PAIN UPDATE

Obstructive sleep apnea in a patient with orofacial pain secondary to cervical fixation

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CLINICAL PROBLEM

Orofacial pain symptoms associated with obstructive sleep apnea (OSA) include morning headaches and facial pain.[1] The upper airway is blocked in OSA when the muscles of the throat relax during sleep, causing the soft tissues in the back of the throat to collapse. OSA also can result from a physical obstruction that has the potential to compress the airway. The placement of anteriorly positioned cervical fixation has this capability.

CLINICAL PRESENTATION

We describe the case of a 58-year-old woman who was referred by her general dentist to the Center for Temporomandibular Disorders and Orofacial Pain at the Rutgers School of Dental Medicine with chief symptoms of pain in the right temporomandibular joint (TMJ), a limited mandibular range of motion, and headaches. Additional symptoms included loud snoring, difficulty breathing, and gasping for air while asleep. She was not referred for evaluation of a sleep disorder.

In 2006, the patient experienced pain in the right TMJ and along the lower border of the mandible that appeared to have evolved from a motor vehicle accident. She underwent arthroplasty, meniscectomy, and eminectomy of the right TMJ. She reported developing facial paralysis on the right side after this surgery. She became emotionally depressed and reported having weekly headaches "that feel like a tight band around my head" lasting for several hours. Her headaches were worse on awakening, and she reported them as 10 of 10 on a visual analog scale. Her headaches decreased as the day would progress.

Her problems were exacerbated after a second motor vehicle accident in 2009. She subsequently had herniated cervical disks diagnosed, and a metal plate with 3 screws was placed to stabilize the lower cervical spine (Figures 1 and 2, Table). The patient reported snoring loudly and having difficulty breathing during sleep after the cervical fixation. Panoramic and lateral cephalographic radiographs, initially obtained for assessment of her facial pain symptoms, showed that an anteriorly positioned cervical fixation device impinged on her trachea, resulting in airway constriction. In addition, during intraoral examination, we found anatomic markers consistent with OSA. Therefore, a complete history and results of a dental and orofacial pain examination revealed not only a temporomandibular disorder (TMD) but also previously undiagnosed OSA.

MEDICAL HISTORY

The patient was hospitalized for a transient ischemic attack in 2014 and for tachycardia in 2015. No sequelae for the transient ischemic attack were reported. She admitted to having anxiety and to having refused a referral for psychological evaluation. Her medications included 60 milligrams of delayed-release dexlansoprazole, 25 mg of mirabegron, 5 mg of solifenacin, 100 mg of docusate, diclofenac sodium topical gel 1%, 10 mg of cyclobenzaprine, 50 mg of metoprolol succinate, 10 mg of escitalopram, and loteprednol etabonate ophthalmic suspension.

A detailed history included poor sleep, tiredness, and headache on awakening. She reported a "lump" in her throat impairing her ability to breathe when she placed her head in specific positions, which would awaken her from sleep. The Epworth Sleepiness Scale (ESS) is the tool most commonly used to measure excessive daytime sleepiness. It is used in both clinical and research settings. This questionnaire consists of 8 everyday situations that provide measurement of sleepiness, ranging from 0 to 3.[2] This patient's ESS score was 20.[2] A score higher than 18 is considered severe and requires medical attention.[3]

We evaluated the TMJs consistent with the diagnostic criteria for TMD.[4] The patient's mandibular range of motion was limited and painful. We noted an uncorrected deviation (3-4 millimeters) of the mandible to the right on opening. We perceived crepitus in both joints.

