



Tinnitus and temporomandibular disorders: a literature discussion and case report

Zumbido e disfunções temporomandibulares: uma discussão da literatura e relato de caso

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Abstract

Tinnitus describes the sensation of any sound perceived in the head or in the ears without any evident stimulus. It may be associated with any form of sensorineural hearing impairment but difficulty in hearing is the major determinant of tinnitus, followed by aging and exposure to noise. Somatic tinnitus is a type of subjective tinnitus in which the frequency or intensity is altered by body movements such as clenching the jaw, turning the eyes, or applying pressure to the head and neck. This article reviews the literature about the possible etiologic factors associated with tinnitus, including temporomandibular disorders, while also presenting a clinical case in which tinnitus could be associated with myofascial pain. Adequate diagnosis and treatment planning of the clinical case are presented and discussed as well.

Keywords: Temporomandibular disorders. Tinnitus. Myofascial pain. Etiology. Management.

Resumo

O zumbido é descrito com uma sensação de qualquer tipo de som percebido na cabeça ou ouvidos sem nenhum estímulo evidente. Pode estar associado com alguma forma de deficiência auditiva neurosensorial, mas a dificuldade de audição é o maior fator determinante do zumbido, seguido do envelhecimento e da exposição a ruídos. O zumbido somático é um tipo de zumbido subjetivo cuja frequência ou intensidade é alterada por movimentos do corpo, como fechamento da mandíbula, movimento dos olhos ou aplicação de pressão na cabeça e

pesçoço. Este artigo faz uma revisão da literatura sobre os possíveis fatores etiológicos associados ao zumbido, incluindo disfunções temporomandibulares, além de apresentar um caso clínico no qual o zumbido pode ser associado à dor miofascial. O adequado diagnóstico e o plano de tratamento do caso clínico também são apresentados e discutidos.

Palavras-chave: *Disfunção temporomandibular. Zumbido. Dor miofascial. Etiologia. Tratamento.*

Introduction

Tinnitus is defined as a phantom auditory perception of sound without corresponding acoustic or mechanical correlates in the cochlea. Tinnitus describes the sensation of any sound perceived in the head or in the ears without any evident stimulus. Tinnitus represents one of the most common and distressing otologic problems and can cause psychological disorders that may interfere with patient's quality of life (1). It has a high prevalence (15% in the general population and 33% in the elderly) and causes considerable morbidity, which may interfere with sleep, concentration, emotional balance and social life of subjects. On the other hand, the pathophysiological complexity and subjectivity of tinnitus reduces the interest of ENT (Ear, Nose and Throat) doctors in this symptom (2).

The association between tinnitus and hearing loss has been well described. According to different reports, 85% to 96% of the patients with tinnitus present with some level of hearing loss and only 8% to 10% present normal audiometry. In this last group, the isolated presence of tinnitus suggests that it may be a primary symptom of diseases that are only diagnosed after the onset of hearing loss. The origin of tinnitus in these cases is still more obscure than in those with concomitant hearing loss. These patients form a significant sample due to the characteristics attributed to tinnitus, and not to the hearing loss that follows the other cases (2).

Although tinnitus is subjective, for clinical purposes it is subdivided into subjective and objective tinnitus, the latter describing those few incidents in which the sound is detected or potentially detectable by another observer. Somatic tinnitus is a type of subjective tinnitus in which the frequency or intensity is altered by body movements such as clenching the jaw, turning the eyes, or applying pressure to the head and neck (1). Tinnitus is considered to be chronic when it has been present for more than

three months without signs of spontaneously resolving (3).

Most people occasionally experience tinnitus, and most experience it in silent soundproofed rooms. The British national study of hearing found that 10% of adults had prolonged spontaneous tinnitus – that is, tinnitus “usually lasting for longer than five minutes”; 1% experienced severe annoyance due to tinnitus, and in 0.5% of adults tinnitus severely reduced the ability to lead a normal life. The same study ascertained that 7% of the population in the United Kingdom presented to their general practitioners with tinnitus and 2.4% were referred to hospitals (4, 5).

Noise-induced tinnitus can be acute or chronic. Acute tinnitus can last from a few minutes to a few weeks after noise exposure. In some cases, tinnitus has a gradual onset and several years can pass before an intermittent, low-intensity tinnitus becomes bothersome. Spontaneous remission by natural habituation is experienced by more than three-quarters of sufferers. Habituation occurs within the CNS, whereas adaptation involves a peripheral sensory organ. When the condition worsens, the tinnitus intensity increases over time but its pitch tends to remain stable. If tinnitus persists for more than two years, it is considered permanent and irreversible (5).

