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Literature Review

Hidden Clues: Radiographic Incidental Findings That Could Compromise MAD Therapy Success or Tolerance for OSA - A Targeted Literature Review

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Abstract

Background: Mandibular advancement devices (MADs) are widely used for the treatment for patients with mild to moderate obstructive sleep apnea (OSA), particularly for those who cannot tolerate continuous positive airway pressure (CPAP) therapy, the current gold standard. These appliances function by advancing the mandible during sleep to maintain upper airway patency and reduce respiratory events. Although MAD therapy can be effective, its success is highly dependent on anatomical, musculoskeletal, and systemic conditions that affect airway dynamics and mandibular function.

Aim: This review aims to identify and discuss clinical and radiographic findings that may complicate the use of MADs. Conditions such as nasal obstruction, temporomandibular joint (TMJ) disorders, craniofacial anomalies, cervical spine changes, and certain systemic diseases may affect appliance tolerance, retention, and overall therapeutic effectiveness. Radiographic findings, including TMJ osteoarthritis, subchondral cysts, and structural nasal abnormalities, may serve as important diagnostic indicators when evaluating patients for oral appliance therapy.

Discussion: A comprehensive assessment that includes medical history, clinical examination, and appropriate imaging is essential prior to initiating MAD therapy. In many cases, interdisciplinary collaboration with ENT specialists, rheumatologists, sleep physicians, or orofacial pain specialists may be necessary to optimize treatment outcomes. Recognizing these anatomical and radiographic challenges allows clinicians to improve patient selection, tailor appliance design when necessary, and reduce the risk of complications.

Conclusion: Overall, a personalized, evidence-based approach is essential for maximizing the effectiveness and long-term success of MAD therapy in patients with obstructive sleep apnea.

Keywords: *Mandibular advancement device (MAD); Obstructive sleep apnea (OSA); Temporomandibular joint (TMJ); Radiographic findings; Craniofacial anatomy; Nasal airway obstruction; Oral appliance therapy; Temporomandibular joint disorders (TMD); CBCT imaging; Anatomical contraindications.*

Introduction

A mandibular advancement device (MAD) has become an increasingly popular and effective non-invasive option for managing mild to moderate obstructive sleep apnea (OSA), particularly in patients who are intolerant of continuous positive airway pressure (CPAP) or not candidates for surgical correction of OSA. By repositioning the mandible in a forward, vertically open position during sleep, MADs aims to maintain upper airway patency and reduce apneic events, snoring, sleep fragmentation, respiratory pauses, and daytime somnolence. However, the success of MAD therapy is not universal and may be significantly influenced by a variety of anatomical, musculoskeletal, and systemic factors.

Patients with craniofacial anomalies, temporomandibular joint (TMJ) disorders, sinonasal pathology, or systemic musculoskeletal diseases often present with structural or functional challenges that can affect not only the fit and comfort of MAD, but also their safety, efficacy, and long-term tolerability.

This section aims to highlight several clinical and radiographic findings that may complicate or contraindicate the use of MAD. Understanding the implications of these conditions is critical for providers involved in OSA management using oral appliance therapy. A thorough medical history, a detailed clinical examination, and appropriate imaging are essential to identify such conditions early in the treatment planning phase. In certain cases, multidisciplinary collaboration with ENT specialists, neurologists, orofacial pain specialists, rheumatologists, and sleep physicians may be necessary to ensure that MAD therapy is both safe and effective.

Ultimately, the goal is to adopt a patient-centered, evidence-informed approach that accounts for individual anatomical and physiological nuances—maximizing therapeutic outcomes while minimizing risk.

Methods

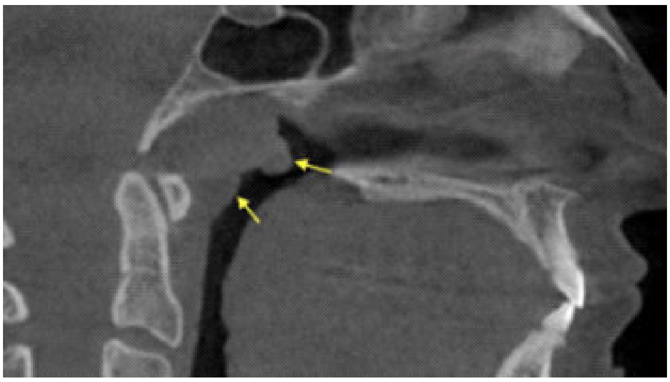
This study was conducted at a specialized orofacial pain and dental sleep medicine center. A retrospective review was performed of all radiographic imaging obtained over a one-month period for patients who presented for an initial consultation for evaluation of mandibular advancement device (MAD) therapy as a potential treatment for obstructive sleep apnea (OSA).

Radiographic assessment focused on non-odontogenic findings, including TMJ morphology and degenerative changes, craniofacial skeletal characteristics, airway-related observations, and other incidental findings of potential clinical significance.

Inclusion criteria consisted of patients undergoing initial evaluation for oral appliance therapy with available radiographic imaging cone-beam computed tomography (CBCT) as part of their diagnostic work-up. Patients with incomplete imaging records or those not evaluated for MAD therapy were excluded.

