

Treatment and follow-up of referred otic symptomatology in 23 patients with diagnosed temporomandibular disorders

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Abstract

Reports of the cause and effect relationship between temporomandibular disorders and orofacial-otic symptoms are polemic. The current research had two aims: to explore the possible link between temporomandibular disorders and otic-orofacial referred symptomatology and to emphasize their diagnostic importance. One hundred and sixty-five patients with temporomandibular dysfunction and otic symptoms but without ear, nose, paranasal sinuses and throat pathology, were referred from an otolaryngology clinic to an orofacial pain centre. Twenty-three patients (20 females and 3 males, age range 14–66 years, median age 35 years) with otic symptoms and headache associated with temporomandibular disorders were selected as fulfilling the inclusion criteria for the research. In the follow-up, each patient received an intra-oral device and the treatment outcomes were evaluated monthly by visual analogue scales (VAS) for five months. During this time, a reduction of their symptomatology was observed with complete relief (headache and vertigo), partial symptom alleviation (tinnitus, otic fullness and otalgia) or without symptom alleviation (subjective hearing loss) in some patients. The time for resolution of symptoms varied with each patient. Team work (odontology-otolaryngology) and detailed evaluation criteria based on a completely structured interview and physical examination are necessary for the diagnosis and treatment of these symptoms, closing the wide breach existing between these health disciplines.

Key words: *tinnitus, otic fullness, vertigo, dizziness, otalgia, headache*>

Introduction

Temporomandibular disorders (TMD) are a sub-classification of muscle-skeletal disorders and include a wide range of orofacial conditions with multiple aetiologies. TMD mask a great variety of subjective signs and symptoms. These can be referred and isolated or combined and be from the temporomandibular joint (TMJ), the masticatory muscles, the cervical muscles and associated structures. These symptoms appear similarly in adults and in children (1,2). The prevalence of TMD is 1.5 to 2 times higher in women than in men (3,4). The aetiology of the TMD can be the resumption of macrotrauma and microtrauma. Bruxism (microtrauma) plays a meaningful role in TMD and orofacial referred symptoms, although many researchers have considered the association of bruxism and TMD inconclusive (5,6).

TMD symptoms can be primary and referred and can be expressed as otic symptoms, myalgia, TMJ arthralgia, facial pain, craniosinus pain and headache. The ear is one of the anatomical structures often involved with TMD patients, frequently identifying otological symptomatology. Among these, tinnitus, otic fullness, subjective hearing loss, vertigo and otalgia are noted more in patients with TMD than in the general population (7–16). Williamson (17) had to validate otological symptoms in patients with TMD, and reported that none of them had an otological disorder when examined by specialists (8).

Problems with the ear are absent in over 50% of the patients who express otic complaints (9). Lam et al. (10) confirmed that the prevalence of referred otic pain and other otic symptoms of non-otological origin in patients with TMD varies from 3.5% to 42%. The prevalence of tinnitus in patients with TMD seems to be higher than that found in the

general population (10,11). The frequency of tinnitus in patients with TMD varies from 33% to 76% (12–14). Epidemiological studies have shown that in an industrial society, 31% of all adults have had tinnitus at some time, 14.2% suffer from tinnitus often, and 2.4% have the worst degree of severity (15–17). The prevalence of dizziness (a sense of turning, spinning or rocking without balance) in patients with TMD ranges from 40% to 70% and that of vertigo (illusion of movement in space) from 5% to 40%. (18–21).

The relationship between TMD and headache is important. Vascular and tension headaches in TMD are common and highly associated since they share common nociceptive pathways (22,23). The concept that migraine is a primary vascular phenomenon (Wolf encephalic vasospasm) has been studied and does not have a firm foundation. This has opened research into the relationship with its aetiology among migraines and TMD. Night bruxism is found in 75% of the migraine patients who suffer these attacks (24–26).

In this research, we take the first step in finding the association between TMD and referred orofacial and otic symptoms which reinforces the importance of the clinical evaluation method in patients with TMD and craniofacial complaints using a synthesized sign and symptom diagnostic chart.

Material and methods

Study population

Patients from an otolaryngology clinic with otic symptoms without ear, nose, paranasal sinuses and throat pathology were sent to our centre. These patients were treated ultra-conservatively with NSAIDs, antivertiginous and muscle relaxant drugs and additionally self-care instructions one month before remissions. The patients were sent owing to the absence of any alleviation of their complaints measured by a VAS on a 1–10 scale.

