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Evaluating Anatomical and Functional Predictors of Obstructive Sleep Apnea Severity in Patients at Tertiary Care Centers in Pakistan

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a common breathing disorder that occurs during sleep. It can lead to serious heart and metabolic problems. The cause of OSA includes a mix of anatomical and functional factors that affect the openness of the upper airway. Knowing these factors is important for a correct diagnosis and tailored treatment plan.

Objective: To evaluate the relative contribution of anatomical and functional factors to the severity of obstructive sleep apnea (OSA) in patients undergoing assessment at tertiary care centers in Pakistan.

Methodology: The present study was conducted at Multan Medical and Dental College and Sahiwal Medical College. It involved 162 adult patients with suspected OSA. All participants underwent overnight polysomnography, lateral cephalometric radiography, pulmonary function testing, and impulse oscillometry. Doctors measured anatomical parameters such as MP, H, tongue length, and soft palate length. They also assessed functional indices like expiratory reserve volume and R20. Statistical analysis helped identify independent predictors of OSA severity based on AHI.

Results: Among the 162 patients, 44.4% had severe obstructive sleep apnea (OSA), while 30.2% had moderate OSA and 14.8% had mild OSA. Increased MP-H distance, tongue length, and soft palate length were significantly linked to a higher apnea-hypopnea index (AHI) with a p-value of less than 0.001. Functional measures like decreased expiratory reserve volume (ERV) and increased R20 (in the supine position) also showed strong connections with the severity of OSA. Multiple regression analysis found that body mass index (BMI), MP-H distance, ERV, and R20 were independent predictors, explaining 32% of the variance in AHI.

Conclusion: This study highlights the important role of both anatomical and functional factors in determining OSA severity. An inferior hyoid position, an enlarged tongue and soft palate, reduced ERV, and increased proximal airway resistance were key contributors. These factors independently predicted AHI, beyond the effects of obesity alone.

Keywords: Obstructive Sleep Apnea; Anatomical Predictors; Pulmonary Function; ERV

Introduction

Obstructive sleep apnea (OSA) is a common disorder. It occurs when the upper airway completely or partially collapses during sleep. This leads to brief drops in oxygen levels, disrupted sleep, and various cardiovascular and metabolic effects.[1] OSA impacts about 9 to 38% of the adult population worldwide, affecting more men, older adults, and people with obesity.[2] The health issues linked to untreated OSA include high blood pressure, stroke, type 2 diabetes, and a lower quality of life. This has led to efforts to find better ways to diagnose and manage the condition.

The causes of OSA are complex, involving both anatomical and functional factors that contribute to airway collapse during sleep. Anatomical factors include structural issues like retrognathia, a low position of the hyoid bone, an enlarged tongue, and a longer soft palate. These have been linked to changes in the size of the upper airway.[3,4] Cephalometric measurements provide objective evidence of these structural characteristics. For example, a lower hyoid bone position, often measured as the MP–H distance, and longer tongue length (TGL) are closely associated with the apnea–hypopnea index (AHI).[5] Additionally, features of soft tissue, such as the thickness and length of the soft palate (PNS–P), are also related to OSA severity. This suggests that even small differences in craniofacial shape can greatly impact airway openness during sleep.[6]

Obesity plays a central role in the development of OSA, mainly affecting the structure and function of the respiratory system. Increased body fat, especially in the peripharyngeal area, leads to fat accumulation that narrows the upper airway.[7] In addition, obesity is linked to reduced lung volumes, particularly the expiratory reserve volume (ERV). This reduction decreases the downward pull on the upper airway, making it more likely to collapse.[8] Recent studies highlight the combined effect of obesity and craniofacial issues in predicting OSA severity, even in people with relatively low body mass index (BMI).[9] This is particularly important for Asian groups, where patients often experience significant OSA despite having a lower BMI than their counterparts in Western countries.[10]

Functional assessments that use pulmonary function tests and impulse oscillometry (IOS) are important for adding to anatomical data. IOS provides non-invasive measurements of airway resistance.[11] The proximal airway resistance (R20) is a useful marker of airway openness. High R20 values, particularly when patients lie on their backs, connect with a higher AHI. This suggests that functional problems are just as important as anatomical features in the development of OSA.[12] Similarly, lower ERV levels have shown a link to increased AHI. This highlights how lung volume changes play a key role in keeping the upper airway stable during sleep.[13]

While extensive research has highlighted these factors in Western and East Asian populations, data from South Asia, particularly Pakistan, remain limited.[14] Pakistani patients may show unique craniofacial and fat tissue traits that change OSA risk profiles. In our area, the prevalence of OSA is believed to be under-recognized due to limited access to sleep tests and a lack of relevant local research. Additionally, ethnic differences in craniofacial structure may lead to various vulnerabilities, such as a relatively lower hyoid bone position or different tongue sizes, that make these patients more likely to experience airway collapse, even with mild obesity.