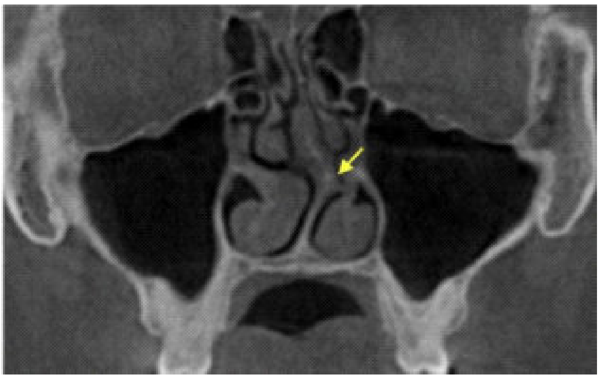
Based on the most common radiographic findings identified in the clinical review, a targeted literature search was conducted to assess their relevance to MAD therapy and OSA management.

1. Adenoid Hypertrophy



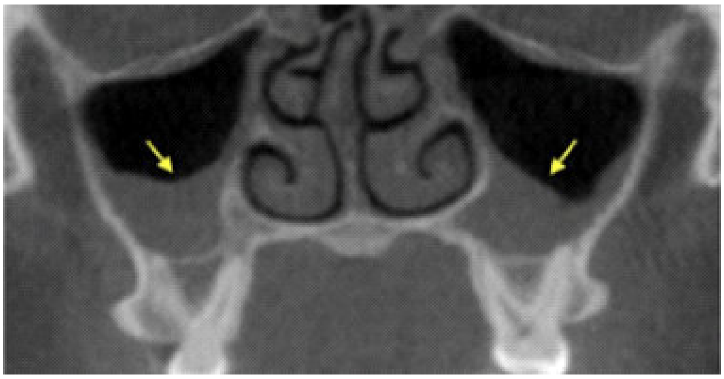
- **Etiology:** The adenoids are a collection of lymphoid tissue along the posterior wall and roof of the nasopharynx. Hypertrophy of these tissues may result from retention of secretions, recurrent inflammation, infections, allergies, or other types of antigen exposure, which can lead to the obstruction of the nasopharynx. [1,3]
- **Image:** Cone-beam computed tomography (CBCT) was obtained in this case; however, cephalometric radiography remains the gold standard for diagnosing adenoid hypertrophy. Additional imaging that may aid in the diagnosis includes magnetic resonance imaging (MRI). [1,3]
- **Effect:** Enlargement of the adenoids can cause nasopharyngeal obstruction, leading to persistent nasal breathing difficulties, and may be clinical manifestations such as malocclusion, a high palate, snoring, and sleep apnea. [3]
- **Impact on MAD Therapy:** MAD depends on nasal breathing for effectiveness and tolerance. Patients with adenoid hypertrophy may exhibit mouth breathing, which may be associated with mechanical obstruction and/or chronic inflammatory processes in the nasopharynx, leading to reduced MAD compliance and efficacy. [3]
- **Management:** Prior to initiating MAD therapy, referral to an ENT specialist for evaluation and possible adenoidectomy may be indicated in patients with sleep interruption due to nasal airway obstruction persisting for at least three months, such as obstructive sleep apnea syndrome (OSAS). [3,5]

2. Deviated Nasal Septum



- **Etiology:** Deviated nasal septum (DNS) has a multifactorial etiology involving intrinsic and extrinsic factors. Intrinsic causes include congenital or developmental abnormalities of the septal cartilage, whereas extrinsic causes result from trauma, scar contracture, or asymmetric osseocartilaginous attachments that displace the septum from the midline. [6]
- **Image:** CBCT was used in this case; however, computed tomography (CT) and magnetic resonance imaging can be considered for the diagnosis of DNS. [6]
- **Effect:** Deviation of the nasal septum from the midline can result in obstruction of nasal airflow, reduced nasal patency, sinusitis, and impaired breathing. [6]
- **Impact on MAD Therapy:** Similar to adenoid hypertrophy, DNS may promote oral breathing, increasing airway collapsibility and decreasing device comfort and retention; as a result, successful treatment with MAD is significantly less likely in patients with nasal abnormalities. [7]
- **Management:** Referral to an ENT specialist for evaluation. Septoplasty, septorhinoplasty, nasal decongestant, antihistamines, or corticosteroid therapy may be considered prior to or alongside MAD usage. [6]

3. Sinusitis (Chronic or Recurrent)



- **Etiology:** This condition is multifactorial and includes genetic predisposition, immunodeficiency factors. Anatomic variations such as concha bullosa, nasal septal deviation, and a displaced uncinate process, which have been suggested as potential risk factors for developing chronic sinusitis (CS). In addition, environmental exposures, including pollutants, smoke, allergens, viruses, fungi, and bacteria, may contribute to the onset and progression of the disease. [11]

