ORIGINAL ARTICLE

"Cochlear conductive hearing loss" in patients with Meniere's disease

Meniere hastalarında "koklear iletim tipi işitme kaybı"

Sertaç YETİŞER, MD., Mustafa KERTMEN, MD.

Objectives: Physiological studies with experimental models of endolymphatic hydrops in Meniere's disease demonstrate some pressure changes at the level of round and oval window. Interestingly, conductive type hearing loss was observed in some patients during Meniere episodes. A close auditory follow-up of these patients may provide a better understanding of hydrodynamic changes of the hydrops and micromechanics of the inner ear.

Patients and Methods: Eighty-four patients (mean age 34.3 years; range 17 to 77 years) with Meniere's disease were enrolled in the study. Duration of Meniere symptoms ranged between six months to 22 years (mean 6.2 years). Longitudinal follow-up of patients was planned for three years. Bone and air conduction thresholds were averaged and the differences between the thresholds were calculated.

Results: Twenty-four patients (28.4%) demonstrated air-bone gap at least at one frequency, the differences being 18.3, 11.8, 2.8, and 11.1 dB at 0.5, 1, 2, and 4 kHz, respectively. The average air-bone gap was 11.5 dB. The difference was the greatest at 0.5 kHz and the least at 2 kHz.

Conclusion: Low-frequency conductive hearing loss may be detected in Meniere's disease, which is apparently not indicative of middle ear pathology. High incidence of conductive involvement in patients who have had a recent episode of vertigo may indicate a distortion of vibratory movement of the stapes.

Key Words: Audiometry; endolymphatic hydrops; hearing loss, conductive; Meniere's disease/physiopathology.

Amaç: Endolenfatik hidropsun deneysel modellerindeki fizyolojik çalışmalar Meniere hastalığında oval ve yuvarlak pencere etrafında basınç değişimleri ortaya çıktığını göstermektedir. Bazı hastalarda Meniere atakları sırasında iletim tipi işitme kaybının gözlenmesi ilginç bir bulgudur. Bu hastaların yakından işitsel takipleri ataklar sırasında ortaya çıkan hidrodinamik değişimleri ve iç kulak mikromekaniğini daha iyi anlamamıza yardımcı olabilecektir.

Hastalar ve Yöntemler: Bu çalışma için 84 Meniere hastası (ort. yaş 34.3 dağılım 17-77) incelemeye alındı. Meniere hastalığının ortalama süresi altı ay ile 22 yıl arasında dağılım göstermekteydi (ort. 6.2 yıl). Hastaların üç yıl süreyle longitudinal takipleri planlandı. Kemik ve hava yolu eşiklerinin ortalamaları çıkarıldı ve eşikler arasındaki farklar hesaplandı.

Bulgular: Yirmi dört hastada (%28.4) en az bir frekansta hava-kemik yolu açıklığı gözlendi. Hava-kemik yolu aralığı analizinde sırasıyla 0.5, 1, 2, and 4 kHz frekanslarda 18.3, 11.8, 2.8 ve 11.1 dB fark görüldü. Ortalama hava-kemik yolu aralığı 11.5 dB idi. En büyük fark 0.5 kHz, en düşük fark 2 kHz'de idi.

Sonuç: Meniere hastalığında düşük frekans işitme kaybı görülebilir ve bu durum orta kulak patolojisiyle ilgili değildir. Bu durumun yakın dönemde vertigo atağı geçirmiş olan hastalarda sıkça izlenmesi stapes vibratuar mekaniğinin bozulması ile açıklanabilir.

Anahtar Sözcükler: Odyometri; endolenfatik hidrops; işitme kaybı, iletim tipi; Meniere hastalığı/fizyopatoloji.

Meniere's disease is known to induce sensorineural pathology with recruitment which fluctuates initially and then changes into permanent hearing loss progressively with the evolution of the disease.[1] However, audiograms of some patients suffering from Meniere's disease show an unexplained conductive component.[2] The cause of the hearing impairment associated with Meniere's disease is of course associated with the inner ear endolymphatic hydrops but the mechanism has not been fully understood. Proposed hypotheses are: endolymphatic overpressure, mixing of high K^+ endolymph with perilymph and flow of perilymph from scala vestibuli towards scala tympani with the mixing of the two perilymphs which are similar but not identical in composition.[3]

PATIENTS AND METHODS

This prospective study was conducted at Gulhane Medical School, Department of Otolaryngology, outpatient clinic. Eighty-four patients (mean age 34.3; range 17 to 77 years) with Meniere's disease who were followed for three years were included in the study. None of these patients had middle or inner ear surgery before. Duration of Meniere's symptoms in pastmedical history ranged between 6 months to 22 years (mean duration of symptoms; 6.2 years). Patients were subjected to analysis of past medical history, complete otolaryngological and neuro-otological examination including audiological (air and bone conductions for pure-tones of 125 to 8000 Hz, speech reception threshold, speech discrimination score, middle ear impedance, counterlateral and ipsilateral reflex measurement, otoacustic emission and ABR, if necessary), vestibular (electronystagmography, vestibulospinal tests etc.) and radiological (temporal bone computed tomography scanning) evaluation. Audiometric test battery included pure-tone audiometer (AC-30, IAC, Denmark), stapes reflex threshold and tympanometry (Amplaid 775, Italy). Patients with abnormal ear drum or auditory canal, history of draining ear and abnormal tympanometry (other than Type-A) were excluded. An informed consent was obtained from each patient.

