Clinical Study

Is It Necessary to Do Temporal Bone Computed Tomography of the Internal Auditory Canal in Tinnitus with Normal Hearing?

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Objective. To investigate the compression of the vestibulocochlear nerve in the etiology of the tinnitus in the normal hearing ears with temporal bone computed tomography scans. Methods. A prospective nonrandomized study of 30 bilateral tinnitus and 30 normal hearing patients enrolled in this study. Results. A total of 60 patients (ages ranged from 16 to 87) were included. The tinnitus group comprised 11 males and 19 females (mean age 49.50 ± 12.008) and the control group comprised 6 males and 24 females (mean age 39.47 ± 12.544). Regarding the right and left internal acoustic canals measurements (inlet, midcanal, and outlet canal lengths), there were no significant differences between the measurements of the control and tinnitus groups (P > 0.005). There was no narrowness in the internal acoustic canal of the tinnitus group compared with the control group. High-frequency audiometric measurements of the right and left ears tinnitus group at 8000, 9000, 10000, 11200, 12500, 14000, 16000, and 18000 Hz frequencies were significantly lower than the control group thresholds (P < 0.05). There was high-frequency hearing loss in the tinnitus group. Conclusion. There were no anatomical differences in the etiology of tinnitus rather than physiological degeneration in the nerves.

1. Introduction

Tinnitus is the perception of sound without an external stimulus. The prevalence of tinnitus varies between 3 and 30% of all population [1]. Tinnitus is a symptom of many diseases rather than a unique disease. Nonpulsatile form is more frequent in tinnitus. Tinnitus can originate from any part of the auditory system [2].

Most of the time the etiology of nonpulsatile tinnitus is not known. Hearing loss is the most frequent known etiology [3]. Symmetric hearing loss is observed in patients with tinnitus due to noise exposure. Although tinnitus is often seen in patients with hearing loss, it can also be seen in patients with normal hearing. For this reason it is not known whether tinnitus arises from the cochlea, the hearing nerve, or the central nervous system.

The severity of tinnitus varies from mild to severe and can be bad enough to interfere with a person’s daily activities even leading to distress, depression, and suicide by reducing the quality of life [4].

In order to find out the etiology of tinnitus, a good history, physical examination, radiological diagnosis, and audiological examinations are very important. Many systemic diseases such as hyperthyroidism, hypertension, and hypercholesterolemia are proven in the etiology, but the pathophysiology is still unknown [5].

As we know very well, tinnitus can be seen due to the pressure of the acoustic neuromas, cerebellopontine angle tumors, and vascular lesions, such as vascular loop to the eight cranial nerve reported in the literature. The development of the tinnitus can be observed due to nerve edema, degeneration, and compression in the canal. Accordingly, the
pathological conditions that affect the width of the canal can lead to tinnitus due to compression.

This study evaluated the diameter of internal acoustic canal in physiologically impaired tinnitus patients as the etiology may be due to anatomical differences of the temporal bone.

2. Material and Methods

This study was performed in 30 bilateral tinnitus patients who were referred to the outpatient clinic and 30 patients without any ear disease between 2011 and 2012. Microscopic ear examination and a complete audiological examination were performed. The study group had no symptoms and signs other than tinnitus. In the physical examination, they had normal external ear canal and tympanic membrane. Patients with normal hearing thresholds in audiometric tests at the octave frequencies of 250–4000 Hz were included in the study group. The patients with any ear complaints other than tinnitus such as chronic serous otitis media, chronic otitis media, trauma history, and external ear problems were excluded from the study. Also the patients with hyperlipidemia, hypertension, hyperthyroidism, and other systemic diseases which may cause vestibular toxicity were excluded.

Both tinnitus and control groups had high-frequency audiogram at 8000, 9000, 10000, 11200, 12500, 14000, 16000, 18000 and 20000 Hz frequencies. All patients had temporal bone computed tomography imaging. The internal auditory canal inlet, mid-canal, and outlet canal lengths were measured at the most distinctive cross-section of the seventh and eighth cranial nerves bifurcation (Figure 1). Patients who were admitted to our outpatient clinic other than ear disease with the temporal bone computed tomography results were taken as control group. Informed consent and ethical approval have been taken from all the participants. Measurements of internal auditory canal inlet, mid-canal, and outlet canal lengths were compared between the groups.

