



The incidence of vestibular disorders among the patients suffering from otosclerosis

MT Nasretdinova¹, KHE Karabaev²

¹ Samarkand Medical Institute, Department of Otorhinolaryngology, Uzbekistan

² Tashkent Pediatric Medical Institute Department of Otorhinolaryngology, Uzbekistan

Abstract

The aim of the investigation was to detect the occurrence of vestibular symptoms among patients suffering from otosclerosis, as well as their dependence on the form of otosclerosis. The study included 90 patients with a confirmed diagnosis of "otosclerosis" and the absence of concomitant diseases that can cause vestibular symptoms. Patients were interviewed and carefully examined. It was found that 16.7% of patients had vestibular asymmetry, which exceeds the rate of vestibular imbalance in the General population. The majority of patients with vestibular complaints and symptoms were in the group with unilateral sensorineural hearing loss, which, however, may not be a marker of unilateral vestibular deficit.

Keywords: otosclerosis, vestibular symptoms and complaints, dizziness, ataxia

Introduction

The prevalence of otosclerosis among the adult population is 0.3%, vestibular complaints (dizziness and imbalance of the body) are made by 10-15% of patients [1, 2]. At the moment the development of medical science the relationship of vertigo and otosclerosis is not completely proven; most authors expressed the view that the damage of the ampullar receptors enzymes released by osteosclerotic process in the cochlea of the so-called "cochlear" form of otosclerosis. The same process is the basis for the death of the hair cells of the inner ear and the appearance of the sensorineural component of hearing loss in this disease [2-5].

Since the middle of the XX century, when there was enough data on the mechanism of functioning of the vestibular analyzer and the possibilities of detecting its dysfunction, many attempts were made to link the development of vestibular symptoms with the progression of the otosclerotic process. The data of these studies are very contradictory: while in earlier publications the connection was traced quite clearly, later works contain more skeptical results. So, in 1966 B. McCabe included episodic dizziness in one of the mandatory symptoms when describing the syndrome of "otosclerotic inner ear" (otosclerotic inner ear syndrome) [6]. Similar data are present in many works of that time [7-9].

At the end of the XX century there was a evidential approach to the diagnosis of peripheral dizziness with a statement of the presence of asymmetry of the vestibular function. So, A. Morawiec-Bajda *et al.* in 2000, using the electronistagmographic method, a vestibular asymmetry was found in 18.8% of patients with otosclerosis in the absence of one in patients of the same age in the control group [10]. However, M. Toupet and *et al.* [11] indicate that in the population of patients with vestibular disorders, the percentage of patients with otosclerosis does not differ from the world population. The authors do not find signs of peripheral vestibular asymmetry even in the presence of monaural sensorineural hearing loss and typical for the true

vertigo complaints. These findings led them to conclude that there was no statistically reliable link between the otosclerotic process and dizziness.

This trend is associated with the discovery of new nosologies. So, benign paroxysmal positional vertigo at the moment is the main cause of recurrent episodes of rotational vertigo. Dehiscence upper semicircular canal helps to explain the relationship of hearing loss, tinnitus and dizziness. A more rigorous approach to the diagnosis of peripheral vestibular dizziness - recording the presence of vestibular asymmetry in vestibulometric studies - also reduces the percentage of patients with a diagnosis of true vertigo in patients with otosclerosis.

We conducted our own study on the prevalence of vestibular imbalance as a cause of dizziness in patients with otosclerosis. The aim of the investigation was to determine the prevalence of vestibular disorders in patients with otosclerosis; to identify the relationship between vestibulopathic syndrome, the presence of otosclerosis and its form (tympanal, cochlear).

Patients and methods

The study was conducted on the basis of Samarkand medical Institute at the Department of otorhinolaryngology. The study included 90 patients admitted for surgical treatment with the diagnosis "otosclerosis".