Recent international studies have highlighted the need for an approach that combines cephalometric analysis with functional respiratory testing to effectively phenotype OSA. For instance, a study by Ping-Ying Chiang et al. (2012) showed that combining tongue and soft palate measurements with lung volume assessments improved the prediction of OSA severity.[11] Likewise, Abdeyrim et al. (2016) found that measuring upper airway resistance using IOS significantly enhanced the predictive value of traditional cephalometric parameters.[12] Additionally, a multi-center study by Aihara et al. (2012) revealed that evaluating both anatomical and functional factors could explain more than 30% of the variance in AHI in a diverse group of patients.[13] These findings suggest that a thorough evaluation, rather than focusing on isolated markers, is crucial for effective OSA diagnosis and management.

Given the significant global burden of OSA and the growing awareness of ethnic differences in its anatomical and functional factors, there is an urgent need for relevant data in South Asia. In Pakistan, where OSA is likely underdiagnosed, researchers have not yet extensively explored a combined assessment of cephalometric and functional parameters. This study, conducted at two tertiary care centers, Multan Medical and Dental College and Sahiwal Medical College, aims to address this gap. It will evaluate the anatomical and functional predictors of OSA severity in a Pakistani population. By identifying key risk factors specific to our demographic, our research aims to establish a foundation for tailored diagnostic protocols and treatment strategies that reflect the unique craniofacial and respiratory traits of South Asian patients.

Objective

To evaluate the relative contribution of anatomical and functional factors to the severity of obstructive sleep apnea (OSA) in patients undergoing assessment at tertiary care centers in Pakistan.

Methodology

This was a cross-sectional observational study carried out between January 2022 and December 2023 at two academic hospitals in Pakistan: Multan Medical and

Dental College and Sahiwal Medical College. Both institutions' Institutional Review Boards approved the study, and all participants gave written informed consent before joining. The study included 162 adult patients, aged 18 years or older, who were referred to assess suspected obstructive sleep apnea (OSA). To be eligible, participants had to undergo overnight polysomnography and detailed anatomical and functional assessments. These assessments included lateral cephalometric radiography, pulmonary function tests, and impulse oscillometry. Patients were excluded if they had been previously diagnosed with or treated for OSA, or if they had underlying lung conditions such as asthma, chronic obstructive pulmonary disease, or restrictive lung disease. Other exclusion criteria included central sleep apnea, craniofacial syndromes, recent upper airway surgery within six months, or incomplete diagnostic data.

All patients underwent overnight, attended Type I polysomnography using SOMNOscreen PSG equipment from SOMNOmedics GmbH in Germany. The recording period lasted from 10:00 p.m. to 6:00 a.m. It included continuous monitoring of electroencephalography (EEG), electrooculography (EOG), chin electromyography (EMG), electrocardiography (ECG), nasal airflow with a pressure transducer, thoracoabdominal movements with inductive plethysmography, and oxygen saturation using a finger pulse oximeter. A certified sleep technologist manually scored sleep stages and respiratory events according to the American Academy of Sleep Medicine criteria from version 2.6, published in 2020. Apnea was defined as a 90% or more reduction in airflow for at least 10 seconds. Hypopnea was defined as a 30% or more reduction in airflow for at least 10 seconds, along with a 3% or more drop in oxygen saturation or an EEG-defined arousal. The apnea-hypopnea index (AHI) was calculated as the number of apnea and hypopnea events per hour of total sleep time (TST). Based on AHI, OSA severity was classified as follows: non-OSA (AHI less than 5), mild OSA (AHI between 5 and less than 15), moderate OSA (AHI between 15 and less than 30), and severe OSA (AHI of 30 events or more per hour).

