RELATIONSHIP BETWEEN SOMATIC TINNITUS AND TEMPOROMANDIBULAR JOINT DYSFUNCTION SIGNS AND SYMPTOMS

ABSTRACT

Background and Aim: To investigate the possible relationship(s) between somatic tinnitus and temporomandibular joint dysfunction symptoms.

Subjects and Methods: This study was carried out on a total of 318 randomly-selected patients (mean age 44.35±16.38), who admitted to the Department of Oral Diagnosis and Radiology for routine dental treatment between November 2005 and 2008. 151 of the patients had tinnitus complaint, whereas 167 of them were symptom-free. The patients were evaluated for the use of prostheses, bruxism, otalgia, loss of posterior teeth and findings of temporomandibular joint (TMJ) dysfunction as a possible cause of tinnitus. The TMJ examination also included: TMJ sounds; TMJ pain during palpation; pain on masticatory muscles on palpation; and deviation and limitation on mouth opening.

Results: It was observed that tinnitus was significantly associated with age, bruxism, otalgia, loss of posterior teeth, pain in masticatory muscles, TMJ pain, and existence of TMJ sounds (p<0.05). On the other hand, no significant relationship was observed between tinnitus and the use of partial dentures, and deviation during jaw movements (p>0.05).

Conclusion: The results of this study demonstrate that TMJ symptoms and the complaint of somatic tinnitus should be taken into account during differential diagnosis.

Key words: Bruxism, Otalgia, Somatic Tinnitus, Temporomandibular Joint Dysfunction
INTRODUCTION

Tinnitus may be defined as an auditory sensitivity which occurs without any external stimulus. It is not considered as a disease, but rather as a symptom that may occur due to various causes such as; cochlear lesions, acoustic nerve diseases, exposure to high sound volume, atoxic medicaments, trauma arteriosclerosis, vascular diseases, cervical pathologies, ear infections, foreign body or plug in the helix, nasal allergy, temporomandibular joint (TMJ) disease and depression.\(^1\)\(^-\)\(^4\)

Despite several attempts to describe the mechanism(s) of tinnitus through various theories, the pathophysiology of tinnitus has yet to be elucidated.\(^5\) Through an epidemiological search, it may be observed that 31% of the affected adults may sometimes experience tinnitus, while 14.2% of them would experience the condition frequently, and 2.4% of them would experience it significantly.\(^2\) It has been demonstrated that tinnitus adversely affects the quality of life in 20% of the patients, and 60% of them have presented with symptoms of depression.\(^6\)

The relationship between TMJ and tinnitus have been initially defined by Costen,\(^7\) who suggested that the auriculotemporal branch of the trigeminal nerve and chorda tympani branch of the facial nerve are compressed due to the disposition of the mandibular condyle to the posterior. Consequently, dysfunction of the Eustachian tube and changes in tympanic pressure may occur, which will result in temporomandibular joint pathologies that are accompanied with tinnitus, otalgia, auditory insufficiency and aural fullness.\(^7\)

Today, the correlation between the TMJ diseases and tinnitus has been confirmed by many studies.\(^2\)\(^-\)\(^8\)\(^-\)\(^10\) Recent epidemiological studies show that complaints of tinnitus, the mastication system and cervical muscular system pain are accompanied with TMJ diseases.\(^3\)\(^1\)\(^1\)\(^2\)\(^1\)\(^2\) Further, degeneration of the TMJ disc is associated with tinnitus and masticator muscle pain, and is indicated as the TMJ dysfunction symptom.\(^13\)\(^-\)\(^15\)

It has also been shown that the frequency of tinnitus is high in patients with myofacial pain.\(^16\) Different approaches have been made to describe the correlation between the TMJ disease and tinnitus. Some of these studies have suggested that dysfunction of the craniomandibular system causes hyperactivity in masticatory muscles, and the increase in this activity, specifically in veli palatini and tensor timpani muscles may be the cause of tinnitus. Alternatively, the afferent somatic stimulation on the trigeminal nerve during bruxism may cause tinnitus by creating stimulation on the ventral cochlear nucleus and superior olivery complex.\(^17\) It has also been suggested that psychological stress induces both mechanisms.\(^18\) In light of previous studies, complaints of tinnitus can be classified in two basic categories with respect to its location: 1. Otic tinnitus that originates from the inner ear or acoustic nerve pathologies; and 2. Somatic tinnitus that originates from the head-neck pathologies other than the ear.\(^9\) Otic tinnitus may originate as a consequence of peripheral, central or peripheral-induced central event. Peripheral originated otic tinnitus is associated with the damage of the cochlear hair cell.\(^19\) It has been suggested that molecular movement and joint sound are increased upon removal of the streocilia from the tectorial membrane and this is perceived as tinnitus.\(^19\) According to another theory, the interruption of the normal, regular spontaneous afferent transmission of the cochlear nerve results in tinnitus.\(^9\)