The criteria for inclusion in the study:

1. No other pathology of middle and inner ear that can cause a combination of hearing loss and vestibulopathies syndrome or be an underlying cause of dizziness (chronic middle purulent otitis, glamurnye tumor; benign paroxysmal positional vertigo, perilymphatic fistula, dehiscence upper semicircular canal, Meniere's disease, bilateral vestibulopathy the result of taking ototoxic drugs, etc.);
 2. Absence of signs of Central lesion of vestibular analyzer.
- The patients were divided into 3 groups depending on the type of hearing loss. In the 1st group (n=30) consisted of patients

with only conductive type of hearing loss. In the 2nd group (n=30) - with unilateral sensorineural hearing loss (increasing the threshold of sound perception over the bone above 20 dB at speech frequencies). The 3rd group consisted of patients (n=30) with bilateral sensorineural hearing loss. All patients were asked to complete a questionnaire indicating the characteristics of vertigo and trigger factors: the availability of atasia and ataxia, the presence of oscillopsia. We recommended that patients take for "dizziness" only episodic rotational sensation lasting from several minutes to several hours, which is typical for vestibular peripheral dizziness. From anamnesis it is known that no patient who participated in the study did not use ototoxic drugs. All examined persons underwent microscopy to exclude inflammatory processes and other pathology.

To confirm the presence of vestibular asymmetry and eliminate other causes of peripheral vestibular dizziness we used the following Protocol vestibulometric examination:

- The presence, direction, amplitude and the extent of sensible and latent spontaneous nystagmus;
- Research of the oculomotor reactions (with the exception of the Central causes of vertigo): the ability to slow object tracking, accuracy and speed of saccades, the presence of convergence, symmetry of movements of the eyeballs during extreme abduction of the eye, the lack of rebound of nystagmus. Patients with pathological test results are not included in the study;
- Test quick turn of the head (head-thrust test - HTT) is an indicator of the oppression of the Vestibulo-ocular reflex (VOR) and the functions of the vestibular apparatus on the side that you are turning heads;
- Test shaking heads (head-shake test - HST) is a test for inducing vestibular nystagmus in the presence of hidden (compensated) vestibular asymmetry;
- Study abnormalities of the body in posture Romberga;
- Fukuda test (step-test) - a test that reveals the presence of vestibular asymmetry when walking the patient in place with his eyes closed;
- Rotational test-quantitative evaluation of vestibular asymmetry;
- Caloric test - a quantitative assessment of vestibular asymmetry;
- Study of dynamic visual acuity (DOSES) - elimination of bilateral vestibular dysfunction;
- Study of subjective vertical-exclusion of otolith syndrome as the cause of vestibular complaints;
- Fistula symptom and a Valsalva maneuver - avoid the presence perilymphatic fistula and dehis-therfore, the upper semicircular canal as a cause of hearing loss and vestibular complaints;

Maneuver Dix-Hospice the exception of benign paroxysmal positional vertigo as the cause of vestibular complaints.

A quantitative assessment of the vestibular asymmetry, we considered only the difference in duration of induced nystagmus in excess of 20%.

The conclusion about the presence of peripheral vestibular imbalance was made on the basis of a set of test results.

The study relates to the 4th level (without an internal control group) with the category of proof of class C.

For comparison of parametric criteria between groups

(calorific and rotational tests) we used student's t-test and nonparametric criteria (all other samples) student's t - test. The value of p was determined by the table. As for all biomedical research, the value of $p < 0.05$ showed a statistically significant difference between the compared results with probability $> 95\%$ [1].

The data obtained by filling in the questionnaires and performing vestibulometric examination are given in the table. All patients with dizziness complaints and coordination disorders had evidence of vestibular asymmetry. In some patients, hidden vestibular asymmetry is detected, which is revealed only in provocative tests. However, a statistically significant difference between compensated and decompensated forms of vestibular imbalance has not been found, which confirms the inseparable link between the manifestation of symptoms of vestibular dysfunction and clinically detected asymmetry.

In our study, the prevalence of vestibular symptoms in patients with otosclerosis coincides with the data of the literature, amounting to 13.3%. In group 1 (tympanic otosclerosis), the percentage of patients with vestibular asymmetry and complaints differs significantly from other groups (3.3%), approaching the value found among healthy people (Φ 5%). Probably, the mechanism of development of sensorineural hearing loss in otosclerotic process is the basis of the same cytotoxic effect on the cells of the vestibular apparatus. Vestibular asymmetry in the 2nd and 3rd groups differed significantly from that in the 1st group ($p < 0.05$). The absence of differences between the 2nd and 3rd groups (monoaural and binaural cochlear forms) in the prevalence of vestibular complaints and asymmetry leads to the conclusion that unilateral sensorineural hearing loss is not a marker of unilateral lesion of the vestibular part of the same inner ear. The rate of damage of ampular receptors does not coincide with the rate of degeneration of hair cells in otosclerosis.

