Undiagnosed Obstructive Sleep Apnea in Patients with Dentofacial Deformity Undergoing Orthognathic Surgery: A Prospective Cohort Study

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Abstract

Objectives: The primary aim of this study was to determine the prevalence of previously undiagnosed obstructive sleep apnea (OSA) among patients with jaw deformities scheduled for corrective jaw surgery. The secondary aim was to describe changes in objective and subjective sleep parameters following surgery.

Methods: This prospective study was conducted among patients who had not previously been diagnosed with OSA and were scheduled for jaw corrective surgery between 2021 and 2022. Home Sleep Apnea Testing was used alongside the Epworth Sleepiness Scale and the STOP-BANG questionnaire. Consented patients underwent HSAT preoperatively and at a median of 5.7 months postoperatively (interquartile range 3.8–7.4 months). Jaw corrective surgery was performed by experienced surgeons at a tertiary maxillofacial surgery referral center.

Results: A total of 64 patients (24 males, 40 females; mean age 28.23 years) completed the baseline assessment. We identified previously undiagnosed OSA in 43.75% (95% CI 32.3–55.9%). Thirty-nine patients completed postoperative testing; of these, 25 (64.1%) had a baseline AHI below 5 events/h, and only one had moderate-to-severe disease. Within this subgroup, STOP-BANG scores improved significantly ($p < 0.001$), and sleep efficiency increased from 80.1% to 87.3% (mean difference +7.2 percentage points, 95% CI +3.1 to +11.2; $p = 0.001$). However, the AHI did not change (mean difference +0.79 events/h, 95% CI −1.24 to +2.82; $p = 0.434$). The one patient with severe OSA improved from an AHI of 33.6 to 4 events/h.

Conclusions: Previously undiagnosed OSA is common among patients presenting for jaw corrective surgery, supporting routine preoperative screening in this population. In a subgroup composed predominantly of patients without OSA or with only mild OSA, AHI did not change after surgery, although sleep efficiency and symptom scores improved. These secondary findings should not be extrapolated to patients with moderate-to-severe disease, few of whom completed postoperative testing.

Keywords: Sleep apnea, Jaw Deformities, Orthognathic Surgery, Jaw Corrective Surgery, Apnea-Hypopnea Index, Home Sleep Apnea Testing.

Introduction

Obstructive sleep apnea (OSA) is a prevalent chronic condition with multiple contributing factors, characterized by repeated partial or complete collapse of the upper airway during sleep, leading to periodic oxygen desaturation and disruption of the sleep cycle.[1,2] OSA is a heterogeneous disorder with a complex, multifaceted pathophysiology, including impaired anatomy, a low arousal threshold, ventilatory control instability, and reduced upper airway muscle tone.[1,3]

Obesity is commonly cited as the primary cause of OSA, and research indicates that many individuals have anatomical predispositions, such as enlarged tonsils, a retrusive jaw, and obstructed nasal cavities, that contribute

to upper airway collapse.[1,3,4] It has been reported that most OSA patients remain undiagnosed or untreated.[5,6] Sleep surgeons also consider anatomical factors as potential therapeutic targets for skeletal surgery.[3]

Sleep quality is important and recognized as an integral cornerstone of preventive healthcare.[7,8] OSA is associated with cardiovascular diseases, stroke, and risk factors for motor vehicle accidents (MVAs) and work-related accidents, all of which are linked to increased mortality and medical costs.[2,4,6,8–12] The death rate among untreated severe OSA patients is 3.8 times higher than that of the general population.[13] The standard treatment for OSA is continuous positive airway pressure (CPAP); however, adherence is often poor.[5,10,12,14] CPAP adherence ranges from 17% to 85%.[8,10,12,14,15] In the Omani population, Al-Abri et al. examined acceptance of and adherence to CPAP among patients undergoing polysomnography and identified factors influencing adherence in this setting.[16] This variability in adherence is part of what sustains interest in skeletal surgical options for selected patients.

Several surgical approaches have been used to manage OSA, ranging from soft- tissue procedures to skeletal osteotomies. Among these, maxillomandibular advancement (MMA) produces the greatest enlargement of the posterior airway space at multiple levels. It is regarded as the most effective skeletal procedure for patients with moderate-to-severe disease who are intolerant of continuous positive airway pressure. Reported success and cure rates for MMA, based on changes in the apnea–hypopnea index, range from 85.5%–100% and 4.8%–43.5%, respectively.[3,6,8,10–12,17–20] Most studies examining MMA outcomes for OSA focus on objective metrics such as the apnea- hypopnea index (AHI), respiratory disturbance index, oxygen desaturation levels, and changes in upper airway volume.[4,5,10,11,18,21] While improving these indicators is crucial, patients perceive treatment as successful when daytime symptoms, including excessive moodiness, stress-related fatigue, headaches, and cognitive function, improve.[4,8,21]

Although MMA is well established in the surgical management of moderate- to severe OSA, this evidence derives almost exclusively from cohorts in whom OSA was the indication for surgery. Most orthognathic procedures performed worldwide, however, are undertaken for dentofacial deformity, malocclusion, and facial aesthetics, and are performed in patients who are not routinely screened preoperatively for sleep- disordered breathing.

This distinction is clinically important. Patients with dentofacial deformity, particularly those with mandibular retrognathia and skeletal Class II patterns, share an anatomical risk profile for upper airway collapse, suggesting a substantial burden of unrecognized OSA in a young, otherwise healthy population. Furthermore, orthognathic procedures undertaken for occlusal indications include both advancement and setback movements, the latter of which may reduce the posterior airway space and has been associated with de novo sleep-disordered breathing. To our knowledge, the prevalence of undiagnosed OSA in patients presenting for orthognathic surgery has not previously been reported in an Omani or wider Gulf population.