Loss of the tonic afferent input causes removal of the inhibition in the acoustic structures of the brain stem and, consequently, the occurrence of tinnitus.\(^9\) Other theories describing otic tinnitus are localized on the neural sources of the central tinnitus or central neural dysfunction.\(^11\) With the advent of functional imaging techniques; the activation of tinnitus has been detected in many sections of the brain. Accordingly, central activation changes leading to tinnitus are associated with a number of areas from inferior colliculus to acoustic cortex, which are the other elements of the central auditory system on higher levels.\(^11\) Persistent and dense tinnitus, which is seen after complete incision of the eighth cranial nerve, is a consequence of the aberrant central neural activity. On the other hand, the tinnitus accompanied by acoustic trauma and age related hearing loss causes peripheral originated central damage, namely, the damage in the periphery induces central damage.\(^12\) A search of the literature reveals that the relationship between TMJ and somatic tinnitus has not been clarified. Hence, the aim of the present study was to investigate the possible relationship(s) between somatic tinnitus and complaints of the temporomandibular joint.

SUBJECTS AND METHODS

This study was carried out on a total of 318 randomly-selected patients who were admitted to the Department of Oral Diagnosis and Radiology for routine dental treatment between November 2005 and 2008. The mean age of the patients was 44.35±16.38 (13-84 ages). The patients were selected on a voluntary basis of having tinnitus complaints or not. All participants were white Caucasians, with no racial
or ethnic diversity among the study population. Emergency cases were not included, since such patients presented with chief complaints that could interfere with the proper diagnosis of tinnitus (i.e., trauma, acute pain and swelling). The dental and otorhinolaryngologic examinations of each patient were performed by a specialist in oral diagnosis and a specialist in otorhinolaryngology and audiology, respectively. Patients with underlying pathologies which may cause otic tinnitus (i.e., hearing loss, the use atoxic drugs, history otologic disease, ear surgery, and head and/or ear trauma) and those using full removable dentures were excluded from the study. The tinnitus complaints of 318 patients were recorded as “yes” or “no” without taking into account its direction or intensity.

Thereafter, the TMJ of patients was examined by a prosthodontist. The patients were evaluated for the use of prostheses, bruxism, otalgia, loss of posterior teeth and findings of TMJ dysfunction, as a possible cause of tinnitus. The TMJ examination also included: TMJ sounds (being classified as click, pop and crepitation, without the use of a stethoscope); TMJ pain during palpation; pain on masticatory muscles (temporal, masseter, medial and lateral pterygoid) on palpation; and deviation and limitation on mouth opening. A standard pressure was applied during palpation of the TMJ and masticatory muscles. Based on above findings, the patients with or without tinnitus complaints were further subgrouped according to the existence or absence of posterior teeth loss, bruxism, use of partial dentures, otalgia and TMJ examination findings.

All data were analyzed statistically using Statistical Package for the Social Sciences (SPSS, V.16, SPSS Inc. Chicago, IL, U.S.A.). Cross-tabulations, t-test and chi-square test were used for comparison of the data at significance level of p<0.05.

RESULTS

The statistical relationship among the studied parameters is presented in Table 1. Statistical analysis of the data showed that the frequency of somatic tinnitus significantly increased as the age of the patient increased (p<0.05). 151 of the patients had tinnitus complaint, whereas 167 of them were symptom-free. 58% of the patients were female, and 51.9% of them had tinnitus complaints. Among the male patients (42%), 40.6% had the complaint of tinnitus. The frequency of tinnitus were significantly in women than in men (cross-tabulation and chi-square test, p<0.05). 71.4% of the patients with otalgia had the complaint of tinnitus, while 28.6 % of them were symptom-free, leading to a significant relationship otalgia and tinnitus (p<0.05). 62.1%of the patients with posterior teeth loss had symptoms of tinnitus, yielding a significant relationship between the loss of posterior teeth and complaint of tinnitus (p<0.05). However, 37.6% of the patients without posterior teeth loss also presented with the same complaint. Likewise, 65.6% of the patients with TMJ sounds had tinnitus complaints, and thus, a significant relationship was observed between the existence of TMJ sounds and tinnitus (p<0.05). On the other hand, 39.9% of the patients without TMJ sounds also had tinnitus complaints. Within the study population, limited mouth opening was not observed. Among 18.6% of the patients with deviation on mouth opening, 50.8% had the complaint of tinnitus; while 46.3% of the patients without deviation had also tinnitus complaints. No significant relationship was observed between the deviation and tinnitus (p>0.05). 51.1% of the patients using partial dentures had the complaint of tinnitus, while 44.1% of those not using partial prosthesis also presented with the same complaint. No significant relationship was observed between the use of partial prostheses and tinnitus complaints (p>0.05).