2.1. Statistical Analysis. In the statistical model, gender (male/female), age group, measurements of internal acoustic canal, and frequencies (250, 500, 1000, 2000, 4000, 8000, 9000, 10000, 11200, 12500, 14000, 16000, 18000, and 20000 Hz) were evaluated as the main factors. For statistical analysis, SPSS 17.0 V software was used to assess the findings of the study. Descriptive statistical methods (mean, standard deviation) as well as Student’s t-test for the comparison of quantitative data showing the parameters of the normal distribution were used for the determination of difference between the groups. The significance levels were set as \( P < 0.05 \) and 95% confidence interval.

3. Results

A total of 60 patients were included in this study. The ages ranged from 16 to 87. The tinnitus group comprised 11 males and 19 females (mean age 49.50 ± 12.008) and control group comprised 6 males and 24 females (mean age 39.47 ± 12.544) (Table 1). Tinnitus and the control group did not differ significantly by gender (\( P = 0.152 \)) (Table 2).

Regarding the right and left internal acoustic canals measurements (inlet, mid-canal, and outlet canal length), there were no significant differences between the measurements of the control and tinnitus groups (\( P > 0.005 \)) (Table 3).

Tinnitus group was evaluated according to internal acoustic canal measurements and there was no significant difference between the right and left canals (\( P > 0.05 \)) (Table 4).

High-frequency audiometric measurements of the right and left ear tinnitus group at 8000, 9000, 10000, 11200, 12500, 14000, 16000, and 18000 Hz frequencies were significantly lower than the control group thresholds (\( P < 0.05 \)). There was significant decrease at 20000 Hz frequency in the control group (\( P < 0.05 \)) (Table 5).

4. Discussion

Every nerve fiber has an electric discharge, even at rest, where this represents the spontaneous activity of the nerve. In patients with tinnitus, there is an increase in this spontaneous activity. As a result, hyperactive cilia or hyperactive nerve fibers may appear and the nerve fibers perceive sounds that cannot be heard under normal conditions within central auditory and nonauditory structures [6, 7]. This could explain the persistence of tinnitus after total hearing amputation.

Table 1: The mean age of the patients included in the study.

| Age    | Mean | Std. deviation | Std. error |
|--------|------|----------------|------------|
| Tinnitus group | 49.50 | 12.008 | 2.192 |
| Control group | 39.47 | 12.544 | 2.290 |

Table 2: The distribution by gender of the patients in the tinnitus and control groups.

| Male | Female | Total |
|------|--------|-------|
| Tinnitus | 11 (36.7%) | 19 (63.3%) | 30 (100.0%) |
| Control | 6 (20.0%) | 24 (80.0%) | 30 (100.0%) |
| Total | 17 (28.3%) | 43 (71.7%) | 60 (100.0%) |

Pearson Chi-square = 2.052; \( P = 0.152 \).
Table 3: Comparison of measurements of the internal auditory canal of the tinnitus and control groups.

| Group          | Mean  | Std. deviation | Std. error | t   | P*  |
|----------------|-------|----------------|------------|-----|-----|
| Right canal inlet | Tinnitus | 53,30          | 12,609     | 2,302 | −0,333 | 0,741 |
|                | Control | 54,37          | 12,235     | 2,234 |       |      |
| Right mid-canal | Tinnitus | 46,67          | 10,933     | 1,996 | 1,067 | 0,291 |
|                | Control | 43,93          | 8,800      | 1,607 |       |      |
| Right canal outlet | Tinnitus | 29,03          | 5,055      | 0,923 | 0,510 | 0,612 |
|                | Control | 28,00          | 9,879      | 1,804 |       |      |
| Right canal length | Tinnitus | 80,13          | 14,178     | 2,589 | −0,156 | 0,877 |
|                | Control | 80,70          | 14,042     | 2,564 |       |      |
| Left canal inlet | Tinnitus | 58,10          | 14,320     | 2,614 | 0,282 | 0,779 |
|                | Control | 56,93          | 17,546     | 3,203 |       |      |
| Left mid-canal | Tinnitus | 45,37          | 8,739      | 1,596 | −0,794 | 0,431 |
|                | Control | 47,13          | 8,496      | 1,551 |       |      |
| Left canal outlet | Tinnitus | 30,73          | 6,253      | 1,142 | −0,851 | 0,398 |
|                | Control | 32,73          | 11,255     | 2,055 |       |      |
| Left canal length | Tinnitus | 77,43          | 14,330     | 2,616 | −0,823 | 0,414 |
|                | Control | 80,60          | 15,453     | 2,821 |       |      |

*Independent samples t-test.

