Medicine guidelines.¹ Cutting of the airflow through at least two breath cycles was considered as apnea. The apnea seen with continuous or increased inspiratory effort was defined as obstructive; if there is no respiratory effort, it is defined as the central apnea. A mixed apnea was defined in the absence of airflow associated with periods with and without inspiratory effort. Apnea in adults is scored when there is a drop in the peak signal excursion by $\geq 90\%$ of pre-event baseline using an oronasal apnea sensor, for ≥ 10 s. Hypopnea in adults is scored when the peak signal excursions drop by $\geq 30\%$ of pre-event baseline using nasal pressure or an alternative sensor, for ≥ 10 s in association with either $\geq 3\%$ arterial oxygen desaturation or an arousal. Obstructive and mixed types of hypopneas and apneas are included into study. Apnea-hypopnea index (AHI) of the patients was noted. Patients whose hypopnea ratio was more than 50% were taken to the hypopnea group and patients whose apnea ratio was more than 50% were taken to the apnea group. Minimum oxygen saturation values were also recorded.

CT analysis

All patients were scanned using a 64-multidetector CT (MDCT) device (Ingenuity Core 64 MDCT, Philips, Holland)

Figure 1. Measurements of the HPL, SPL, A-HPSP, SPT-max and VL on midsagittal tomographic imaging. A-HPSP, hard-palate-softpalate angle; HPL, hard palate length; SPL, soft palate length; SPT-max, maximum thickness of the soft palate; VL, velopharynx length.

with 120 kV, 294 mA and 215 field of view in a supine position. 1 mm slices of multiplanar reconstruction images in axial, coronal and sagittal sections were obtained by using bone algorithm. The airway parameters were measured by means of the software (Philips Intellispace Portal). All measurements were performed by two radiologists. First, sagittal sections were reoriented using tilt or rotation on coronal images to find the correct midsagittal plane of upper airway. Hard palate length (HPL, cm) and soft palate length (cm) was measured as the distance from the anterior nasal spine to posterior nasal spine and the posterior nasal spine to uvula tip on the midsagittal plane, respectively. The angle between these lines was determined as hard palate-soft palate angle (A-HPSP). SPT-max was measured for the maximum thickness of the soft palate on midsagittal plane (Figure 1). Superior and inferior borders of velopharynx were calculated for velopharynx length (VL, cm). In the midsagittal plane, the line between anterior nasal spine and the inferior margin of the clivus is superior border of VL. As the parallel to the superior border, the line passing through the inferior margin of the soft palate is inferior border of VL (Figure 1). The superior border of the soft palate was measured as superior boundary of velopharynx and the inferior border of the uvula was assessed as inferior boundary of velopharynx in the axial slices. The pharyngeal walls formed the lateral and posterior boundaries. The anterior boundary of velopharynx was formed by the soft palate. Velopharynx parameters were measured according to these boundaries by using region of interest in the axial plane. Region of interest was obtained by operator defined region in all 1 mm slices of axial images because of irregular contours of the upper airway (Figure 2). Minimal cross-sectional airwa

Figure 2. Measurement of the velopharynx cross-sectional airway area in axial tomographic imaging.

y area of velopharynx (V-mCSA, cm²) represented the smallest cross-sectional airway area in all axial images. The lateral and anteroposterior dimension on the minimal cross-sectional airway area defined as minimal lateral airway dimension of velopharynx (V-mLAT, cm) and minimal anteroposterior airway dimension of velopharynx (V-mAP, cm). The sum of each cross-sectional area in all 1 mm slices of axial images taken accounted for velopharynx airway volume (VAV, cm³). The ratio of VAV to VL represented the average cross-sectional airway area of velopharynx (V-aCSA, cm²).