There are some studies that actually evaluated hypothesis that would link tinnitus and TMD. For example, Komori et al. (6) examined autopsy specimens of 12 Japanese adult cadavers by a superior approach through the middle cranial fossa. They found a bilateral ligament that they called the discomalleolar, which passed through the petrotympanic fissure and attached the neck of the malleus to the Temporomandibular Joint (TMJ) articular disc. They proposed that this ligament might be a factor in explaining the existence of the ENT symptoms associated with TMD, since it connects the mandibular lingula and TMJ disc to the middle ear. These findings

form an anatomic basis for the clinical relationship between TMD and ENT symptoms.

Attention and affective components of tinnitus anxiety and phobias emerge in the limbic system; moreover, the psychosocial dimension may trigger tinnitus states. Such broad limbic organization is the most important CNS modulator component that is able to transform (positively or negatively) the whole organism's homeostasis (pain, concentration, temperature, muscle hyperactivity, memory, motivation, vegetative, hormonal, endocrine, feed, sleep, and other circadian rhythms) as a response to survival instinct stimulus. Mild tinnitus could be someone's worst experience (high priority attention) but also the least significant incident (low priority attention) for the same person, depending on personal biopsychosocial reaction and its negative emotional reinforcement (7).

Literature discussion

Etiology - TMD and Tinnitus

Tinnitus does not represent a disease itself but instead is a symptom of a variety of underlying diseases. Otologic causes include noise-induced hearing loss, presbycusis, otosclerosis, otitis, impacted cerumen, sudden deafness, Meniere's disease, and other causes of hearing loss. Infectious causes include otitis media and sequelae of Lyme disease, meningitis, syphilis, and other infectious or inflammatory processes that affect hearing. Some oral medications, such as salicylates, nonsteroidal anti-inflammatory drugs, aminoglycoside antibiotics, loop diuretics, and chemotherapy agents can cause tinnitus (1).

ENT symptoms, such as deafness, dizziness, tinnitus, and earache, are common in patients with temporomandibular disorders (TMD) (3).

Temporomandibular disorders (TMD) are characterized by various signs and symptoms of pain and dysfunction in the temporomandibular joint (TMJ) and/or the masticatory musculature. The signs and symptoms of TMD can manifest in areas of the face and neck; the temporal, occipital, and frontal areas of the head; and the preauricular and auricular areas. In addition to pain and dysfunction, many patients with TMD also complain of aural symptoms. The most commonly reported aural symptoms in TMD patients are ear pain, tinnitus, vertigo/dizziness, and

subjective hearing loss. The prevalence of tinnitus in the TMD population appears to be greater than that found in the general population. Studies of the general population have revealed a median prevalence of all forms of tinnitus in the range of 15% to 20%, with the prevalence increasing with age. The frequency of tinnitus among patients attending TMD clinics has been reported as varying from 33% to 76% (8-10).

Approximately two-thirds of individuals with tinnitus can modulate the loudness or pitch of their tinnitus by voluntary or external manipulations of the jaw, movements of the eyes, or pressure applied to head and neck regions, including the temporomandibular joint (somatic tinnitus). In some cases, the necessary manipulations reported were forceful; while in others, less vigorous manipulations could produce the changes in perceived loudness and/or pitch of the tinnitus. In addition, there is an increased prevalence of somatoform disorders in individuals with tinnitus, as well as reports of tinnitus occurring after dental pulpalgia that resolved after endodontic therapy. Tinnitus is also associated with upper craniocervical imbalances, such as prolapsed intervertebral disks or instability of the craniocervical junction. Similarly, tinnitus occurs more frequently in patients who have craniocervical mandibular disorders, such as temporomandibular joint syndrome or temporomandibular disorders (TMD) (1, 7).

In another study, of the 344 subjects who had TMD, 59.9% complained of aural symptoms, versus 29.2% of the 432 patients without TMD. Of the subjects with ear pain, tinnitus, vertigo, or perceived hearing loss, 67%, 64.1%, 65.2%, and 62.2% had TMD, respectively. Subjects with aural symptoms were significantly more likely to be female, to consider themselves in poor health, and smoke. They also seemed to have TMD, orofacial pain, headaches (temporal, occipital, or frontal), neck and shoulder pain, altered vision and sensation, sleep disturbances, loss of appetite, memory loss, or low energy. Clinical findings indicated that pathognomonic signs of TMD were associated with an increased risk of aural complaints in this patient population. A significantly greater negative impact on normal life functions was found in subjects exhibiting aural symptoms versus those who only had TMD complaints (8).

