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Evaluation of Central Obesity, Increased Body Mass Index, and its Relation to Oropharyngeal Airway Space Using Lateral Cephalogram in Risk Prediction of Obstructive Sleep Apnea

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Abstract

Introduction:

Maxillofacial radiologists play a significant role in detecting airway changes using radiographic tools. Clinical examination parameters and lateral cephalogram parameters play a vital role in diagnosing obstructive sleep apnea (OSA) and dreadful consequences.

Aim and Objectives:

This study aims to evaluate central obesity, increased body mass index (BMI), and its relation to oropharyngeal airway space using lateral cephalogram in risk prediction of OSA. Objectives are to measure central obesity and BMI.

Methodology:

BMI is measured using World Health Organization guidelines to measure oropharyngeal airway space and the tongue and soft palate area using lateral cephalogram to predict OSA's risk using Berlin's questionnaire. Age group between 18 and 60 years with 20 individuals in each group will be present in the study.

Conclusion:

Feedback

Cephalometric upper airway space and soft-tissue variables in different BMI groups were compared, and it was found that there was a decrease in SPAS, MAS with an increase in BMI, and in patients with BMI <24, there was narrower nasopharynx and oropharynx.

KEYWORDS: *Obstructive sleep apnea, Berlin's questionnaire, body mass index, central obesity, lateral cephalogram, sleep medicine*

INTRODUCTION

Ideal BMI and waist circumference are physical characteristics that determine overall health status in an individual. Worldwide obesity has tripled since the early 70s. In 2016, a collective population aged 17 years and above comprised of 1.9 billion adults and more tended to be obese/overweight. Obesity affected more than 650 million people over the last decade. Most of the world's population live in countries where obesity causes increased mortality rate than people who are underweight. Under the age of 5, about 41 million children; and over 340 million of the population aged between 5 and 19 years were overweight or obese in 2016.[1] The technological era at present yields the human race to a moribund lifestyle. Central obesity is an epidemiological decrepitude that poses numerous threats for various systems in the human body. The abdomen serves as a predominant site for fat deposition besides the neck, trunk, and limbs. The consequences of weight gain include slowed metabolism to conduct daily life activities and renders the individual with overall decreased quality of life. Sleep apnea is a well-established consequence of increased body mass. Various investigations support the diagnostics of obstructive sleep apnea.

Maxillofacial radiologists have helped observe transformation in superior airway space anatomy in individuals affected with obstructive sleep apnea. Lateral cephalogram serves as one such simple yet effective diagnostic aid. Aberrations of the anatomical arrangement encasing the pharynx can bestow to the condition; because an augmentation in mass inside the enclosure of bony structure have a propensity to restraint the size of pharyngeal lumen. Besides, the inferences of Berlin's questionnaire give an insight into the risk of experiencing OSA. The facts above preceded the present study's aims to evaluate central obesity, Increased BMI, and its relation to oropharyngeal airway space using lateral cephalogram in risk prediction of obstructive sleep apnea (OSA). Objectives are to measure central obesity and BMI using the WHO guidelines according to December 2008 to measure oropharyngeal airway space and tongue and soft palate area using lateral cephalogram to predict OSA's risk using Berlin's questionnaire.

MATERIALS AND METHODS

Individual consent was obtained from all individuals recruited in the study. The study took place in the Oral Medicine and Radiology Department, Vivekananda dental college for women, Namakkal district. This study follows a prospective cross-sectional design. Study samples were classified into two groups, age group between 18 and 60 years with 20 individuals in each group, and cases are individuals with central obesity and higher body mass index (BMI); controls are individuals with normal BMI and ideal waist circumference. Age and gender matching of study subjects were done. Individuals in both the study groups were subjected to height,

weight, waist circumference measurement, and exposure to lateral cephalogram to assess oropharyngeal airway space. Berlin's OSA risk questionnaire served as a chair-side diagnostic aid by an oral medicine specialist.

Inclusion criteria were individuals with central obesity based on WHO obesity criteria – waist circumference ≥ 102 cm for men, ≥88 cm for women; BMI 30.0–40. Healthy individuals are patients with normal BMI (18.5–24.9) with waist circumference ≤102 cm for men, ≤88 cm for women. Risk of OSA using Berlin's questionnaire.