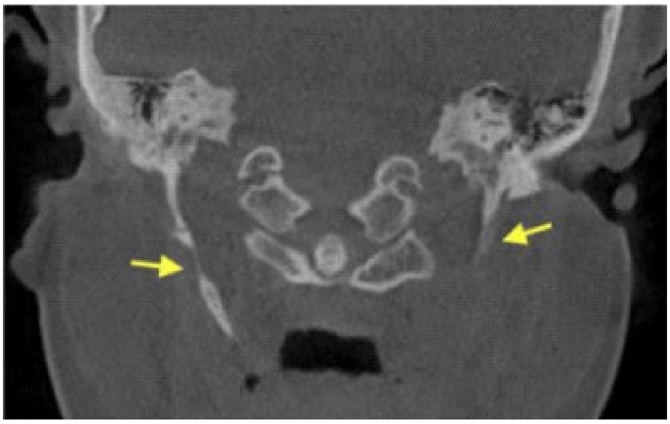
- **Image:** Cone-beam computed tomography (CBCT) was performed during the diagnostic process for this case; however, computed tomography (CT) remains the primary imaging modality for diagnosing chronic sinusitis as it provides detailed 3D images of sinus inflammation and obstruction. [10,11]

- **Effect:** Chronic sinusitis (CS) is a common inflammatory condition characterized by persistent symptomatic inflammation of the sinonasal cavities lasting for more than three months [10]. CS may cause facial pain, congestion, pressure, mouth breathing, and temporomandibular (TMJ) loading associated with pain and dysfunction. [10,12]

- **Impact on MAD Therapy:** Nasal obstruction and mouth breathing may reduce patient comfort, compliance, and efficacy of mandibular advancement devices (MADs). Sinus-related facial pain may mimic or exacerbate temporomandibular disorder (TMD) symptoms during MAD use. [12]

- **Management:** Management may include treatment of the underlying sinus inflammation or infection with saline irrigation, topical corticosteroid therapy, systemic corticosteroid, or oral doxycycline or an ENT referral before starting MAD therapy. [10]

4. Stylohyoid Ligament Ossification (Eagle's Syndrome)



- **Etiology:** Eagle's syndrome is a rare condition characterized by the elongation and calcification of the styloid process of the temporal bone and calcification of the ligaments associated stylohyoid ligaments. [14-16]

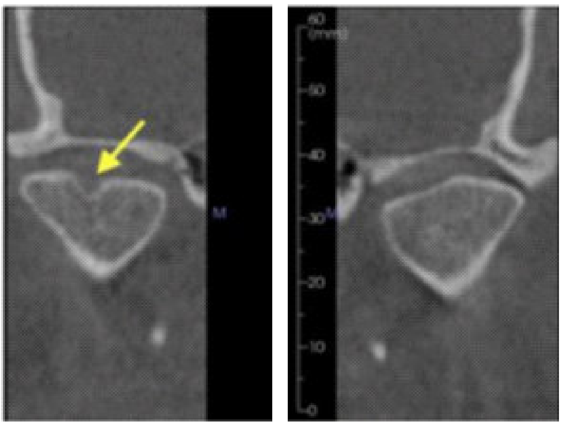
- **Image:** Cone-beam computed tomography (CBCT) aided in the identification of this finding; however, computed tomography (CT) is considered the imaging modality of choice. Additionally, 3D reconstruction, angiography, and panoramic radiography are valuable tools for diagnosing Eagle's syndrome. [13-16]

- **Effect:** Eagle's syndrome is associated with compression of adjacent neurovascular structures, which can cause pain with head and neck movement, swallowing, or jaw activity. [13]

- **Impact on MAD Therapy:** Mandibular advancement may aggravate pain, as forward displacement of the mandible can increase tension on the stylohyoid complex, potentially reducing tolerance to MADs. [13]

- **Management:** Management may include conservative pain control with analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs), as well as adjunctive therapies including anticonvulsants, antidepressants, local injections, and manipulation. [15] Surgical intervention, primarily styloidectomy (removal of the elongated styloid process) [15,16], may be considered prior to MAD fitting.

5. Bifid Mandibular Condyle



- **Etiology:** Bifid mandibular condyle (BMC) is considered an extremely rare disorder and may have multiple possible causes, including trauma, infection, vascular, developmental, teratogenic, or endocrine origins. [17-19]

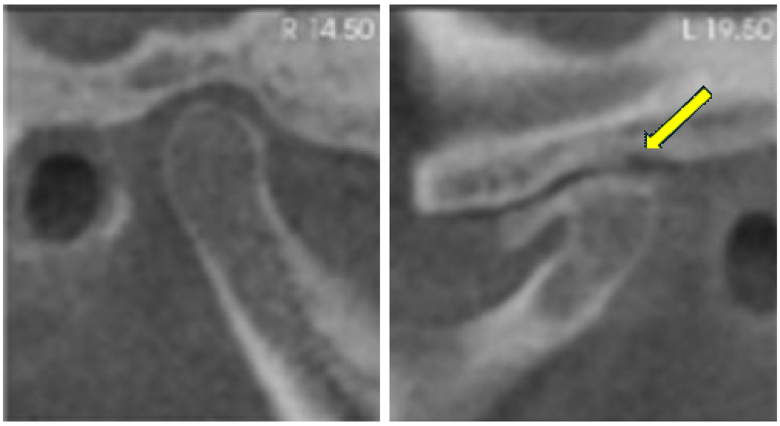
- **Image:** Cone beam computed tomography (CBCT) of the maxillofacial region was performed in this case and is considered the preferred modality for evaluating the bony anatomy and articulations of the temporomandibular joint (TMJ) [19]. Magnetic resonance imaging (MRI) is used to evaluate the soft tissues, cartilage, and disc within the joint space. BMC may also be incidentally identified on panoramic radiographs or other head and neck imaging studies. [17-19]

