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# Impact of Mandibular Advancement Devices on Temporomandibular Disorders and Quality of Life in Obstructive Sleep Apnea Syndrome Patients: A Retrospective Study

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## Highlights

- Mandibular advancement devices improved sleep-related outcomes and quality of life in patients with obstructive sleep apnea syndrome.
- No worsening of temporomandibular disorder symptoms was observed after six months of MAD therapy.

## Abstract

Background: Obstructive sleep apnea syndrome (OSAS) is a prevalent sleep-related breathing disorder associated with significant systemic complications and reduced quality of life. Mandibular advancement devices (MADs) represent an established alternative therapy for patients who cannot tolerate continuous positive airway pressure (CPAP). However, concerns remain regarding their potential effects on temporomandibular disorders (TMD). Materials and Methods: This retrospective exploratory study analyzed clinical records of 26 patients (mean age 55.4 ± 5.8 years) with polysomnography-confirmed OSAS and baseline TMD-related symptoms treated with a custom-made monobloc MAD. Clinical parameters were evaluated at baseline (T0) and after approximately 6 months of therapy (T1). Outcomes included apnea–hypopnea index (AHI), Epworth Sleepiness Scale (ESS), Fonseca Anamnestic Index, and health-related quality of life assessed using the SF-36 questionnaire. Repeated measures ANOVA and linear regression analyses were performed. Results: After six months of MAD therapy, a significant reduction in AHI was observed (30 ± 13.76 vs. 10.87 ± 3.9; $p < 0.00001$). Daytime sleepiness significantly decreased (ESS: 9.31 ± 3.53 vs. 3.38 ± 1.77; $p < 0.00001$). TMD symptom severity also decreased significantly according to the Fonseca Index (33.85 ± 17.74 vs. 10.00 ± 8.94; $p < 0.00001$). Quality of life scores improved significantly (SF-36: 41.15 ± 9.52 vs. 65.38 ± 5.82; $p < 0.00001$). Linear regression analysis showed no significant association between changes in AHI and changes in TMD symptoms, ESS scores, or quality of life. Conclusions: Within the limitations of this retrospective study, MAD therapy was not associated with symptom aggravation of temporomandibular disorders in patients with pre-existing TMD symptoms. Significant improvements in respiratory parameters, daytime sleepiness, and quality of life were observed after six months of therapy.



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## 1. Introduction

According to the definition provided by the Ministry of Health, Obstructive Sleep Apnea Syndrome (OSAS) is a sleep-related breathing disorder characterized by repeated episodes of complete or partial obstruction of the upper airways during sleep. These episodes are associated with clinical signs and symptoms that can lead to significant systemic dysfunctions, resulting in a reduced quality of life and considerable economic burden [1].

A definitive diagnosis of OSAS is established through polysomnography when the apnea–hypopnea index (AHI), defined as the number of apneas and hypopneas per hour of sleep, exceeds 5 events per hour [2]. OSAS affects individuals of all ages, with a prevalence ranging between 1.2% and 5.7% in the pediatric population. In adults, the condition affects 9% of women and 24% of men [2,3].

Obesity, age, and sex are recognized as significant risk factors for OSAS; male sex, in particular, is an independent risk factor, with a male-to-female prevalence ratio of approximately 1.5:1 [4]. Additionally, the risk of OSAS is closely associated with body mass index (BMI), with the incidence of the disorder increasing proportionally as BMI rises. This is primarily due to the narrowing of the upper airways caused by excess adipose tissue [4]. OSAS is classified based on the number of apnea–hypopnea episodes per hour of sleep using the Apnea–Hypopnea Index (AHI). Mild OSAS is defined by an AHI between 5 and 15, moderate OSAS by an AHI between 16 and 30, and severe OSAS by an AHI greater than 30.

During apneic events, blood oxygen levels drop rapidly, leading to hypoxia, cortical arousals, and changes in intrathoracic pressure caused by respiratory effort. These events result in increased blood pressure and cardiac activity.

