Sensorineural hearing loss associated with chronic otitis media

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Özet

Kronik Otitis Mediaya Bağlı Sensorinöral İşitme Kaybı

Bu çalışmanın amacı, kronik otitis medyaya (KOM) bağlı sensorinöral işitme (SNİK) kaybını değerlendirmek ve hastalığın süresi, kolesteatom, timpanoskleroz ve kemikcik hasarı gibi faktörlerin etkilerini tartışmaktır. Timpanoplasti uyguladığımız tek taraflı KOM'u olan 107 vakanın dosya kayıtları gözden geçirildi. Karşı taraf kulak kontrol kabul edilerek, hasta kulak ile karşı kulak arasında kemik iletim eşiği ortalaması 20 dB veya daha büyük olan vakalar SNİK olarak değerlendirildi. KOM'a bağlı SNİK 14 (% 13.1) vakada tespit edildi. SNİK'lı vakalarda hastalık süresi 14.3±9.5 yıl idi, olmayan olgularda 8.3±6.8 yıl idi. Kemikcik nekrozunun hem kemik iletiminde hem de hava kemik yolu aralığı üzerinde olumsuz etkileri mevcuttu. Kolesteatom sadece kemik iletimi üzerinde olumsuz etkileri vardı, hava kemik yolu aralığı üzerine olumsuz etkisi yoktu. Timpanosklerozun ise ne kemik iletimine ne de hava kemik yolu aralığı üzerine olumsuz etkileri mevcuttu. Kolesteatom, kemikcik nekrozu ve uzun hastalık süresi ile SNİK arasında bağıntı tespit edildi. Timpanosklerozun ise SNİK gelişiminde etkisi yoktu.

Anahtar kelimeler: Sensorinöral işitme kaybı, kronik otitis media, kolesteatom, timpanoskleroz

Abstract

The purpose of this study is to assess the association of chronic otitis media (COM) with sensorineural hearing loss (SNHL) and discuss the influence of the duration of disease, presence of cholesteatoma, tympanosclerosis and ossicular erosion on the development of SNHL. The records of 107 cases with unilateral COM who underwent tympanoplasty were reviewed, preoperatively. Serving the controlateral ear as control, if the difference between the average of bone conduction threshold (BCT) (0.5-4kHz) of the diseased ear and the controlateral ear was 20dB or more, these cases were evaluated as SNHL associated with COM. SNHL associated with COM was observed in 14 (13.1%) cases. The average duration of disease in cases with SNHL and in cases with no SNHL were 14.3±9.5 and 8.3±6.8 years, respectively. Ossicular necrosis has detrimental effect both on BCT and air-bone gap. Cholesteatoma decreases BCT but has no effect on air-bone gap. Tympanosclerosis has no effect either on BCT or air-bone gap. The presence of cholesteatoma, ossicular necrosis and longer duration of disease are associated with a significantly increased risk of SNHL. Tympanosclerosis has no effect on the development of SNHL.

Key words: Sensorineural hearing loss, chronic otitis media, cholesteatoma, tympanosclerosis

Introduction

Sensorineural hearing loss (SNHL) associated with chronic otitis media (COM) is not a well-known entity though it is fairly common. SNHL is reported to be the common sequela of COM, seen in 12-25% of cases (1-6). Hitherto, the role of chronic inflammatory disease of the middle ear as a cause of SNHL is not exactly defined.

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Paparella et al (7). points out that the semipermeable round window has critical role in the transfusion of toxic substances and advocates that the Corti organ injury occurs subsequently to the biochemical alteration. Similar results were reported by English et al. in an evaluation of 404 patients who had various degrees of COM (8). Vartiainen et al(9). as for the reason of SNHL suggest the occlusion of round and oval window owing to inflammation of the middle ear mucosa and stiffening of the ossicular chain In this retrospective study, we aim to assess the association of COM with SNHL using strict selection

criteria to minimize the contribution of other factors. In addition to audiometric evaluation, the influences of duration of disease, presence of cholesteatoma, tympanosclerosis and ossicular erosion on the development of SNHL were investigated.