Al-Nuumani et al. described an Omani patient with temporomandibular joint ankylosis, mandibular retrognathia, and severe sleep apnea, showing how skeletal restrictions can cause airway obstruction.[22] While severe cases are obvious, milder, undiagnosed sleep apnea might exist in patients seeking correction of dentofacial deformities.

The primary aim of this study was therefore to determine the prevalence of previously undiagnosed OSA among patients with dentofacial deformity scheduled for jaw corrective surgery in our population. The secondary aim was to describe changes in objective and subjective sleep parameters following surgery in this cohort.

Methods

A prospective cohort study was conducted from 2021 to 2022 at Al-Nahdha Hospital, a tertiary maxillofacial surgery referral center. The study included all male and female patients with dentofacial deformity scheduled for corrective jaw surgery who had not previously been evaluated for OSA.

Patients previously diagnosed with OSA, medically compromised patients, and those diagnosed with a craniofacial syndrome were excluded. In addition, patients were excluded if their clinical data were incomplete or their home sleep apnea tests (HSATs) were technically inadequate. An AHI $\geq$ 5 events/h is considered indicative of OSA. The study's data and variables include gender, age, body mass index (BMI), AHI, total sleep time, sleep efficiency, minimal desaturation SpO2, STOP-BANG, snoring index, Epworth Sleepiness Scale (ESS), and type of surgery.

No a priori sample size calculation was performed. All eligible patients attending the maxillofacial clinic during the recruitment period were invited, with the final sample determined by those who consented. For the primary prevalence outcome, the 64 participants provided a 95% confidence interval of approximately ±12

percentage points around the observed proportion. The secondary analysis was limited to participants with paired pre- and postoperative assessments and was considered exploratory.

Home Sleep Apnea Testing (HSAT) type III was used to conduct sleep studies for all included patients, and scoring was based on the American Association of Sleep Medicine (AASM) criteria (2017).[5,23] Based on AASM guidelines, an AHI $\geqslant$ 5 is considered mild OSA, AHI $\geqslant$ 15 moderate OSA, and AHI > 30 severe OSA.[24,25]

Validated clinical questionnaires are used to assess the risk of OSA and daytime sleepiness, including the STOP-BANG and ESS.[8,26] The STOP-BANG questionnaire is an 8-question survey that includes four subjective and four objective items.[25] It comprises Snoring, Tiredness, Observed apnea, and high blood pressure (STOP), as well as Body mass index, Age, Neck circumference, and Gender (BANG). The ESS is a self-report questionnaire with 8 questions; each is scored on a 0–3 scale. These questionnaires are based on patients' responses.

The surgeon conducted clinical evaluations of the participants, including history and physical examinations, and a specialized sleep nurse provided instructions for HSATs and data collection. The patients were screened with the STOP-BANG and ESS questionnaires. All consenting patients underwent HSAT level III (SOMNO-touch Resp, Germany) preoperatively and 3–12 months postoperatively. The protocol specified postoperative testing at six months; in practice, the interval was a median of 5.7 months (interquartile range 3.8–7.4; range 3.0–12.0), reflecting scheduling disruptions during the COVID-19 pandemic. Within a week before surgery, patients were contacted and instructed to perform HSAT level III either at home if they lived near the hospital or on the day of admission if they were traveling from a distance (>50 km). All patients underwent presurgical orthodontic treatment, and general anesthesia was used for surgery. In coordination with orthodontists, surgical planning was based on the presenting jaw deformities, using study models and articulators to create intermediate and final splints. Highly experienced surgeons performed jaw corrective surgeries using the same osteotomy methods (Table 1).

Table 1: Types of jaw corrective surgery procedures.

For two jaw surgeries, a maxillary Le Fort I osteotomy was performed first through an incision extending from one first premolar to the other. This resulted in a horizontal osteotomy line extending from the pyriform rim to the posterior zygomatic buttress. The maxilla was positioned using an intermediate interocclusal splint and fixed with four titanium miniplates, each with four monocortical screws (KLS Martin, Freiburg, Germany). For the mandible, a bilateral sagittal split osteotomy (BSSO) with a low medial cut (Posnick modification) was performed, and the mandible was positioned with a final interocclusal splint. Osteotomized fragments were fixed with two titanium miniplates and four monocortical screws per miniplate (KLS Martin, Freiburg, Germany). The genioplasty osteotomy was designed to ensure extensive involvement of the lingual cortex, including a sufficient portion of the genioglossus and geniohyoid muscles. Fixation was achieved with a prebent chin plate or bicortical screws (Fig. 1). Patients were placed on a guiding elastic postoperatively.

Types of Surgery	**n**
Not operated	5
Genioplasty	4
Mandible Advancement	5
Mandible Advancement + Genioplasty	14
Mandible Setback	1
Mandible Setback + Genioplasty	1
Maxilla Impaction	1
Maxilla Advancement	6
Maxilla Impaction + Genioplasty	3
Maxilla Advancement + Genioplasty	3
Maxilla Advancement + Down-grafting	1
Maxilla Advancement + Mandibular Setback	3
Maxilla Advancement & Down-grafting + Genioplasty	1
Maxilla Advancement + Mand. Advancement	2
Maxilla Advancement & Impaction + Genioplasty	1
Maxilla Advancement + Mand. setback + Genioplasty	2
Maxilla Impaction + Mand. Advancement + Genioplasty	4
Maxilla Advancement + Mand. Advancement + Genioplasty	1
Maxilla Advancement & impaction + Mand. Advancement + Genioplasty	6



Figure 1: The clinical picture of a patient who underwent bilateral sagittal split osteotomy (BSSO) and genioplasty, fixed with L-Shaped plates for BSSO advancement and bicortical screws for the genioplasty.

The patient repeated HSAT 3–12 months postoperatively. A senior sleep technician analyzed all sleep studies at the sleep physiology center at Sultan Qaboos University Hospital (SQUH) in accordance with AASM guidelines.