Statistical analysis

For description of all continuous variables, mean and standard deviations were shown in Tables 1 and 2. In addition, median,

minimum and maximum values were used in Table 2. Student's *t*-test was used to compare minimum oxygen saturation, AHI, NC, ESS, apnea and hypopnea ratio for OSA. ANOVA was used to compare age, BMI means among the three groups. *X*² test was to compare sex distributions among groups. A Kruskal–Wallis test was used to upper airway tomographic data between control and OSA groups. Kruskal–Wallis—*post-hoc* test after Conover was used testing for pairwise differences. Statistical analyses were performed using MedCalc for Windows, v. 15.0 (MedCalc Software, Ostend, Belgium). Maximum Type I error rate was accepted as 5%.

Pearson's correlation test was used for determination of linear relationship between upper airway tomographic measurements and BMI, AHI, minimum oxygen saturation, apnea ratio, hypopnea ratio for OSA and control group.

To compare interobserver reliability, κ statistics were performed. κ -values < 0 were interpreted as poor agreement, between 0.0–0.20, 0.21–0.40, 0.41–0.60, 0.61–0.80 and 0.81–1.00 were accepted as slight agreement, fair agreement, moderate agreement, substantial agreement, and perfect agreement, respectively.

RESULTS

54 patients (17 apnea predominant and 37 hypopnea predominant) and 53 controls completed the trial. All patients and controls were >18 years old. The demographic and polysomnographic data of OSA patients and control group age, gender, BMI (m kg⁻²), NC, AHI, apnea rate (%), hypopnea rate (%), minimum oxygen saturation (%) and ESS scores were shown in Table 1.

All upper airway morphologic parameters of both apnea group and hypopnea group were significantly different from control groups, except A-HPSP and HPL. V-mCSA, V-mLAT and V-mAP measurements of apnea group were significantly lesser

Table 1. Demographic and polysomnographic data of OSA patients and control group

Variable	Apnea (n = 17) (mean ± SD)	Hypopnea (n = 37) (mean ± SD)	Control group (n = 53) (mean ± SD)	<i>p</i> -value
Age (years)	53.6 ± 12.6	47 ± 10.6	46.2 ± 13.1	0.09
Gender M n (%)	13 (76.5)	23 (62.2)	28 (52.8)	0.19
F n (%)	4 (23.5)	14 (37.8)	25 (47.2)	
BMI (m/ kg ⁻²)	32.4 ± 4.4	32.2 ± 6.1	28.9 ± 3.6	0.04 ^a
NC (cm)	46.7 ± 9.2	45.4 ± 7.3	–	0.63
AHI	60.49 ± 20.71	36.62 ± 23.99	–	0.001 ^a
Apnea (%)	76.56 ± 13.72	19.47 ± 15.59	–	<0.001 ^a
Hypopnea (%)	23.44 ± 13.72	78.46 ± 15.49	–	<0.001 ^a
O ₂ Sat (%)	74.17 ± 9.99	80.83 ± 9.02	–	0.05 ^a
ESS	13.2 ± 4.64	8.35 ± 5.95	–	0.02 ^a

AHI, apnea–hypopnea index; BMI, body mass index; ESS, Epworth sleepiness scale; F, female; M, male; NC, neck circumference; O₂ Sat, minimum oxygen saturation; OSA, obstructive sleep apnea; SD, standard deviation.

^aAccording to Student's *t*-test; mean values of AHI, apnea (%), hypopnea (%), O₂ sat (%) and ESS show statistically significant differences between apnea and hypopnea group (*p* < 0.05). Statistically significant *p*-value. According to One Way ANOVA test; average value of BMI among the groups are statistically different (*p* < 0.05). According to the ANOVA *post-hoc* Tukey test; the difference between control and apnea and hypopnea group causes a statistical difference.