Table 1

Symptom parameter	1 st group	2 nd group	3 rd group
Episodes of true dizziness	2	1	2
Explicit spontaneous nystagmus	2	3	1
HTT test	9	7	9
HST- nystagmus	6	8	8
Deviation in the Romberg pose	2	3	1
The asymmetry of the reaction in a caloric test	8	5	6
Established vestibular imbalance	1	5	3
Total	30	30	30

As can be seen from the table, the most sensitive tests in assessing the vestibular function are the study of latent nystagmus (Frenzel glasses), HTT, HST, step test. To quantify the asymmetry, it is preferable to use bithermal caloric test. More common vestibular complaint is episodic peripheral vertigo, not a seizure disorders of body balance.

In 2 patients from group 3, signs of bilateral inhibition of the peripheral part of the vestibular analyzer with characteristic symptoms were found: oscillopsia, loss of DOSES and bilateral fallout of THIEVES. Despite the absence of a statistically significant difference on these grounds between the groups, it is logical to assume that long-term binaural

cochlear otosclerosis can lead to bilateral peripheral vestibulopathy, but it is not statistically proved due to a small sample.

Summary

1. The prevalence of complaints characteristic of peripheral vestibulopathic syndrome and clinically detected signs of peripheral vestibular asymmetry among patients suffering from otosclerosis is 13.3 and 16.7%, respectively. This figure significantly exceeds the average indicators in the healthy population, which indicates the relationship otosclerotic process vestibulopathies syndrome.
2. Unilateral sensorineural hearing loss is not a marker of unilateral lesion of the vestibular part of the same inner ear. The rate of damage of ampular receptors does not coincide with the rate of degeneration of hair cells in otosclerosis.
3. With the tympanic form of otosclerosis, the risk of developing a true vertigo does not differ from a healthy population, with the cochlear form, the probability increases regardless of mono - or biourality of sensorineural hearing loss.

References

1. Altman F, Glasgold A, Macduff JP. The evidence of otosclerosis as related to race and sex Ann ORL. 2007; 76:377-392.
2. Causse JR, Uriel J, Berges J. The enzymatic mechanism of the otospongiotic disease and NaF action on the enzymatic balance. Am J Otol. 2012; 3(4):297-314.
3. Causse J, Bel J, Michaux P, *et al.* Vertigo and osteospongiosis. Rev Laryngol Otol Rhinol Bord. 2012; 91(1):120-136.
4. Malik A, *et al.* Hypertension-related knowledge, practice and drug adherence among inpatients of a hospital in Samarkand, Uzbekistan //Nagoya journal of medical science. 2014; 76(3-4):255.
5. Mckenna M. Pathophysiology of otosclerosis Otol Neurotol. 2001; 22:249-257.
6. Schuknecht HF, Kirchner JC. Cochlear otosclerosis: Fact or fan-tasy. Laryngoscope. 2004; 84:766-782.
7. Kasimov S, *et al.* Haemosorption In Complex Management Of Hepatargia //The International Journal of Artificial Organs. 2013; 36(8):548.
8. Kucherenko VZ. Application of methods of statistical analysis. M: GEOTAR-Media, 2007, 134-142.
9. Piaget F, Charachon R, Michel JFR. Spontaneous vertigo and otospongiosis Otorhinolaryngol Audiophonol Chir Maxillofac. 2009; 18(5):315-318.
10. Toupet M, Grayeli AB, Sterkers O. Audiovestibular function in patients with otosclerosis and balance disorders. Otol Neurotol. 2009; 30(8):1085-1091.
11. Shambaugh GE. JR. The diagnosis and treatment of active cochlear otosclerosis J Laryngol Otol. 2011; 85:301-314.
12. Shamsiev AM. *et al.* Surgical treatment of septicopyemic form of acute hematogenous osteomyelitis in children Vestnik khirurgii imeni II Grekova. 2010; 169(6):51-53.