Such occurrences represent a significant risk factor for psychiatric, cardiovascular, cerebrovascular, and metabolic disorders, thereby increasing overall morbidity [5].

Recent evidence has further highlighted the systemic consequences of OSAS and the potential benefits of its treatment on cardiovascular outcomes. In particular, Dieltjens et al. demonstrated that treatment with mandibular advancement devices may contribute to reverse left ventricular hypertrophic remodeling in patients with OSAS, suggesting that oral appliance therapy may exert beneficial effects beyond respiratory improvement, including potential cardioprotective benefits [6]. Moreover, OSAS has been associated with several health complications, including an increased risk of motor vehicle accidents, workplace incidents, and reduced work performance, all of which further contribute to the socioeconomic burden of the disease [7–9].

In Italy, the social cost of this condition is exceedingly high, amounting to €2.9 billion, encompassing both direct and indirect healthcare costs, including traffic and workplace accidents as well as productivity losses (data from the Ministry of Health in the document approved at the State-Regions Conference on 12 May 2016, taking into account the national and international guidelines and recommendations currently available). The substantial social impact of OSAS elevates it to a public health emergency. Early diagnosis is therefore essential to mitigate the costs associated with morbidity. Direct healthcare costs related to diagnosis and treatment (consultations, diagnostic tests, therapies) are estimated to represent only 6% of the total costs. In contrast, healthcare costs arising from the failure

to identify and prevent comorbidities account for 49% of the total. Non-healthcare costs, which make up the remaining 45%, are distributed as follows: motor vehicle accidents (24%), workplace accidents (12%), and productivity losses (9%). OSAS significantly and negatively affects quality of life [9].

Continuous Positive Airway Pressure (CPAP) is considered the most effective and widely used treatment for OSA, but long-term adherence remains limited, with rates hovering around 50% [10–12].

In addition to CPAP and oral appliance therapy, surgical approaches such as maxillomandibular advancement (MMA) have also been proposed for selected patients with moderate to severe OSAS. A recent systematic review and meta-analysis by Trindade et al. confirmed that MMA surgery can significantly reduce the apnea–hypopnea index and improve airway patency, supporting its role as an effective treatment option when conservative therapies fail or are not tolerated [13]. For patients who cannot tolerate CPAP, those with primary snoring, mild or moderate OSAS, or those who prefer alternative solutions, mandibular advancement devices (MADs) represent a valid option [14].

Recent systematic reviews and meta-analyses have further confirmed the effectiveness of MAD therapy in the management of OSAS. For example, Yong et al. reported significant reductions in the apnea–hypopnea index and improvements in sleep-related outcomes among patients treated with mandibular advancement devices, reinforcing their role as a reliable alternative for individuals who are unable to tolerate CPAP therapy [15].

These devices work by mechanically advancing the mandible, increasing the oropharyngeal space, and preventing the tongue from collapsing against the pharyngeal wall, thereby significantly reducing snoring and obstructive respiratory events. The Orofacial Pain Prospective Evaluation and Risk Assessment (OPPERA) prospective cohort study reported that individuals presenting symptoms suggestive of obstructive sleep apnea had a significantly increased risk of developing first-onset temporomandibular disorders (TMD) [16]. However, recent studies have shown that MADs do not exacerbate TMD and can be safely used even in patients with pre-existing masticatory disorders [17].

Therefore, the aim of this study was to evaluate the effects of mandibular advancement device (MAD) therapy on temporomandibular disorder (TMD) symptoms and quality of life in patients affected by obstructive sleep apnea syndrome (OSAS).

The null hypothesis of the study was that treatment with mandibular advancement devices does not significantly influence TMD symptoms or the quality of life of patients with OSAS.

The specific objectives of this study were:

- To evaluate the progression of clinical signs and symptoms of temporomandibular disorders (TMD) in patients with OSAS treated with mandibular advancement devices.
- To assess changes in patients' quality of life by comparing data collected before and after MAD therapy.

