Comparison of Cephalometric Variables in Non-obese and Obese Patients with Obstructive Sleep Apnea

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ABSTRACT

Objective: To compare the cephalometric variables of obese (body mass index (BMI) ≥ 30) and non-obese (BMI < 30) Turkish male patients with obstructive sleep apnea syndrome (OSAS).

Materials and Methods: OSAS diagnosed 85 patients who were obese [n = 37; mean age (±SE), 49.41 ± 1.54 year] and non-obese [n = 48; mean age (±SE) 46.92 ± 1.39 year] were included in the study. The cephalometric measurements and polysomnographic data of the patients were compared and a discriminatory analysis was performed.

Results: The apnea-hypopnea index (AHI) was significantly higher in obese patients (p < 0.01). Bimaxillary protrusion was found in obese patients (p < 0.05). The non-obese patients with AHI ≥ 30 had an increased mandibular plane angle In the stepwise discriminant analysis done separately in obese and non-obese patients according to AHI; only the hyoid bone position was included in the model in obese patients and the estimated success of discrimination of AHI's level (<30 and ≥30) was 70.3%. Age, anterior face and posterior face height were included to the model in non-obese patients and the estimated success of discrimination was found as 79.2%.

Conclusion: Craniofacial morphology has an effect on the severity of OSAS. If the craniofacial morphology tends toward a worsening of OSAS with obesity, the severity of the OSAS increases.

Key Words: Cephalometry, obesity, obstructive sleep apnea syndrome, polysomnography

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Introduction

Obstructive sleep apnea syndrome (OSAS) is characterized by loud snoring and periodic breathing with repetitive apneas, hypopneas, arousals leading to fragmented sleep, and excessive daytime sleepiness. It is a common condition, affecting at least 2% of adult females and 4% of adult males (1). Several causes for OSAS have been suggested. It appears to result from a variable combination of anatomical and pathophysiological factors, some of which may be due to genetics (2, 3).

Although there is no consistent evidence that the differences in the upper airway anatomy have a substantial impact on the occurrence of OSAS, (4) the etiology of OSAS includes abnormalities in both the pathophysiology and anatomy of the airway and associated facial structures (5). The factors that cause obstruction of the upper airway include anatomical and functional factors, and anatomical factors such as obesity and craniofacial deformity are thought to play a major role (6, 7). However, the determinants of upper airway collapse are multiple, airway anatomy being only one such determinant. It has been suggested that bony craniofacial abnormality is more important as a risk factor in non-obese than in obese OSAS patients (8, 9). This may be the reason why some OSAS subjects are of normal weight and physique. Furthermore, the etiology of OSAS in obese patients may be different from that in non-obese patients (9, 10).

Obesity, being a risk factor for OSAS, may affect upper airway morphology in the absence of bony craniofacial abnormality. In obese patients who have a distribution of body fat mainly over the upper part of their body, the resistance of the upper airway during sleep tends to be very high (11, 12). It was demonstrated that the closing pressures of the pharynx were distinctively higher in apneic patients than in normal subjects matched for age and body mass index (BMI) (13). However, the possible influence of age and BMI on cephalometric measurements has frequently been neglected (10). It has also been suggested that the discrepancy in the cephalometric measurements may depend on race (14). A previous study suggested that there may be differences between Asians and Caucasians in the degree to which obesity and craniofacial anatomy serve as risk factors, and that the etiology of OSAS in...
obese patients may differ from that in non-obese patients (15). It is also thought that Turkish OSAS patients may present some different cephalometric features from those of other Caucasian patients due to racial physical differences.

The objectives of the present study were to compare the cephalometric variables of obese and non-obese Turkish male patients with OSAS. Another objective was to clarify the relationship between cephalometric variables and obesity to predict the severity of AHI.

Materials and Methods

Study population

Eighty-five Turkish adult male patients with OSAS were recruited from patients who complained of habitual snoring, apneas, and daytime sleepiness, and were referred to the Sleep Disorders Center at Suleyman Demirel University. All patients were examined by one of two pulmonologists experienced in sleep medicine, and they underwent an overnight polysomnography. Cephalometry was performed on all patients the morning after the polysomnography. The patients were recruited on the basis of the following inclusion criteria: dentate in both jaws, apnea-hypopnea index (AHI) ≥5 events per hour (events.h\(^{-1}\)) during an overnight polysomnographic study, and no apparent craniofacial deformities. The patients were excluded if there was any previous history of pharyngeal surgery, orthognathic surgery, or poor general dental health. This study was approved by the local research ethics committee, and informed consent was obtained from all patients before the polysomnographic study.