Exclusion criteria include patients who are suffering from malocclusion, airway problem, large adenoids, tonsils, sleep disorders, those under sleep medication, alcohol and smoking habits.

Measurement protocol[2] waist circumference is measured during tailing of a natural breathing cycle, the mid-point at peak of the iliac crest and the lowest margin of the hindmost palpable rib in the mid-axillary line leveled perfectly parallel to the floor. The stretch-resistant tape was used to make the measurements, is sheathed encircling the subject, but not to the range that the tape feels strangling.

Hoffen HO-18 digital electronic liquid crystal display personal body fitness weighing Scale was used to measure weight, and the height was assessed using ICS height measure stature meter. Computing the BMI is worked out as a weight measure in kilograms that will be divided by height in meter square.

Measurements of Linear airway space as shown in Figure 1, Airway space and soft tissue perimeter as represented in Figure 2 were done according to[3] using Digmizer software (Version 5.4.7, © 2005-2021 Medcalc Software Ltd, Belgium).

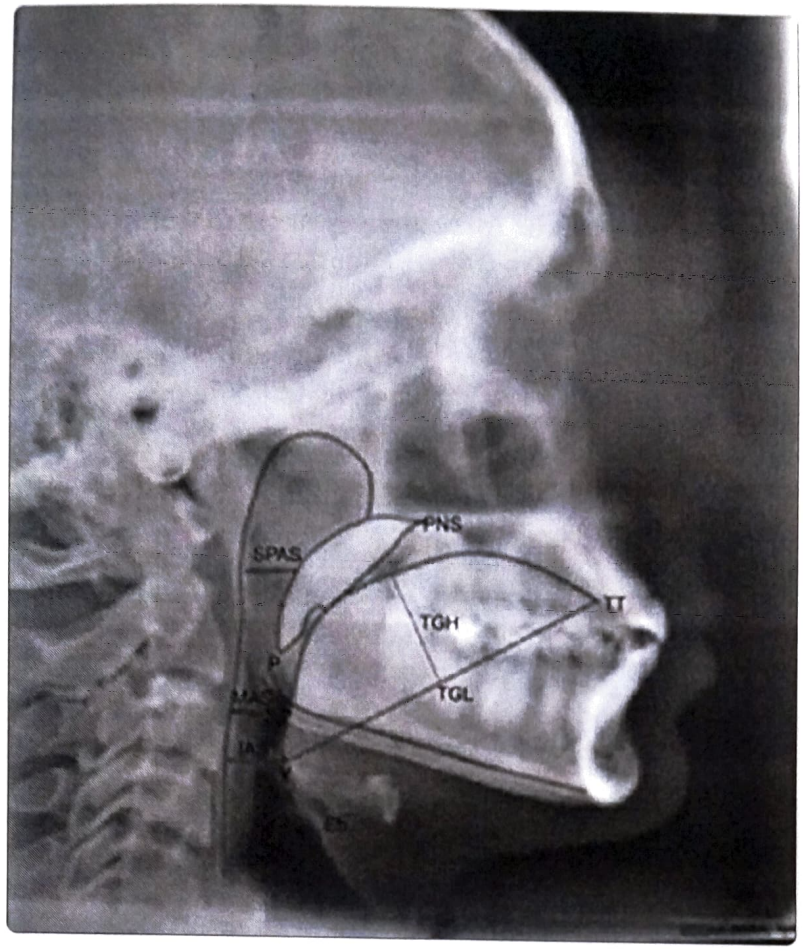


Figure 1

Linear measurement of superior airway space and soft tissue

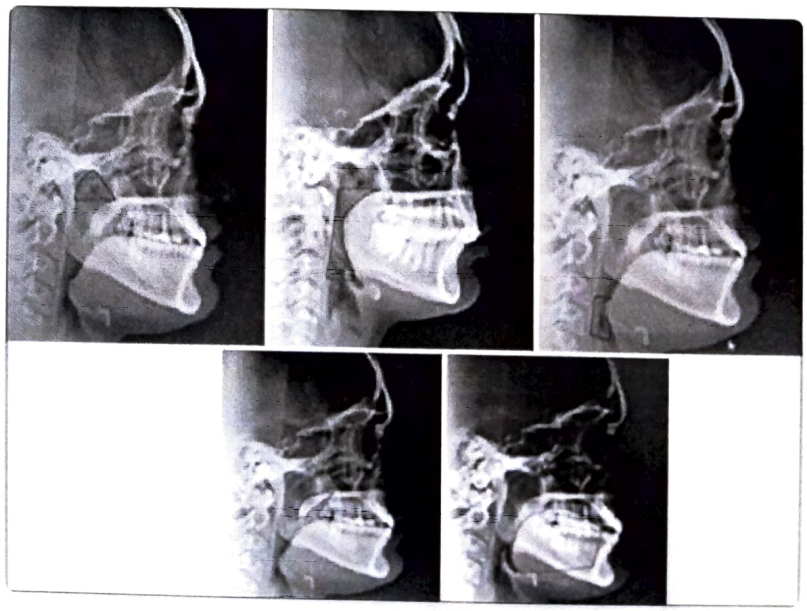


Figure 2

Measurement of the superior airway and soft-tissue area

1. Superior posterior airway space (SPAS): It is deliberated from a spot on the posterior contour of the soft palate's anterior half and to the pharyngeal wall's closest point
2. Middle airway space (MAS): Considered from the convergence point of the posterior border of tongue and inferior border of the mandible to the closest point on the posterior pharyngeal wall
3. Inferior airway space (IAS) (mm): It is the measurement allying the posterior pharyngeal wall and the point of intersection of the tongue with hyoid bone, i.e., the convergence point of epiglottis and base of the tongue (V) to lower pharyngeal wall
