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Superior Semisirküler Kanal Dehissansında Distorsiyon Ürünü Otoakustik Emisyon Ölçüm Değerlerinin Araştırılması

Investigation of distortion product otoacoustic emission values in superior semicircular canal dehiscence

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ÖZ

***GİRİŞ ve AMAÇ**: Süperior semisirküler kanal dehissansı vertigo ile başvuran hastalarda saptanabilen bir hastalıktır. Klinikte vertigo, otofoni,iletim tipi işitme kaybı, nistagmus saptanabilir. Otoakustik emisyon dış tüylü hücrelerin aktivitesine bağlı oluşan, koklea fonksiyonunun monitörizasyonunda kullanılan non-invaziv bir testtir. Biz çalışmamızda superior semisirküler kanal dehissansı olan hastalarda otoakustik emisyon değerlerini araştırdık.*

***YÖNTEM ve GEREÇLER**: Hastalar iki gruba ayrıldı. Tek taraf dehissans olan hastaların test kulakları(1.grup), karşı sağlam kulaklar ile, bilateral dehissans olan hastaların kulakları(2.grup), kontrol grubunun (3. grup) aynı taraf kulakları ile distorsiyon product ve sinyal-gürültü oranı ölçümleri yapılarak karşılaştırıldı.*

***BULGULAR**: Distorsiyon ürünü otoakustik emisyon ölçümü sonrası amplitüd değerleri ve sinyal gürültü oranı değerleri esas alınarak, 1-8 kHz' de değerler karşılaştırıldı. Tek taraf dehissans olan olgularda, karşı sağlam kulak ile test kulağı karşılaştırıldığında amplitüd ve sinyal-gürültü oranı değerlerinin test kulağında,sırasıyla 1 ve 6 kHz'de anlamlı olarak düştüğü saptandı ($p<0,05$). Bilateral dehissans olan ikinci grupta, test kulağı kontrol grubu ile kıyaslandığında, amplitüd ve sinyal gürültü oranı değerlerinin test kulağında tüm frekanslarda anlamlı olarak düştüğü saptandı ($p<0,05$).*

***TARTIŞMA ve SONUÇ**: Süperior semisirküler kanal dehissans saptanan hastalarda, kokleadaki dış titrek tüylü hücrelerin etkilendiği gözlense de otoakustik emisyonun tanıya katkısından söz edilemez.*

***Anahtar Kelimeler**: Superior semisirküler kanal dehissansı, distorsiyon ürünü, sinyal gürültü oranı*

ABSTRACT

***INTRODUCTION:** Superior semicircular canal dehiscence has been found in patients with vertigo symptoms. Patients may have otophonia,conductive hearing loss,nistagmus with vertigo.Otoacustic emission is a non-invasive test used for monitoring cochlea function due to outer hair cell activity. In our study we investigated otoacustic emission results in superior semicircular canal dehiscence patients.*

***METHODS:** Patients were divided into two groups. Patients with unilateral superior semicircular canal dehiscence were compared with the opposite normal side of their ear, using distortion product and signal-noise ratio values at 1-8 kHz. Patients with bilateral semisircular canal dehiscence were compared with control group patient's ears.*

***RESULTS:** Distorsion product otoacustic emission measurements and signal noise ratio values were significantly decreased at 1 kHz and 6 kHz in unilateral patients respectively($p<0.05$),and significantly decreased at 1-6 kHz in bilateral superior semicircular canal dehiscence patients($p<0.05$).*

***DISCUSSION AND CONCLUSION:** We showed the damage of the outer hair cell with emission measurements in superior semicircular canal dehiscence patients but we can not mention about its contribution to diagnosis.*

***Keywords:** Superior semicircular canal dehiscence, distorsion product, signal noise ratio*

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INTRODUCTION

Superior semicircular canal dehiscence (SSCD) is a peripheral vestibulopathy which although has been recently described, many studies have been conducted about it and is characterized by the absence of the bone on the superior semicircular canal (1). SSCD was described for the first time by Minor et al. (1). The incidence of SSCD has been reported as 0.7% by autopsy studies, and 1% by imaging studies in normal population without any complaint (2). It is diagnosed with determination of dehiscence of the bone on the superior semicircular canal (SSCC) on high-resolution computed tomography (CT) in patients with vestibular complaints due to voice or pressure changes or hearing loss (Figure 1). Vestibular membranous labyrinth is abnormally open in the middle cranial fossa, and the dehiscence functions like a third window (3). After the motion of stapes, acoustic energy propagates to the cranial cavity via conduction from the oval window to the cochlea. The dehiscence disrupts the voice conduction in the cochlea, causing conduction type or mixed type hearing loss (4). Endolymph motion combined with intracranial pressure alterations affect protruded membranous canal and subsequently the ampulla of superior semicircular canal in a retrograde way, causing vertigo and nystagmus through afferent vestibular nerve (5). Normal middle ear functions and normal acoustic reflexes were found in these patients (6). Dehiscence may be focal or diffuse. It has been reported that the size of dehiscence may be associated with symptom severity (3). If the symptoms are very severe surgical intervention may be considered, and and the dehiscence area is closed with fibrin glue, fascia, or bone plug (3). However, most of the patients with semicircular canal dehiscence reject surgery.