Initial clinical evaluation

The patients were evaluated by a complete clinical and verbal questionnaire in relation to cranio-cervico-facial pain and otic symptomatology (Table I). This guide uses otic symptom diagnosis because of the multidimensional character of its symptomatology. Otic symptoms, such as otalgia, vertigo, otic fullness and hearing loss sensation were recorded and classified from subjective complaints by each patient based on a structured interview. TMD examinations (articular, muscular and associated structures) were performed according to the American Academy of Orofacial Pain (AAOP) guidelines (27). Headaches were diagnosed by a physician

according to the International Classification of Headache Disorders (28). TMD was diagnosed in the patients, and it was assigned to the questionnaire through a direct interview of the patient and prior to treatment procedures.

Patient selection

Twenty-three patients (20 females and 3 males, age range 14–66 years, median age 35 years) were selected according to the inclusion criteria of this study. The 23 patients were selected from 165 patients sent with ‘muscular compromise’, otic symptoms and headache from a private otolaryngology clinic. Patients were strictly selected by the inclusion criteria, ensuring no involvement of additional musculoskeletal and health variables that could influence results and disperse the focus of the study.

Inclusion criteria:

- Complete and fully erupted natural teeth (excluding third molars).
- Absence of intraoral pathology: mucous, peri-odontal, dental or osseous.
- Absence of orthodontic treatment or preceding treatment to TMD.
- Complaints of headaches.
- Patient verbal report of spontaneous or evoked pain or muscle fatigue in head and/or neck.
- Positive report of tenderness to muscle palpation in head and/or neck.
- Chronic symptomatology for at least six months.
- Combination of otic symptoms consisting of tinnitus (mild or moderate), otic fullness, subjective hearing loss, earache and vertigo.
- Absence of diagnosed deafness.
- Absence of medical or mental involvement.
- Not under treatment for: medical, audiological, odontological, psychiatric, physical therapy, acupuncture.
- Neither TMJ disorders, nor opening mouth impediment.
- Absence of skeletal growing disorders.
- Absence of cranial traumatic injuries and cervico-facial painful syndromes.

Exclusion criteria:

If, during the follow-up, the patients presented one of the pathologies whose absence was in the inclusion criteria, they were excluded. Not following the indications given during the follow-up, as well as abandoning the treatment, excluded the patients. However, this situation did not occur.

Table I. Diagnosis approach of otic signs and symptoms related to TMD.

Symptom	Characteristics	Signs presentation
Vertigo	Intensity	Mild (dizziness), moderate (spinning vertigo), severe (severe spinning vertigo).
	Presentation (d/w/m/y format)	Evolution (how long ago it started), frequency (paroxistic, constant), and duration (how long it is perceived: hours, minutes, and seconds).
	Evoked while:	Standing up, during vehicle transportation, head movement, chewing, headache, macrotrauma.
	Collateral effects	Nausea, vomiting, syncope, amnesia, temporary loss of sight, dismay, aura, headache.
Tinnitus	Intensity	Mild (only heard in silence), moderate (heard in light environmental sound), severe (heard during talking or moderate-severe environmental sound and interferences with sleep).
	Localization	Left ear, right ear, both ears.
	Objective type(true sound perceived)	Seconds ticking on a clock, blood flow pulse, clicking or low-pitched buzzing.
	Subjective type (false sound perceived)	Ring, tone, cricket, water running in the sink, river water, cascade water, motor, chickadee, bell, wind, air escaping.
	Presentation (d/w/m/y format)	Evolution (how long ago it started), frequency (paroxistic, constant), and duration (how long it is perceived: hours, minutes, and seconds).
	Evoked during:	Macrotrauma, head movement, forced eye closure, chewing, teeth clenching or grinding, posterior occlusion, left or right jaw movement, maximum mouth opening, jaw protrusion, right or left head turning, periauricular palpation, forehead counterforce, occipital counterforce, right or left temple counterforce, opening mouth counterforce, left or right jaw movement counterforce, muscle palpation. ----- <i>Muscle palpation* reaction by finger pressure response, graded arbitrary from 0 to 4: Grade 0 (no pain), Grade 1 (light pain reported verbally), grade 2 (spontaneous palpebral response pain), grade 3 (escape pain), grade 4 (escape, scream and cry)</i>
Otic fullness	Presentation (d/w/m/y format)	Evolution (how long ago it started), frequency (paroxistic, constant), and duration (how long it is perceived: hours, minutes, and seconds).
	Localization	Left ear, right ear, both ears.
	Collateral effects	Tympanic stretching sensation, popping, earache, patulous Eustachian tube.
Otalgia	Location	Left ear, right ear, both ears. Auricular, pre-auricular, post-auricular, diffuse.
	Presentation (d/w/m/y format)	Evolution (how long ago it started), frequency (paroxistic, constant), and duration (how long it is perceived: hours, minutes, and seconds).
	Evoked or triggering	During: speaking, yawning, swallowing, smiling, drinking, cold weather, hot weather, macrotrauma.
	Pain quality (paraesthesia associated)	Numbness, tingling, scratching, pulsation.
	Pain quality (disaesthesia associated)	Neuralgia, pricking, tickling, burning.
Subjective hearing loss	Presentation (d/w/m/y format)	Evolution (how long ago it started), frequency (paroxistic, constant), and duration (how long it is perceived: hours, minutes, and seconds).
	Localization	Left ear, right ear, both ears.
	Evoked	Environmental noise, macrotrauma.
	Character	Fluctuating presentation, progressive beginning, sudden beginning, paroxistic hyperacusis.