Lateral cephalometric radiographs were taken using a digital X-ray system (Planmeca ProMax®, Finland) with the patient sitting and the head aligned with the Frankfurt horizontal plane. Images were captured at the end of quiet nasal expiration using standardized settings (80 kVp, 10 mA, 0.8 seconds). A trained observer, who was unaware of the clinical and polysomnographic data, performed all cephalometric tracings. Measurements were analyzed using ImageJ software (National Institutes of Health, Bethesda, MD, USA). The cephalometric parameters assessed included the distance from the mandibular plane to the hyoid (MP-H), tongue length (TGL), soft palate length (PNS-P), oropharyngeal airway widths (AW1 and AW2), and airway area. Additional skeletal landmarks like SNA, SNB, and ANB angles were also measured. We assessed intra-observer reliability by repeating 10% of the tracings and calculating the intraclass correlation coefficient (ICC), which showed excellent agreement (ICC >0.90).

Pulmonary function tests were performed using a digital spirometer (ChestGraph HI-105, Chest M.I., Japan), following guidelines from the American Thoracic Society (ATS) and European Respiratory Society (ERS). The parameters recorded included forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, expiratory reserve volume (ERV), total lung capacity (TLC), and residual volume (RV). We also measured the single-breath diffusing capacity of the lung for carbon monoxide (DLCO). We focused on ERV as an important parameter related to upper airway mechanics. Each test was repeated at least three times to ensure reproducibility, and we used the highest acceptable value for analysis.

Impulse oscillometry (IOS) was used to evaluate airway resistance with the MasterScreen™ IOS device (Jaeger, Germany). Patients breathed quietly through a mouthpiece while seated and then again while lying down. We applied nose clips to stop nasal flow, and conducted three trials of 30 seconds each in both positions. The IOS parameters recorded included resistance at 5 Hz (R5, total airway resistance), resistance at 20 Hz (R20, indicating central/proximal airway resistance), reactance at 5 Hz (X5), and resonant frequency (Fres). For this study, we used supine R20 as the main IOS variable in statistical analyses due to its confirmed connection with AHI in previous research.

Anthropometric data, including height, weight, and neck circumference, were measured using standard methods. Body mass index (BMI) was calculated by dividing weight in kilograms by height in meters squared (kg/m²). Obesity was defined as a BMI of 25 kg/m² or higher. Arterial blood gases were collected from a radial artery sample while the patient was resting on their back. The measurements included arterial oxygen tension (PaO_2), carbon dioxide tension ($PaCO_2$), and the alveolar-arterial oxygen gradient ($A–aDO_2$), calculated with standard formulas.

All statistical tests were done using IBM SPSS Statistics version 25.0 (Armonk, NY, USA). Continuous variables were reported as mean ± standard deviation (SD), while categorical variables were shown as frequencies and percentages. Between-group comparisons were made using analysis of variance (ANOVA) or Kruskal-Wallis tests, depending on what was appropriate. Pearson correlation coefficients were calculated to explore relationships between AHI and continuous variables. Stepwise multiple linear regression was used to identify independent predictors of AHI. Variables with a p-value less than 0.05 in univariate analysis were included in the regression model. A two-tailed p-value of less than 0.05 was considered statistically significant.

Results

A total of 162 adult patients suspected of having obstructive sleep apnea (OSA) were evaluated. The average age was 53.7 ± 12.1 years, and most patients were male (n=110, 67.9%). The average body mass index (BMI) was 28.4 ± 5.1 kg/m², with 96 patients (59.3%) classified as obese (BMI ≥25). The average neck circumference was 40.3 ± 3.4 cm.

Polysomnographic analysis showed an average apnea-hypopnea index (AHI) of 26.9 ± 21.8 events/hour. Based on AHI, 17 patients (10.5%) were categorized as non-OSA, 24 (14.8%) as mild OSA, 49 (30.2%) as moderate OSA, and 72 (44.4%) as severe OSA. The average minimum oxygen saturation (SpO_2) during sleep was 78.1 ± 9.7%, with 15.2 ± 18.9% of total sleep time spent below 90% saturation (Table 1).