Data were entered into a Microsoft Excel database (version 16.0, Microsoft Corp., Redmond, Washington, USA), and statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS), version 25.0 (IBM Corp., Armonk, New York, USA). Descriptive statistics summarized patients' characteristics. Continuous variables were presented as mean ± standard deviation, and categorical variables as frequency and percentage (Table II).

The primary outcome was OSA prevalence, defined as an AHI ≥5 events/h. Secondary outcomes included changes in AHI, oxygen desaturation index, minimum oxygen saturation, sleep efficiency, snoring index, STOP-BANG score, and Epworth Sleepiness Scale score between preoperative and postoperative assessments.

Paired comparisons were performed using the paired t-test. We assessed normality of paired differences using the Shapiro–Wilk test; we used the Wilcoxon signed-rank test as a sensitivity analysis when appropriate and reported both results. Mean differences are presented with 95% confidence intervals, and paired effect sizes as Cohen's d. The 95% confidence interval for OSA prevalence was calculated using the Wilson method. Statistical significance was set at $p < 0.05$. OSA was diagnosed according to AASM criteria.

Results

We approached 120 patients with jaw deformities who attended the maxillofacial clinic. Only sixty-four participants agreed to participate and were included in the study at baseline. Of the 64 patients, 39 were included in the second-phase analysis because they had completed HSAT postoperatively (Figure 1). The mean age of patients was 28.23 ± 6.59 years (range: 19 to 45 years); 40 patients (62.5%) were female; and their preoperative BMI was 25.75 ± 5.67 kg/m2. The types of surgeries performed are listed in Table I.

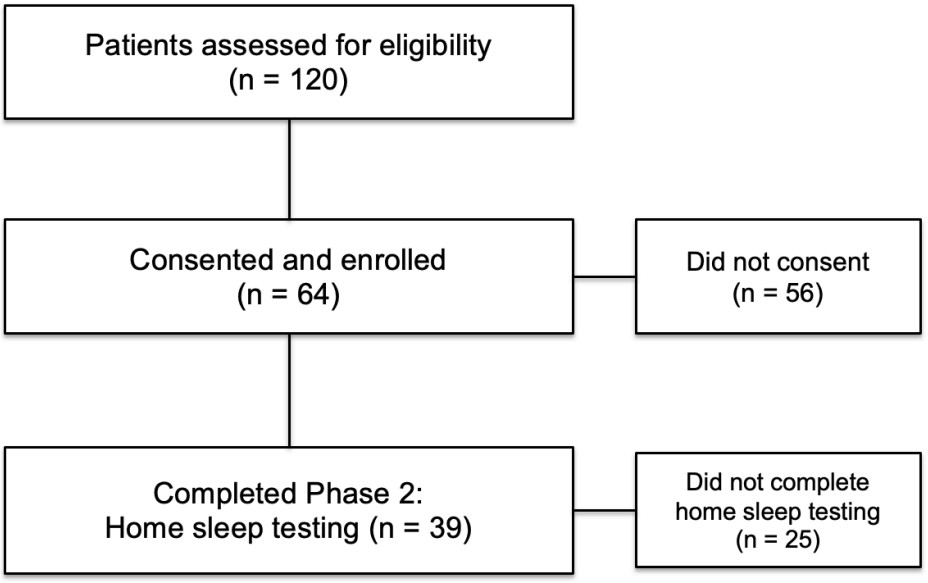


Figure 1: Flow diagram of participant recruitment, enrolment, and completion of home sleep testing.

Table 2: Demographic and clinical characteristics of the included patients (N= 64). Data are presented as numbers (%) except for age and BMI, which are reported as mean ± SD. Apnea-Hypopnea Index (AHI). Body Mass Index (BMI).

Clinical characteristics	**N (%)**
Age (years (mean ± SD))	28.23 ± 6.59
Age distribution (years)	
18 – 20	5 (7.8%)
21 – 30	40 (62.5%)
31 – 40	16 (25%)
41 – 50	3 (4.6%)
BMI, Kg/m^2 (preoperatively)	25.75 ± **5.67**
Prevalence of AHI at a cutoff of ≥ 5	
< 5	36
5 – 14.9	21
15 – 29.9	4
≥ 30	3
Sex distribution	64
Male	24 (37.5%)
Female	40 (62.5%)
Skeletal Patterns	
Skeletal type I	0
Skeletal type II	47 (73.4%)
Skeletal type III	17 (26.6%)

Among the 39 participants analyzed for surgical outcomes, 25 (64.1%) had an AHI <5 events/h, 13 (33.3%) had mild OSA, none had moderate OSA, and one (2.6%) had severe OSA. Thus, the analyzed subgroup was

predominantly composed of participants without OSA or with mild disease and was more heavily weighted toward normal and mild disease than the original cohort of 64 participants.

Forty-seven patients were deemed to have presurgical skeletal class II malocclusion, while 17 patients were considered to have presurgical skeletal class III. A wide range of orthognathic surgeries were performed, including MMA, mandibular advancement with genioplasty, maxillary impaction, and genioplasty. Mandibular advancement with genioplasty was the most performed surgery, followed by MMA, including genioplasty and maxillary advancement. Table II presents the remaining patient characteristics.

Paired-samples t-tests were used to compare pre- and post-scores for the STOP-BANG and ESS. STOP-BANG scores showed a significant reduction in mean postoperative scores (preoperative 3.28 ± 1.57; postoperative 1.92 ± 1.22; mean difference −1.36, 95% CI −1.96 to −0.76; Cohen's d = −0.74; $p < 0.001$). No significant difference was noted between pre- and post-ESS scores ($p = 0.65$) (Table III). Body mass index decreased significantly following surgery, from a mean of 25.41 kg/m² to 24.79 kg/m² (mean difference −0.62 kg/m², 95% CI −1.06 to −0.17; paired t-test $p = 0.008$).