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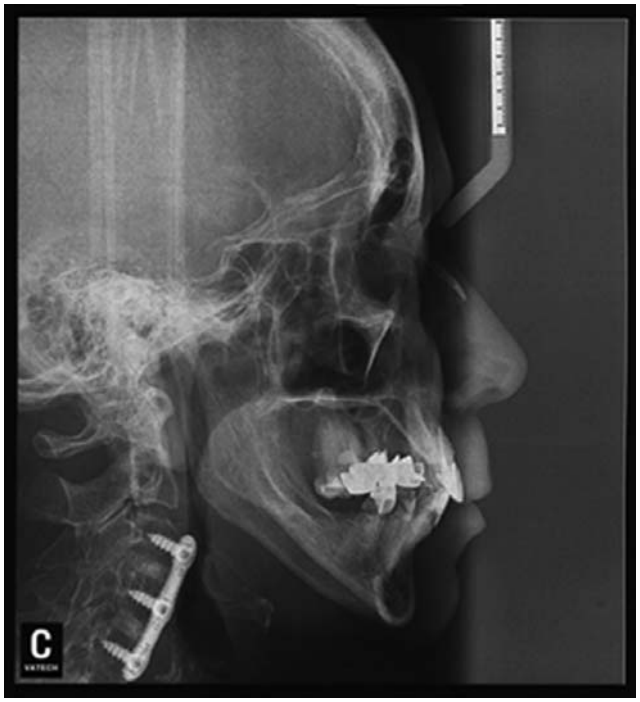

Figure 1. Lateral cephalogram of the cervical fixation impinging on the trachea, leading to obstruction of the upper airway.

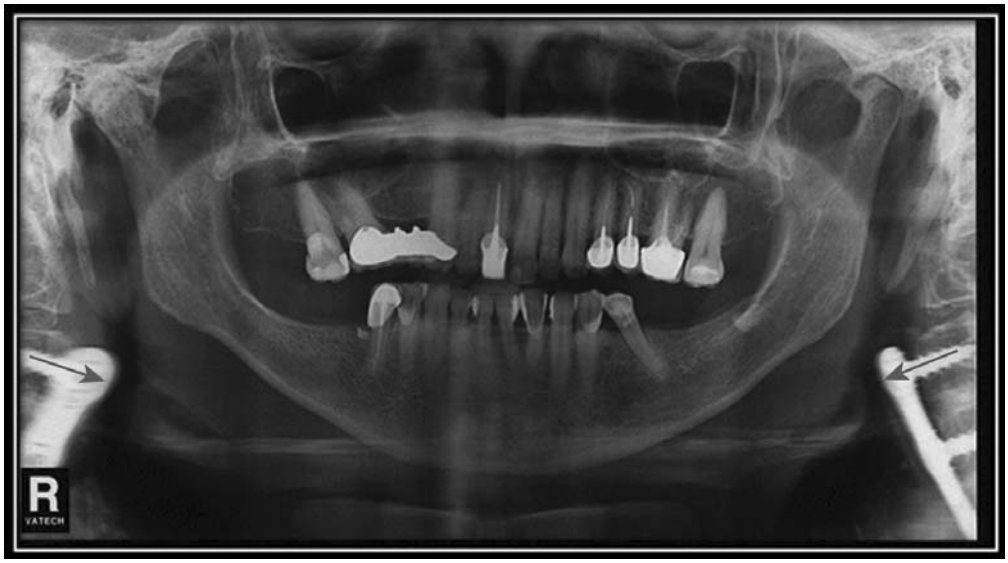

Figure 2. Panoramic radiograph of the cervical fixation impinging on the trachea (arrows), leading to obstruction of the upper airway.

A palpatory examination of the cervical and masticatory musculature revealed mild to moderate tenderness throughout the head and neck. Palpation of the temporalis, masseter, sternocleidomastoid, posterior cervical, trapezius, suboccipital, and lateral pterygoid muscles reproduced familiar facial pain and headache. However, the patient commented that the localized headache secondary to muscle palpation was not the same as the daily headache experienced on awakening. The patient had a moderate forward head posture with restriction of extension, rotation, and side bending.

The patient's Mallampati score was IV. This score refers to the opening of the airway on the basis of the relationship of the soft palate to the base of the tongue. The higher the score, I through IV, the more likely the patient has OSA (Figures 3 and 4).[5] The patient's tongue was scalloped (Figure 5). The patient had had multiple missing teeth replaced with fixed and removable prostheses.

Results of the cranial nerve examination were within normal limits, with the exception of a motor deficiency of cranial nerve VII. The patient had difficulty raising her eyebrow, a drooping of the lips ipsilaterally, and a twitching of the muscles of facial expression.