The OSA population was further classed into non-obese and obese OSA groups by BMI, using the cut-off point of <30 and ≥30 kg.m\(^{-2}\) (16). Each group was also classified into two subgroups according to the AHI, using the cut-off point of <30 and ≥30 events.h\(^{-1}\) (17). The characteristics and polysomnographic data for the patients are presented in Table 1.

Cephalometric analysis

A standard lateral cephalogram was obtained during the end-expiration phase for each subject with the teeth in centric occlusion and without swallowing. A cephalostat was used to keep the subject's head in such a position that the Frankfort horizontal plane was parallel to the ground during exposure. Each radiograph was scanned into digital format at 300 dpi resolution, stored in TIFF format, and transferred to Dolphin imaging software (Dolphin, Chatsworth, California, USA) for digitization of landmarks. Each image was digitally traced twice by the same investigator (H.A.), who did not know the clinical status of the patient, and the mean value of the two most proximate measurements was used for the statistical analyses to ensure reliability. In each lateral cephalogram, 14 landmark points were identified (Figure 1). A total of 17 variables related to both craniofacial skeletal and soft tissue morphology were measured as angular (degrees) or linear (millimeters). The angles and linear measurements and their abbreviations are given in Figure 2.

Figure 1. Landmarks on lateral cephalograms: S: sella; N: nasion; Or: orbitale; ANS: anterior nasal spine; PNS: posterior nasal spine; A: point A; B: point B; G: gnathion; Me: menton; Go: gonion; Hy: hyoid bone; PA: tip of uvula; Po: porion; Ba: basion

staged according to the Rechtschaffen and Kales (20) criteria by two trained sleep clinicians who were blind to the clinical characteristics of each patient.

Apnea was defined as the cessation of airflow for 10 sec or longer. Hypopnea was defined as an airflow reduction of at least 50% over preceding epochs or an amplitude reduction of at least 50% of the thoracoabdominal belts (also compared to prior epochs), together with the presence of either arterial oxygen desaturation equal to or greater than 3%, or an arousal. AHI was determined by the frequency of these events per hour during sleep time based on the results of the overnight polysomnography. The patients with an AHI of 5 or higher were considered to have OSAS (17).
The patients had a mean age of 48.0±1.04, with a mean BMI of 30.53±0.61 kg.m⁻², had a mean AHI of 33.82±2.92 events.h⁻¹ (n=85) ranging from 5 to 109.3, which was significantly higher in obese patients (Mann Whitney U test, p<0.01). Thirty-three of the 85 patients had an AHI≥30 events.h⁻¹. Overall, 48 of the 85 patients were non-obese and 37 were obese. The mean neck circumference was 40.47±0.30 cm. We found that the largest neck circumferences were more prevalent in the obese patients. The anthropometric, cephalometric and polysomnographic variables of the study group are shown in Table 1.

The ANOVA results of the demographic and polysomnographic parameters and cephalometric measurements: The AHI-BMI interaction and the differences between BMI factor means and AHI factor means were not statistically significant for age, inferior airway space, middle airway space, maximum soft palate thickness (Max.spt), mandibular plane angle (SNGoGn), SNA°, SNB°, ANB°, anterior face height, ramus height, uvula length and lower face height. Although the differences between the mean waist circumference (p<0.01), height (p<0.05), maxillary length (p<0.01), mandibular body length (p<0.05), posterior face height (p<0.01), and middle face length (p<0.05) of the BMI factor were statistically significant, the AHI-BMI interaction and the differences between the mean levels of the AHI factor were not statistically significant. There were statistically significant differences between the mean levels of the AHI factor for the arousal index (p<0.01), awake mean SaO₂% (p<0.05), lowest SaO₂% (p<0.01), and Hy-MPPerp (p<0.05). However, the AHI-BMI interaction and the differences between the mean levels of the BMI factor were not statistically significant (Table 2).