- **Effect:** BMC is an anatomical variation that can alter the temporomandibular joint mechanics and symmetry. In most cases, it presents with a mediolateral of the condylar heads. [17]

- **Impact on MAD Therapy:** BMC can be asymptomatic or develop symptoms such as pain, swelling, noise, hypomobility, joint locking, mandibular deflection, joint luxation, or ankylosis [17]. Approximately 60% of cases demonstrate concurrent osteoarthritic changes, which may further reduce tolerance to mandibular advancement therapy. Additionally, uneven mandibular advancement and altered occlusion, may increase the risk of TMJ symptoms during MAD use. [18]

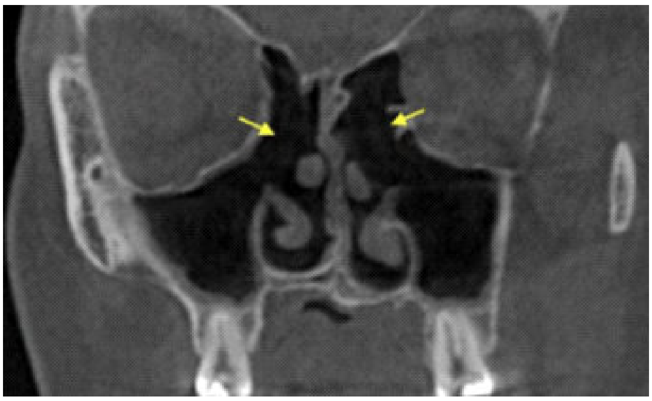
- **Management:** In symptomatic patients with pain, joint sounds, or hypomobility, occlusal splint therapy should be considered to stabilize the joints prior to initiating MAD therapy. Surgical consultation may be indicated in cases of progressive ankylosis or severe hypomobility (e.g., maximal opening of approximately 21mm opening), with referral for procedures such as arthroplasty or condylectomy when appropriate before considering MAD use. [19]

6. Osteoarthritic Changes of the Temporomandibular Joint (TMJ)



- **Etiology:** TMJ osteoarthritis is a degenerative disorder characterized by progressive deterioration of the articular cartilage and remodeling of the subchondral bone of the mandibular condyle. [20] Inflammatory mediators, mechanical stress, and abnormal bone remodeling contribute to compromised joint function. Contributing factors may include bruxism, malocclusion, trauma, disc displacement, internal derangement, and age-related cartilage degeneration. [20,21]
- **Image:** Cone-beam computed tomography (CBCT) remains the preferred modality for evaluating osseous TMJ changes, including condylar flattening, osteophyte formation, cortical erosion, and subchondral sclerosis. Magnetic resonance imaging (MRI) complements CBCT by assessing soft tissue components, particularly disc position and joint effusion. [23]
- **Effect:** Clinical manifestations range from mild discomfort to severe pain and include crepitus, stiffness, limited mouth opening, and mandibular deviation. In advanced stages, the condition may affect occlusion and masticatory efficiency and can exacerbate discomfort during sleep when mandibular advancement devices are used. [21,23]
- **Impact on MAD Therapy:** Mandibular advancement devices (MADs) reposition the mandible anteriorly, which may increase mechanical stress on the TMJ. Patients with pre-existing osteoarthritis may experience increased pain or fatigue; however, with careful device adjustment and monitoring, therapy can often be continued. [22]
- **Management:** Management typically begins with conservative measures, including nonsteroidal anti-inflammatory drugs (NSAIDs), occlusal splints, physiotherapy, and behavioral modifications aimed at reducing joint load. If these approaches are unsuccessful, minimally invasive procedures such as arthrocentesis or intra-articular injections may be considered to control inflammation and preserve joint function. [24]

7. Nasal Lateral Wall Deficiency (Post-Surgical or Congenital)



- **Etiology:** Nasal lateral wall deficiency involves structural weakness of the lateral nasal wall, leading to airway collapse and obstruction. Congenital cases may result from underdeveloped cartilage or abnormal nasal anatomy. [25] Acquired causes include post-surgical changes from rhinoplasty, septoplasty, or turbinate reduction, as well as trauma, aging, or connective tissue disorders. [25,26]