Table 4: The difference between measurements of the right and left internal acoustic canal.

| Group                      | Mean  | Std. deviation | t  | P*  |
|----------------------------|-------|----------------|----|-----|
| Right-left canal inlet     | 0,734 | 0,466          |    |     |
| Right-left mid-canal       | 0,509 | 0,613          |    |     |
| Right-left canal outlet    | −1,378| 0,174          |    |     |
| Right-left canal length    | −1,158| 0,252          |    |     |

*Independent samples t-test.

Tinnitus is classified as objective tinnitus (tinnitus can be heard) and subjective tinnitus which is perceived by the patient.

Today, the aim of the subjective tinnitus therapy is to increase the tolerance with sound enrichment or cognitive behavior therapy [8, 9]. Sound maskers, tinnitus-retraining therapy, and cognitive behavioral therapy are applied to relieve the symptoms caused by tinnitus. However, there is no exact solution in the treatment of tinnitus.

We investigate the anatomic reasons in the etiology of the tinnitus in the normal hearing ears. While the young population has usually normal hearing in the studies with tinnitus, hearing loss was found increasingly in the elderly. This also proves the nerve degeneration in the auditory pathways. But why are these not seen in everyone? So we evaluated the anatomy of the temporal bone by selecting patients without systemic diseases to find out if there was an anatomic difference in the tinnitus etiology.

Patients in the study group had bilateral tinnitus. Most of the patients cannot describe the localization, the time, the duration, and the severity of the tinnitus. Females were more in the tinnitus group, consistent with the literature. There was no statistically gender difference between the tinnitus group and selected control group accordingly (P > 0,05).

Vestibulocochlear nerve and the facial nerve enter the temporal bone through the internal auditory canal. The width of the internal acoustic canal varies from person to person. The etiology of tinnitus was influenced by many factors. But the pathophysiology is still unknown. Sometimes, all
due to compression [14]. Exocytoses and osteomata are cerebellopontine angle and may be presented with tinnitus in the internal auditory canal [12, 13]. This shows that nerve compression is effective in the etiology of tinnitus.

In cases of transverse fractures of the temporal bone, the labyrinth is involved more frequently than in longitudinal fractures. This may cause hearing loss and tinnitus [16]. There have been attempts to establish relation between vestibulocochlear nerve compression site and the character of symptoms. But there is still no definitive data [7, 17].

For this purpose, the internal auditory canal diameters (inlet, mid-canal, and outlet canal lengths) were evaluated in this study. We investigated whether narrowness in any of these locations may be the cause of tinnitus. The results showed that there were no significant differences in the measurements of internal canal between control and tinnitus groups (P < 0.05). Also comparison of the measurements within the tinnitus groups did not show any significant difference in the right and left internal acoustic canals (P < 0.05).

Normal hearing thresholds can be seen in tinnitus. However, normal hearing thresholds do not necessarily indicate the absence of cochlear damage. The state of cochlea can be judged with audiogram. Therefore, cochlear damage in study group was evaluated with high-frequency audiogram. There was a significant decrease in high-frequency audiometry at 8000, 9000, 10000, 11200, 12500, 14000, 16000, and 18000 Hz frequencies even if normal audiograms at 500, 1000, 2000, and 4000 Hz in the tinnitus group (P < 0.05). This showed neural degeneration rather than an anatomic variation or nerve compression in the internal auditory canal. Temporal bone tomography of tinnitus patients with normal hearing at the speech frequencies (500, 1000, 2000, and 4000 Hz) did not give any additional information.

Temporal bone imaging allows fine depiction of labyrinth abnormalities related to neoplastic, inflammatory, ischemic, degenerative, or traumatic disorders [13, 18]. Magnetic resonance imaging is the best for soft tissue masses and intracranial evaluation. However, this study shows that, in normal hearing patients with tinnitus, temporal bone imaging did not give any valuable information regarding tinnitus.

5. Conclusion

As a conclusion, if there is a complaint of tinnitus in patients with normal hearing, temporal bone tomography does not give us valuable information and it is not cost-effective to perform it. However, if tinnitus is accompanied by hearing loss, there may be underlying acoustic neuromas or Meniere's disease. Therefore, a CT or MRI can be performed in all hearing loss patients. It was concluded that tinnitus occurred in patients with normal hearing due to pathophysiological degeneration other than anatomical variations in internal acoustic canal.

Conflict of Interests

None of the authors have any conflict of interests.