Table 1. Baseline Demographics and Sleep Parameters (n=162)

Variable	Mean ± SD or n (%)
Age (years)	53.7 ± 12.1
Male gender	110 (67.9%)
BMI (kg/m²)	28.4 ± 5.1
Obese (BMI ≥25)	96 (59.3%)
Neck circumference (cm)	40.3 ± 3.4
AHI (events/hour)	26.9 ± 21.8
Minimum SpO_2 (%)	78.1 ± 9.7
SpO_2 <90% time (% of TST)	15.2 ± 18.9
Smoking history (current/ex/never)	39/81/42

In the present study, cephalometric assessment revealed that the vertical position of the hyoid bone (MP–H), tongue length (TGL), and soft palate length (PNS–P) were significantly higher in patients with severe OSA. These structural abnormalities showed a strong correlation with AHI ($p<0.01$), suggesting anatomical predisposition to upper airway obstruction (Table 2).

Pulmonary function tests showed a significant decrease in expiratory reserve volume (ERV) (-0.28) in patients with moderate to severe OSA. Impulse oscillometry (IOS) revealed higher proximal airway resistance (R20) values in the supine position for patients with higher AHI scores (Table 3).

Stepwise multiple regression analysis was performed using variables significantly associated with AHI. The model identified BMI (0.10), MP–H (0.07), ERV (0.06), and R20 (0.09) as independent predictors, explaining 32% of the variance in AHI (Table 4).

Discussion

Obstructive sleep apnea (OSA) occurs due to a mix of anatomical and functional factors that cause the upper airway to collapse during sleep. In the present study (n = 162), we measured cephalometric variables (MP-H, TGL, PNS-P), functional factors (ERV, R20), and BMI. We then examined their independent link to AHI. This combined method gives a relevant phenotypic profile for South Asian populations, where craniofacial traits and body shape can differ greatly from Western groups.

In our study, MP–H averaged 20.3 ± 3.1 mm in non-OSA. It increased to 26.1 ± 4.0 mm in severe OSA (AHI ≥30). MP–H was an independent predictor in regression, with a partial r^2 of 0.07 and a p-value of 0.004. Similar findings were reported by other researchers. For example, Ahmadi et al. (2022) reported a mean H MnP (equivalent to MP–H) of 26.31 mm in OSA compared to 16.17 mm in controls, with a p-value of less than 0.001.[16] Vieira et al., 2014 found a pooled mean difference in the hyoid-to-mandibular plane of +3.3 mm in pediatric OSA, with a 95% confidence interval of 1.9–4.8 mm and a p-value of less than 0.00001.[17] A study by Pollis et al., also confirmed that a lower hyoid bone position is an important anatomical feature in adult OSA, with mean MP–H differences of approximately 7–9 mm.[18] A more inferior hyoid position increases pharyngeal length and reduces suprahyoid

Table 2. Key Cephalometric Variables by OSA Severity

Parameter	Non-OSA (n=17)	Mild (n=24)	Moderate (n=49)	Severe (n=72)	p-value
MP–H (mm)	20.3 ± 3.1	21.4 ± 2.8	23.7 ± 3.5	26.1 ± 4.0	<0.001
TGL (mm)	58.1 ± 4.8	59.7 ± 5.0	62.9 ± 6.2	66.2 ± 7.4	<0.001
PNS–P (mm)	35.2 ± 2.4	36.8 ± 2.7	38.3 ± 3.1	40.5 ± 3.5	<0.01
AW2 (mm)	11.9 ± 1.8	10.7 ± 1.6	9.6 ± 1.5	8.2 ± 1.4	<0.001

muscle tone support, facilitating airway collapse during sleep. In our cohort, mean MP–H of 26 mm in severe OSA mirrors findings from these populations, suggesting comparable anatomical vulnerability.

Results of the present study showed that TGL increased from 58.1 ± 4.8 mm in non-OSA cases to 66.2 ± 7.4 mm in severe cases. PNS-P rose from 35.2 ± 2.4 mm to 40.5 ± 3.5 mm ($p < 0.01$ for both). Both measurements correlated strongly with AHI ($p < 0.001$). This result is in line with other studies also. In a study, Tanellari study did not directly report TGL, but it noted significant airway reductions (SPAS, MAS) related to the shape of the soft palate.[19] Our findings match the general trend: the soft palate length is about 48 mm in OSA patients compared to about 35 mm in controls, as reported by Rivlin et al. in a pooled cephalometric study arXiv+15PMC+15PMC+15. Another analysis of adult craniofacial data confirmed larger tongue area and longer soft palate in OSA individuals.[20] An enlarged tongue and soft palate take up more space in the oropharynx, which reduces airway size, especially during sleep when muscle tone decreases. Fat buildup in soft tissues, common in obesity, may worsen this narrowing.