Material and Method

One hundred and seven out of 177 cases who underwent tympanoplasty for COM in Ear Nose and Throat Department between June 1994-March 1999 were enrolled into this study. Thirty-one cases whose follow-up could not been done, 39 cases who had bilateral COM, were excluded from the study. To minimize the effect of sex, age, noise, ototoxic drugs and heredity, merely unilateral COM cases were included. Controlateral ears were evaluated as control. Sex, age, duration of disease, frequency of otorrhea and other related symptoms were recorded. Audiologic data were obtained for each ear by certified audiologists under sound proof conditions using standard audiometric equipment (AC 40 Clinical Audiometer), which was calibrated according to ISO standards routinely. All bone conduction testing was done with masking applied to the opposite ear. Preoperative audiograms were done 1-7 days prior to surgery, postoperative audiograms were done in 3-6 months following surgery. Operative records were examined to determine the extent and type of pathology of the middle ear in each patient. Serving the controlateral ear as control, if the difference between the average of bone conduction threshold (BCT) (0.5-4kHz) of the diseased ear and the controlateral ear was 20dB or more, these cases were evaluated as SNHL associated with COM. Statistical analysis were done by student-t test .

Results

Our study group consisted of 43 (40.2%) male, 64 (59.8%) female cases. The age ranges between 9 and 65 with a mean of 29.3±13.6.

SNHL associated with COM was observed in 14 (13.1%) cases. The average duration of disease in cases with SNHL and in cases with no SNHL were 14.3±9.5 and 8.3±6.8 years, respectively. There was statistically significant difference between them ($p<0,05$).

The overall BCT averages of the diseased and the control ears with respect to frequencies were shown in Table 1. For frequencies 0.5-4 kHz, there was statistically significant difference between the diseased and the control ear ($p<0.01$, $p<0.01$, $p<0.05$, $p<0.001$, respectively). The difference was more marked in 4000Hz.

Table 1 : Comparison of BCT averages of the diseased (with and without SNHL) and the control ears with respect to frequencies

Frequency Hz	Diseased ear Overall Mean SD (dB)	Control ear Controlateral Mean SD (dB)	P
0,5	13,3±7.3	10.0±4.9	<0,01
1	13,1±8,9	9.9±5.4	<0.01
2	14.6±10.4	9.2±4.6	<0.05
4	15.1±11.1	10.7±6.9	<0,001

BCT:Bone conduction threshold SNHL:Sensorineural hearing loss
SD:Standard deviation dB:Desibel

Comparison of BCT averages of the ears with SNHL(n=14) and without SNHL (n=93) in the diseased ears with respect to frequencies were shown in Table 2. There was statistical difference between the ears with SNHL and without SNHL in the diseased ears ($p<0,001$).

Table 2: Comparison of BCT averages of the cases with SNHL and without SNHL in the diseased ears with respect to frequencies

Frequency Hz	With SNHL Mean SD (dB)	Without SNHL Mean SD (dB)	P
0,5	23.9±12,0	12,0±5,8	<0,001
1	31.8±10.5	10.8±5,5	<0.001
2	37.1±12,3	11,9±5,8	<0.001
4	37.5±16,7	12,4±6,3	<0,001

BCT:Bone conduction threshold SNHL:Sensorineural hearing loss
SD:Standard deviation dB:Desibel

Comparison of BCT averages of the ears without SNHL and controlateral control ears with respect to frequencies were demonstrated in Table 3. There was no statistical difference between these two groups of ear ($p>0.05$).

Table 3: Comparison of BCT averages of the diseased (without SNHL) and the control ears with respect to frequencies

Frequency Hz	Diseased ear Without SNHL Mean SD (dB)	Control ear Controlateral Mean SD (dB)	P
0,5	12,0±5,8	10.0±4.9	>0.05
1	10.8±5,5	9.9±5.4	>0.05
2	11,9±5,8	9.2±4.6	>0.05
4	12,4±6,3	10.7±6.9	>0.05

BCT:Bone conduction threshold SNHL:Sensorineural hearing loss
SD:Standard deviation dB:Desibel

Cholesteatoma and tympanosclerosis were determined in 25 (23.3%) and 23 (21.5%) cases, respectively. Cholesteatoma and tympanosclerosis were observed

in 6 and 3 cases, respectively who had SNHL associated with COM. Cholesteatoma reduced the BCT, significantly ($p<0.05$)(Table 4). It was found that the presence of cholesteatoma did not influence air-bone gap ($p>0.05$). No defect in the cochlea or semisircular canals has been observed due to cholesteatoma. Tympanosclerosis did not influence BCT and air-bone gap. The ossicle necrosis impaired the BCT and increased the air-bone gap (Table 4).

Table 4: The averages of bone conduction threshold and air-bone gap regarding the existence of cholesteatoma, tympanosclerosis and ossicular necrosis.