4. Tongue length (mm): It is a measure connecting tongue tip (TT) and the deepest point of the epiglottis that denotes its base (Eb)
5. Tongue height (mm): The linear distance between a point on the most significant curvature of the tongue dorsum and the base of a line drawn at a right angle to the TT-V line
6. Soft palate length (posterior nasal spine [PNS] – P) (mm): The straight PNS and P.

1. Nasopharynx (mm^2): The perimeter formed by a line between R and PNS, an adjunct of the palatal plane to the posterior pharyngeal wall, and the posterior pharyngeal wall
2. Oropharynx (mm^2): The area traced by the caudal most border of the nasopharynx, the posterior surface of the soft palate and tongue, a line related to the palatal plane through the point Et, and the posterior pharyngeal wall
3. Hypopharynx (mm^2): The area contoured by the inferior border of the oropharynx, the posterior surface of the epiglottis, a line resembling the palatal plane through the point C4, and the posterior pharyngeal wall
4. Tongue (mm^2): The area formed by the dorsal configuration of the tongue surface and lines that connect TT, retrognathion, hyoidale (H), and Eb
5. Soft palate (mm^2): The area that delineates the soft palate outline that emerges and terminates at PNS through P.

Statistical analysis

Student's *t*-test was used for assessing significance connecting the case and control variables; the Chi-square test was applied to find the association of Berlin's risk allying the groups. The level of statistical significance was kept at $P > 0.05$.

Results

Table 1 shows information on the sample size distribution between the case and controls. Standard deviation was observed as 2.68 in controls and 2.98 in case.

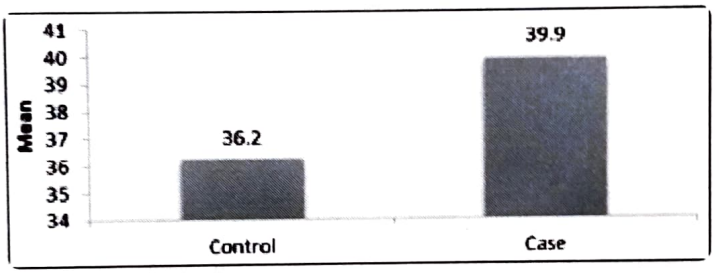
Table 1

Sample size distribution between the case and controls

Group	*n*	Mean	SD	SE	*t*	*P*
Control	20	36.20	11.97	2.68	0.92	0.361
Case	20	39.90	13.31	2.98		

SD: Standard deviation, SE: Standard error

Graph 1 presents the mean age distribution, which is 36.2 years in the control and 39.9 years in the case group.