Table 2. Comparison of upper airway tomographic data between OSA and control groups

Variable	OSAS (n = 54)				Control (n = 53)			
	Apnea (n = 17)		Hypopnea (n = 37)		(Mean ± SD)		Median (min-max)	
VL (cm)	(Mean ± SD)	Median (min-max)	(Mean ± SD)	Median (min-max)	(Mean ± SD)	Median (min-max)		p-value
VL (cm)	3.71 ± 0.55	3.62 (2.8-4.1)	3.52 ± 0.52	3.6 (1.9-4.5)	3.25 ± 0.55	3.2 (2.2-4.6)		0.002 ^a
SPL (cm)	4.16 ± 0.59	4.1 (3.07-5.6)	3.93 ± 0.51	3.9 (2.5-4.9)	3.71 ± 0.45	3.6 (3-5)		0.003 ^a
SPT-max (cm)	1.11 ± 0.26	1.09 (0.7-1.7)	1.07 ± 0.21	1.1 (0.9-1.6)	0.86 ± 0.14	0.8 (0.6-1.2)		<0.001 ^a
HPL (cm)	4.89 ± 0.67	4.83 (3.9-6.02)	4.92 ± 0.44	4.9 (3.9-5.7)	5.14 ± 0.42	5.1 (4.2-5.9)		0.08
AHPSP	128.06 ± 8.16	131 (108-142)	124.38 ± 9.18	124 (107-144)	125.38 ± 6.82	125 (108-139)		0.27
V-mCSA (cm ²)	0.21 ± 0.16	0.17 (0-0.44)	0.65 ± 0.38	0.58 (0.14-2)	1.14 ± 0.5	1.08 (0.35-3.32)		<0.001 ^a
V-mLAT (cm)	0.86 ± 0.73 ^b	0.64 (0-2.3)	1.2 ± 0.42	1.09 (0.45-2.19)	1.71 ± 0.52	1.61 (0.7-3.4)		<0.001 ^a
V-mAP (cm)	0.21 ± 0.13	0.2 (0-0.5)	0.54 ± 0.22	0.53 (0.2-1.17)	0.71 ± 0.33	0.6 (0.3-2)		<0.001 ^a
VAV (cm ²)	4.09 ± 1.79	4.02 (1.65-7.85)	4.08 ± 1.42	4.05 (2.05-7.06)	7.45 ± 2.47	6.99 (3.3-15.77)		<0.001 ^a
V-aCSA (cm ³)	1.12 ± 0.54	0.98 (0.4-2.24)	1.28 ± 0.67	1.16 (0.54-3.8)	2.27 ± 0.59	2.23 (1.26-3.94)		<0.001 ^a

A-HPSP, hardpalate-softpalate angle; HPL, hard palate length; SD, standard deviation; SPL, soft palate length; SPT-max, maximum thickness of the soft palate; VaCSA, velopharynx average cross-sectional airway area; VAV, velopharynx airway volume; VL, velopharynx length; VmAP, velopharynx minimal anteroposterior airway dimension; VmCSA, velopharynx minimal cross-sectional airway area; VmLAT, velopharynx minimal lateral airway dimension.

^aStatistically significant *p*-value. According to Kruskal-Wallis test; the mean values of V-mCSA (cm²), V-mLAT (cm) and V-mAP (cm) are statistically significant difference among the three groups (*p* < 0.05). According to the Kruskal-Wallis *post-hoc* test after Conover test, the difference between mean values of the control and apnea and hypopnea groups and also between mean values of the apnea and hypopnea group causes statistical difference. According to Kruskal-Wallis test; The mean values of VL (cm), SPL (cm), SPT-max (cm), VAV (cm³) and V-aCSA (cm²) are statistically significant difference among the three groups (*p* < 0.05). According to the Kruskal-Wallis *post-hoc* test after Conover test, the difference between mean value of the control and apnea and hypopnea group causes statistical difference.

^bThe values of V-mCSA, V-mLAT and V-mAP for a patient in tomographic imaging because of complete obstruction of the upper airway. This extreme measurement may cause that standard deviations are large for means, especially for V-mLAT value (mean ± SD; 0.86 ± 0.73) in apnea group.