Table 3: Comparison of pre- and postoperative sleep parameters and questionnaire scores.

Parameter	n	Pre-operative mean (SD)	Post-operative mean (SD)	Mean difference (95% CI)	Cohen's d	p (paired t)	p (Wilcoxon)
AHI (events/h)	39	5.63 (5.62)	6.42 (5.08)	+0.79 (−1.24 to +2.82)	0.13	0.434	0.104
ODI (events/h)	37	2.55 (3.47)	2.99 (2.82)	+0.44 (−0.96 to +1.84)	0.11	0.528	0.096
Minimum SpO_2 (%)	38	91.8 (3.3)	92.7 (2.5)	+0.9 (−0.4 to +2.3)	0.24	0.155	0.289
Total sleep time (min)	39	332 (71)	334 (93)	+3 (−35 to +40)	0.02	0.890	0.655
Sleep efficiency (%)	39	80.1 (10.9)	87.3 (7.2)	+7.2 (+3.1 to +11.2)	0.57	0.001	<0.001
Snoring index	38	8.54 (19.32)	3.42 (6.36)	−5.12 (−11.85 to +1.60)	−0.25	0.131	0.514
BMI (kg/m²)	39	25.41 (5.91)	24.79 (5.72)	−0.62 (−1.06 to −0.17)	−0.45	0.008	0.005
ESS	39	8.49 (3.46)	8.00 (4.11)	−0.24 (−1.32 to +0.84)	−0.07	0.651	0.390
STOP-BANG	39	3.28 (1.57)	1.92 (1.22)	−1.36 (−1.96 to −0.76)	−0.74	<0.001	<0.001

AHI = apnea–hypopnea index; ODI = oxygen desaturation index; BMI = body mass index; ESS = Epworth Sleepiness Scale; STOP-BANG = Snoring, Tiredness, observed apnea, blood Pressure, BMI, Age, Neck circumference, Gender; CI = confidence interval. Positive mean differences indicate an increase from preoperative to postoperative assessment. Cohen's d for paired data is the mean difference divided by the standard deviation of the differences. The Wilcoxon signed-rank test is reported alongside the paired t-test because the paired differences for AHI, ODI, snoring index, and ESS were non-normally distributed on Shapiro–Wilk testing; the two tests agreed in direction and significance for every parameter. Sample sizes vary owing to missing values in individual parameters. A p-value < 0.05 was considered statistically significant.

The overall prevalence of previously undiagnosed obstructive sleep apnea among patients with jaw deformity was 43.75% (n = 28; 95% CI 32.3–55.9%). The distribution of OSA severity and patient characteristics is shown in Table II. The study found that 56.25% of patients with jaw deformity had no OSA (n=36), followed by 32.81% with mild OSA (n=21). The t-test showed no statistically significant difference between males and females ($P > 0.05$). Moreover, only one patient, who initially presented with severe OSA (AHI 33.6) and a high-risk STOP-BANG score, had a minimum preoperative SpO_2 desaturation of 84%. Following surgery, the patient's condition improved markedly, with the AHI reduced to 4, a low-risk STOP-BANG score, and a minimum SpO_2 desaturation of 93%. The patient had a skeletal class II deformity and was treated with mandibular advancement (13 mm) and genioplasty (8 mm chin advancement) (Fig. 2). Another patient developed moderate OSA after maxillary advancement and mandibular setback. Most operated cases had either mild OSA (AHI=5-15) or no OSA (AHI<5).

Objective sleep parameters before and after surgery are presented in Table III. AHI showed no significant change (mean difference, +0.79 events/h; 95% CI, −1.24 to +2.82; paired t-test, p = 0.434; Wilcoxon signed-rank test, p = 0.104), with the confidence interval excluding a clinically meaningful improvement. Sleep efficiency increased from 80.1% to 87.3% (mean difference, +7.2 percentage points; 95% CI, +3.1 to +11.2; p = 0.001; Cohen's d = 0.57). No significant changes were observed in oxygen desaturation index, minimum oxygen saturation, total sleep time, snoring index, or ESS.

Using an AHI threshold of 15 events/h, one participant had OSA preoperatively and a different participant postoperatively. The participant with severe OSA improved from an AHI of 33.6 to 4 events/h, whereas another participant developed OSA, with AHI increasing from 4.5 to 20.5 events/h following maxillary advancement with mandibular setback.

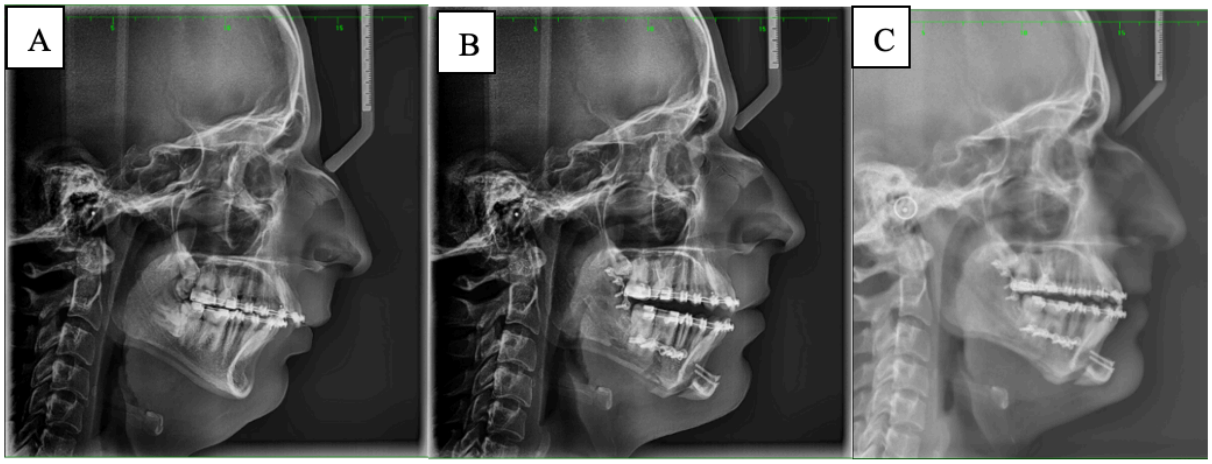


Figure 2: Lateral Cephalometric radiograph. A. Preoperatively. B. Postoperatively, 1 day. C. Postoperatively, 6 months.