Otoacoustic emission (OAE) is an objective test technique reflecting functional status of the cochlea. These emissions occur due to the activity of the outer hair cells, and thus reflect only motor function of the cochlea. Otoacoustic emission is widely used in hearing screening of newborns and in the use of ototoxic drugs (7). Evaluation of otoacoustic emission in patients with semicircular canal dehiscence has been rarely reported in the literature. In this study, we aimed to investigate whether there were changes in distortion product otoacoustic emission (DPOAE) findings in patients with semicircular canal dehiscence.

MATERIAL AND METHODS

A total of 40 patients were included in this study with 27 patients who presented to ENT clinic due to complaints of vertigo between May 2007 and May 2009, had no any otologic complaint, and diagnosed as SSCD with high-resolution temporal computed tomography, and 13 healthy volunteers who had no any otologic complaint. The ethic approval was received from the local ethics committee before beginning of the study (29/11/2007 – 009/297).

Patients included in the study were informed about the study, and gave written consents. Detailed medical histories of the patients were received, and the patients were assessed with otolaryngologic examination. The patients were questioned about whether they had a history of trauma (physical trauma, barotrauma, acoustic trauma), and those with a history of trauma were excluded from the study. A full neurologic examination was carried out. The presence of vertigo due to sound and nystagmus (Tullio phenomenon), and dizziness due to pressure (Hennebert sign) was studied. Fine section temporal computed tomography of 1 mm section-interval was taken on the coronal and axial planes without using intravenous contrast agent in patients with clinical symptoms. Sagittal reformation was not made. Patients with opening at the longitudinal axis in the superior semicircular canals were included in the study. Absence of dehiscence in the control group was demonstrated with temporal CT. Patients with SSCD detected , were divided into two groups. Fourteen patients with unilateral SSCD were assigned to Group 1, and 13 patients with bilateral SSCD were assigned to Group 2. A total of 13 volunteers who were healthy according to the anamnesis, physical examination and imaging findings constituted the control group (Group 3). Weber and Rinne tests were performed. As a standard procedure, pure tone audiogram was carried out between 250 and 8000 Hz with air conduction, and between 250 and 4000 Hz with bone conduction. Speech discrimination scores were measured. Static acoustic admittance was measured with a tympanometry. Presence of

acoustic reflex was determined with narrow band at 80-100 dB at 1000 Hz. DPOAE was used to monitor the responses in the outer hair cells and specific frequency changes. DPOAEs were measured with Vivosonic Integrity device using a probe modified to fit with the external auditory canal. After proper position of the device was confirmed with proper indicator in the device and appropriate configuration of stimulus waveform, the measurement was started with this assembly. DPOAEs (2f1 – f2 cubic distortion product components) were measured at General Diagnostic mode. The ratio of f2 to f1 (f2/f1) was kept as 1.22. Stimulus intensity was taken as L1 for f1 frequency, and L2 f2. L1-L2 was kept at the level of 10 dB SPL (L1=65, L2=55). The results were expressed in the geometric mean of the primary tones (f1 and f2). OAEs were stimulated using two separate speakers for two stimuli (f1 and f2). DPOAEs were measured with a microphone inserted in the external auditory canal at a frequency of 2f1 – f2, and recorded in the geometric means of f1 and f2 at the frequencies of 1000, 1500, 2000, 3000, 4000, 6000, and 8000 Hz. DPOAE amplitude values of 3 dB higher than the noise threshold were considered significant. The measurement was carried out in a room where noise level did not exceed 50 dB.

Evaluation of DPOAE results was based on 2f1-f2 cubic distortion products, DPOAE amplitude values (DP) and signal to noise ratio (SNR) that occurred in geometric mean of f1 and f2, namely at frequency bands of 1000, 1500, 2000, 3000, 4000, 6000, and 8000 Hz. In our study these ratios were individually averaged for each patient. DP and SNR values were compared with the opposite normal ear in patient group with unilateral dehiscence, and with DP and SNR values obtained from the control subjects in patient group with bilateral dehiscence. DP and SNR values were separately averaged for each group, and frequency curves were plotted. At the end of the study, pure tone threshold avarage values and mean values of DPOAEs at 1-8 kHz frequencies obtained from the patients were tabulated.