## 2. Materials and Methods

### *2.1. Study Design and Ethical Approval*

This retrospective exploratory study analyzed clinical records of patients treated at the Orthodontics and Gnathology Outpatient Clinic of the Unit of Dentistry and Odontostomatology, "G. Martino" University Hospital, Messina, Italy.

The study protocol was approved by the local Bioethics Committee of Messina. All patients had previously provided written informed consent for treatment and for the use of anonymized clinical data for research purposes.

### *2.2. Eligibility Criteria*

#### 2.2.1. Inclusion Criteria

Patients were eligible for inclusion if they met all of the following criteria:

1. Age between 40 and 65 years at baseline (T0).
2. Diagnosis of Obstructive Sleep Apnea Syndrome (OSAS) confirmed by baseline polysomnography.
3. Presence of temporomandibular disorder-related symptoms at baseline, assessed using the Fonseca Anamnestic Index.
4. Baseline body mass index (BMI) $\leq 35$ kg/m$^2$.
5. Absence of major craniofacial malformations or skeletal anomalies contraindicating mandibular advancement device (MAD) therapy.
6. Availability of complete paired data at baseline (T0) and at approximately 6 months of follow-up (T1), including:
   - Apnea–Hypopnea Index (AHI);
   - Epworth Sleepiness Scale (ESS);
   - SF36 questionnaire;
   - Fonseca Anamnestic Index.

#### 2.2.2. Exclusion Criteria

Patients were excluded if they presented with:

1. Central sleep apnea.
2. Severe neurological disorders potentially affecting sleep or pain perception.
3. Severe or uncontrolled psychiatric disorders that could compromise the reliability of self-reported questionnaires.
4. Severe dental conditions preventing the use of a mandibular advancement device.

### *2.3. Data Collection and Patient Assessment*

Clinical and demographic data, including age and sex, were retrospectively extracted from medical records. As part of routine clinical practice, all patients completed standardized questionnaires evaluating daytime sleepiness (Epworth Sleepiness Scale), temporomandibular disorder-related symptom severity (Fonseca Anamnestic Index), and health-related quality of life (SF36).

Baseline polysomnography was used to confirm the OSAS diagnosis and determine disease severity. Follow-up sleep study data (T1) were obtained from the same sleep laboratory and were performed with the mandibular advancement device in situ.

Baseline assessment (T0) corresponded to the initiation of MAD therapy, while follow-up assessment (T1) was conducted approximately 6 months after treatment initiation. All included patients completed the follow-up period, and no drop-outs were recorded.

#### Study Population

A total of 26 patients (12 males and 14 females) were included in the study. The mean age was 55.4 $\pm$ 5.8 years, ranging from 40 to 65 years. The mean follow-up period was approximately 6 months.

### *2.4. Mandibular Advancement Device Fabrication and Delivery*

All patients were treated with a custom-made monobloc mandibular advancement device fabricated following a standardized digital workflow.

Dental impressions of both arches were obtained using an intraoral scanner (TRIOS® 4, 3Shape, Copenhagen, Denmark). Mandibular protrusion was registered using a George Gauge (Great Lakes Dental Technologies, Tonawanda, NY, USA) and set at approximately

60–70% of the patient's maximum comfortable protrusion, as documented in the clinical records.

Digital models were generated from STL files and 3D-printed using a stereolithographic printer (Form 3B, Formlabs, Somerville, MA, USA) with biocompatible resin. Upper and lower thermoplastic splints were fabricated from medical-grade polyurethane sheets (Erkodur®, 1.5 mm thickness) using a pressure molding system (Erkoform-3D Motion, Erkodent, Pfalzgrafenweiler, Germany). Following a brief acclimatization period, the splints were definitively fixed in the predetermined protrusive position using a biocompatible light-curing resin, resulting in a non-titratable monobloc device.

The MAD was prescribed for nightly use throughout the entire sleep period. According to clinical records, all patients reported regular nocturnal use of the device during the observation period.

All fabrication and delivery procedures complied with applicable ISO 13485 standards for intraoral medical devices.