Graph 1

Mean age distribution in groups

Table 2 shows males' dispersion about 50% in the case and 40% in the control group. The female population was about 40% in cases and 60% in the controls.

Table 2

Gender distribution among groups

Gender	Gender		Total
	Male, *n* (%)	Female, *n* (%)	
Control	10 (50)	10 (50)	20
Case	8 (40)	12 (60)	2
Total	18 (45)	22 (55)	40

Highlights mean value of 21.82, denoting normal BMI in controls and 30.37, suggesting the BMI of overweight in the case group in Table 3. A standard deviation of 11.18 in control and 7.30 among the case population. Inferences in BMI and waist circumference comparison between the case and controls proved to be relevant with a $P = 0.001^{**}$.

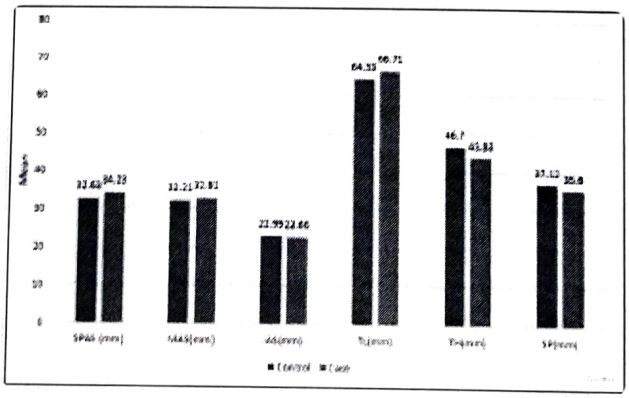
Table 3

Body mass index and waist circumference of case and controls

Feature	Group	*n*	Mean	SD	SE	*t*	*P*
BMI	Control	20	21.82	1.48	0.33	9.98	0.001**
	Case	20	30.37	3.53	0.79		
WC (cm)	Control	20	84.55	11.18	2.50	6.15	0.001**
	Case	20	102.90	7.30	1.63		

BMI: Body mass index, SD: Standard deviation, SE: Standard error, WC: Waist circumference, **Suggestive of high statistical significance

Although there was no statistical significance regarding the linear measurement of upper airway space and soft tissue, there was a mean increase in 3 out of 6 parameters contemplated. A mean enlargement in the SPAS, MAS, and length of tongue were observed in the case group compared to the controls. The values were 34.23 mm, 32.81 mm, 66.71 mm, distinctively given in Graph 2.



Graph 2

Straight measurement of superior airway space and the soft tissue

$P = 0.002^{**}$ in the nasopharynx airway space area suggests a statistically significant difference, which proves increased nasopharynx area in cases with a mean value of 2186.90, whereas the controls were about 1632.03 as given in Table 4. However, a mean value increase in hypophar-

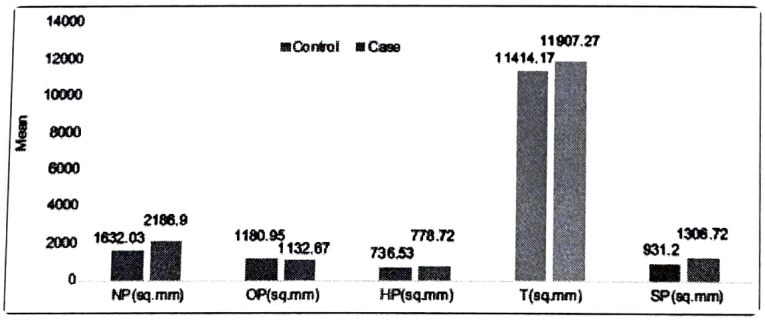
ynx, tongue, and soft palate areas was found among the case group, as seen in Graph 3. This finding suggests a gross increase in the oropharynx's soft tissue area and its associated structure in the case group.