Tensor tympani muscle physiology and function in the middle ear have been indistinct, even when their dysfunction and anatomical relationships may

explain a group of confused otic symptoms during conventional clinical evaluation. Middle ear muscles share a common embryological and functional origin with chewing and facial muscles. These muscles share a functional neurological and anatomical dimension with the stomatognathic system. These muscles' increased tonicity ceases to be a phenomenon having no logical connections. Tinnitus, vertigo, otic fullness sensation, hyperacusia, hypoacusia and otalgia are not only primary hearing organ symptoms. They should be redefined and related to the neighboring pathologies which can produce them. There is a need to understand TMD and craniofacial referred symptomatology from neurophysiologic and muscle-skeletal angles contained in the stomatognathic system. Common symptomatology is frequently observed in otic symptoms and TMD during daily practice; this should be understood by each discipline from a broad, anatomical and clinical perspective (11).

The intimate nexus between dysfunctional masticatory activity, such as bruxism, TMD and emotional stress, can lead to the cause-effect relationship between them. TMD etiology thus has a multifactor origin involving environmental, physiological and behavioral agents. TMD produces contraction and tension of the masticatory muscles and reflex contraction in tensor veli palatini and tensor tympani muscles due to common motor innervation from the trigeminal mandibular branch (V3) (12). Ear pain can be felt by tympanic membrane tension due to constant tensor tympani muscle contracture. Such tension can produce dysacusia (a more general tinnitus symptom) accompanied by strange or hallucinatory acoustic sensations perceived as rings, clicks, pops, whispers or friction noises (11).

Other Etiologic Factors

Tinnitus could also be explained as an overexcitation of hearing structures in the same way as allodynia can be an overexcitation of neural structures in the somatosensory system. A case report was presented in the literature linking tinnitus with a subtype of neuropathic pain, postherpetic neuralgia (PHN). In this particular patient, tinnitus developed together with PHN and increased in intensity in proportion to the amount of PHN pain. Tinnitus was responsive to PHN treatment and disappeared with PHN recovery, which led the authors to hypothesize

that tinnitus was a symptom associated with the neuropathic pain in this patient (12).

Conductive hearing impairment with tinnitus occurs after middle ear surgery and myringoplasty. It also occurs in chronic suppurative otitis media and is a common feature of otosclerosis. Temporary tinnitus may be due to wax in the ear canal, otitis media, Eustachian tube catarrh, drug ingestion or toxemia, or to recent exposure to noise (3).

Tinnitus is known to occur as a concomitant of most of the dysfunctions that involve the human auditory system, and it is postulated that the etiology of tinnitus is diverse and that different activation circumstances can be present. Little is known about the pathophysiology and there is no known drug or curative therapy currently available, though considerable research effort has been expended in this regard (13).

Management

Tinnitus treatments can be divided into two categories: 1) those aimed at directly reducing the intensity of tinnitus and 2) those aimed at relieving the annoyance associated with tinnitus. The former include pharmacotherapy and electrical suppression, and the latter include pharmacotherapy, cognitive and behavioral therapy, sound therapy, habituation therapy, massage and stretching, and hearing aids (1). We could also include the treatment directed towards temporomandibular disorders and associated tinnitus into this second category (8-10).

Since the late 1970s, prosthetic treatment involving hearing aids, tinnitus maskers, or combination instruments have been widespread in tinnitus management. Maskers look similar to hearing aids and produce a broad band of noise. The instruments generate an external, constant sound that can be controlled by the patient, allowing the patient to concentrate rather than be distracted by sounds from televisions, etc. They divert attention and thereby make tinnitus less troublesome (3).

One study (14) focused on the effect of Melatonin in conjunction with Sulodexide as a treatment method for tinnitus. Overall, 102 patients suffering from tinnitus were evaluated in a prospective randomized controlled study conducted in a tertiary care ENT department. After randomization, 34 patients were treated with Melatonin and Sulodexide, another 34

were treated with Melatonin alone, while the remaining 34 (control group) were managed without treatment in order to evaluate spontaneous variations in the quality of tinnitus. Patients were assessed prospectively with the Tinnitus Handicap Inventory and Acufenometry, both pre-treatment and post-treatment. Among the patients studied, better results with both Tinnitus Handicap Inventory and Acufenometry were found in the group that received Melatonin and Sulodexide compared to those receiving Melatonin alone. No improvement was observed in the control group. In conclusion, Melatonin in combination with Sulodexide was, in their opinion, a viable treatment option for patients suffering from central or sensorineural tinnitus.