Discussion

This cohort study aimed to determine the proportion of patients with OSA among those with jaw deformities in the local population and to examine the outcomes of jaw corrective surgery. The study found that the overall prevalence of OSA in the local population was 43.75% among patients aged 19-45. Few studies have summarized OSA prevalence across different age ranges, including systematic analyses. Zasadzinska-Stempniak et al. reported that in the general adult population (aged 18– 30 years), the prevalence of OSA among jaw deformities was 16%. In contrast, another systematic review reported that OSA in the general adult population, using a cutoff of ≥5 events/h, ranged from 9% to 38%.[1,27]

The prevalence of OSA among patients with jaw deformities varies significantly across populations and definitions of OSA.[2,23,27] The proportion of OSA may be higher than indicated by the selected population and the AHI definition.[2,23,27] In advanced age groups, the overall prevalence was 84%.[1] In the reported literature, OSA prevalence increased with age, whereas the majority of patients in our study were young adults (<45 years).[5,27] In addition, the prevalence of OSA is higher among men (13–33%, 50%) than among women (6–19%, 23%), which is similar to the findings of this study.[5,27–29] In our cohort, OSA was identified in 23% of men and 20% of women. However, there were no statistically significant differences in OSA prevalence between males and females in our sample (P> 0.05).

Al-Abri et al. studied Omani individuals using polysomnography, finding sex differences in the apnea–hypopnea index that were linked to body mass index and age.[30] The absence of a significant sex difference in our mostly young, small-sample group may be due to size, narrow age range, and different reasons for presentation, focusing on dentofacial deformities rather than sleep issues.

Recognizing and treating OSA is crucial for many reasons, including improving quality of life (QOL).[23] Improving the patient's QOL would eventually reduce the risk of chronic consequences of untreated OSA and lower motor vehicle accident rates.[5,23]

HSAT is considered a reliable tool for screening and diagnosing OSA.[29] The postoperative follow-up sleep study period varied across studies; some patients were followed for 3 months, others for more than 6 months, and others over a year.[13] Another study used a 10-day postoperative sleep study follow-up.[31] The initial plan for this study was to perform postoperative HSAT follow-up after six months. Contrary to initial expectations, the

postoperative sleep study follow-up period was conducted after 3 months in some cases and up to 12 months in others due to COVID-19 pandemic restrictions during the study period. Moreover, the outcome of the postoperative sleep study follow-up might be influenced by soft-tissue swelling, especially in the early postoperative period.[13] Abbasi et al. reported different postoperative sleep study timings, with follow-up at 1 month and 6 months.[29] The variability in follow-up interval is a limitation. Participants tested at the lower end of this range may still exhibit postoperative soft-tissue edema and altered neuromuscular function. In contrast, those tested later are more likely to reflect a stable skeletal and functional result. This heterogeneity may have contributed to variance in the postoperative measurement.

The study examined other potential variables that could influence surgical outcomes, including age, sex, minimum desaturation, and snoring index. No significant postoperative change was observed in these variables, although body mass index decreased significantly, as reported above. Most of the patients' symptoms have improved significantly postoperatively, as indicated by STOP-BANG scores. The ESS self-report assessment was used to assess daytime sleepiness. Few patients presented with excessive daytime sleepiness (ESS score > 10). The STOP-BANG and ESS assessments in this study did not correlate with AHI outcomes. Similarly, Lipford et al. reported that ESS is not associated with AHI at mild to moderate OSA.[32]

The significant reduction in STOP-BANG score without a corresponding change in AHI warrants cautious interpretation. STOP-BANG incorporates anthropometric and demographic factors, and the significant reduction in BMI (mean difference, −0.62 kg/m²; $p = 0.008$) may have contributed to the lower score. In contrast, ESS, which does not include anthropometric variables, remained unchanged ($p = 0.651$), as did AHI. The baseline ESS of 8, below the threshold of 10, also limited potential for improvement.[25]

The predominance of participants with AHI <5 and the single participant with moderate-to-severe OSA limited the ability to detect objective changes. Notably, this participant's AHI improved from 33.6 to 4 events/h, consistent with the established efficacy of MMA. Furthermore, HSAT may underestimate AHI, particularly in mild disease. Finally, although skeletal advancement enlarges the airway, non-anatomical factors such as muscle tone, arousal threshold, and ventilatory stability may also influence airway patency and may not be substantially altered by surgery.

Craniofacial deformity is considered a major cause of OSA.[4,16,17] Different factors, including abnormal anatomy, airway resistance, and individual patient anatomy, impact OSA severity.[20,33] Orthognathic surgeries aim to improve facial aesthetics and anatomical discrepancies, improve functional morphology, and change airway volume.[13,19,21] Hence, this would change the position of the skeletal pattern and soft tissues, including the pharyngeal airway space and the hyoid bone, ultimately improving anatomical condition, sleep quality, and oxygen saturation.[8,13,18,20,21,34] Since orthognathic surgery is considered a treatment option for moderate-to-severe OSA, the AHI score used was≥ 15 events/h.[5,8,10]