In non-parametric analysis: the mean SaO₂ Des. (%) was significantly different between the mean ranks of the AHI<30 events.h⁻¹ and AHI≥30 events.h⁻¹ in obese and non-obese patients (Mann-Whitney U test, p<0.01); but weight, neck circumference, ESS, superior airway space, and posterior face height parameters were not statistically significant.

Relationship between AHI, BMI, neck and waist circumferences: AHI was found to be significantly correlated with BMI (r=0.49, p<0.01), neck circumferences (r=0.39, p<0.01) and waist circumferences (r=0.40, p<0.01) in the study. Also a positive correlation was found between BMI and neck circumferences (r=0.62, p<0.01) and waist circumferences (r=0.76, p<0.01) in the study.

Relationship between AHI and the cephalometric measurements: While there was a positive relationship between AHI and mandibular body length (r=0.33, p<0.05) in non-obese patients with AHI<30 events.h⁻¹, AHI was correlated negatively with SPAS (r=-0.34, p<0.05) and posterior-anteri-or face height (r=-0.73, p<0.05) in non-obese patients with AHI<30 events.h⁻¹. AHI was significantly correlated with IAS (r=0.78, p<0.01) in obese patients with AHI<30 events.h⁻¹, HyMPPerp (r=0.45, p<0.05) and ANS-Me (r=0.44, p<0.05) in obese patients with AHI≥30 events.h⁻¹.

Relationship between BMI and the cephalometric measurements: BMI showed a positive significant correlation with IAS (r=0.39, p<0.05), SPAS (r=0.36, p<0.05) and MAAS (r=0.45, p<0.01) in non-obese patients with AHI<30 events.h⁻¹. However, cephalometric measurements did not significantly correlate with BMI in the non-obese patients with AHI≥30 events.h⁻¹. In obese patients with AHI<30 events.h⁻¹, there was a positive correlation with maxillary length (r=0.69, p<0.01), posterior-anterior face height (r=0.57, p<0.05) and a negative correlation with SNGoGn (r=0.62, p<0.05). A positive correlation was found between BMI and SNGoGn (r=0.57, p<0.05) and posterior-anterior face height (r=0.47, p<0.05) in obese patients with AHI≥30 events.h⁻¹.
In the stepwise discriminant analysis done separately in obese and non-obese patients according to the AHI:

The age, anterior face height, and middle face length were included in Fisher’s linear discriminant function in non-obese patients. The results obtained from the discriminant analysis are shown in Table 3a. The total result for this discrimination was 79.2%. If the diagnosis was made using parameters other than AHI, the estimated success of discrimination was 79.2%. The estimated successes were distributed as follows: AHI<30 events.h⁻¹ at 76.3%, AHI≥30 events.h⁻¹ at 90% (Table 3b).
Hy-MPPerp was included in Fisher’s linear discriminant function in obese patients. The results obtained from the discriminant analysis are shown in Table 4a. The total result for this discrimination was 70.3%. If the diagnosis was made using parameters other than AHI, the estimated success of discrimination was 70.3%. The estimated successes were distributed as follows: AHI<30 events.h⁻¹ at 71.4%, the AHI≥30 events.h⁻¹ at 69.6% (Table 4b).

### Discussion

The main finding of this study is that the craniofacial morphology and BMI have an effect on the severity of OSAS. There are obvious sex differences in the craniofacial skeletal characteristics that contribute to OSAS severity and, to evaluate OSAS severity, different anthropometric and cephalometric measurements should be used for men and women (12). Furthermore, it has been reported that there are racial differences between the prevalence and characteristics of OSAS (21). Thus, the male patients were included in the study and the cephalometric features of Turkish male patients with OSAS were evaluated.

Although OSAS, being a progressive disease, increases with age (22) the possible influence of age on cephalometric measurements has been frequently neglected (10). However, Sakakibara et al. (10) could not determine any correlation be-