ESS and clinical evaluation results indicated possible OSA with both physical and iatrogenic causes. On the basis of the chief symptoms and clinical findings, we referred the patient to a medical specialist in pulmonology. The specialist performed an overnight polysomnographic test. The patient's apnea-hypopnea index (AHI), used to assess the severity of OSA, was 18.4 (considered moderate, on a scale in which 0 is normal and ≥30 is severe), and the oxygen desaturation rate was 12%, helping to confirm the diagnosis of OSA. The physician prescribed a continuous positive airway pressure (CPAP) device. The CPAP device, the primary treatment for OSA, provides CPAP through a nonrebreathable mask worn during sleep. The pressure maintains the airway patency, overcoming the respiratory disturbance.[6]

PATHOPHYSIOLOGY

There are 3 types of sleep apnea. These are central sleep apnea, OSA, and mixed sleep apnea.[7] Central sleep apnea results from a temporary cessation of signals from the brain to the respiratory muscles.[8] OSA results from a decreased or complete cessation of air flow despite an ongoing effort to breathe. A partial reduction (hypopnea) or complete pauses (apnea) in breathing occur during sleep, and these last at least 10 seconds. This stoppage leads to a severe reduction in oxygen levels during which the brain induces an abrupt arousal to restore normal breathing.[9] OSA is categorized as mild

TABLE

Chronology.

YEAR	EVENT
2006	Motor vehicle accident Bilateral temporomandibular disorder with anterior disk displacement and myogenous pain Conservative therapy was ineffective
2007	Eminectomy and meniscectomy
2009	Motor vehicle accident Exacerbation of previous temporomandibular disorder Cervical injuries Cervical fusion at C5 through C7 with an anteriorly positioned plate Difficulty breathing and snoring begins
2010	Slow onset of headache, tiredness, and difficulty breathing, especially during sleep Temporomandibular disorder progressively worsens No specific treatment
2016	Referred to the Center for Temporomandibular Disorders and Orofacial Pain at the Rutgers School of Dental Medicine

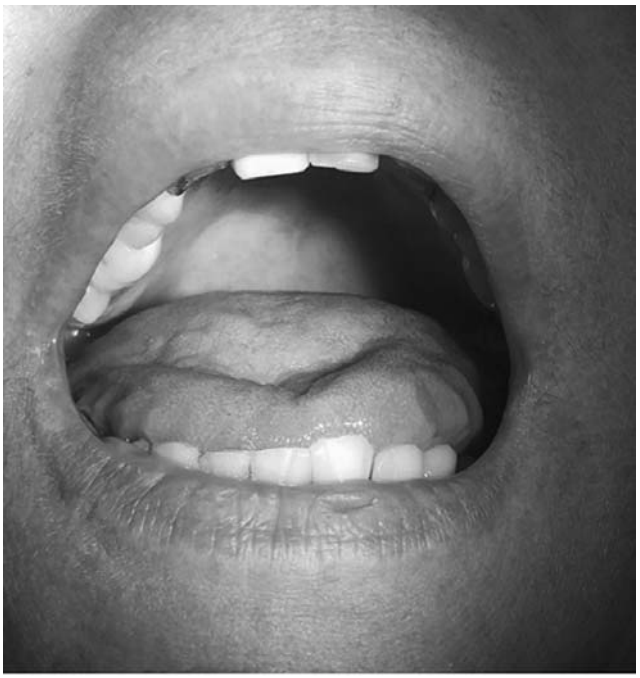

Figure 4. Mallampati class IV. Photograph of the uvula hidden behind the base of the tongue. The upper oropharynx cannot be seen.

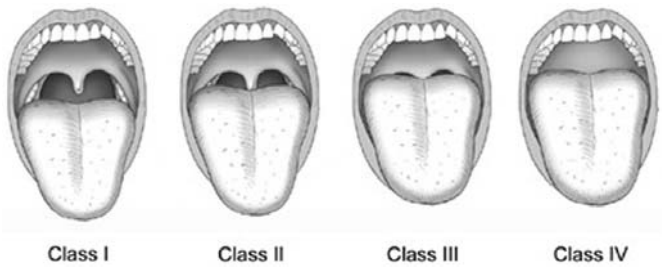

Figure 3. Mallampati airway classification. During assessment, the patient is instructed to open his or her mouth as wide as possible while protruding the tongue as far as possible. Patients are instructed not to emit sounds during the assessment. Class I: soft palate and entire uvula visible. Class II: soft palate and portion of uvula visible. Class III: soft palate visible (may include base of uvula). Class IV: soft palate not visible. *Reproduced with permission of the publisher from Nuckton and colleagues.*[5]