Muscles palpated: temporal, auricular (anterior, superior, posterior), masseter superficial portion, masseter deep portion, lateral pterygoid (functional measure), medial pterygoid, anterior digastric belly, posterior digastric belly, occipitofrontal, esternocleidomastoid, trapezius, semispinalis, splenius, levator anguli scapule, scalenus, inferior belly of the omohyoid, sternohyoid, superior belly of the omohyoid, thyrohyoid, middle constrictor of the pharynx, mylohyoid, genioglossus.

Treatment and follow-up

The patients were instructed not to take analgesic, anti-inflammatory, muscle relaxant, anti-vertiginous, oestrogen replacement, anti-conception, anti-depressant, anxiolytic, sedative-hypnotic, anti-migraine and anticonvulsive drugs during the course of the study.

After the clinical evaluation, each of the selected patients received a NTI-tss (K010876 patent) device (29–31). This device, a prefabricated plastic matrix, was rebased and adapted with acrylic material in accordance with the manufacturer's recommendations and patient's individual requirements. This device reduces chewing agonist muscular activity such as an anterior bite stop (32,33) comparable to Lucia's inclined plane (34), but also provides support for all the extreme excursive movements of the jaw on a flat disclosing element that permits contact only with the anterior teeth (central incisors), avoiding contact with canines, premolars and molars (35).

Treatment for each patient was to use the device while sleeping and all waking hours, except meals, obtaining the longest effect. The patients were evaluated at monthly intervals for five months. A visual analogue scale (VAS) was used in each control accompanying a clinical evaluation (36). The patients were instructed to estimate the degree of change on each of the studied orofacial symptoms over an individual VAS on a 1–10 scale.

The VAS is a method of direct parametric estimation, validated and widely used as a reliability measuring instrument for painful and non-painful symptoms. In each control the painful symptoms (otalgia and headache) and non-painful symptoms (tinnitus, otic fullness, subjective hearing loss and vertigo) were measured to compare the current

symptom with the original symptom. To avoid some potential inaccuracy in self-report, the original symptom characteristics (evolution, presentation, localization, intensity, type, frequency, duration, character and collateral effects) were recalled for each patient before they scored the monthly treatment change on a VAS scale.

Results

Twenty-three patients (20 females and 3 males, age range 14–66 years, median age 35 years) were selected according the inclusion criteria from 165 patients (97 females and 45 males, age range 12–75 years, median age 39 years). A general description of the patients is listed in Table II.

There was a major proportion of female to male patients with TMD and craniofacial symptoms. Each of these 23 patients measured every one of the symptoms presented to them by a VAS over a 5-month period. During this time, a reduction of their symptomatology was observed with complete relief observed (headache and vertigo) and partial symptom alleviation (tinnitus, otic fullness and otalgia) or no symptom alleviation (subjective hearing loss) in some patients.