Table 4

Measurement of the superior airway and soft-tissue parenchyma

Area	Group	*n*	Mean	SD	SE	*t*	*P*
NP (mm^2)	Control	20	1632.03	315.20	70.48	3.34	0.002**
	Case	20	2186.90	673.68	150.64		
OP (mm^2)	Control	20	1180.95	365.82	81.80	0.36	0.721
	Case	20	1132.67	474.88	106.19		
HP (mm^2)	Control	20	736.53	295.98	66.18	0.38	0.707
	Case	20	778.72	399.97	89.44		
T (mm^2)	Control	20	11,414.17	3121.67	698.03	0.49	0.626
	Case	20	11,907.27	3232.96	722.91		
SP (mm^2)	Control	20	931.20	305.57	68.33	0.58	0.565
	Case	20	1306.72	2873.96	642.64		

SD: Standard deviation, SE: Standard error, NP: Nasopharynx, OP: Oropharynx, HP: Hypopharynx, T: Tongue, SP: Soft palate, **Suggestive of high statistical significance



Graph 3

Measurement of the superior airway and soft-tissue parenchyma

Table 5 shows Berlin's questionnaire for the risk of obstructive sleep apnea, about 65% in the case group, and about 30% among the control group belonged to the high-risk category. There was a statistical implication with a $P = 0.027$*.

Table 5

High and low risk of obstructive sleep apnea using Berlin's questionnaire

Group	Berlin's risk		Total	χ^2	*P*
	Low, *n* (%)	High, *n* (%)			
Control	14 (70)	6 (30)	20	4.91	0.027*
Case	7 (35)	13 (65)	20		
Total	21 (52)	19 (48)	40		

*Suggestive of high statistical significance

Discussion

The present study found correspondence between central obesity and the mean value of most of the parameters in subjects related to high risk to OSA. This finding was following results observed by Li *et al.*,[4] their study showed critical variation transpired betwixt controls and subjects with OSA in the proportions of parapharyngeal fat pad ($P > 0.001$), fat of soft palate ($P > 0.01$). Notable imbalance in fraction of the parapharyngeal fat pad to the total volume of total lateral pharyngeal soft tissues ($P = 0.004$) was detected.

The exact pathophysiology of obstructive sleep apnea syndrome (OSAS) is not annotated thoroughly. However, the morbidity repercusses preceding a mixture of inclining anatomical and neuromuscular factors. Obesity can accord to OSAS in assorted course of actions; the convictions include involuntary concretion of the superior airway by extrinsic fat accumulation in the neck, or through intrinsic fat invasion into the upper airway.[5] A characteristic fat tissue called the parapharyngeal fat pad exists encompassing the superior pharyngeal airway. The upper airway's collapsibility is being affiliated to fat tissue concentrated in the lateral pharyngeal wall as reported by several studies.[6,7,8,9]

In a study by Wosu Adaeze *et al.*, high OSA risk was also accompanied by higher WC (β: 7.32; standard error: 1.54, $P < 0.001$) and HC (P: 5.00; standard error: 1.41, $P < 0.05$).[5] The mechanisms whereby obesity contributes to the pathogenesis of OSA are poorly understood. However, the findings were similar to the results of the present study with a $P = 0.027$*. Obesity accompanies most patients with OSA and is scrutinized a significant risk stake for its upshot.

Deformities of soft tissue adjoining the pharyngeal airway can subscribe to OSA as shown from results in Table 6; it can be because of fat aggregation inside the enclosure conditioned by bony structure at the pharynx level; that restrains the capacity of the pharyngeal lumen. Therefore, patients with OSA have more fat tissue adjoining the pharyngeal cavity in comparison to their BMI paralleled controls. Fats deposited around the upper airway may provide the collapsibility of retro-palatal and retro-glossal airway in the case group.