57.1% of the patients with bruxism had the complaint of tinnitus, yielding a significant relationship between both conditions (p<0.05). Similarly, a significant relationship between masticatory muscle pain during palpation and tinnitus complaints was observed (p<0.05). 89.7% of patients with pain on TMJ during palpation had tinnitus complaints. A significant relationship was detected between TMJ pain and tinnitus complaint (p<0.05).

When all the data were pooled together, it was observed that tinnitus was significantly associated with bruxism, otalgia, loss of posterior teeth, pain in masticatory muscles, TMJ pain, and existence of TMJ sounds (p<0.05). On the other hand, no significant relationship was observed between tinnitus and the use of partial dentures, and deviation during jaw movements (Figures 1 and 2, p>0.05).

DISCUSSION

Attempts to describe the origin of somatic tinnitus have been made through neural, vascular and craniocervical theories. Initially, Moller et al.\textsuperscript{12} suggested that the lemniscal or non-auditory neural stimulations can induce tinnitus. Lockwood et al.\textsuperscript{20} utilized positron emission computerized tomography
Table 1. A summary of parameters investigated in the present study.

<table>
<thead>
<tr>
<th></th>
<th>TINNITUS</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>No</td>
<td>p value</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>40.29+ 15.83</td>
<td>48.85+ 15.85</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>36 %</td>
<td>47 %</td>
<td>0.030</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>64 %</td>
<td>53 %</td>
<td></td>
</tr>
<tr>
<td>Partial prosthesis</td>
<td>Use</td>
<td>51.1 %</td>
<td>48.9 %</td>
<td>0.130</td>
</tr>
<tr>
<td></td>
<td>No use</td>
<td>55.9 %</td>
<td>44.1 %</td>
<td></td>
</tr>
<tr>
<td>Bruxism</td>
<td>Yes</td>
<td>57.1 %</td>
<td>42.9 %</td>
<td>0.022</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>43.6 %</td>
<td>56.4 %</td>
<td></td>
</tr>
<tr>
<td>Otalgia</td>
<td>Yes</td>
<td>71.4 %</td>
<td>28.6 %</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>41.2 %</td>
<td>58.8 %</td>
<td></td>
</tr>
<tr>
<td>Posterior tooth loss</td>
<td>Yes</td>
<td>62.9 %</td>
<td>37.1 %</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>37.6 %</td>
<td>62.4 %</td>
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<tr>
<td>Masseter muscle pain during palpation</td>
<td>Yes</td>
<td>87 %</td>
<td>13 %</td>
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<tr>
<td></td>
<td>No</td>
<td>44.4 %</td>
<td>55.6 %</td>
<td></td>
</tr>
<tr>
<td>Temporal muscle pain during palpation</td>
<td>Yes</td>
<td>89.7 %</td>
<td>10.3 %</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>41.6 %</td>
<td>58.4 %</td>
<td></td>
</tr>
<tr>
<td>Medial pterygoid muscle pain during palpation</td>
<td>Yes</td>
<td>73.1 %</td>
<td>26.9 %</td>
<td>0.005</td>
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<tr>
<td></td>
<td>No</td>
<td>45.2 %</td>
<td>54.8 %</td>
<td></td>
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<tr>
<td>Lateral pterygoid muscle pain during palpation</td>
<td>Yes</td>
<td>75 %</td>
<td>25 %</td>
<td>0.001</td>
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<tr>
<td></td>
<td>No</td>
<td>44.4 %</td>
<td>55.6 %</td>
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<tr>
<td>TMJ pain during palpation</td>
<td>Yes</td>
<td>89.7 %</td>
<td>10.3 %</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>41.6 %</td>
<td>58.4 %</td>
<td></td>
</tr>
<tr>
<td>Deviation during jaw movements</td>
<td>Yes</td>
<td>52.5 %</td>
<td>47.5 %</td>
<td>0.314</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>46.3 %</td>
<td>53.7 %</td>
<td></td>
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<tr>
<td>TMJ sounds</td>
<td>Yes</td>
<td>65.6 %</td>
<td>34.4 %</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>39.9 %</td>
<td>60.1 %</td>
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</table>
(PECT) in order to describe the mechanism of somatic tinnitus, and observed a positive correlation between the orofacial movements, the blood flow in the temporal lobe and hippocampus. Levine proved the increased frequency of tinnitus following trauma of the head and neck.