Complaints of patients with Meniere's were reviewed and vertigo was staged according to the AAO & HNS, 1995 guidelines.[1] The frequency, duration, severity and type of vertigo were noted. Audiological test battery was reviewed at three-month-intervals or at episodes of vertigo. Conductive component was investigated at main frequencies (0.5, 1, 2 and 4 kHz) of pure-tone audiogram and was noted if there was any hearing loss during longitudinal follow-up of each patient. Bone and air conduction thresholds were then averaged and the difference between the thresholds was calculated. Aural fullness was particularly questioned and its relation to conductive involvement was investigated. The results were statistically analyzed with Pearson correlation analysis.

RESULTS

Patients were evaluated at three (35%) to six month (65%) intervals regularly, or whenever they had any vestibular problems. Twenty-four patients (29%) demonstrated air-bone gap at one or more than two frequencies at least at two interviews during follow-up. That is, the majority of them had basically neurosensorial type hearing loss with shifting to the conductive component on some occasion. Analysis of air-bone gap in patients with conductive hearing loss demonstrated that the difference was 18.3, 11.8, 2.8 and 11.1 dB at 0.5, 1, 2 and 4 kHz frequencies, respectively. The average air-bone gap in these patients was 11.5 dB. The difference was greatest at 0.5 and it was the least at 2 kHz. There was no clear correlation between the duration of the disease and the frequency of conductive involvement. However, 15 of these patients (62.5%) had an episode of Meniere's within two weeks prior to audiological analysis.

Twenty-six of 84 patients (31%) had aural fullness defined at least twice during the interviews (Those with a sense of aural fullness once or before were not included and those with aural fullness at the interview and audiological evaluation were noted) (Table I). However, when the connection between the sense of aural fullness and conductive hearing component was analyzed, no correlation was found since only six of the patients (7%) had both aural fullness and conductive type hearing loss. Twenty-one patients (25%) had variable (basically neurosensorial but conductive on some occasions) or fluctuant (sometimes better than before) hearing loss. However, only 11 patients had both aural fullness and fluctuant hearing loss.

DISCUSSION

Maintenance of homeostasis of inner ear fluids and biochemical integrity of the inner ear tissue are essential for proper functioning of auditory and

TABLE I

ANALYSIS OF MENIERE'S PATIENTS WITH LOW FREQUENCY HEARING LOSS AND AURAL FULLNESS

Meniere's patients		Patients with LF-CHL		Patients with aural fullness		Patients with both aural fullness and LF-CHL	
n	%	n	%	n	%	n	%
84	100	24	29	26	31	6	7

		Patients with Variable and fluctuant HL				Patients with both aural fullness and fluctuant HL	
n	%	n	%			n	%
84	100	21	25			11	13

HL: Hearing loss; LF-CHL: Low frequency conductive hearing loss.

vestibular end organs. The most prominent changes in temporal bone histopathology of Meniere's disease are hydrops-like extension of the endolymphatic spaces resulting with neurosensorial hearing loss. Endolymphatic space is larger and endolymphatic pressure is higher in hydropic ears.[4] Kawase et al.[5] measured the effect of pressure on cochlear microphonics (CM) in experimentally induced hydropic ears in the guinea pig and demonstrated that CM depression was greater at low frequencies, as the result of pressure due to mechanical effect on the intracochlear partition. Valk et al.[6] experimentally analyzed the effect of acute inner ear pressure changes on low-level distortion product otoacoustic emissions in guinea pigs and demonstrated that the reversible amplitude changes were closely related with the pressure changes which could be explained by a change in endocochlear stiffness. It is clear that an increase in endocochlear pressure not only changes the hydrodynamics but also affects the auditory function. However, conductive hearing loss is not common in the clinical course of Meniere's disease. Muchnik et al found that 32.5% of patients with Meniere's disease in their series had low frequency air-bone gap.[2] Longitudinal follow-up of patients with Meniere's disease demonstrated that this incidence was 29% in the present study.

It has been proposed that conductive hearing loss is the result of inner ear hyperpressure exerted against medial surface of the stapes footplate caused by an increase in endolymphatic volume since the fluids in the cochlea are incompressible. Stapes footplate mobility is only dampened and not fixed, thus stapedial reflex may still be elicited. Pressure changes in one compartment are immediately trans-mitted to the other. Gout et al.[7] analyzed multi-frequency impedence measurement in patients with Meniere's disease and found increased stapes annular ligament resonant frequency. Studies with experimentally induced hydrops in animals show that increase in static pressure of the endolymph lead to cochlear conductive loss.[8] The presence of a conductive component in a patient can be misinterpreted as a middle ear ventilation problem or an ossicular dismotility. It has been reported that some of the patients with temporal bone congenital abnormality such as large vestibular aqueduct syndrome and superior semicircular canal dehiscence syndrome may present with conductive type hearing loss simulating otosclerosis.[9-11] The hypothesis for the conductive hearing loss in a dehissent canal is that the third mobile window leads shunting air-conducted sound away from the cochlea elevating air conduction threshold or increasing the difference in impedance between the oval and round windows, thus improving the threshold for bone-conducted sound.