A complete alleviation of symptoms for headache and vertigo was observed. In two patients hearing loss remained with no response to treatment. Tinnitus symptoms remained with three patients, with partial alleviation. One patient remained with otic fullness and earache, with partial alleviation. According to these results the cumulative percentage of relief for subjective hearing loss was 77.78%, for tinnitus 84.21%, for otic fullness 93.33%, for earache 93.34%, and 100% for headache and vertigo.

Table II. Patients' characteristics and distribution of initial symptoms.

	Male		Female	
	<i>n</i>	%	<i>n</i>	%
Sex	3	13.04	20	86.96
Initial symptoms				
T. headache	3	13	20	87
Vertigo	3	13.6	19	86.4
Tinnitus	3	15.8	16	84.2
Both	1	14.	6	85.7
Left	2	28.6	5	71.4
Right	0	0	4	100
Ootalgia	0	0	15	100
Otic fullness	3	20	12	80
M. headache	2	16.7	10	83.3
S. hearing Loss	0	0	9	100
Age	Average (range) 21 (14–31)	SD 8.8	Average (range) 37.6 (16–66)	SD 12.1

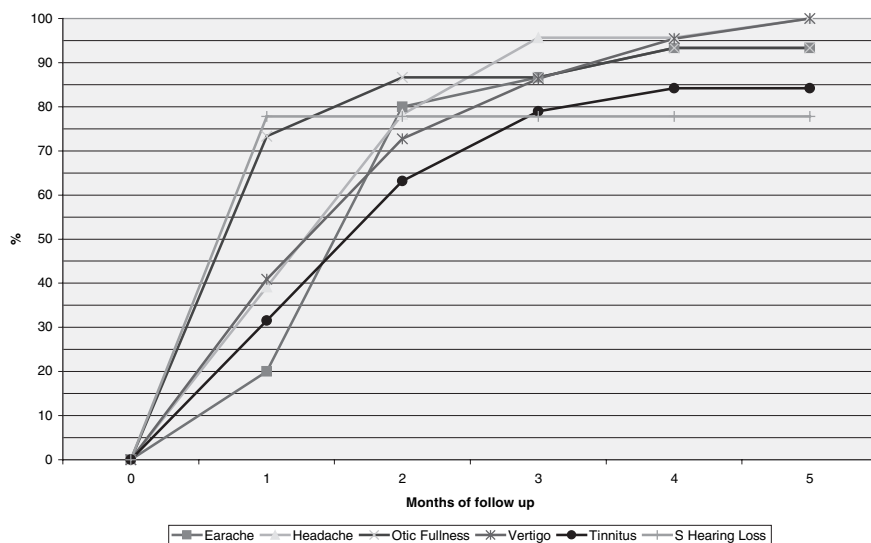


Figure 1. Cumulative percentage of relief.

The cumulative percentage of relief is present in Figure 1. 2 shows the individual reported relief of each symptom on a VAS during treatment follow-up.

Discussion

It must be recognized that the unique cause of otic symptoms such as tinnitus, vertigo, hearing loss, otalgia and fullness is still vague. These otic symptoms can originate in the hearing system (primary symptoms), but are habitually a symptom of an associated disease (secondary symptoms). Among several suggested diseases with origin in the ear that include psychological pathologies, viral cranial neuropathy, intracranial vascular anomalies (unstable blood flow), cerebrovascular disease, Menière's disease, ear and head trauma, chronic myringitis, impacted cerumen, infections, ototoxic drugs, acoustic neuroma, multiple sclerosis, noise exposure, otosclerosis and presbycusis, TMD also appears to play a relevant role. Also, in the aetiological explanation of otic symptoms, associated structures such as the pterygoid hamulus of the soft palate, the hypopharynx and larynx zone, the stylohyoid and stylomandibular complex, the temporalis tendon, the sphenopalatine ganglion, the paranasal sinuses, the thyroid, parotid gland and dental structures, as well as possible cervical lesions should be included in the exclusionary diagnostic approach. Tinnitus and vertigo, as well as other otic symptoms, have been implicated in patients with a compromised systemic condition such as systolic and diastolic pressure levels but with no correlation demonstrated (37,38). The aforementioned symptoms, tinnitus and vertigo, and otalgia may be mediated through the vagus nerve, driving the exploration of distal

conditions such as angina, tracheobronchial disease and aneurysmal dilatation of the thoracic arteries.