### *2.5. Follow-Up and Tolerability*

Patients were monitored through routine clinical follow-up visits, and information regarding comfort, dental issues, and temporomandibular symptoms was recorded in the medical charts.

Transient adverse effects were reported during the initial phase of MAD use. These included mild hypersalivation and transient muscular discomfort, typically resolving within the first 3–4 days of treatment. No adverse events required treatment discontinuation or device modification, and no clinically significant temporomandibular complications were documented.

### *2.6. Study Outcomes*

The primary outcomes of the study were changes from baseline (T0) to follow-up (T1) in:

- Apnea–Hypopnea Index (AHI);
- TMD-related symptom severity assessed by the Fonseca Anamnestic Index;
- Health-related quality of life assessed by the SF36 questionnaire.

Secondary outcomes included changes in daytime sleepiness assessed by the Epworth Sleepiness Scale.

#### 2.6.1. Statistical Analysis

To ensure adequate statistical power, a power analysis was conducted using the reference value of temporomandibular disorders (TMD) obtained through Fonseca's Anamnestic Index. The mean TMD score was 33.80 at T0 and 11.66 at T1, with a standard deviation of 17.95. With the significance level ($\alpha$) set at 0.01 for greater precision, the minimum required sample size to detect a significant difference was 23 subjects. To enhance the robustness of the results, a total of 26 patients were included in the study.

This slight increase ensures greater reliability of the analyses and improves the generalizability of the findings.

#### 2.6.2. Descriptive Statistics

A descriptive statistical analysis was performed on the initial sample at T0 and after six months of treatment (T1). The analysis included the following variables:

- Age;
- Sex;
- Body Mass Index (BMI);

- Presence of temporomandibular disorders (TMD) (evaluated with Fonseca's Anamnestic Index);
- Apnea-hypoapnea index (AHI);
- Mallampati classification;
- Quality of life (measured using the SF-36 questionnaire);
- Daytime sleepiness (assessed using the Epworth Sleepiness Scale—ESS).

To assess the normality of variable distribution, the Shapiro–Wilk test was conducted. The results confirmed a normal distribution, allowing for the use of parametric statistical tests. (Table 1).

**Table 1.** Baseline and Post-Treatment Clinical Data.

| Variable | Value T0 | Value T1 |
|---|---|---|
| Age (years, mean ± SD) | 55.4 ± 5.8 | 55.4 ± 5.8 years |
| Sex, (%) | Male: 12 (46.2%)<br>Female: 14 (53.8%) | Male: 12 (46.2%)<br>Female: 14 (53.8%) |
| BMI (kg/m$^2$, mean ± SD) | 28.6 ± 4.3 kg/m$^2$ | 29.76 ± 3.5 kg/m$^2$ |
| TMD (Fonseca test, *n* (%)) | Positive: 26 (100%) | Positive:15 (60%) |
| OSAS (AHI, mean ± SD) | 30 ± 13.76 | 10.87 ± 3.9 |
| Mallampati Classification, *n* (%) | I:0 II:0 III:12 (46.2%)<br>IV:14 (53.8%) | I:0 II:0 III:12 (46.2%)<br>IV:14 (53.8%) |
| Quality of Life (SF-36, mean ± SD) | 41.15± 9.5 | 65.38± 5.8 |
| ESS (Epworth Sleepiness Scale, mean ± SD) | 9.30 ± 3.5 | 3.38± 1.76 |

2.6.3. Comparative Analysis Between Time Points (T0, T1)

Repeated measures ANOVA revealed a statistically significant effect of time (T0 vs. T1) for all outcome variables.

AHI showed a marked reduction after 6 months of treatment ($F(1,25) = 220.60$, $p < 0.00001$), indicating a significant improvement in sleep apnea severity.

Daytime sleepiness, assessed by the Epworth Sleepiness Scale (ESS), significantly decreased from baseline to follow-up ($F(1,25) = 56.75$, $p < 0.00001$).

TMD symptoms, evaluated using the Fonseca Index, were significantly reduced at T1 compared with T0 ($F(1,25) = 43.41$, $p < 0.00001$).