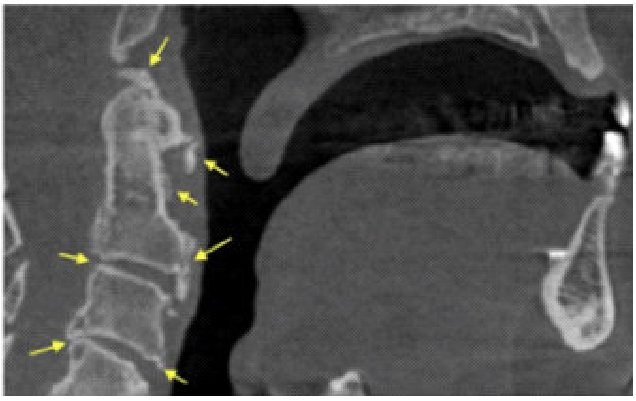
- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate the osseous anatomy of the nasal lateral wall. While CBCT remains a cornerstone for mapping these osseous deficiencies, magnetic resonance imaging (MRI) provides an essential window into the cartilage, soft tissues, and dynamic changes of the nasal valve. [27,28]

- **Effect:** Patients commonly present with nasal obstruction, mouth breathing, snoring, increased inspiratory effort, fatigue, and sleep disruption. Chronic nasal obstruction may exacerbate OSA symptoms or reduce response to MAD therapy. [25]

- **Impact on MAD Therapy:** Effective MAD therapy depends on adequate nasal airflow. Lateral wall collapse may limit tolerance to MADs and reduce therapeutic efficacy. Pre-treatment evaluation of nasal airway function is therefore essential. [25]

- **Management:** Initial management may include nasal dilators or external support devices. In more severe cases, surgical interventions such as cartilage grafts, lateral wall suspension, or structural rhinoplasty may be needed in severe cases to restore nasal valve stability and improve airflow. [29]

8. Cervical Spine Osteoarthritis



- **Etiology:** Cervical spine osteoarthritis is associated with age-related cartilage degeneration, chronic mechanical stress, trauma, microinjury, and cervical alignment changes. [30,31]

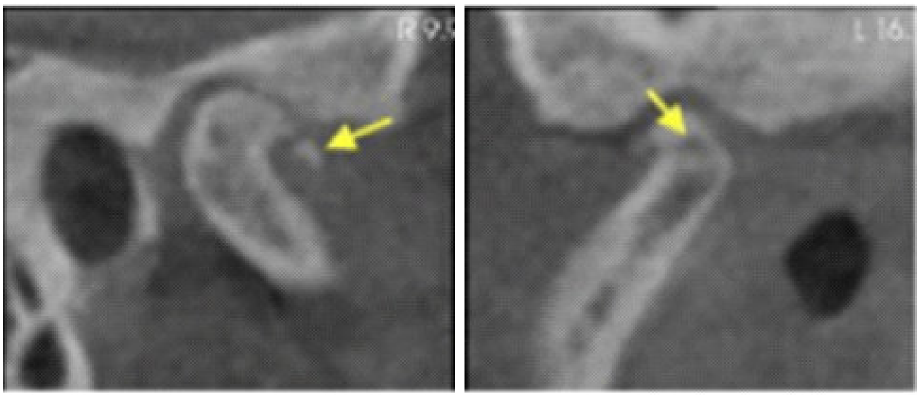
- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate the osseous structures of the cervical spine. Magnetic resonance imaging (MRI) may be used to assess soft tissue components, including intervertebral discs, joint structures, and inflammatory changes. [33]

- **Effect:** Cervical spine osteoarthritis may present with neck pain, stiffness, radiculopathy, myelopathy, reduced range of motion, and functional limitations. These symptoms may negatively impact sleep quality and reduce adherence to MAD therapy. Chronic cervical pain may further disrupt sleep and increase fatigue. [32]

- **Impact on MAD Therapy:** Limited cervical mobility due to pain or stiffness may reduce tolerance to MADs, complicate mandibular protrusion, and contribute to muscle fatigue. Cervical muscle tension may also reduce comfort and adherence, ultimately affecting the effectiveness of OSA therapy. [33]

- **Management:** Conservative management may include physical therapy, manual therapy, traction, pharmacologic pain control, and image-guided cervical facet injections. Surgery intervention is typically reserved for severe cases with neurologic compromise or instability. Pre-treatment evaluation of cervical spine status is recommended in patients undergoing MAD therapy. [33]

9. Joint Mice and Subchondral Cysts of the TMJ



- **Etiology:** Joint mice and subchondral cysts of the temporomandibular joint can develop in the setting of chronic joint changes, although they arise through different mechanisms. Joint mice are most associated with synovial chondromatosis, in which the synovial membrane undergoes changes that lead to the formation of cartilaginous nodules which may detach and become loose bodies within the joint. [70] In contrast, subchondral cysts are associated with degenerative joint disease, where ongoing mechanical stress and cartilage breakdown allow synovial fluid to enter the subchondral bone, leading to cyst formation. [34]
- **Image:** Cone-beam computed tomography (CBCT) is a preferred imaging modality for evaluating osseous changes, including calcified loose bodies, bony erosions, and subchondral cysts. Additionally, magnetic resonance imaging (MRI) is essential for detecting non-calcified bodies, joint effusion, and early cystic changes. [35,37,38,70]
- **Effect:** Patients may present with preauricular pain, swelling, trismus, crepitus, and joint sounds, commonly associated with mechanical obstruction from intra-articular loose bodies. In contrast, subchondral cysts may contribute to deeper bone pain and progressive structural deterioration. Joint effusion and synovial inflammation may further increase symptom severity. [35,36]
- **Impact on MAD Therapy:** Pain and mechanical restriction associated with intra-articular loose bodies (joint mice) and subchondral cysts may reduce tolerance to the mandibular protrusion required for MAD therapy. Limited mandibular motion can restrict adequate advancement, while underlying inflammation and degenerative changes may contribute to muscle fatigue and decreased adherence to treatment. [36]
- **Management:** Management depends on symptom severity. Initial treatment may include analgesics, anti-inflammatory medications, and therapeutic jaw exercises. In cases involving persistent symptoms or mechanical obstruction from intra-articular loose bodies, surgical interventions such as arthroscopy, synovectomy, or open excision may be indicated. Subchondral cysts are typically managed conservatively, focusing on symptom control and limiting joint deterioration. [34,38,39]