Two-jaw surgery is widely practiced worldwide and yields highly favorable outcomes.[29] Multiple studies have shown a strong correlation between jaw surgery and OSA.[8,11] MMA is considered the first-line treatment for moderate-to-severe OSA, including severe OSA, by increasing the upper airway diameter at multiple levels and reducing airway collapse.[8,11,14] Although orthognathic surgery has complications and side effects, it is considered safe and effective.[8,11] One patient with severe AHI was successfully cured after orthognathic surgery (mandibular advancement and genioplasty), with an AHI of 4 events/h. Despite numerous studies supporting the outcomes of MMA for the management of OSA, our data show no significant difference in AHI pre- and post-operatively, since the majority of our patients had normal to mild OSA.[15]

Reported MMA success rates derive primarily from cohorts with moderate-to-severe OSA in whom OSA was the surgical indication.[13] In the meta-analysis by Holty and Guilleminault, the mean preoperative AHI among 627 adults was 63.9 events/h, compared with 5.63 events/h in our analyzed subgroup.[14] Our cohort was selected for dentofacial deformity and predominantly had no or mild OSA; therefore, these populations are not directly comparable. Our findings neither support nor contradict the MMA literature; notably, the single participant with severe OSA achieved an outcome consistent with previous reports.

On the other hand, many studies have reported the development of OSA following orthognathic surgery, including cases with skeletal class III.[21–23] The incidence of OSA after Le Fort I advancement, bilateral sagittal split osteotomy setback, and maxillary advancement was 19.2%, 8.57%, and 0.7%, respectively.[13] The first reported case of OSA in skeletal class III after mandibular setback was described by Guilleminalt et al.[8,17] In our study, a single skeletal class III patient developed moderate OSA after maxillary advancement and mandibular setback (postoperative sleep study follow-up was 3 months). Yamada et al. reported that the risk of obstruction may increase during the first month after surgery due to swelling and a reduction in pharyngeal space, a finding similar to that of Abbasi et al.[7,24] The development of OSA in skeletal class III patients who underwent orthognathic surgery depends on surgical and patient factors, such as high BMI, older age, and reduced pharyngeal airway volume. [7,8] Nevertheless, no solid evidence establishes a causal relationship between orthognathic surgery,

the development of OSA in skeletal class III, and changes in upper airway dimensions that predispose the individual to OSA.[7,8,16]

One objective parameter improved significantly: sleep efficiency increased by a mean of 7.2 percentage points (95% CI, 3.1–11.2; p = 0.001), representing a moderate effect. As sleep efficiency reflects sleep continuity and is influenced by arousals and respiratory events, this finding suggests that surgery may improve sleep quality through mechanisms not captured by AHI alone. It also provides an objective correlate of the reported symptomatic improvement. However, this exploratory finding requires confirmation with polysomnography, which allows direct assessment of sleep staging and arousal indices.

Previously undiagnosed OSA was identified in 43.75% of patients presenting for jaw corrective surgery. Despite their young age (mean age, 28.2 years) and relatively normal BMI (mean, 25.75 kg/m²), these patients had not previously been investigated for sleep-disordered breathing. Unrecognized OSA may increase perioperative respiratory risk, particularly with airway manipulation, postoperative edema, intermaxillary fixation, and opioid analgesia.

Importantly, OSA in this cohort was undiagnosed rather than asymptomatic and could potentially have been identified using a brief, validated screening questionnaire, supporting systematic preoperative screening.

Preoperative identification may also inform surgical planning, as different skeletal movements can have opposing effects on pharyngeal airway dimensions. Notably, the participant who developed moderate postoperative OSA underwent mandibular setback. These findings support incorporating validated OSA screening, with objective testing when indicated, into routine preoperative orthognathic assessment.

Objective sleep assessment used type III HSAT rather than attended polysomnography because institutional capacity was insufficient for serial testing of a prospective cohort of this size. As sleep is not directly recorded, AHI is calculated using total recording time rather than total sleep time, which can potentially underestimate the true index. The absence of sleep staging and arousal indices also prevents assessment of respiratory effort-related arousals and sleep fragmentation. These limitations may reduce sensitivity, particularly for mild OSA, thereby underestimating its true prevalence.

Of the 64 participants who completed baseline assessment, 39 (61%) completed postoperative testing. Attrition occurred predominantly during the COVID-19 pandemic due to quarantine requirements and restrictions on non-urgent hospital visits. Thus, the postoperative analysis involved a smaller cohort with limited power to demonstrate objective improvement, representing the main limitation of the surgical component.

The cohort included various procedures, including advancement, setback, impaction, and combined movements, which may have different or opposing effects on posterior airway dimensions. Pooling these procedures may therefore have obscured directionally opposite changes. Notably, the only participant who developed moderate OSA postoperatively underwent maxillary advancement with mandibular setback. Subgroup sizes were insufficient for meaningful comparisons, highlighting the need for adequately powered studies stratified by the direction of skeletal movement.

Different levels of nasal and pharyngeal structures were not assessed because the study focused on previously undiagnosed OSA. Strengths of the study include its prospective design, comprehensive objective and subjective assessment, and focus on patients with jaw deformity who had not previously been evaluated for sleep-disordered breathing.

Conclusion

Previously undiagnosed OSA was found in 43.75% of patients undergoing jaw surgery, supporting routine preoperative sleep screening in this population. In the subgroup with postoperative testing, sleep efficiency and STOP-BANG scores improved, whereas the AHI remained unchanged. Most had no or mild OSA; only one with moderate-to-severe disease showed marked improvement. These results do not oppose MMA as a treatment for moderate-to-severe OSA. Further prospective studies with polysomnography, standardized follow-up, and stratification are needed. The accuracy of OSA diagnosis, follow-up protocols, and patient factors should be considered to improve outcomes.

Disclosure

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Ethical Approval

Ethical approval was granted by the Research and Ethics Committee, Al-Nahdha Hospital (MOH/ANH/RC/2018). All patients provided written informed consent.