Quality of life, measured by the SF-36 questionnaire, significantly improved after treatment ($F(1,25) = 601.36$, $p < 0.00001$).

Detailed descriptive statistics and ANOVA results are reported in Table 2.

**Table 2.** ANOVA Analysis of Clinical Variables Pre- and Post-Therapy.

| Outcome | T0 (Mean ± SD) | T1 (Mean ± SD) | F (1,25) | *p*-Value |
|---|---|---|---|---|
| AHI | 30± 13.76 | 10.87 ± 3.90 | 220.60 | <0.00001 |
| ESS | 9.31 ± 3.53 | 3.38 ± 1.77 | 56.75 | <0.00001 |
| Fonseca Index | 33.85 ± 17.74 | 10.00 ± 8.94 | 43.41 | <0.00001 |
| SF-36 (QoL) | 41.15 ± 9.52 | 65.38 ± 5.82 | 601.36 | <0.00001 |

Overall, these results indicate that MAD therapy produced clinically meaningful improvements across all evaluated domains. In particular, the marked reduction in AHI reflects a substantial improvement in sleep apnea severity, while the decrease in ESS scores indicates reduced daytime sleepiness. At the same time, the significant reduction in Fonseca Index scores suggests an improvement in TMD symptoms, and the increase in SF-36 scores reflects a relevant enhancement in patients' perceived quality of life.

2.6.4. Regression Analysis

Model assumptions were evaluated using the Shapiro–Wilk test. Linear regression results are reported as F statistics, coefficients of determination ($R^2$), correlation coefficients, and *p*-values. Statistical significance was set at $p < 0.05$ (Table 3).

**Table 3.** Linear regression analyses showed no significant association between changes in AHI and changes in Fonseca Index, ESS, or SF-36 scores.

| Dependent Variable (Y) | Independent Variable (X) | $R^2$ | Correlation (r) | F (1,24) | *p*-Value |
|---|---|---|---|---|---|
| ΔFonseca Index | ΔAHI | ~0.000 | 0.011 | 0.003 | 0.958 |
| ΔESS | ΔAHI | ~0.000 | −0.013 | 0.004 | 0.950 |
| ΔSF-36 | ΔAHI | 0.003 | 0.055 | 0.072 | 0.791 |

Linear regression analysis was performed to explore whether the magnitude of improvement in obstructive sleep apnea severity (ΔAHI) was associated with changes in TMD symptoms (ΔFonseca Index), daytime sleepiness (ΔESS), or quality of life (ΔSF-36). The analysis showed no significant relationships between the reduction in AHI and the changes observed in the other clinical outcomes. In particular, the coefficient of determination ($R^2$) was close to zero for all models, indicating that changes in AHI explained virtually none of the variability in TMD symptoms, ESS scores, or quality-of-life measures. Similarly, correlation coefficients were extremely low, and *p*-values were not statistically significant ($p > 0.05$), suggesting that improvements in TMD symptoms and patient-reported outcomes occurred independently of the magnitude of respiratory improvement. The present data suggest that the beneficial effects of MAD therapy on TMD symptoms, daytime sleepiness, and quality of life may not be solely attributable to the reduction in apnea severity, but may involve additional mechanisms such as improved mandibular stabilization, neuromuscular adaptation, or patient-related factors.

## 3. Discussion

This retrospective study evaluated the progression of temporomandibular disorder (TMD) symptoms and quality of life in patients with obstructive sleep apnea syndrome (OSAS) treated with mandibular advancement devices (MADs). The results indicate that MAD therapy did not exacerbate TMD-related symptoms. On the contrary, patients showed a significant reduction in TMD symptom severity, together with clinically relevant improvements in sleep-related outcomes and perceived quality of life following treatment. The management of OSAS requires a multidisciplinary approach in which dental professionals, particularly orthodontists, play an important role. Dentists may contribute to the early identification of OSAS and may act as sentinel clinicians, especially in patients presenting with orofacial symptoms. Evidence from the OPPERA study [16], reported a coexistence between TMD and OSAS, suggesting that dentists may detect early signs of sleep-disordered breathing before systemic symptoms become evident. However, the mechanisms underlying this association remain unclear, and it is still uncertain whether one condition precedes the other or whether a bidirectional relationship exists.