10. Prominent External Occipital Protuberance

- **Etiology:** Prominent External Occipital Protuberance (PEOP) develops as an adaptive osteogenic response to chronic mechanical stress and sustained traction on the nuchal ligament and muscle attachments (trapezius and semispinalis capitis). This increased biomechanical load results from forward head posture (FHP) and upper cervical hyperextension, which serve as compensatory mechanisms in OSA patients to maintain airway patency. [42-44]

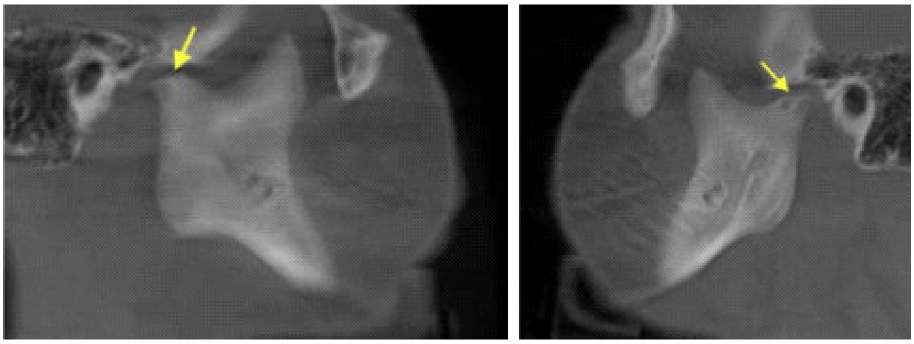
- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate this finding and provides detailed assessment of osseous structures. Additionally, lateral cervical radiographs were utilized to assess sagittal alignment and cervical posture, offering critical insight into the biomechanical factors that may influence mandibular advancement device (MAD) therapy. [40,41,43]

- **Effect:** This finding is often incidental and asymptomatic; however, it may be associated with neck pain, posterior skull tenderness, postural strain, and altered cranio-cervical mechanics. [40,42]

- **Impact on MAD Therapy:** Forward head posture or cervical imbalance associated with PEOP may influence mandibular positioning and patient comfort during MAD therapy. Patients with significant postural deviations may experience neck strain or reduced tolerance to mandibular advancement. Evaluation of cranio-cervical posture is recommended prior to initiation of therapy. [43]

- **Management:** Management of PEOP in the context of OSA requires a multidisciplinary approach. Mandibular advancement devices (MADs) may improve airway patency and reduce reliance on compensatory forward head posture. To complement this, postural rehabilitation and cervical retraction exercises are indicated to mitigate the chronic tensile stress on the nuchal ligament and occipital attachments. [44]

11. Juvenile Idiopathic Arthritis (JIA) Involving the TMJ



- **Etiology:** Juvenile idiopathic arthritis involving the temporomandibular joint is characterized by autoimmune synovial inflammation leading to chronic inflammatory changes affecting the condylar cartilage and surrounding structures. [45–48]

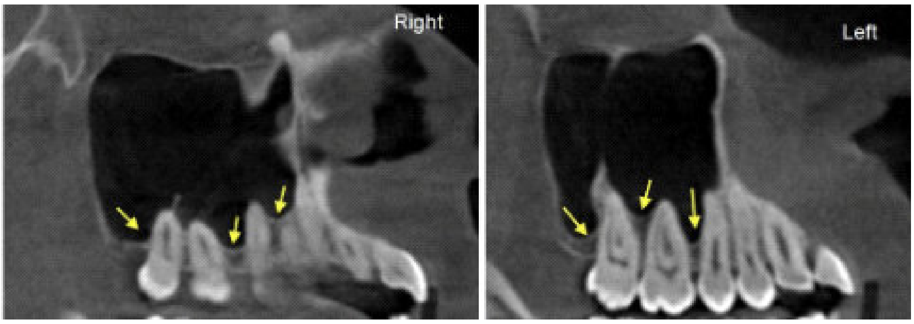
- **Image:** Cone-beam computed tomography (CBCT) was used in this case to evaluate osseous structures. Magnetic Resonance Imaging (MRI) remains the reference standard for detecting active TMJ arthritis, particularly with gadolinium enhancement. [46–49]

- **Effect:** Chronic synovitis may lead to condylar resorption, mandibular hypoplasia, retrognathia, facial asymmetry, malocclusion, and reduced mandibular range of motion. Growth disturbances are particularly relevant in pediatric patients, where TMJ damage can alter normal craniofacial development. [45–48]