(5-15), moderate (15-30), or severe (more than 30), depending on the AHI.[9] Mixed sleep apnea is a combination of obstruction of the airway and blocked signals to the respiratory muscles from the brain. Report results have suggested that patients with mixed sleep apnea fail to breathe even after the airway is opened or with CPAP.[7]

Of importance to the general dentist is that OSA is often comorbid or associated with chronic headache and musculoskeletal pain associated with TMDs and orofacial pain.[10] In results from a 2013 prospective cohort study, Sanders and colleagues[11] showed a significant association between the signs and symptoms of OSA and TMD, suggesting a bidirectional relationship. OSA is considered a medical risk factor for systemic hypertension. It is associated with headaches, diffuse muscle pain, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, increased rates of motor vehicle accidents, sleepiness, cognitive impairment, loss of memory, impaired quality of life, and increased mortality.[12] Although most study investigators do not differentiate between primary snoring and mild OSA, snoring is an important social problem.

The primary cause of OSA is weakness of the pharyngeal muscles, leading to a collapsed airway, but it also may result from mechanical compression. Cervical fixation devices can affect airway impingemnt.[13,14] In results from a retrospective study in 2008, Patel and colleagues[15] showed various complications from cervical fixation surgeries, including dysphagia, odynophagia, and esophageal and tracheal compromise due to instrument migration resulting in a fatal outcome.[16] In 2007, Yoshida and colleagues[14] discussed cervical fixation techniques to avoid airway obstruction. Therefore, a history of cervical fixation for any patient with OSA must be given consideration as a potential causative factor.

TREATMENT

This patient initially sought care for a TMD. She also received a diagnosis of OSA. She became intolerant of the CPAP, and her physician referred her for an evaluation for an intraoral mandibular advancement device to

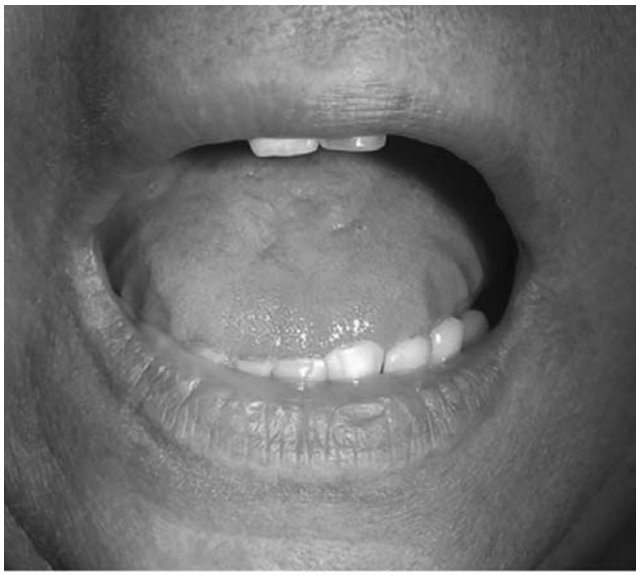

Figure 5. Photograph of a scalloped tongue.

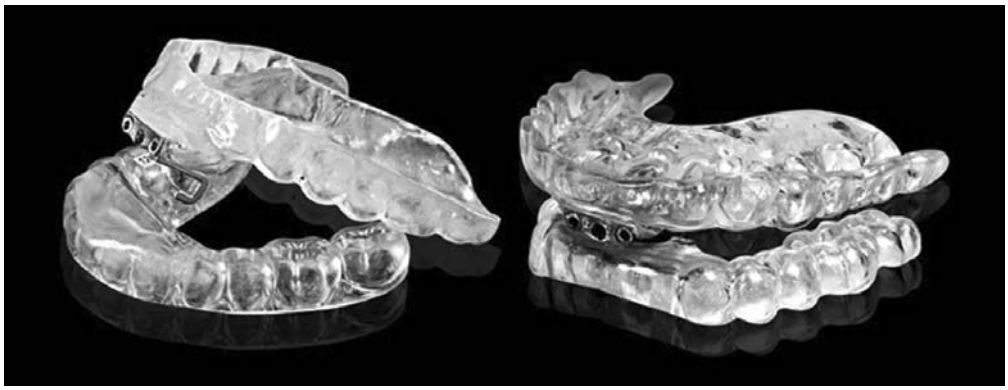

Figure 6. Photograph of the Thornton Adjustable Positioner 3 (right) and Thornton Adjustable Positioner 3 Elite (left). *Reproduced with permission from Airway Labs*.

maintain the patency of her airway. This device also can stabilize the TMJs. These appliances are provided by dentists only with a prescription from a physician and typically are used when CPAP is not tolerated.