Figure 3. Schematic representation of upper airway VmCSA values of the apneic and hypopneic OSA groups and control group as boxplot. OSA, obstructive sleep apnea; VmCSA, velopharynx minimal cross-sectional airway area.

than of hypopnea group ($p < 0.001$). VAV measurements were decreased in both groups comparing to controls. But there were no significant difference between apneic and hypopneic group in terms of decrease in volumes. Comparison between upper airway tomographic data for hypopnea and apnea and also for control groups was shown in Table 2. Boxplot graphics of V-mCSA, V-mLAT and V-mAP values in control, apnea and hypopnea groups were given in Figures 3–5, respectively.

Correlation between tomographic measurements and BMI, AHI, O₂ Sat, apnea ratio, hypopnea ratio for OSA and control groups were studied: There was a positive weak correlation between V-mCSA, V-mLAT, V-mAP, parameters and hypopnea

Figure 4. Schematic representation of upper airway VmLAT values of the apneic and hypopneic OSA groups and control group as boxplot. OSA, obstructive sleep apnea; VmLAT, velopharynx minimal lateral airway dimension.

Figure 5. Schematic representation of upper airway VmAP values of the apneic and hypopneic OSA groups and control group as boxplot. OSA, obstructive sleep apnea; VmAP, velopharynx minimal anteroposterior airway dimension.

rate ($r = 0.452$, $p = 0.003$; $r = 0.330$, $p = 0.033$ and $r = 0.565$, $p < 0.001$ respectively). There was a negative weak correlation between V-mCSA, V-mLAT and V-mAP parameters and apnea rate ($r = -0.487$, $p = 0.001$; $r = -0.364$, $p = 0.018$ and $r = -0.569$, $p < 0.001$ respectively). There was a negative weak correlation between BMI, AHI and O₂ sat ($r = -0.454$, $p = 0.001$, $r = -0.688$, $p < 0.001$). We found positive weak correlation between apnea rate and AHI ($r = 0.508$, $p < 0.001$), whereas negative correlation between hypopnea rate and AHI ($r = -0.528$, $p < 0.001$). For interobserver reliability, the κ -values were 0.815, 0.834 and 0.812 for VL, V-mCSA and VAV, respectively.

DISCUSSION

A sleep study can document the presence and severity of OSA by recognizing apnea, hypopnea, and respiratory event-related arousals.¹ For detecting the severity of the disease, assessing the number of respiratory events per hour is mandatory. Hypopnea shows dynamic upper airway obstruction, apnea shows static upper airway obstruction. Many anatomical and physiological factors take role in the pathogenesis of respiratory events in this syndrome. Objective assessment of upper airway morphology in OSA would be useful in understanding the reason of bringing out hypopnea vs apnea. The objective of this study was to investigate the association between upper airway morphology and apnea vs hypopnea predominance in patients with OSA. There has been no study in the literature showing the relationship between the types of respiratory events and upper airway morphology.

CT imaging allows us to assess the volume of soft tissues and anatomical structures, as a quick and noninvasive diagnostic tool. MDCT of the upper airway can provide valuable data about dynamic patterns of collapse in OSA with high temporal and spatial resolution.^{4,5} With its low radiation dose in contrast to MDCT, cone beam CT has been developed as an effective tool to evaluate upper airways, in which images are obtained in three dimensions and so as volumetric data. With cone beam CT,

reliable studies have been carried out in the areas of diagnosis and treatment of upper airways and dental diseases, both in children and adults.^{6,7} Although CT is a non-invasive technique, but contains ionizing radiation effects. It is important to use low-dose protocols in studies performed especially in children due to radiation exposure.⁸ MDCT is a highly reliable method for quantitative assessment of changes in upper airway patency.^{9,10} In this retrospective study, we compared upper airway and its surrounding structures' MDCT findings to evaluate whether there are morphological differences in adult OSA patients with apnea vs hypopnea predominance.