Available clinical trials vary in methodological rigor and have been performed for a considerable number of different drugs (15). None of the investigated drugs have provided replicable long-term reduction of tinnitus intensity in the majority of patients, in excess of placebo effects. Accordingly, there are no FDA or EMEA approved drugs for the treatment of tinnitus. However, despite the lack of evidence, a large variety of different compounds are prescribed off-label (16). A large variety of drugs from different pharmacological classes is currently prescribed for tinnitus, despite the fact that for a large number of them no data from clinical trials is available. Ginkgo biloba contains bioactive flavonoids with vasoactive and antioxidant properties and has been proposed for the treatment of a wide range of disorders, including tinnitus. Even though some studies have suggested beneficial effects of ginkgo on tinnitus, particularly in patients with short duration symptoms, there is a growing body of evidence from large, double-blind, placebo-controlled clinical studies clearly indicating that ginkgo is no more effective in alleviating tinnitus symptoms than placebo (16).

In 1935, lidocaine was found to suppress tinnitus following nasal administration. Since then, many clinical studies have confirmed transient tinnitus suppression by intravenous administration in 40–70% of tinnitus subjects. The effect seems to be dose dependent with suppression of tinnitus occurring at free arterial plasma concentrations from 1.75 to 3.5 $\mu\text{mol/L}$, whereas concentrations above 3.5 $\mu\text{mol/L}$ may induce tinnitus (17).

Besides their antidepressant properties, tricyclics have been shown to be highly efficient for the treatment of chronic pain. This is of interest because

of the proposed etiological similarities between tinnitus and neuropathic pain (12). These drugs would include nortriptyline, amitriptyline and trimipramine (16).

Stomatognathic treatment addressing masticatory muscle relaxation by using removable interocclusal plastic appliances seems to be able to eliminate or attenuate otic symptoms triggered or exacerbated by TMDs. Oral devices attempt peripheral relaxation of muscle hyperactivity triggered during anxiety and depression. These devices have been used individually or as part of varied treatment including physiotherapy, self-care management and acupuncture. However, several discrepancies limit research into this management device's methodology, making them prone to error and bias due to lack of method standardization. This variety among methods includes the sample-size, diagnostic criteria, oral appliance, combination of oral appliance with another mode of treatment, non-validated questionnaires, absence of control group, and mailing the clinical evaluation, which makes the results difficult to interpret. The placebo effect must be considered in TMD-otic symptom treatment results (18).

Another study (19) tried to determine a clinically valid method for identifying which otologic symptoms would have a higher probability of improving as a result of satisfactory TMD symptom improvement. Two hundred TMD patients with coexisting tinnitus, otalgia, dizziness, and/or vertigo were asked about their otologic symptom characteristics and associations and were given clinical tests, which were speculated to predict otologic symptom response from TMD therapy. The subjects received conservative TMD therapy in a manner thought to be most advantageous for their disorders. These potential assessment instruments were then evaluated for their ability to predict otologic symptom improvement. After satisfactory TMD symptom improvement was obtained, the percentage of subjects reporting significant improvement or resolution of their tinnitus, otalgia, dizziness, and vertigo was 83%, 94%, 91%, and 100%, respectively. Tinnitus, otalgia, and dizziness showed a significant correlation for improvement with younger age. There was also a significant correlation shown in patients with tinnitus and otalgia improvement, who related that the otologic symptoms began when the TMD symptoms began and that they were worse when the TMD symptoms were worse, and it was related to stress. Patients with more severe

TMD symptoms showed a significant correlation with dizziness improvement. The author concluded that asking TMD patients with coexisting otologic symptoms these specific questions would help practitioners identify which otologic symptoms have a higher probability of benefiting from TMD therapy.