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**Table 2. AHI-BMI interaction table showing the statistically significant differences**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Diagnosis</th>
<th>BMI&lt;30 kg.m⁻²</th>
<th>BMI≥30 kg.m⁻²</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm) (p&lt;0.05)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>171.05±5.09</td>
<td>170.21±4.66</td>
<td>170.64±0.98</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>173.30±4.52</td>
<td>167.30±8.96</td>
<td>170.30±1.19</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>172.18±1.12</td>
<td>168.76±1.06</td>
<td></td>
</tr>
<tr>
<td>Waist Circumference (cm) (p&lt;0.01)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>92.39±11.97</td>
<td>109.0±8.37</td>
<td>100.69±1.79</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>98.60±8.22</td>
<td>110.48±13.08</td>
<td>104.54±2.16</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>95.49±2.03</td>
<td>109.74±1.94</td>
<td></td>
</tr>
<tr>
<td>Arousal Index (arousals.h⁻¹) (p&lt;0.01)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>19.89±12.84</td>
<td>13.35±9.89</td>
<td>18.12±12.27</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>36.90±10.38</td>
<td>30.67±18.87</td>
<td>32.56±16.84</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>23.42±14.11</td>
<td>24.12±17.95</td>
<td></td>
</tr>
<tr>
<td>Awake mean SaO₂% (p&lt;0.05)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>94.05±1.83</td>
<td>94.29±1.54</td>
<td>94.17±0.27</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>93.90±1.29</td>
<td>92.65±1.72</td>
<td>93.28±0.32</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>93.98±0.30</td>
<td>93.47±0.29</td>
<td></td>
</tr>
<tr>
<td>Lowest SaO2% (p&lt;0.01)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>83.08±5.77</td>
<td>81.50±6.61</td>
<td>82.29±1.19</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>76.50±9.94</td>
<td>72.52±9.53</td>
<td>74.51±1.44</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>79.79±1.35</td>
<td>77.01±1.29</td>
<td></td>
</tr>
<tr>
<td>Maxillary length (mm) (p&lt;0.01)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>55.22±3.83</td>
<td>58.32±3.88</td>
<td>56.77±0.56</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>53.87±1.72</td>
<td>58.84±3.52</td>
<td>56.36±0.68</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>54.54±0.64</td>
<td>58.58±0.61</td>
<td></td>
</tr>
<tr>
<td>Mandibular body length (mm) (p&lt;0.05)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>90.40±092</td>
<td>93.80±1.51</td>
<td>92.10±0.89</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>87.82±1.79</td>
<td>91.29±1.18</td>
<td>89.56±1.07</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>89.11±1.01</td>
<td>92.55±0.96</td>
<td></td>
</tr>
<tr>
<td>Hy-MPPerp (mm) (p&lt;0.05)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>22.19±0.99</td>
<td>19.10±1.64</td>
<td>20.65±0.96</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>23.09±1.94</td>
<td>24.39±1.28</td>
<td>23.74±1.16</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>22.64±1.09</td>
<td>21.75±1.04</td>
<td></td>
</tr>
<tr>
<td>Posterior face height (mm) (p&lt;0.01)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>90.52±1.11</td>
<td>94.90±1.84</td>
<td>92.71±1.07</td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>64.18±1.50</td>
<td>67.84±1.27</td>
<td>62.15±1.30</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>90.07±1.22</td>
<td>94.79±1.16</td>
<td></td>
</tr>
<tr>
<td>Middle face height (mm) (p&lt;0.05)</td>
<td>AHI&lt;30 events.h⁻¹</td>
<td>96.83±0.75</td>
<td>98.06±1.24</td>
<td></td>
</tr>
<tr>
<td></td>
<td>AHI≥30 events.h⁻¹</td>
<td>94.07±1.47</td>
<td>97.74±0.97</td>
<td>97.44±0.73</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>95.45±0.83</td>
<td>97.92±0.79</td>
<td>95.92±0.88</td>
</tr>
</tbody>
</table>

*a: the differences between diagnosis. A: the differences between BMI. Hy-MPPerp: distance of hyoid bone to mandibular plane*
Obesity, which tends toward excessive soft tissue in the upper airway, might be related to OSAS. It has been demonstrated that BMI and neck size statistically significantly increased with the severity of OSAS (12) which also seems to be in agreement with our data (p<0.01). Although the previous studies showed a correlation between the neck circumference and OSAS (23, 24). Dahlqvist et al. (25) reported that the neck circumference was not independently related to the AHI. In the present study, the neck circumference and also waist circumference were correlated with the AHI and increased with the BMI.