Among the therapeutic options endorsed by the Ministry of Health, continuous positive airway pressure (CPAP) remains the gold standard for the treatment of OSAS, particularly for reducing excessive daytime sleepiness [18,19]. However, limited patient adherence to CPAP has encouraged the use of alternative therapies. Mandibular advancement devices have emerged as a valid therapeutic option for patients who are unable to tolerate CPAP, as they advance the mandible and tongue, thereby maintaining upper airway patency during

sleep (11). Although CPAP remains more effective in reducing respiratory events, MAD therapy is often associated with greater comfort and adherence, supporting its role within individualized treatment strategies [20].

In the present study, MAD therapy was associated with significant improvements in OSAS-related parameters, daytime sleepiness, and health-related quality of life, without exacerbating pre-existing TMD symptoms. These findings are consistent with previous studies indicating that MAD treatment does not worsen TMD symptoms and can be safely prescribed in patients with concomitant OSAS and TMD [18,21].

The potential risk that MAD therapy may aggravate TMD symptoms or negatively influence occlusion has been widely debated. Some authors have considered TMD an absolute contraindication to MAD therapy, arguing that forced mandibular protrusion—particularly when exceeding 50% of maximum protrusive capacity—may overload the masticatory muscles and temporomandibular joint (TMJ), potentially leading to discomfort or dysfunction [22,23].

However, the literature remains partially controversial. Some studies have suggested that prolonged mandibular advancement during sleep may induce biomechanical stress on the TMJ and masticatory muscles, particularly during the initial adaptation phase of therapy. This may result in transient muscular discomfort, joint sounds, or fatigue of the masticatory muscles in susceptible individuals. Nevertheless, most reports indicate that these symptoms are typically mild and tend to resolve spontaneously with continued appliance use and appropriate clinical monitoring [24].

In addition to mechanical factors related to mandibular advancement, systemic conditions may also contribute to temporomandibular joint involvement. For example, inflammatory diseases such as rheumatoid arthritis have been associated with structural and functional alterations of the TMJ. Recent research has highlighted the relevance of serological markers in the assessment of temporomandibular disorders in patients with rheumatoid arthritis, emphasizing the multifactorial etiology of TMJ pathology and the potential interaction between systemic inflammatory processes and temporomandibular dysfunction [25].

In addition to potential muscular or articular discomfort, long-term MAD therapy has been associated with dental and occlusal changes. Several longitudinal studies have reported progressive reductions in overjet and overbite, proclination of mandibular incisors, retroclination of maxillary incisors, and modifications in occlusal contact patterns [26].

These changes are believed to result from the continuous mechanical forces exerted by the appliance on the dentition during nightly mandibular advancement. Although such occlusal modifications may become detectable during long-term therapy, they are generally considered clinically manageable when patients undergo regular dental follow-up [23].

Nevertheless, several investigations have challenged the assumption that MAD therapy increases the risk of TMD. Martínez-Gomis et al. reported no increase in TMD prevalence after five years of MAD therapy despite mandibular advancement of approximately 70%, and even observed an improvement in joint sounds over time [27]. Similarly, Pérez et al. found no persistent new-onset TMD symptoms in MAD-treated patients, and pre-existing symptoms did not worsen [28].

Imaging studies have further supported the safety of MAD therapy for the temporomandibular joint. Magnetic resonance imaging investigations have shown that MAD use does not adversely affect TMJ structures, even in patients with anterior disc displacement, with or without reduction. No significant association has been found between mandibular advancement and the onset or worsening of arthrogenic or myogenic pain in patients with pre-existing TMD.