One study investigated the presence of symptoms and signs of TMD in patients with tinnitus and evaluated the effect of TMD treatment on tinnitus in a long-term perspective in comparison with a control group of patients on a waiting list. One-hundred-and-twenty patients with tinnitus were subjected to a clinical examination of the masticatory system and whether they had co-existing TMD to TMD treatment. Ninety-six patients had TMD, most frequently localized myalgia. Seventy-three of these completed the treatment and responded to a questionnaire two years later. Fifty patients with tinnitus who were on the waiting list served as a control group. Eighty percent of the patients had signs of TMD, most commonly myofascial pain. Forty-three percent of the patients reported that their tinnitus was improved at the 2-year follow-up, 39% that it was unchanged, and 17% that it was impaired compared to before the treatment. Twelve percent of the subjects in the control group reported that their tinnitus was improved compared to two years previously, 32% that it was unchanged, and 56% that it was impaired. The difference between groups was significant. The results of this study showed that TMD symptoms and signs are frequent in patients with tinnitus and that TMD treatment has a good effect on tinnitus for a long term perspective, especially in patients with fluctuating tinnitus (20).

The literature contains descriptions of a variety of psychological methods, including cognitive approaches aimed at affecting attitudes, thoughts, and beliefs. In combination with behavioral strategies, such as relaxation training and biofeedback, these methods form the basis of self-control or coping techniques. Methodological limitations, such as lack of proper controls or small samples, often preclude firm conclusions as to whether any treatment effect is due to specific factors. The therapeutic effect in itself is great in any treatment of tinnitus and patients often express relief when they see a professional who has more than 10 minutes for listening and reassurance (3).

Although patients suffer from tinnitus for relatively short periods of time, if they have catastrophic

thoughts such as 'they cannot escape from their tinnitus', 'tinnitus is a terrible disease', or 'they have no control over their tinnitus', they may have more emotional and functional distresses or become more anxious or depressed. Cognitive characteristics such as catastrophic thoughts in relation with tinnitus play an important role in distresses caused by tinnitus (21). Cognitive characteristics mediate between the severity of tinnitus and distresses from tinnitus, and between personality traits of patients and annoyances from tinnitus. Thus, cognitive interventions such as Cognitive Behavioral Therapy are useful for psychological adaptation (22).

A different study (23) concluded that younger subjects with TMD were more likely to report that their tinnitus improved than were older subjects. Most patients with TMD are between the ages of 20 and 40 years old, whereas most patients with tinnitus are between 40 and 80 years old (24, 25). It appears that the cause of the tinnitus in the younger patients in their study tended to be related to TMD, whereas the cause in older patients tended to be from another source. This study corroborated the findings of previous studies that suggested that patients with more severe tinnitus are less likely to experience improvement after TMD therapy (26). They also concluded that no single question or test consistently identified those patients with TMD and coexisting tinnitus, or who would experience improvement in, or resolution of their tinnitus. However, patients with TMD who have the greatest likelihood of experiencing tinnitus improvement would be those that are younger (the age range in this population was from 18 to 67 years old); their tinnitus occurs less frequently, that is, on a monthly basis rather than on a constant basis. Their tinnitus also lasts for a shorter time, that is, for seconds rather than continuously; their hearing is normal; they have pain in their ipsilateral ear; their tinnitus began approximately when their TMD symptoms began, their tinnitus is worse when their TMD symptoms are worse; their tinnitus appears to be related to stress; they experience changes in tinnitus (such as intensity) when they move their jaw; their tinnitus is not related to loud noise and their tinnitus is reproduced or intensified when they clench their back teeth as hard as possible for one minute (but they stop sooner if the pain becomes intolerable) (23).

According to Sanchez et al. (2), patients with tinnitus and normal audiometry represent a rare group. Clinical characteristics of tinnitus (duration of dis-

ease, location, type, frequency of onset) in these patients are similar to those in subjects with tinnitus and hearing loss. However, the interference caused by the concentration and emotional balance was significantly lower in their study, which did not occur in relation to sleep and social activity interference.

In a recent review (1), the authors commented that massage and stretching of the neck and masticatory muscles have been associated with significant improvement in tinnitus. Patients with somatic tinnitus can have symptoms of cervical spine disorders, including head, neck, and shoulder pain as well as limitations in sideways bending and rotation. Treating jaw and neck disorders have beneficial effects on tinnitus. Injecting lidocaine into jaw muscles, such as the lateral pterygoid, also reduces tinnitus.