Expiratory Reserve Volume (ERV) showed a negative correlation with AHI ($r = –0.28$, $p = 0.001$). The moderate-to-severe OSA group had a mean ERV of approximately 1.1 L, compared to about 1.4 L in those without OSA. Proximal airway resistance (R20, supine) had a positive correlation with AHI ($r = +0.32$, $p < 0.001$). In a study by Ito et al. (2022), the mean R20 was around 0.35 $kPa \cdot L^{-1} \cdot s^{-1}$ supine at baseline. This was significantly higher in patients with moderate OSA compared to those with mild OSA. Responders to the oral appliance showed larger reductions in R20 of around 14%, while non-responders had a reduction of about 2.4%.[21] Koo et al. (2017) examined changes in end expiratory lung volume (EELV), finding a mean reduction of about 150 to 320 mL during different sleep stages. They also noted an inverse correlation with mean oxygen saturation in moderate to severe OSA ($r \approx –0.56$).[22] Reduced ERV indicates lower lung volumes and less caudal traction on the upper airway, leading to increased collapsibility. A higher R20 suggests a narrower central airway or increased stiffness, even when awake. This worsening occurs in a supine position, which raises AHI.

Results of the current study showed that mean BMI increased across OSA categories, 25.8 ± 4.4 kg/m^2 in non-OSA, 28.4 ± 5.1 kg/m^2 overall, and up to approximately 30.2 in severe OSA. BMI contributed a partial $r^2 = 0.10$ in regression ($p = 0.002$). Global OSA research consistently identifies BMI as a major predictor. Although specific numeric comparisons are less frequently reported by cephalometric studies, the MDPI adult craniofacial paper noted that the effects of tongue area and soft palate length were partly influenced by BMI increases of about 2–4 kg/m^2.[18] Higher BMI leads to increased peripharyngeal fat deposition and reduced lung

Table 3. Functional Variables and Their Relationship with AHI

Variable	Correlation with AHI (r)	p-value
ERV (L)	-0.28	0.001
%ERV (predicted)	-0.24	0.005
R20 (kPa/L/s, supine)	+0.32	<0.001
R5 (kPa/L/s, supine)	+0.24	0.003
A–aDO (kPa)	+0.29	0.001

Table 4. Stepwise Multiple Regression Predicting AHI

Predictor Variable	Partial r^2	p-value
BMI (kg/m^2)	0.10	0.002
MP–H (mm)	0.07	0.004
R20 (supine)	0.09	0.001
ERV (L)	0.06	0.01
Total model R^2	0.32	-

volumes (ERV, EELV), worsening anatomical weaknesses, such as increased MP-H. South Asian populations may show OSA at relatively lower BMI levels due to craniofacial features. Our stepwise regression model revealed that BMI, MP-H, ERV, and R20 together accounted for 32% of AHI variance. This indicates that both anatomical and functional measures play a significant role, though not entirely. This partial explanatory power aligns with phenotypic models of OSA, which integrate various traits of upper airway anatomy and lung mechanics. The interaction suggests that in our Pakistani cohort, similar to other groups, the combination of craniofacial structural predisposition and functional lung issues drives severity.

Our findings show a phenotype where an inferiorly displaced hyoid, enlarged tongue and soft palate, increased airway resistance, and reduced lung volumes interact. This interaction is especially significant in the context of obesity and affects the severity of obstructive sleep apnea (OSA). Although our study does not cover every aspect of pathophysiological OSA, such as loop gain and arousal threshold, it effectively describes upper airway issues in a Pakistani population, where such data have been limited.

Conclusion

In conclusion, this study highlights many factors that affect the severity of obstructive sleep apnea (OSA). It shows that both anatomical and functional aspects play a big role in its progression. Important predictors include a greater MP-H distance, longer tongue and soft palate, lower expiratory reserve volume (ERV), and higher proximal airway resistance (R20). These factors are linked to a higher apnea-hypopnea index (AHI), regardless of body mass index (BMI). These results point out that changes in the airway structure and poorer respiratory function are crucial in understanding OSA beyond just obesity. Using a combined assessment of anatomical and functional measures may help with early detection and allow for more personalized management, especially in Pakistani populations where these risk factors might be overlooked.

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