Wilcoxon Test and Mann-Whitney test were used for statistical analysis of the data obtained. Our parameters were expressed as mean ± SD and supported with the tables. All analyses were carried out using SPSS 15.0 for Windows at 95% confidence interval. P values < 0.05 were considered statistically significant. No any treatment was scheduled for the patients because of the challenging surgical applications and rejection of treatment by the patients.

RESULTS

The patients with dehiscence detected in the SSC on temporal bone CT , aged between 26 and 61 (mean 47 ± 13.85). Of the patients, 24 were female and 3 male. Female to male ration was 8/1. The pathology was in a single ear in 14 patients with 11 being at the left and 3 at the right side. Dehiscence was bilateral in 13 patients. During physical examination, sight of the patients was deviated 20-30 degrees from the middle line, and eye related nystagmus was sought. Nystagmus was identified in 16 patients (59%). One patient (4%) developed vertigo in the test performed with applying pressure to the external auditory canal. Vertigo was increasing in 9 patients (33%) upon movements that increase intracranial pressure such as lifting heavy things and pushing, while 63% of the patients had sensation of imbalance, and 15% patients had history of vertigo with loud sound. Conduction type hearing loss (CTHL) was detected in the affected side of 8 patients (57%) with unilateral dehiscence. Hearing loss was marked at the frequencies of 500-1000 Hz. Air-bone gap was detected only in two of the patients with bilateral dehiscence at 1000 Hz frequency. Considering all patients, CTHL was detected by 37%. Carhart's notch was not found in any patient. Tympanometry was type A in all patients. All patients had acoustic reflex. Dehiscence between 4-9 mm on temporal bone CT was radiologically detected. None of the patients had otosclerosis or inner ear pathology other than SSCD on CT. Mean pure tone thresholds at 1-8 kHz was found as 14.64 at right and 17.76 in Group 1, and 17.44 at right and 17.3 in Group 2 (Table 1). DP and SNR outcomes of DPOAE at seven distinct frequencies are as follows:

Table 1. Mean 1-8 kHz pure tone thresholds.

	Right ear	Left ear
Unilateral SSCD	14,64	17,76
Bilateral SSCD	17,44	17,30
Control group	16,05	16,05

1.Results of the comparison of DP and SNR values for both ears in Group 1 patients (unilateral dehiscence) using Wilcoxon Signed-Rank test (two paired samples):

DP value was significantly low at 1 kHZ ($p<0.05$) (Table 2),

Table 2. Mean DP values in Group 1 with unilateral dehiscence

Frequencies (kHz)	Test ear DP values (n=14)	Control ear DP values (n=14)	P values
1	-2.34±9.36	4.02±7.13	**0.022**
1,5	1.77±8.78	4.29±7.76	0.177
2	-1.45±8.85	-1.47±10.18	0.826
3	-7.42±11.63	-4.6±10.54	0.510
4	-9.03±11.38	-6.34±11.32	0.397
6	-11.52±8.18	-7.0±9.27	0.096
8	-14.46±5.44	-14.92±11.09	0.925

SNR value was significantly low at 6 kHz ($p<0.05$) (Table 3).

Table 3. Mean SNR values in Group 1 with unilateral dehiscence

Frequencies (kHz)	Test ear SNR values (n=14)	Control ear SNR values (n=14)	P values
1	6.77±8.14	11.13±8.58	0.074
1,5	14.13±6.69	11.23±9.41	0.331
2	11.44±9.48	10.79±10.81	0.551
3	11.44±7.34	12.64±8.03	0.975
4	9.84±9.93	11.38±9.76	0.730
6	11.03±7.02	15.38±5.94	**0.009**
8	10.66±6.01	9.93±11.04	0.925

SNR, Signal to noise ratio

2.Ill right ears were compared with the healthy right ears of the control group, and the ill left ears with the healthy left ears of the control group in Group 2 patients (bilateral dehiscence) using Mann-Whitney test. Results of the comparison of DP and SNR values:

Decrease in DP values was significant for the right ears at all frequencies except for 1000 and 4000 kHz ($p<0.05$), and at all frequencies for the left ears in patients with bilateral dehiscence ($p<0.05$) (Tables 4 and 5).