References

[1] J. A. Henry, K. C. Dennis, and M. A. Schechter, "General review of tinnitus: prevalence, mechanisms, effects, and management," Journal of Speech, Language, and Hearing Research, vol. 48, no. 5, pp. 1204–1235, 2005.
[2] R. Schaette and D. McAlpine, "Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model," Journal of Neuroscience, vol. 31, no. 38, pp. 13452–13457, 2011.
[3] C. Nicolas-Puel, T. Akbaraly, R. Lloyd et al., "Characteristics of tinnitus in a population of 555 patients: specificities of tinnitus induced by noise trauma," International Tinnitus Journal, vol. 12, no. 1, pp. 64–70, 2006.
[4] R. Prestes and D. Gil, "Impact of tinnitus on quality of life, loudness and pitch match, and high-frequency audiometry," International Tinnitus Journal, vol. 15, no. 2, pp. 134–138, 2009.
[5] H. Kaźmierczak and G. Doroszewska, "Metabolic disorders in vertigo, tinnitus, and hearing loss," International Tinnitus Journal, vol. 7, no. 1, pp. 54–58, 2001.
[6] S. B. Lapsiwala, G. M. Pyle, A. W. Kaemmerle, F. J. Sasse, and B. Badie, "Correlation between auditory function and internal auditory canal pressure in patients with vestibular schwannomas," Journal of Neurosurgery, vol. 96, no. 5, pp. 872–876, 2002.
[7] C. J. Wuertenberger and S. K. Rosahl, "Vertigo and tinnitus caused by vascular compression of the vestibulocochlear nerve, not intracanalicular vestibular schwannoma: review and case presentation," Skull Base, vol. 19, no. 6, pp. 417–424, 2009.
[8] A. Londero and A. Chays, "Tinnitus treatment: neurosurgical management," Neurochirurgie, vol. 55, no. 2, pp. 248–258, 2009.
[9] H. Hesser, V. Westin, S. C. Hayes, and G. Andersson, "Clients' in-session acceptance and cognitive defusion behaviors in acceptance-based treatment of tinnitus distress," Behaviour Research and Therapy, vol. 47, no. 6, pp. 523–528, 2009.
[10] P. N. Patel, S. Connor, S. Brew, and M. J. Gleeson, "An arteriovenous malformation within the internal acoustic meatus and cerebellopontine angle cistern," Journal of Laryngology and Otology, vol. 125, no. 12, pp. 1275–1278, 2011.
[11] D. Sade, D. K. Lee, R. A. Prayson, G. B. Hughes, and J. H. Lee, "Intraosseous cavernous angiomia of the petrous bone," Skull Base, vol. 19, no. 3, pp. 237–240, 2009.
[12] H. Ryu, S. Yamamoto, K. Sugiyama, S. Nishizawa, and M. Nozue, "Neurovascular compression syndrome of the eighth cranial nerve. Can the site of compression explain the symptoms?" Acta Neurochirurgica, vol. 141, no. 5, pp. 495–501, 1999.
[13] A. K. Rohlf, R. Burger, C. Viebahn et al., "Uncommon lesions in the internal auditory canal (IAC): review of the literature and case report," Journal of Neurological Surgery. Part A, vol. 73, pp. 160–166, 2012.
[14] G. Maglìulo, F. Zardo, S. Bertin, R. D’Amico, and V. Savastano, “Meningiomas of the internal auditory canal: two case reports,” *Skull Base*, vol. 12, no. 1, pp. 19–26, 2002.

[15] F. M. Baik, L. Nguyen, J. K. Doherty, J. P. Harris, M. F. Mafee, and Q. T. Nguyen, “Comparative case series of exostoses and osteomas of the internal auditory canal,” *Annals of Otology, Rhinology and Laryngology*, vol. 120, no. 4, pp. 255–260, 2011.

[16] L. Heid, C.-F. Claussen, M. Kersebaum, E. Nagy, G. Bencze, and B. Bencsik, “Vertigo, dizziness, and tinnitus after otobasal fractures,” *International Tinnitus Journal*, vol. 10, no. 1, pp. 94–100, 2004.

[17] M. K. Schwaber and J. W. Hall, “Cochleovestibular nerve compression syndrome. I. Clinical features and audiovestibular findings,” *Laryngoscope*, vol. 102, no. 9, pp. 1020–1029, 1992.

[18] M. Kang and E. Escott, “Imaging of tinnitus,” *Otolaryngologic Clinics of North America*, vol. 41, no. 1, pp. 179–193, 2008.