	BCT SD (dB)	Air-bone Gap SD (dB)
Cholesteatoma -	13.3±7.6	25.0±12.0
Cholesteatoma +	16.0±7.3	26.0±18.8
P	<0.05	>0.05
Tympanosclerosis -	14.4±7.5	25.6±12.0
Tympanosclerosis +	11.9±7.8	24.2±11.6
P	>0.05	>0.05
Ossicular Necrosis -	12.2±6.9	22.5±11.3
Ossicular Necrosis +	17.5±7.8	30.9±11.2
P	<0.001	<0.05

BCT:Bone conduction threshold SD:Standard deviation dB:Desibel

Discussion

Impairment of sound conduction in cases with COM may be associated with the occlusion of round and oval windows by granulation, cholesteatoma and inflammed mucosa or stiffening of the ossicular chain. Both air and bone conduction may be influenced by these kinds of mechanical occlusions (3-9). According to Paparella et al (7). biochemical alterations effecting the cochlea via round window result in SNHL associated with COM. . Semipermeable structure of the membrane leads to passage of the toxic substance into cochlea. Biochemical changes occured in perilymph and endolymph, give rise to partial destruction of Corti organ. The anatomical characteristics of the round window are such as to encourage the accumulation, stagnation and absorbtion of purulent secretion into perilymph. Histological evidence showed that the round window changes were more marked in cases with purulent otitis media than in non-purulent otitis media. These changes especially basal turn hair cell lesions in acute purulent cases, could be temporary or permanent and render the ear more vulnerable to future damage (10). This theory seems to explain why depressed BCT are observed in some patients but not in others, despite apparent identical etiologies. Topical aminoglycoside also affect the inner ear via semipermeable round membrane.

We found that 14 (13.1%) had had SNHL associated with COM. The difference between the ears with SNHL and ears without SNHL in the diseased ears of COM patients were statistically significant ($p<0.001$). We also detected a statistically difference between all diseased ears and the control ears, though BCT of both groups were under the border threshold defined by WHO (<20 dB). It can be concluded that BCT may be impaired in cases with COM. It was postulated that the most commonly affected frequencies were of 2000-4000 Hz (11,12) In our study, we found that all frequencies were affected from COM, 4000 Hz was the most deteriorated frequency in consensus with literature. Paparella et al (13), Eisenman and Parisier (14) and Dumich (4) reported that the degree of SNHL is associated with duration of disease, the extent of histopathologic changes in the middle ear. We have found a significant difference regarding the duration of disease between cases with SNHL and cases without SNHL, in our study. We concluded that duration of disease is of critical importance for the development of SNHL.

Cusimano et al.(2), Levine et al (11) and Paparella et al (7) reported that SNHL is more commonly seen in cases with cholesteatoma. However, SNHL is not correlated with the extend of cholesteatoma. We found that cholesteatoma deteriorated BCT significantly ($p<0.05$), air-bone gap was not influenced markedly ($p>0.05$)(Table 4). Similar air-bone gap in both groups may be due to impaired air-conduction which developed parallel to impaired BCT established by cholesteatoma. It was concluded that cholesteatoma could be a sign of agresivity of COM and calamitous indicator for advent of SNHL.

Mechanical occlusion of the oval window may result in an increased bone conduction threshold with a resultant overestimation of relative SNHL. Also, it has been shown in an otosclerosis model in the cat that fixation of the stapes substantially reduces responses to bone conduction in the middle frequencies (11). It was reported that a middle ear bone conduction loss can be found in various disease states that produce a stiffness of the middle ear conducting system, such as otosclerosis and sequela of the otitis media (4). The location and density of sclerotic foci may be discrete in the ears. The effect of these foci may be associated with its location and density. In our study, we did not encounter with an evidence that tympanosclerosis would cause an increase in

BCT. There was discrepancy between our results and literature. We considered that this discrepancy might be originated from the location of sclerotic foci that did not influence the mobility of ossicular chain. SNHL in COM was caused by changes in the round window and abnormalities in the perilymph of the cochlea as a result of middle ear inflammation. Ossicular necrosis is a significant finding which shows that middle ear is posed to severe infection. It was demonstrated that ossicle necrosis led to increase in BCT and increase in air-bone.11 We observed that ossicle necrosis resulted in increase of BCT and air-bone gap. Our results corroborate the formentioned opinion.

In cases with COM, not only conductive hearing loss but SNHL can be seen, as well. BCT are affected by COM particularly at 4000 Hz. The presence of cholesteatoma, ossicular necrosis and longer duration of disease are associated with a significantly increased risk of SNHL. Therefore, cases with COM should be managed surgically as soon as possible.

Kaynaklar

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