Low frequency involvement in Meniere's patients has been investigated. Horner claimed that the higher K^+ concentration arriving from the scala vestibuli into the scala tympani at the apex of the cochlea via the helicotrema is likely to be toxic to hair cell and auditory nerve fiber function. The mixing of the two perilymphs will result in the deterioration of low frequency sensitivity, provoke low frequency tinnitus and in the long term cause spiral ganglion cell degeneration at the apex of the cochlea. Whether hydrops begins at the apex is not clear, but it is apparent that the apical portion of the cochlea is initially affected in that way or another. The feeling of

fullness in the ear might be the result of the decreasing perilymph volume in the scala vestibuli which could give rise to inner ear conductive loss. However, no correlation was found between the presence of aural fullness and the incidence of low-frequency hearing loss in the present study.

Conductive hearing loss can be seen in some patients with vestibular problems.[12] Some of the patients with otosclerosis may present vestibular problems of some kind and at least 4% of those may have classical Meniere's symptoms. Cochlear hydrops in otosclerosis has been demonstrated before.[13] However, the causal relationship of these two entities remains controversial.[14-15] The low frequency involvement in patients with Meniere is somewhat different from otosclerosis and is basically related with alterations in pressure. This may be due to saccular hydrops extending to the stapes inner surface or increased hydrostatic pressure preventing stapes vibratory movement as a response to the auditory stimuli. In conclusion, we would like to point out to the low frequency conductive hearing loss in Meniere's, which is apparently not indicative of middle ear pathology, as demonstrated after close and longitudinal follow-up of patients with Meniere's, which can otherwise be simply overlooked.

REFERENCES

1. Monsell EM. New and revised reporting guidelines from the Committee on Hearing and Equilibrium. American Academy of Otolaryngology-Head and Neck Surgery Foundation, Inc. Otolaryngol Head Neck Surg 1995;113:176-8.
2. Muchnik C, Hildesheimer M, Rubinstein M, Arenberg IK. Low frequency air-bone gap in Meniere's disease without middle ear pathology. A preliminary report. Am J Otol 1989;10:1-4.
3. Horner KC. Old theme and new reflections: hearing impairment associated with endolymphatic hydrops. Hear Res 1991;52:147-56.
4. Ito S, Fisch U, Dillier N, Pollak A. Endolymphatic pressure in experimental hydrops. Arch Otolaryngol Head Neck Surg 1987;113:833-5.
5. Kawase T, Kobayashi T, Takasaka T, Shinkawa H. The effects of pressure on cochlear microphonics in experimentally induced hydropic ears in the guinea pig. Eur Arch Otorhinolaryngol 1990;247:364-7.
6. Valk WL, Wit HP, Albers FW. Effect of acute inner ear pressure changes on low-level distortion product otoacoustic emissions in the guinea pig. Acta Otolaryngol 2004;124:929-36.
7. Gout MC, Negrevergne M, Portmann D, Lahrizia R, Dauman R, Darrouzet V, et al. Multi-frequency impedance measurement and Meniere's disease. Analysis of the results of TEFLAG [Article in French] Rev Laryngol Otol Rhinol (Bord) 1999;120:317-21.
8. Horner KC, Cazals Y. Evolution of recruitment at different frequencies during the development of endolymphatic hydrops in the guinea pig. Arch Otorhinolaryngol 1988;245:103-7.
9. Nakashima T, Ueda H, Furuhashi A, Sato E, Asahi K, Naganawa S, et al. Air-bone gap and resonant frequency in large vestibular aqueduct syndrome. Am J Otol 2000;21:671-4.
10. Halmagyi GM, Aw ST, McGarvie LA, Todd MJ, Bradshaw A, Yavor RA, et al. Superior semicircular canal dehiscence simulating otosclerosis. J Laryngol Otol 2003;117:553-7.
11. Mikulec AA, McKenna MJ, Ramsey MJ, Rosowski JJ, Herrmann BS, Rauch SD, et al. Superior semicircular canal dehiscence presenting as conductive hearing loss without vertigo. Otol Neurotol 2004;25:121-9.
12. Yetiser S, Kertmen M, Yildirim A. An analysis of correlation between the unusual location of the jugular bulb and audiovestibular symptoms. Auris Nasus Larynx 2004;31:369-77.
13. Johnsson LG, Pyykko I, Pollak A, Gleeson M, Felix H. Cochlear vascular pathology and hydrops in otosclerosis. Acta Otolaryngol 1995;115:255-9.
14. Shea JJ Jr, Ge X, Orchik DJ. Endolymphatic hydrops associated with otosclerosis. Am J Otol 1994;15:348-57.
15. Paparella MM, Mancini F, Liston SL. Otosclerosis and Meniere's syndrome: diagnosis and treatment. Laryngoscope 1984;94(11 Pt 1):1414-7.