Interestingly, although significant improvements were observed in TMD symptoms, daytime sleepiness, and quality of life, linear regression analyses did not demonstrate a significant association between the magnitude of apnea–hypopnea index (AHI) reduction and the observed clinical improvements. This finding suggests that the beneficial effects of MAD therapy on TMD symptoms and patient-reported outcomes may not be solely dependent on the reduction in respiratory events but may reflect a more complex and multidimensional therapeutic effect [17,27,29].

These findings indicate that the improvement in TMD symptoms observed in the present study cannot be explained solely by the reduction in respiratory events. In fact, the very low coefficient of determination ($R^2$) observed in the regression models suggests that changes in AHI accounted for only a negligible proportion of the variability in TMD symptoms, daytime sleepiness, and quality-of-life scores. This result suggests that additional mechanisms—such as mandibular stabilization during sleep, neuromuscular adaptation of the masticatory system, or improvements in sleep continuity—may contribute to the clinical benefits observed in patients treated with mandibular advancement devices.

The findings of the present study are consistent with previous evidence indicating that MAD treatment for OSAS does not worsen TMD-related symptoms, further suggesting its effectiveness in managing OSAS without evidence of TMJ worsening in this sample or masticatory function [30].

An important strength of the present study is the inclusion of a homogeneous cohort of patients presenting with clinically diagnosed TMD symptoms at baseline. Unlike previous investigations including mixed populations with and without TMD, this design reduces baseline heterogeneity and allows a more precise evaluation of MAD safety in patients with pre-existing temporomandibular disorders.

Nevertheless, this study has several limitations. Its retrospective design limits causal inference and may introduce selection and reporting bias. Furthermore, the use of self-reported questionnaires to assess TMD symptoms and quality of life may not fully capture objective functional changes. Future prospective studies with larger sample sizes and objective clinical assessments are therefore needed to further clarify the long-term effects of MAD therapy on TMD symptoms and patient outcomes.

*Limitations*

This study has several limitations that should be acknowledged. The retrospective design and the absence of a control group limit the ability to establish causal relationships between mandibular advancement device (MAD) therapy and the clinical improvements observed. Consequently, the findings should be interpreted with caution and considered exploratory. In addition, the relatively small sample size may limit the generalizability of the results. Although the a priori power analysis indicated that the study was adequately powered for the primary outcome, the final cohort of 26 patients remains limited, particularly for regression-based analyses. Therefore, the results of the regression models should be interpreted cautiously, and larger prospective studies with greater statistical power are required to further investigate the determinants of treatment response. Another limitation concerns the evaluation of temporomandibular disorder (TMD) symptoms and quality of life, which relied on self-reported questionnaires and may therefore be influenced by subjective perception and response bias. Objective clinical or imaging-based assessments of the temporomandibular joint (such as MRI or CBCT) were not systematically available due to the retrospective nature of the study. Finally, several potential confounding factors—including variations in body mass index, degree of mandibular advancement, patient adherence to therapy, bruxism, and medication use—were not controlled in the statistical analysis. Future prospective investigations, including larger samples,

control groups, and objective clinical assessments, are needed to confirm and expand the present findings.

## 4. Conclusions

- Mandibular advancement device therapy significantly reduced apnea–hypopnea index in patients with obstructive sleep apnea syndrome.
- Daytime sleepiness significantly decreased after six months of therapy.
- Health-related quality of life improved according to SF-36 scores.
- Temporomandibular disorder symptom severity decreased according to the Fonseca Anamnestic Index.
- No worsening of TMD symptoms was observed; however, these findings should be interpreted with caution due to the absence of a control group and objective TMJ assessment.

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## Abbreviations

The following abbreviations are used in this manuscript:

| | |
|---|---|
| BMI | Body Mass Index |
| TMD | Temporomandibular Disorders |
| OSAS | Obstructive Sleep Apnea Syndrome |
| AHI | Apnea–Hypopnea Index |
| ESS | Epworth Sleepiness Scale |
| Δ AHI | Difference Apnea–Hypopnea Index |
| Δ Quality of Life | Difference Quality of Life |
| Δ TMD | Difference Temporomandibular Disorders |

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