- **Impact on MAD Therapy:** Condylar resorption and mandibular hypoplasia may limit achievable mandibular protrusion, reducing the therapeutic efficacy of mandibular advancement devices (MADs). Active inflammation can be exacerbated by continuous protrusive forces, increasing pain, and decreasing tolerance to therapy. [45–48]

- **Management:** During active inflammatory phases, MAD therapy is generally deferred. When indicated, low-force, titratable appliances with gradual advancement and close follow-up may be considered. Multidisciplinary coordination with pediatric rheumatology and orthodontics is essential. [46–49]

12. Pneumatization of the Maxillary Sinuses



- **Etiology:** Etiology includes post-extraction alveolar bone resorption, physiological sinus pneumatization (normal developmental expansion), age-related bone density changes, craniofacial anatomical variation, and periodontal disease-related bone loss. [50–53]

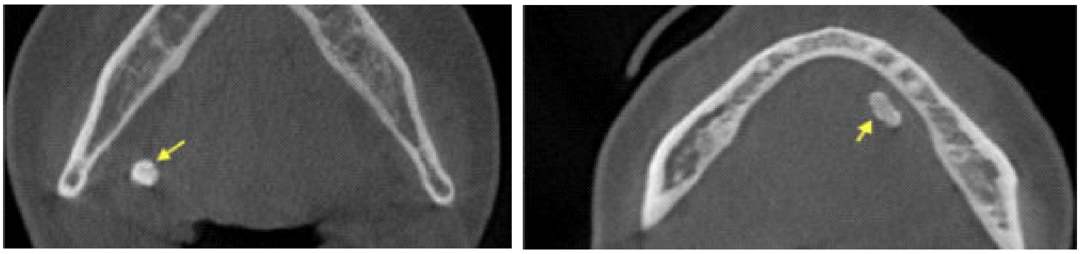
- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate sinus floor extension and residual alveolar bone height and is considered the preferred imaging modality for this purpose. Panoramic radiography may also demonstrate inferior sinus expansion and reduced posterior maxillary bone height. [50–53]

- **Effect:** Maxillary sinus pneumatization is a physiological expansion of the sinus cavity into adjacent alveolar bone, often accentuated after posterior tooth loss. This process may significantly reduce residual ridge height and bone density in the posterior maxilla. [50–53]

- **Impact on MAD Therapy:** Reduction in posterior alveolar bone height may alter maxillary structural support and compromise dental anchorage. Thin sinus floors and reduced bone density can increase local sensitivity and biomechanical vulnerability. Tooth-borne MADs relying on posterior occlusal support may exhibit reduced retention and stability. [50–52]

- **Management:** Appliance design modifications, such as enhanced anterior retention or broader palatal coverage, may improve stability in patients with reduced posterior support. [53]

13. Sialolithiasis of the Submandibular Gland



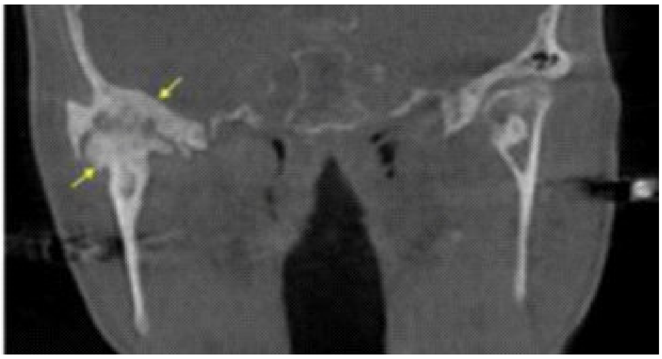
- **Etiology:** Etiology includes salivary stasis or reduced salivary flow, increased calcium and phosphate concentration in saliva, chronic inflammation of the salivary gland, and ductal anatomy favoring retrograde stone formation. [54–57]

- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate this finding. Panoramic and mandibular occlusal radiographs may also demonstrate radiopaque calculi within the submandibular duct. In some cases, calcifications may be identified in the floor of the mouth or submental region, where distinction between submandibular duct sialoliths and other soft tissue calcifications may require further clinical and radiographic correlation. [54–56]

- **Effect:** This condition leads to salivary flow obstruction and patients typically report pain and swelling, particularly during meals. [54–56]

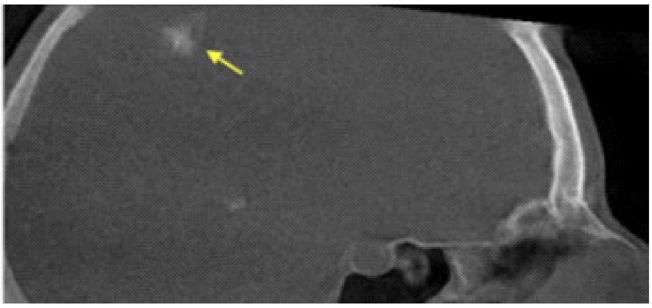
- **Impact on MAD Therapy:** Intraoral appliances may exert pressure on the floor of the mouth, potentially exacerbating obstructive symptoms. Persistent discomfort may reduce compliance and increase the risk of therapy discontinuation. [55]
- **Management:** Obstructive pathology should be treated prior to MAD initiation. Conservative measures such as hydration, gland massage, and sialogogues may be used initially, while minimally invasive techniques such as sialendoscopy may be required for persistent obstruction. [56–57]