References

1. Senaratna CV, Perret JL, Lodge CJ, Lowe AJ, Campbell BE, Matheson MC, et al. Prevalence of obstructive sleep apnea in the general population: A systematic review. Sleep Med Rev. 2017 Aug;34:70–81. doi:10.1016/j.smrv.2016.07.002
2. Mediano O, González Mangado N, Montserrat JM, Alonso-Álvarez ML, Almendros I, Alonso-Fernández A, et al. [Translated article] International consensus document on obstructive sleep apnea. Arch Bronconeumol. 2022 Jan;58(1):T52–68. doi:10.1016/j.arbres.2021.03.027
3. Awad M, Gouveia C, Zaghi S, Camacho M, Liu SYC. Changing practice: Trends in skeletal surgery for obstructive sleep apnea. J Cranio-Maxillofac Surg. 2019 Aug;47(8):1185–9. doi:10.1016/j.jcms.2018.11.005
4. Posnick JC, Adachie A, Singh N, Choi E. "Silent" Sleep Apnea in Dentofacial Deformities and Prevalence of Daytime Sleepiness After Orthognathic and Intranasal Surgery. J Oral Maxillofac Surg. 2018 Apr;76(4):833–43. doi:10.1016/j.joms.2017.09.003
5. Chang JL, Goldberg AN, Alt JA, Mohammed A, Ashbrook L, Auckley D, et al. International Consensus Statement on Obstructive Sleep Apnea. Int Forum Allergy Rhinol. 2023 Jul;13(7):1061–482. doi:10.1002/alr.23079
6. Young T, Blustein J, Finn L, Palta M. Sleep-Disordered Breathing and Motor Vehicle Accidents in a Population-Based Sample of Employed Adults. Sleep. 1997 Aug;20(8):608–13. doi:10.1093/sleep/20.8.608
7. Rosen IM, Kirsch DB, Chervin RD, Carden KA, Ramar K, Aurora RN, et al. Clinical Use of a Home Sleep Apnea Test: An American Academy of Sleep Medicine Position Statement. J Clin Sleep Med. 2017 Oct 15;13(10):1205–7. doi:10.5664/jcsm.6774
8. Goodday RH, Bourque SE, Edwards PB. Objective and Subjective Outcomes Following Maxillomandibular Advancement Surgery for Treatment of Patients With Extremely Severe Obstructive Sleep Apnea (Apnea-Hypopnea Index >100). J Oral Maxillofac Surg. 2016 Mar;74(3):583–9. doi:10.1016/j.joms.2015.07.016
9. Duarte RL, Magalhães-da-Silveira FJ, Oliveira-e-Sá TS, Silva JA, Mello FC, Gozal D. Obstructive Sleep Apnea Screening with a 4-Item Instrument, Named GOAL Questionnaire: Development, Validation and Comparative Study with No-Apnea, STOP-Bang, and NoSAS. Nat Sci Sleep. 2020 Jan;Volume 12:57–67. doi:10.2147/NSS.S238255
10. Furundarena-Padrones L, Cabriada-Nuño V, Brunsó-Casellas J, Garcia-Fernandez RI, Castro-Quintas S, Santos-Zorrozúa B, et al. Correlation between polysomnographic parameters and volumetric changes generated by maxillomandibular advancement surgery in patients with obstructive sleep apnea: a fluid dynamics study. J Clin Sleep Med. 2024 Mar;20(3):371–9. doi:10.5664/jcsm.10874
11. Waite PD, Shettar SM. MAXILLOMANDIBULAR ADVANCEMENT SURGERY: A CURE FOR OBSTRUCTIVE SLEEP APNEA SYNDROME. Oral Maxillofac Surg Clin N Am. 1995 May;7(2):327–34. doi:10.1016/S1042-3699(20)30829-3
12. Dicus Brookes CC, Boyd SB. Controversies in Obstructive Sleep Apnea Surgery. Oral Maxillofac Surg Clin N Am. 2017 Nov;29(4):503–13. doi:10.1016/j.coms.2017.07.005
13. Wei Z, Jiang H, Wang S, Wang Z, Qu B, Hu M. Influencing factors for the development of obstructive sleep apnea after orthognathic surgery in skeletal class III patients: A systematic review. J Cranio-Maxillofac Surg. 2024 Apr;52(4):503–13. doi:10.1016/j.jcms.2024.02.014
14. Holty JEC, Guilleminault C. Maxillomandibular advancement for the treatment of obstructive sleep apnea: A systematic review and meta-analysis. Sleep Med Rev. 2010 Oct;14(5):287–97. doi:10.1016/j.smrv.2009.11.003
15. Semelka M. Diagnosis and Treatment of Obstructive Sleep Apnea in Adults. Vol. 94. 2016;94(5).
16. Al Abri MA, Al-Harmeli A, Al-Habsi M, Jaju D. Acceptance and Compliance of Continuous Positive Airway Pressure in Patients with Obstructive Sleep Apnea: Local Population Survey. Oman Med J. 2020 Nov 15;35(6):e198–e198. doi:10.5001/omj.2020.94
17. Domingues NRAP, Salmen FS, Salmen MD, Sampaio RMF, Sendyk WR, Roman-Torres CVG. Three-Dimensional Evaluation of the Upper Airway after Orthognatic Surgery in Angle Class II and Class III Patients. J Adv Med Res. 2023 Jan 14;8–16. doi:10.9734/jammer/2023/v35i14928
18. Christovam IO, Lisboa CO, Ferreira DMTP, Cury-Saramago AA, Mattos CT. Upper airway dimensions in patients undergoing orthognathic surgery: a systematic review and meta-analysis. Int J Oral Maxillofac Surg. 2016 Apr;45(4):460–71. doi:10.1016/j.ijom.2015.10.018

19. Mattos CT, Vilani GNL, Sant′Anna EF, Ruellas ACO, Maia LC. Effects of orthognathic surgery on oropharyngeal airway: a meta-analysis. Int J Oral Maxillofac Surg. 2011 Dec;40(12):1347–56. doi:10.1016/j.ijom.2011.06.020