Case report

Initial consultation 01/20/2010

A 68-year-old female patient presented to the dental clinic at Marquette University School of Dentistry with a chief complaint of persistent ringing in both ears. She had been experiencing ringing in her ears since October 2009, it started only in the right ear and now persisted in both. The ringing has continued to increase in intensity. The patient explained that when she moved her jaw the ringing was more intense but she was experiencing minimal pain in the TMJ. The ringing caused her to have trouble concentrating at work and interfered with her sleep. She initially went to her ENT doctor because of fluid buildup and ringing in her ears and they prescribed antibiotics, which eliminated the fluid but not the ringing. They went through a series of diagnostic tests and the ENT suggested that she see her dentist about the ringing in her ears. The patient's current medications at the 1st appointment included Lipitor, estrogen, and multivitamins. She also complained of infrequent fatigue or tenderness in the jaw muscles.

Physical exam

Her dental condition included missing teeth numbers 1, 15, 16, 17, 19, 32. Teeth #4 and #13 were rotated 360 degrees and #18 and #31 were lingually inclined. Abfractions were present on teeth # 5, 6,

11, 12 and incisal wear was present on maxillary and mandibular anterior teeth, which could be indicative of sleep bruxism. The patient was aware of her possible sleep bruxism. Her occlusion was found to be class 1 with #14 and #19, #4 and #29, #13 and #20 in posterior crossbite (Figure 1). The TMJ exam revealed a slight deviation to the right when opening and the patient reported an occasional clicking sound in her right joint but no pain. Maximum opening was found to be slightly limited at 35 mm. During digital palpation of the masticatory and cervical muscles, ringing could be increased with functional manipulation of the lateral pterygoid muscle. Lateral and posterior palpation of the TMJ could not elicit the tinnitus or pain. The diagnosis for this patient was myofascial pain (28) with associated tinnitus. Myofascial pain is a regional neuromuscular disorder characterized by the presence of trigger points that when stimulated can cause local and referred pain, but also autonomic symptoms, nausea, dizziness and tinnitus (27). There was no association with the TMJ itself, although there was a disc displacement with reduction on the right side.

Initial Treatment Plan 01/20/2010

It was determined that the best initial treatment was to fabricate an occlusal appliance and recommend the self-care and education program (or counseling), which would include some home physical therapy exercises and other behavior modifications. This program educates patients about their TMD condition, including an explanation about possible contributing factors and the teaching of the resting postural position of the mandible (teeth apart, lips slightly touching and tongue not pushing against the teeth). For more detailed information, please refer to references 28 and 29. She was also instructed to perform simultaneous bilateral mastication and



Figure 1 - Patient's occlusion

not to load any TMJ or masticatory muscles, including the masseter and lateral pterygoid (28). She was asked to apply heat to the masticatory muscles, using a gel pack, up to three times a day for 15 minutes. Stretching exercises for the masticatory elevator muscles were also included in the program. Although she had poor sleep quality and it was recommended that a preventive medication (cyclobenzaprine 10 mg at bedtime) would help with her sleep patterns and relaxation of the muscles (29), she refused to take medications and wanted to try just the oral appliance, physical therapy exercises and the behavioral modification recommendations.

1st follow-up 2/15/2010

A hard acrylic mandibular occlusal appliance (Figure 2) was fabricated and adjusted so that there were even and simultaneous occlusal contacts on all posterior teeth, with less contacts in the anterior teeth (28). The patient was instructed to wear the occlusal appliance every night and all day during the first two weeks of treatment, except for eating. The patient will wear the appliance daily during the first two or three weeks because we want to take the advantage of the appliance as a "behavior reminder" to the patient, making her more aware of the possible parafunctional habits that she can engage in during the day, including clenching, nail biting, lip biting or jaw play (bringing the jaw forward or to the sides, without tooth contact).

2nd follow-up 3/03/2010

After two weeks of wearing the appliance daily and performing the self-care management exercises, she reported a 70% improvement in the ringing in her ears.

3rd follow-up 8/4/2010

The patient reported a recent increase in tinnitus symptoms. The ringing was worse when she protruded her jaw and when the lateral pterygoid was palpated (using functional manipulation). It was recommended that the patient comply with the self-care management based on heat therapy, muscle stretch-



Figure 2 - Mandibular occlusal appliance

ing, and mandibular rest position exercises. Patient confirmed that she was not doing the exercises or using heat anymore. This is a common finding when managing TMD patients. Most of them decrease the number of times they stretch the muscles or apply heat as they get better. The patient was also instructed to wear the occlusal appliance all day for one week.