Table 6

Positive *r* in Pearson's correlation among individuals in case group

Case	Parameter	*r*
TL (mm)	TH (mm)	0.450
NP (mm^2)	OP (mm^2)	0.485
T (mm^2)	SP (mm^2)	0.812

NP: Nasopharynx, T: Tongue, OP: Oropharynx, SP: Soft palate, TL: Tongue length, TH: Tongue height

In the present study, the airways were disposed trivial in the case group than in the control group; however, this dissimilarity was inconsequential in the superior and IASs. Ivanhoe *et al.* stated that discrepancies in assembly in the craniofacial structures that substructure airway, causes tapering in the dimensions of the upper airway in OSA patients than in ordinary people. [10]

Cephalometry has gone beyond the boundaries of its common diagnostic confines, and today it presents as an essential tool to appraise upper airway and diagnosis of the OSA–hypopnea syndrome. However, the gold standard for diagnosing OSA is nocturnal polysomnography; imaging modalities such as computerized tomography and magnetic resonance imaging (MRI) are employed to appraise the craniofacial obstruction site within the upper airway anatomy. [11] The conventional cephalometric method has been the most empirical and commonly used.[12] Cephalometric method is a static, two-dimensional evaluation of the head and neck's dynamic three-dimensional anatomical structures, and it has been proven useful under demonstration. Low cost, impending, and vulnerability to radiation are the primacy of cephalometric studies.[13]

Cephalometric upper airway space and soft-tissue variables in different BMI groups were compared, and it was found that there was a decrease in SPAS, MAS with an increase in BMI, and in patients with BMI <24, there was narrower nasopharynx and oropharynx. The soft palate and tongue area expanded with surging BMI per Lowe *et al.*[14] The certitude that peculiarities of upper airway do not correspond significantly in more obese subjects, as shown in Table 7, other pathophysiological mechanisms have a role to tend to in this disparity. Supplemental catalysts involve increased upper airway collapsibility, fragmented sleep, ventilatory instability, and neurological mechanisms (changes in upper airway dilator muscle activity).

Table 7

Positive *r* in Pearson's correlation among individuals in control group

Control	Parameter	*r*
BMI	TL (mm)	0.445
TL (mm)	TH (mm)	0.883
TL (mm)	SP (mm)	0.477
WC (mm^2)	T (mm^2)	0.648
NP (mm^2)	OP (mm^2)	0.490

BMI: Body mass index, WC: Waist circumference, NP: Nasopharynx, T: Tongue, OP: Oropharynx, SP: Soft palate, TL: Tongue length, TH: Tongue height

Inferences from recent research using three-dimensional MRI exploration, have established that the proportions of superior airway anatomy are augmented in patients with sleep apnea. This amplification is a striking risk factor for sleep apnoea. In this investigation, a case–control was designed to scan upper airway soft-tissue structures in 48 control subjects and 48 patients with sleep apnea. After conducting covariate adjustments, the lateral pharyngeal walls, tongue, and total soft tissue were appreciably enlarged in patients with sleep apnea as to normal subjects. Not only were these structures enlarged in patients with sleep apnea correlated with normal subjects, but there was also a remarkably increased risk of amplifying sleep apnea with increase in the volume of the same corresponding structures of interest. These data furnished reinforce for the adaptations of upper airway anatomy in individuals with sleep-disordered breathing.[15] Discernible anatomic predispositions need more significant upper airway muscle activity levels to conserve an open airway during wakefulness. With onset of sleep, there is a reduction of this neural recompensation, thus providing the foundational cause.

A small population of the study sample is a significant set-back of this study; the second limitation is not being able to include patients diagnosed with polysomnography, which is the gold standard to diagnose obstructive sleep apnea; third consideration is the presence of sagittal skeletal discrepancies that could cause bias in measurement values of the upper airway anatomy.

Conclusion

There are no real debates if obstructive sleep apnea an anatomic disorder. The overriding ingredients for sleep apnea include central obesity, increased BMI, and expansion of the upper airway soft tissue structures. Obstructive sleep apnea; a fosterling condition occurs only during sleep and not during the hours of wakefulness. However, the anatomic compromise allows the reduction in motor activity of airway dilator muscles, which occurs during sleep to have such profound effects. Hence, therapeutic options such as weight loss, pharyngoplasty increase upper airway caliber must be counseled in individuals with high risk for OSA.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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