Obesity, because of neck and soft tissue fat deposition and increased pressure on the neck of submental adipose tissue, may predispose to upper airway obstruction (8). Watanabe et al. (26) also showed that patients with closure of the velopharynx-only type were more obese and had less severe craniofacial abnormalities. In contrast, patients with closure of the combined velopharynx and oropharynx type were less obese and had more severe craniofacial abnormalities. It has been reported that obesity and craniofacial abnormalities contribute synergistically to an increase in the collapsibility of the passive pharyngeal airway (26). However, the upper airway shows anatomic differences during awake and sleep periods, and an occlusion occurs at different levels along the upper airway for patients with OSA. The obstruction begins with fat deposits on the lateral pharyngeal wall, where it cannot be seen in cephalometric graphs (23). The upper airway and soft tissue measurements did not show any statistically significant differences between the mean levels of the BMI and the AHI in the present study.

Studies on sleep-disordered breathing in Europe and America have shown relationships with obesity and craniofacial morphology (26, 27). However, the data in the literature are inconsistent, such that studies evaluating cephalometric anomalies in patients with OSAS have found no clear-cut morphological characteristics (28). Moreover, one study showed that gender and racial variations are present in cephalometric parameters (29). Furthermore, Ishiguro et al. (30) reported that skeletal conditions were probably the factors that contributed most to a further increase in the severity of Japanese male OSAS.

Sadeghianrizi et al. (31) showed that craniofacial morphology differs between obese and normal adolescents. In general, obesity was associated with bimaxillary prognathism and relatively greater facial measurements. The measurements of maxillary length, mandibular body length, posterior face height, middle face length, and lower face height were found increased in obese patients over non-obese patients. The findings in adult men were in accordance with the study of Sadeghianrizi et al. (31). The craniofacial bones are structured during adolescence and do not change in adults (32). It is considered that these measurements cannot be increased by gaining weight in adulthood. Therefore, obese adults might be obese during adolescence and might have a genetic tendency. However, no significant relationship between these measurements and the AHI was found in this study.

Although obesity is a factor very much related to the presence of OSAS, (22) some non-obese patients have severe OSAS (5). The reason for this might be explained by
the comparison of obese patients with AHI<30 events.h⁻¹ and non-obese patients with AHI≥30 events.h⁻¹, as shown in Table 1. Non-obese patients with severe OSAS were characterized by a greater tongue thickness, an inferiorly positioned hyoid bone, a greater mandibular body length, and a longer soft palate (2). In the present study, although the hyoid bone position was not statistically associated with the BMI, there was a strong association with the AHI. As the position of the hyoid bone becomes more inferior, the condition becomes more severe, as mentioned in the literature (12). In the stepwise discriminant analysis, hyoid bone position was included and the estimated success of discrimination of AHI's level was 70.3% in the obese patients. The mandibular body length, maxillary length, and middle face length (sagittal measurements) in non-obese patients with AHI≥30 events.h⁻¹ were found to be less than the ones measured in obese patients with AHI<30 events.h⁻¹. These patients had a divergent face type with increased SNGoGn. The reason the AHI was less than 30 depended on obesity with normal craniofacial morphology in obese patients with AHI<30 events.h⁻¹. The craniofacial morphology affected the severity of OSAS with non-obese patients, and if the craniofacial morphology changed with a tendency toward OSAS with obesity, the severity of OSAS increased more, as shown in Table 1.

One of the major limitations of this study was the lack of cephalograms taken in the supine and sleep positions. The upper airway obstruction usually occurs during sleep, so cephalograms taken in the supine position have the advantage of closely resembling those of the sleep position (33). However, the reproducibility of the head position in the supine position is difficult. In a study that compared supine and upright cephalograms, no significant difference in the skeletal pattern was found (33, 34). The clinical meaningfulness of group comparisons of cephalometric measurements may be questionable, because the traditionally used lateral cephalograms do not provide three-dimensional information (13). However, it has been reported by several investigators that cephalometric measurements had clinical significance. Therefore they can be used for the evaluation of the pathogenesis and pathophysiology of OSAS in a patient, and for the selection of the appropriate treatment modality (34).

Conclusion

The craniofacial morphology and BMI have an influence on AHI. The craniofacial morphology is effective in the severity of OSAS in non-obese patients. If the craniofacial morphology changes favored OSAS in obese patients, the severity of the OSAS increased more.

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Conflict of Interest

No conflict of interest was declared by the authors.

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