Temporomandibular joint dysfunction (TMD) is a term comprising two groups of disorders; actual TMJ anomalies and primer involvement of the masticatory muscles (myofacial pain syndrome). Since it is difficult to discriminate between the latter two groups, it is hard to diagnose. TMD can occur due to trauma, bruxism, and loss of teeth, one sided mastication, degenerative joint diseases, ankylosis, developmental anomalies, myofacial pain syndrome and psychological reasons. Temporomandibular joint symptoms present with joint sounds, pain during TMJ and muscle palpations, limitation on mouth opening, deviation, chronic facial pain and head ache; or auditory symptoms. TMJ pathologies may present with tinnitus, otalgia, auditory insufficiency and aural fullness; as the Eustachian tube dysfunction and tympanic pressure changes occur. Following the Eustachian tube hypothesis of Costen, many researchers have tried to describe the relationship between tinnitus and TMD. According to the tensor tympani hypothesis, masticatory muscles and tensor tympani muscle
are innervated by the trigeminal nerve; and the hyperactivity of these muscles causes tinnitus.23

Previous studies have indicated the relationship between tinnitus and TMJ symptoms without any ear diseases.24,25 In this study, none of the patients had ear disease which might be the actual cause of tinnitus. Our results revealed a significant relationship between TMJ findings and somatic tinnitus.

The vagal and glossopharyngeal nerves directly innervate the inner, medial and external ear canal, while the trigeminal nerve accomplished this through the greater auricular nerve.26 The greater auricular nerve and the facial nerve also innervate the TMJ directly. The close anatomic connection shows the relationship between the neural structures, and the pathologies concerning the entire head and neck complex may cause symptoms on the ear.26

Johansson et al.27 compared tinnitus and disk displacement in the TMJ, and observed that frequency of the symptoms in the areas innervated by the trigeminal nerve was higher in patients with tinnitus than those without it. In the same study, it was found that the complaint of tinnitus is associated with pain complaints of the temporal zone and posterior eye.27 It was also demonstrated that displacement of the disk causes mechanical irritation of the auriculotemporal, lingual and, inferior alveolar nerves or motor nerve branches such as the masseter and deep posterior temporal nerve.27

Phylogenetic and embryogenic approaches suggest that there is a complex neuromuscular relationship between the masticatory muscles and the auditory system.23 This hypothesis has been generated on the theory that the tensor tympani and tensor veli palatine muscles, which function on the opening and closing of the Eustachian tube ostium, are in close relationship with the masticatory muscles during embryonic development. Both muscle groups are innervated by the motor branches of the trigeminal nerve. Consequently, the occurrence of the Eustachian tube dysfunction causes tinnitus.23

Reflex disorders of the tensor tympani and veli palatini muscles and the otomandibular ligaments (disco-malleolar and tympanomandibular) cause ear symptoms.24 Because of the otomandibular ligament, the ear symptoms concerning the temporomandibular joint occur due to the direct stimulation of malleus through the malleolar ligament.25 The present study clearly demonstrates the relationship between the TMJ, peripheral musculature, otalgia and bruxism.

According to the excessive somatic concern (polysymptomatic somatization syndrome) approach, TMJ dysfunction and tinnitus are associated with emotional disorders. Previous studies have shown that emotional stress may cause such complaints by means of somatization.26,28,29 Our study indicated that the pain on TMJ and masticatory muscles and symptoms of bruxism are associated with emotional stress, and thus tinnitus.

Lasiri et al.31 observed that the frequency of tinnitus increases with age and that persistent and chronic tinnitus is frequently experienced above the age of 60. Consistent with their findings, our results confirmed the influence of age on tinnitus, and further establish a significant relationship in an increasing trend.

The present results indicate a relationship between the TMJ, peripheral muscular structures, otalgia and bruxism. This relationship also suggests that the sensation of tinnitus and TMJ pain uses inter-related neural paths.

The results of this study demonstrate that TMJ symptoms and the complaint of somatic tinnitus should be taken into account during differential diagnosis. Nonetheless, the parameters used in the present study are subjective in nature, as with many other studies conducted so far. Further studies are required to elucidate the possible direct and indirect causes of TMJ symptoms and somatic tinnitus.

REFERENCES