There are several possible pathophysiological explanations for the link between otic symptoms and TMD (39), although they are beyond the scope of this case report. This research shows a possible connection between the muscular component of the stomatognathic system and craniofacial symptoms due to the therapeutic alleviation of otic and muscular symptoms in a TMD group of patients. This probable link is established, also, for complete headache relief as a result of the 5-month treatment.

A discriminative history and thorough examination are fundamental to proper diagnosis. Table I categorizes clinical otic symptom in a TMD context. Each of these groups was subdivided by signs and symptoms that differentiate the otic disorders within it. This grouping category helps to establish a differential diagnosis with otic symptoms without a real cause in the inner or middle ear. This additional clinical information is vital in the taxonomy of the otic symptomatology related to TMD based on a structured interview and physical examination. It is not thought that this TMD-otic symptoms scheme explains pathologies such as Menière's syndrome, labyrinthitis or vestibular neuritis, among others, but attempts to assist in its differential diagnosis by exclusion.

There are homogeneous and easily recalled symptoms, such as pain, that are measurable with past experiences. Tinnitus, as hearing loss, otic fullness, and vertigo are difficult to measure when they are to be compared with past non-measured experiences. In relation to hypoacusis, Rubinstein (40) suggested that subjective complaints are not correlated with the objectively assessed level of hearing loss (41,42). In