We chose the Thornton Adjustable Positioner 3 device (Figure 6) because it is effective in the treatment of mild to moderate OSA. It reduces the effect of associated health risks without surgical intervention, CPAP, or medication. The device increases tongue space and allows the lips to seal by holding the mandible in a forward position. The mandible does not shift, which prevents the airway from collapsing. In this case, because of the TMD, there was limited protrusion. The appliance opens the bite with minimal anterior repositioning but is adequate to stabilize the TMJs while at the same time treating the OSA.[1] We also referred the patient back to the surgeon for reevaluation of the cervical fixation device. We provided home care instructions, including jaw exercises, to manage the TMJ pain.

DISCUSSION

OSA is a serious and potentially life-threatening condition with multiple comorbidities. The dentist is often the first-line health care provider in detecting this disorder. It can lower the quality of life significantly and may result in a significant health risk. Because the cause for OSA is observed readily during the dental evaluation, the dental care practitioner must have an awareness of what constitutes screening for OSA and the mechanism for appropriate referral to a medical colleague. Investigators in previous studies have shown that approximately 50% of patients with TMD report poor sleep quality. Smith and colleagues[17] in 2009 reported that approximately 43% of patients with TMD had 2 or more sleep disorders and that 24% of these had OSA, emphasizing the importance of evaluation for OSA.

In 2005, White[13] reported that pharyngeal anatomy and upper airway muscle responsiveness during sleep may contribute to the presence and severity of OSA. In a 1999 study, Sakakibara and colleagues[18] showed that an enlarged tongue, soft palate, and soft tissue around the upper airway can contribute significantly to OSA. Yoshida and colleagues[14] in 2007 reported that improper alignment of cervical fixation of the occipitocervical spine also can lead to OSA because it alters the pharyngeal anatomy. In this case, in addition to a TMD and an anatomic cause for OSA, the patient had cervical fixation that impinged on her airway, creating a mechanical obstruction.

This patient reported snoring, odynophagia, and a reduced range of neck motion. In a 2008 study, Patel and colleagues[15] retrospectively reviewed more than 3,000 cases of anterior cervical spine surgery and reported concomitant odynophagia and reduced range of motion. This patient also was considered for an intraoral mandibular advancement device because of intolerance to CPAP. Results from various studies show the efficacy of mandibular advancement appliances in reducing OSA. The literature supports a reduction in AHI levels, ESS score, and other comorbidities associated with OSA in patients treated with intraoral mandibular advancement devices.[19,20] According to Giannasi and colleagues,[1] long-term use of an intraoral appliance for the management of OSA does not cause any impairment of the TMJs but in turn alleviates the symptoms of TMD.

CONCLUSIONS

This patient sought care for an orofacial pain symptom, and we evaluated her for OSA as part of a routine dental evaluation. We assessed OSA in a dental setting. A consultation with a pulmonology medical specialist helped confirm the diagnosis via polysomnography. A meticulous review of the patient's history, a clinical evaluation, and review of radiographs disclosed multiple causes, including a restricted airway with both physiological and mechanical causes.

A major contributor to this patient's symptoms was the cervical fixation. Proper positioning of cervical fixation is important to avoid postoperative airway obstruction. Cervical fixation can narrow the airway by impinging on the trachea. Adequate preoperative and postoperative imaging can aid in proper diagnosis and management of OSA. In this case, the cause of the patient's OSA was undetected until a dental evaluation, including an intraoral examination of soft tissues, a sleep history, and dental radiographic studies, revealed the cause of her symptoms. We treated TMJ issues concomitantly with the OSA. This and similar cases demonstrate the key diagnostic role of the dental clinician, especially one trained in orofacial pain, oral medicine, or dental sleep medicine. ■

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