Upper airway collapsibility is expected to become severe as weight increases in subjects with sleep disordered breathing. Finkelstein et al analyzed velopharyngeal anatomic structures in OSA patients vs normal subjects by using cephalometric imaging techniques.¹¹ They found that pharyngeal lumen is narrowed as a result of thicker posterior pharyngeal wall and velum, increased pharyngeal length and reduced pharyngeal width in OSA patients. They also showed these changes evolved in a threshold value of BMI and there was a positive correlation between the increasing in BMI and OSA severity. Mathew et al found high hypopnea apnea ratio in extreme obesity.¹² More respiratory event can be expected to be seen in more obese patients. Therefore, OSA group had more obese patients than according to the control group, as expected. We matched the apnea and hypopnea groups in terms of weight; there were no difference between two groups. In addition, two groups matched for age and gender with each other and also with control group.

Almost all upper airway morphologic parameters of both apnea group and hypopnea group were significantly different from control groups. Only A-HPSP and HPL were similar to controls, suggesting hard palate does not take major role in pathogenesis of OSA. Other soft tissues' effect on disease severity was also shown in Finkelstein's study, as mentioned above.¹¹

In our study, airway volumes of velopharynx were decreased in both groups comparing to controls. And this decrease was almost same in both groups. Tang et al studied 58 tonsillectomy and adenoidectomy performed hypopnea versus apnea predominant children: They found no significant difference in disease resolution between two groups.¹³ This result is parallel to our result which decreases in airway volumes were same in both apnea and hypopnea predominant group and there were no difference between them. We thought that not decrease in airway volume but minimum airway crosssectional area defines the respiratory event type.

V-mCSA, V-mLAT and V-mAP measurements seem the most associated parameters with hypopnea versus apnea difference. These values were significantly lower in apnea dominant patients. The higher NCs would be expected to cause more apnea, however, there were no NC difference in the study. The clinical significance of the hypopneas/apneas seems to be related with maximal velopharyngeal narrowing, which is significant in the apneic patients comparing to hypopneics. The results of our study points out that the minimal cross-sectional area defines the

type of respiratory event, but doesn't explain the effects causing in airway narrowing. Many etiopathogenetic factors may be responsible for this narrowing. Possible reasons may be genetic, muscle structure of the patient or lower airway dynamics. In a study conducted with Su et al, increase in lower body positive pressure showed increase in upper airway collapsibility.¹⁴

In a retrospective study, severity of obstruction was shown significantly related to mortality in the severe OSA patients.¹⁵ In a recent study, it is emphasized that OSA patients due to apnea events may be more susceptible to hemodynamic changes.¹⁶ We found that AHI and ESS of apneic group is significantly higher than of hypopnea group in our study. There was positive correlation between apnea rate and AHI. Apneic group had also lower oxygen saturation than that of hypopneic patients. This may suggest us the apnea group is more severe clinical aspect than hypopnea group. However, we cannot give the morphological threshold value for velopharynx cross-sectional area, minimal lateral or anteroposterior airway dimensions related to how much airway constriction change is critical in apnea and hypopnea patients. On the other hand, low number of apnea predominant patients and unequal distribution of patients in two groups (17 apnea vs 37 hypopnea patients) and some extreme values in both groups as outliers (noted in boxplots) are the limitations of this study. Also the possible shifting of tongue or neck disposition during scanning that can impact the dimensions of the airway, is another shortcoming of our study. Future trials including a larger population of both apnea and hypopnea predominant patients would be more helpful in understanding the relationship between the respiratory events and upper airway morphology.

CONCLUSION

The maximum narrowing of velopharyngeal inlet seems to effect the apnea/hypopnea predominance. Both decreases in anteroposterior and lateral dimensions affect the type of respiratory events. Decrease in velopharynx airway volume is significant in OSA patients but doesn't define the apnea/hypopnea predominance. Significant narrowing at cross-sectional area causes more apnea. As minimum crosssectional area of the velopharynx gets lesser, apneas become more common.

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ETHICAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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