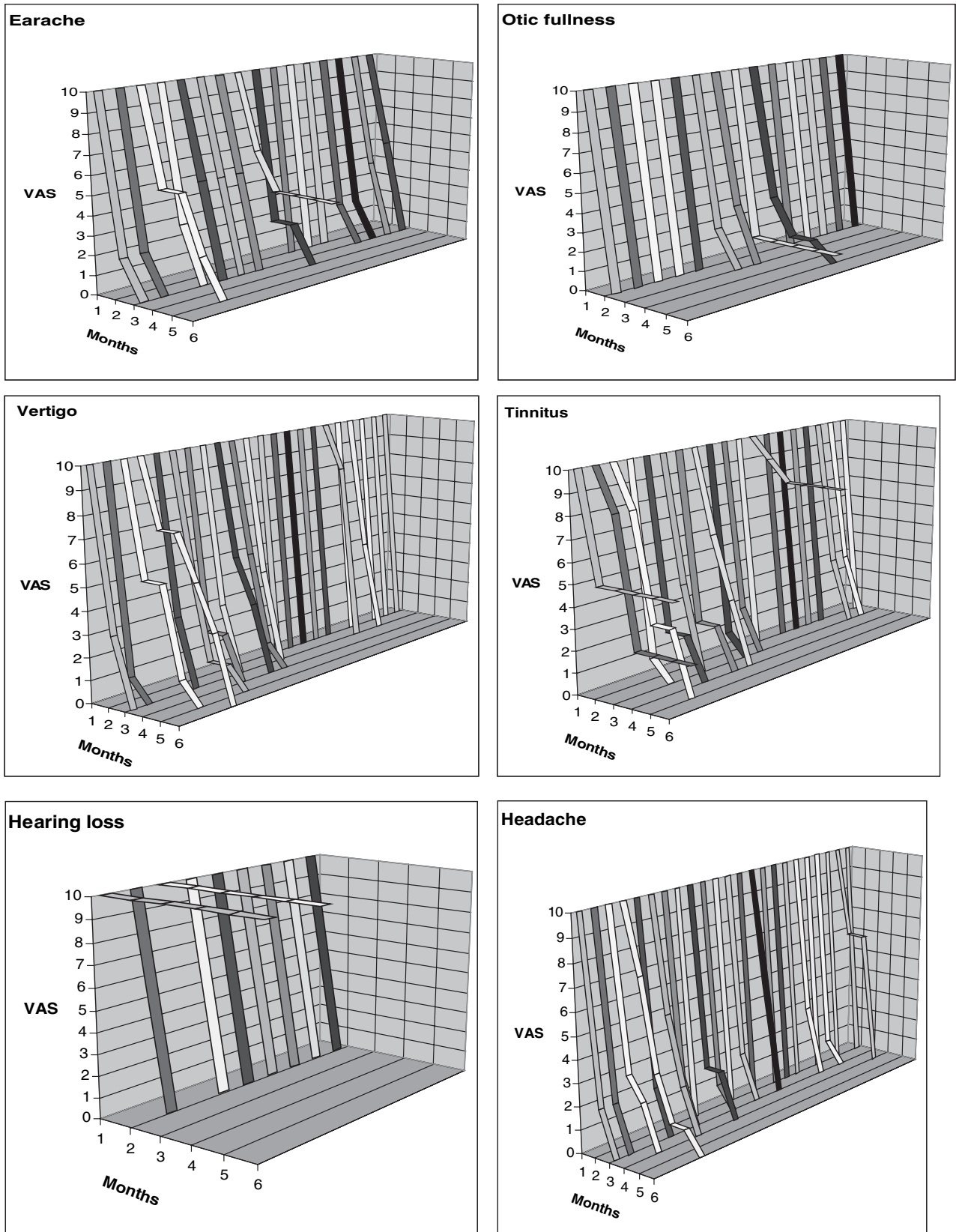


Figure 2a-f. Individual symptom relief on a VAS scale.

this research, these symptoms are semi-quantitatively measured on a VAS scale subsequent to the monthly treatment. The cognitive-emotional dimension of these symptoms perceived by a patient in the past is individual and non-reliable with prior experiences owing to their subjective interpersonal and intrapersonal perceptions. Among these otic symptoms, tinnitus is the most subjective because it cannot be measured or located in the tonotopic tract by electronic equipment. A mild tinnitus could be the worst experience for someone and a less significant incident for the same person at different times. There are several tinnitus questionnaires with high internal consistency and test-retest reliability, but they are created for an evaluation of past and present impediments that the tinnitus produces on the social, emotional, physical aspects in each patient owing to the level of annoyance generated by it (51–58). However, comparing current numerically-measurable tinnitus with past previous non-measurable experiences in relation to its impact on ‘daily living’ must be carefully observed due to its potential inaccuracy in self-reporting.

Otic symptoms such as tinnitus, hearing loss and vertigo can originate due to conductive or sensory-neural causes. The intra-oral device tends to diminish the tensor tympani and tensor veli palatini muscles that generate conductive otic symptoms during their hyperfunctional states. There were three tinnitus patients with partial resolution of their symptomatology and two hearing loss patients with absence of any relief of their symptoms. This can respond to a combined aetiology (conductive and sensorial) for tinnitus and a sensory-neural aetiology for hearing loss. The partial alleviation of earache (one patient) and otic fullness (one patient) appears to respond to more compromised dysfunctional muscular states owing to the fact that these patients have responded without a total remission of their symptoms.

Headache is significantly associated with TMD (found in up to 70% of patients with TMD) (52–59). Pain in the orofacial region frequently triggers headaches or ‘rebound’ headaches secondary to another condition, but there is a scenario in which TMD can be involved in the tensional-vascular headache pathogenic. TMD can be found in 66% of headache patients (53). Otological symptoms such as vertigo, sudden fluctuating hearing loss and tinnitus occur in migraine patients and a correlation has been found between Menière’s disease and migraine, although this link must be interpreted with caution (54). Most of the aetiological theories of migraine now include a trigeminal explanation – vascular, muscular or cortical – and recognize that the muscular tension originates in the pericranial

muscles such as chewing and neck muscles, with implications in the pathogenic of the cluster headache, and common and classic migraines since a peripheral muscle nociception tends to initiate them (55,56).

Limitations of the present study include small sample size, uncontrolled design and the absence of use of audiometry equipment. Otoacoustic emissions (OAEs), acoustic impedance and evoked potentials can be helpful to show a more detailed evaluation of this symptom’s resolution progress. Henderson (50) affirms that there is no correlation between subjects’ complaints (questionnaire responses) of reduced hearing sensitivity or discrimination and audiometric results (audiometric performance). Perception of hearing loss and decreased speech discrimination as well as the change in these symptoms after treatment were not mirrored in the audiograms. Abel and Levine (57) explained how OAEs cannot reflect the auditory perception involvement in all subjects, although some subjects had spontaneous OAEs yet heard nothing, and that the vast majority of subjects with spontaneous OAEs never hear them.

Conclusions

Bearing in mind the limitations of this study, the conclusions which this article can express are:

- The results of the study suggest a relationship between TMD, otic symptoms and headache.
- A population with characteristics similar to the one described can benefit from the treatment with NTI, but randomized studies are required to demonstrate their effectiveness.
- Imperative interdisciplinary management is necessary with previous otolaryngology evaluation.

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