14. Fibrous Dysplasia of the TMJ



- **Etiology:** Fibrous dysplasia is caused by a post-zygotic mutation in the GNAS gene, leading to abnormal bone remodeling and fibro-osseous replacement that may involve the mandibular condyle and alter TMJ structure. [58,60]
- **Image:** Cone-beam computed tomography (CBCT) was used to evaluate this finding and is considered a reliable modality for assessing lesion extent and internal bone architecture. Panoramic radiography can demonstrate characteristic changes in the affected region, while computed tomography (CT) and magnetic resonance imaging (MRI) may be used as adjuncts to assess surrounding soft tissues. [59-61]
- **Effect:** Fibrous dysplasia involving the TMJ may result in altered mandibular movement, abnormal joint loading, and facial asymmetry. [59–61]
- **Impact on MAD Therapy:** Irregular condylar anatomy may limit protrusive movement and create uneven force distribution during mandibular advancement [58–61], which may compromise both the effectiveness and tolerance of MAD therapy.
- **Management:** Customized appliance design to minimize focal stress may be considered. Interdisciplinary consultation with maxillofacial specialists is recommended in extensive or progressive cases. [58–61]

15. Partial Ossification of the Falx Cerebri



- **Etiology:** Partial ossification of the falx cerebri is commonly related to age-associated calcification or physiological ossification. It may also present as an incidental or idiopathic finding and has been associated with systemic disorders of bone metabolism. [62,63]
- **Image:** Cone-beam computed tomography (CBCT) was used to identify this finding; however, computed tomography (CT) is more sensitive for detecting falx cerebri calcification. Magnetic resonance imaging (MRI) may also demonstrate ossification as an incidental intracranial finding. [62,63]
- **Effect:** This finding is typically asymptomatic and unrelated to craniofacial functional structures. [62,63]
- **Impact on MAD Therapy:** Mandibular advancement devices act on the upper airway and craniofacial structures. [69] The falx cerebri is an intracranial structure with no anatomical or functional relationship to these regions; therefore, no impact on MAD therapy is expected.
- **Management:** No modification of MAD therapy is required. If extensive ossification is associated with suspected systemic metabolic conditions, appropriate medical referral may be considered. [67]

Discussions

The findings of this review demonstrate that various clinical and radiographic conditions can affect how patients respond to mandibular advancement device (MAD) therapy. In particular, factors affecting nasal airflow and temporomandibular joint function appear to play a significant role. Reduced nasal airflow may lead to increased reliance on mouth breathing, which can compromise the effectiveness of the appliance. Additionally, joint-related conditions may limit mandibular protrusion or contribute to discomfort during use.

Other findings, including craniofacial structural or cervical spine posture, may also influence appliance fit, comfort, and overall function. Even when not the primary clinical concern, these factors may affect long-term tolerance and treatment outcomes. Accordingly, comprehensive pre-treatment evaluation is essential to guide clinical decision-making and optimize patient management.

This review has several limitations, including its retrospective design and the short observation period, which may limit the generalizability of the findings. Further research with larger samples and longer follow-up would be helpful for a better understanding of the clinical impact of these factors.

Conclusion

Mandibular advancement devices represent an effective and widely utilized treatment modality for selected patients with obstructive sleep apnea. However, their clinical success is not solely dependent on OSA severity or appliance design but is also influenced by individual anatomical variations, temporomandibular joint integrity, cervical and craniofacial biomechanics, nasal airway patency, and underlying systemic conditions.

The spectrum of conditions discussed, including nasal obstruction, TMJ degeneration, craniofacial structural anomalies, inflammatory rheumatologic disease, fibro-osseous disorders, salivary gland pathology, cervical spine changes, and incidental radiographic findings, highlights the multifactorial considerations involved in MAD therapy. Each of these conditions should be carefully evaluated prior to MAD initiation in patients with OSA. While some represent relative contraindications, others may require device modification, conservative titration strategies, patient education, or interdisciplinary collaboration.

Importantly, most of these findings do not preclude oral appliance therapy but rather emphasize the importance of individualized treatment planning. A comprehensive assessment, including detailed medical and dental history, targeted clinical examination, and appropriate imaging when indicated, is essential for identifying potential risk factors early. In selected cases, coordination with otolaryngology, rheumatology, neurology, maxillofacial surgery, physical therapy, or sleep medicine may be necessary to ensure safety and long-term therapeutic stability.

Ultimately, a patient-centered, evidence-informed approach that integrates anatomical evaluation, functional assessment, and interdisciplinary management is fundamental to optimizing MAD outcomes. Through careful screening and personalized modification, clinicians can maximize therapeutic efficacy while minimizing complications and improving long-term adherence in the management of obstructive sleep apnea.

Conflict of Interest

The authors declare no conflict of interest

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