4th follow-up 9/15/2010

The pain in the lateral pterygoid was minimal since she has been doing the stretching exercises. The patient was told that she needs to exercise more, since it could improve her symptoms without prescribing medications. She confirmed that she has the oral parafunction of jaw play, which is one of the causes of her tinnitus. The patient needs to wear the occlusal appliance as much as possible during the day. She did confirm that she was wearing the occlusal appliance every night and during the day at work for the past two weeks. She caught herself clenching her teeth when using the appliance during the day. She also performed the muscle stretching exercises with heat three times daily for the past six weeks. The pain in the lateral pterygoid muscle was minimal since she began doing the stretching exercises, and the ringing had decreased about 80% since her last visit, and was not bothering the patient anymore.

5th follow-up 10/6/2010

At this appointment, the patient had no myofascial pain and the tinnitus was subdued but occa-

sionally bothered her. She explained that the ringing was 90% better than when she first started coming in for treatment. She will continue to do stretching exercises and wear the occlusal appliance during the day as needed. The patient was informed that medications would be the next treatment option.

6th follow-up 10/25/2010

Patient was aware of the relationship of tinnitus and her oral habits such as clenching and jaw play. She was educated about the possible recurrence of tinnitus if she does not control these parafunctional oral habits. She was happy with the treatment, especially because she now had control of her tinnitus and knew the main causes of it. A 90% decrease in the tinnitus was achieved and it was no longer affecting her sleep or daily activities. She was scheduled for a follow-up after 8-10 months.

Conclusions

In this case, we could manage the tinnitus with treatment directed towards a diagnosis of TMD associated with myofascial pain. No invasive therapy was used or needed, such as occlusal adjustments, although the patient had malocclusion. The patient was educated about her problem, so even with a recurrence of tinnitus she would know what to do and how to control it. This is very important in order to decrease anxiety and stress, since this can be also related to tinnitus (1, 19, 23). No medications were used during the treatment because the patient was reluctant to take them.

The recurrent tinnitus could always be associated with factors related to the self-care management program. This would happen when the patient was engaging in oral parafunctions during the day, such as clenching and jaw play, and not doing the stretching exercises and applying heat.

Common symptomatology is frequently observed in otic symptoms and temporomandibular disorders during daily practice; this should be understood by each discipline from a broad, anatomical and clinical perspective. As previously discussed, tinnitus often occurs as a result of insults to the ear, such as noise exposure or administration of specific pharmacologic agents. It can also be caused by ear or head

injuries, some diseases of the ear and ear infections. The identification of the triggering causes may be of more importance for selecting the most adequate pharmacological or other treatment modalities.

However, a multidisciplinary approach between otolaryngology, neurology, and dentistry is vital for establishing a differential diagnosis of the orofacial symptoms seen with TMD and a careful clinical evaluation to rule out the existence of any other pathology. Instrumental assessment can be helpful to show a more detailed diagnosis and evaluation of the symptoms resolution progress. As mentioned previously, the dentist can ask specific questions that would increase the chance to diagnose tinnitus that would respond positively to TMD management. Reproduction, change or increase in the tinnitus during palpation of the masticatory or cervical muscles, or even the TMJ, can also indicate a positive association between tinnitus and TMD.

References

1. Han BI, Lee HW, Kim TY, Lim JS, Shin KS. Tinnitus: characteristics, causes, mechanisms, and treatments. *J Clin Neurol*. 2009;5(1):11-9.
2. Sanchez TG, Medeiros IR, Levy CP, Ramalho Jda R, Bento RF. Tinnitus in normal hearing patients: clinical aspects and repercussions. *Braz J Otorhinolaryngol*. 2005;71(4):427-31.
3. Vesteraager, V. Tinnitus: investigation and management. *BMJ*. 1997;314(7082):728-31.
4. Coles RRA, Davis A, Smith P. Tinnitus: its epidemiology and management. In: Hartvig Jensen J, ed. *Presbycusis and other age related aspects*. Proceedings of the 14th Danavox symposium. Copenhagen: Danavox Jubilee Foundation, 1990.
5. Erlandsson SI, Hallberg LR, Axelsson A. Psychological and audiological correlates of perceived tinnitus severity. *Audiology*. 1992;31(3):168-79.
6. Komori E, Sugisaki M, Tanabe H, Katoh S. Discomaleolar ligament in the adult human. *Cranio*. 1986; 4(4):299-305.
7. Jastreboff PJ. Phantom auditory perception (tinnitus): mechanisms of generation and perception. *Neurosci Res*. 1990;8(4):221-54.