20. Kongsong W, Waite PD, Alshahrani F. Comparison of airway changes after maxillomandibular advancement with or without genial tubercle advancement in obstructive sleep apnea using cone-beam computed tomography. Am J Orthod Dentofacial Orthop. 2022 Nov;162(5):616–25. doi:10.1016/j.ajodo.2021.05.016

21. Ravelo V, Olate G, Unibazo A, De Moraes M, Olate S. Airway Space Changes in Dentofacial Deformity After Two-Jaw Orthognathic Surgery in Class II and Class III Skeletal Conditions [Internet]. 2023 [cited 2024 Apr 6]. Available from: https://www.preprints.org/manuscript/202307.0593/v1 doi:10.20944/preprints202307.0593.v1

22. Al-Nuumani IK, Bakathir A, Al-Hashmi A, Al-Abri M, Al-Kindi H, Al-Macki I, et al. A Triad of Temporomandibular Joint Ankylosis, Mandibular Retrognathia and Severe Obstructive Sleep Apnoea: Case report. Sultan Qaboos Univ Med J. 2018 Dec 19;18(3):e379-382. doi:10.18295/squmj.2018.18.03.019

23. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, et al. Clinical Practice Guideline for Diagnostic Testing for Adult Obstructive Sleep Apnea: An American Academy of Sleep Medicine Clinical Practice Guideline. J Clin Sleep Med. 2017 Mar 15;13(03):479–504. doi:10.5664/jcsm.6506

24. Tufik S, Santos-Silva R, Taddei JA, Bittencourt LRA. Obstructive Sleep Apnea Syndrome in the Sao Paulo Epidemiologic Sleep Study. Sleep Med. 2010 May;11(5):441–6. doi:10.1016/j.sleep.2009.10.005

25. Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire. Chest. 2016 Mar;149(3):631–8. doi:10.1378/chest.15-0903

26. Johns MW. A New Method for Measuring Daytime Sleepiness: The Epworth Sleepiness Scale. Sleep. 1991 Nov 1;14(6):540–5. doi:10.1093/sleep/14.6.540

27. Zasadzińska-Stempniak K, Zajączkiewicz H, Kukwa A. Prevalence of Obstructive Sleep Apnea in the Young Adult Population: A Systematic Review. J Clin Med. 2024 Feb 28;13(5):1386. doi:10.3390/jcm13051386

28. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A, et al. Prevalence of Sleep-disordered Breathing in Women: Effects of Gender. Am J Respir Crit Care Med. 2001 Mar 1;163(3):608–13. doi:10.1164/ajrccm.163.3.9911064

29. Abbasi S, Rahpeyma A, Shooshtari Z, Rezaeetalab F, Vaezi T, Samieirad S. Bimaxillary Orthognathic Surgery Does Not Induce Obstructive Sleep Apnea in Skeletal Class III Patients. J Oral Maxillofac Surg. 2022 Aug;80(8):1340–53. doi:10.1016/j.joms.2022.04.010

30. Al-Abri M, Al-Hashmi K, Jaju D, Al-Rawas O, Al-Riyami B, Hassan M. Gender Difference in Relationship of Apnoea/Hypopnoea Index with Body Mass Index and Age in the Omani Population.

31. Hochban W, Schürmann R, Brandenburg U. Mandibular setback for surgical correction of mandibular hyperplasia — does it provoke sleep-related breathing disorders? Int J Oral Maxillofac Surg. 1996 Oct;25(5):333–8. doi:10.1016/S0901-5027(06)80024-X

32. Lipford MC, Wahner-Roedler DL, Welsh GA, Mandrekar J, Thapa P, Olson EJ. Correlation of the Epworth Sleepiness Scale and Sleep-Disordered Breathing in Men and Women. J Clin Sleep Med. 2019 Jan 15;15(01):33–8. doi:10.5664/jcsm.7564

33. Dos Santos Junior JF, Abrahão M, Gregório LC, Zonato AI, Gumieiro EH. Genioplasty for genioglossus muscle advancement in patients with obstructive sleep apnea-hypopnea syndrome and mandibular retrognathia. Braz J Otorhinolaryngol. 2007 Jul;73(4):480–6. doi:10.1016/S1808-8694(15)30099-9

34. Guilleminault C, Riley R, Powell N. Sleep Apnea in Normal Subjects following Mandibular Osteotomy with Retrusion. Chest. 1985 Nov;88(5):776–8. doi:10.1378/chest.88.5.776

35. Yang HJ, Jung YE, Kwon IJ, Lee JY, Hwang SJ. Airway changes and prevalence of obstructive sleep apnoea after bimaxillary orthognathic surgery with large mandibular setback. Int J Oral Maxillofac Surg. 2020 Mar;49(3):342–9. doi:10.1016/j.ijom.2019.07.012

36. Lee UL, Oh H, Min SK, Shin JH, Kang YS, Lee WW, et al. The structural changes of upper airway and newly developed sleep breathing disorders after surgical treatment in class III malocclusion subjects. Medicine (Baltimore). 2017 Jun;96(22):e6873. doi:10.1097/MD.0000000000006873

37. Uesugi T, Kobayashi T, Hasebe D, Tanaka R, Ike M, Saito C. Effects of orthognathic surgery on pharyngeal airway and respiratory function during sleep in patients with mandibular prognathism. Int J Oral Maxillofac Surg. 2014 Sep;43(9):1082–90. doi:10.1016/j.ijom.2014.06.010

38. Yamada T, Mishima K, Matsumura T, Moritani N, Sugahara T. Respiratory Status during Sleep following Orthognathic Mandibular Setback Surgery. Asian J Oral Maxillofac Surg. 2008 Mar;20(1):12–5. doi:10.1016/S0915-6992(08)80002-7