8. Ciancagalli R, Loreti P, Radaelli G. Ear, nose and throat symptoms in patients with TMD: the association of symptoms according to severity of arthropathy. *J Orofac Pain*. 1994;8(3):293-7.
9. Shore S, Zhou J, Koehler S. Neural mechanisms underlying somatic tinnitus. *Prog Brain Res*. 2007;166:107-23.
10. Lam DK, Lawrence HP, Tenenbaum HC. Aural symptoms in temporomandibular disorder patients attending a craniofacial pain unit. *J Orofac Pain*. 2001;15(2):146-57.
11. Ramirez LM, Ballesteros LE, Sandoval GP. Tensor tympani muscle: strange chewing muscle. *Med Oral Patol Oral Cir Bucal*. 2007;12(2):E96-100.
12. Marinis M, Santili V. Tinnitus in postherpetic neuralgia. *J Headache Pain*. 2010;11(1):83-4.
13. Cima R, Joore M, Maes I, Scheyen D, Refaie AE, Baguley DM, Rilana C, et al. Cost-effectiveness of multidisciplinary management of Tinnitus at a specialized Tinnitus centre. *BMC Health Serv Res*. 2009; 9:29.
14. Neri G, De Stefano A, Baffa C, Kulamarva G, Di Giovanni P, Petrucci G. Treatment of central and sensorineural tinnitus with orally administered Melatonin and Sulodexide: personal experience from a randomized controlled study. *Acta Otorhinolaryngol Ital*. 2009; 29(2):86-91.
15. Murai K, Tyler RS, Harker LA, Stouffer JL. Review of pharmacologic treatment of tinnitus. *Am J Otol*. 1992; 13(5):454-64.
16. Langguth B, Salvi R, Elgoyhen AB. Emerging pharmacotherapy of tinnitus. *Expert Opin Emerg Drugs*. 2009;14(4):687-702.
17. Sanchez TG, Balbani AP, Bittar RS, Bento RF, Camara J. Lidocaine test in patients with tinnitus: rationale of accomplishment and relation to the treatment with carbamazepine. *Auris Nasus Larynx*. 1999;26(4): 411-7.
18. Ramirez LM, Ballesteros LE, Sandoval GP. Topical review: temporomandibular disorders in an integral otic symptom model. *Int J Audiol*. 2008;47(4):215-27.
19. Write EF. Otologic symptom improvement through TMD therapy. *Quintessence Int*. 2007;38(9):e564-71.
20. Tulbberg M, Ernberg M. Long-term effect on tinnitus by treatment of temporomandibular disorders: a two-year follow-up by questionnaire. *Acta Odontol Scand*. 2006;64(2):89-96.
21. Lee SY, Kim JH, Hong SH, Lee DS. Roles of cognitive characteristics in tinnitus patients. *J Korean Med Sci*. 2004;19(6):864-9.
22. Gudex C, Skellgaard PH, West T, Sørensen J. Effectiveness of a tinnitus management programme: a 2-year follow-up study. *BMC Ear Nose Throat Disord*. 2009;9:6.
23. Wright EF, Bifano SL. Tinnitus improvement through TMD therapy. *J Am Dent Assoc*. 1997;128(10):1424-32.
24. Meyerhoff WL, Cooper JC Jr. Tinnitus. In: Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WL, editors. *Otolaryngology*. 3rd ed. Philadelphia: Saunders; 1991. p. 1169-79.
25. National Institutes of Health Technology Assessment Conference Statement. Management of temporomandibular disorders. *J Am Dent Assoc*. 1996;127(11): 1595-606.
26. Bush FM. Tinnitus and otalgia in temporomandibular disorders. *J Prosthet Dent*. 1987;58(4):495-8.
27. Sanita PV, Alencar FGP Jr. Myofascial Pain Syndrome as a contributing factor in patients with Chronic Headaches. *J Musculoskeletal Pain*. 2009;1(1):15-25.
28. Alencar FGP Jr, Becker A. Evaluation of different occlusal splints and counseling in the management of myofascial pain dysfunction. *J Oral Rehabil*. 2009; 36(2):79-85.
29. Herman CR, Schiffman EL, Look JO, Rindal DB. The effectiveness of adding pharmacologic treatment with clonazepam or cyclobenzaprine to patient education and self-care for the treatment of jaw pain upon awakening: a randomized clinical trial. *J Orofac Pain*. 2002;16(1):64-70.

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