Table 4. Mean DP values for the right ear in Group 2 with bilateral dehiscence

Frequencies (kHz)	Test ear DP values (n=13)	Control ear DP values (n=13)	P values
1	3.28 ±8.44	9.35±6.38	0.069
1,5	-1.60±10.65	9.69±9.95	**0.002**
2	-7.14±11.07	6.36±8.32	**0.001**
3	-8.69±13.35	4.46±9.04	**0.015**
4	-7.59±15.22	1.89±8.33	0.158
6	-6.5±10.83	3.84±8.72	**0.029**
8	-14.11±11.86	-0.44±11.13	**0.010**

DP, Distortion product

Table 5. Mean DP values for the left ear in Group 2 with bilateral dehiscence

Frequencies (kHz)	Test ear DP values (n=13)	Control ear DP values (n=13)	P values
1	0.65±5.7	10.53±7.03	**0.0**
1,5	-1.10±8.58	9.61±8.33	**0.002**
2	-4.45±8.75	8.57±6.77	**0.001**
3	-6.4±9.2	3.28±9.52	**0.013**
4	-7.42±11.4	2.79±9.08	**0.029**
6	-10.53±13.29	2.10±9.42	**0.020**
8	-12.9±9.16	-2.64±13.26	**0.033**

DP, Distortion product

Decrease in SNR values was significant for the right ears at 2000, 4000 and 6000 kHz frequencies ($p<0.05$), and at 2000, 3000, 4000 and 6000 kHz frequencies for the left ears in patients with bilateral dehiscence ($p<0.05$) (Tables 6 and 7).

Table 6. Mean SNR values for the right ear in Group 2 with bilateral dehiscence

Frequencies (kHz)	Test ear SNR values (n=13)	Control ear SNR values (n=13)	P values
1	14.94 ±8.27	14.5±8.42	0.701
1,5	13.95±6.49	18.68±8.51	0.091
2	9.55±11.64	17.98±8.64	**0.004**
3	12.13±11.99	18.95±4.79	0.191
4	13.26±10.31	19.87±5.52	**0.029**
6	15.53±8.66	22.2±2.95	**0.015**
8	12.27±11.12	18.06±8.41	0.106

SNR: Signal to noise ratio

Table 7. Mean SNR values for the left ear in Group 2 with bilateral dehiscence

Frequencies (kHz)	Test ear SNR values (n=13)	Control ear SNR values (n=13)	P values
1	12.03±5.46	13.86±6.97	0.427
1,5	12.74±9.17	17.0±8.29	0.118
2	11.54±8.49	19.77±6.06	**0.002**
3	10.57±9.98	19.81±5.56	**0.010**
4	11.9±9.59	19.69±5.74	**0.022**
6	11.27±9.57	20.56±3.49	**0.026**
8	10.96±8.30	15.29±8.96	0.174

SNR: Signal to noise ratio

DISCUSSION

SSCD is a pathology which has been described for the first time by Minor et al., causing signs and symptoms related to hearing and balance impairment, and is characterized by open vestibular membranous labyrinth in the middle cranial fossa (1). In a study, 1000 temporal bones were examined, and the incidence of dehiscence was found as 0.5%. In the same study, the bone on the middle fossa base and superior petrosal sinus was found to be thinner than 0.1 mm by 1.4% (8). Teixido et al. found SSCC in 35 of 6284 patients during 7 years with an incidence of 0.56% (9). However, the incidence of symptomatic SSCD patients is unknown (10).

In our study, the mean age was found as 47, and same value was found in a study by Hillman (4). Minor was found the mean age as 40 years in a study with 17 patients (11).

In various studies, female to male ratio was found close to each other with 4.5 in a study by Mikulec et al. (12), and 8 in our study.

Although Carey reported the bilateral dehiscence quite often (8), Hillman found the rate of bilateral dehiscence as 33% (4), Minor 23% (11), and Mikulec as 25% (12). In our study, rate of bilateral dehiscence was 48%. In the light of the data we obtained, we found that most of the dehiscences (78%) were at the left side in unilateral patients. However, we encountered with quite different rates in the literature (4, 11, 12, 13). In a study, bone wall found to be thinner in the other normal ear of patients with unilateral dehiscence compared with the controls. In addition, it was reported in the same study that the defect should be at least 2 mm of length for the dehiscence being symptomatic (14).

Absence of the bony tissue between middle cranial fossa and SSC causes changes in sound conduction and endolymph motion. This defect functions as a third window in the bone vestibular labyrinth, and make vestibular afferent fibers susceptible to sound and pressure stimuli (5, 11). Vestibular symptoms have been defined in some, and hearing related complaints in other patients, while asymptomatic patients have also been reported (8). In their study with 35 patients, Teixido et al. found the rate of asymptomatic cases as 11%. These patients were randomly identified (9).

The most important symptom in SSCD is vertigo and nystagmus occurring due to high sounds or pressure changes. Pressure change mentioned here is the change occurring in the external auditory canal, middle ear or intracranial area. In the physical examination, these symptoms were investigated with several testing such as high sound, tragal compression, valsalva and straining (5, 11). In their study with 35 patients, Teixido et al. found vestibular symptoms (tullio phenomenon) with high sound by 74%, vestibular symptoms with Valsalva Maneuver by 63%, nystagmus with high sound by 29%, nystagmus with Valsalva Maneuver by 25%, nystagmus with tragal pressure by 16%, chronic imbalance by 57%, and autophonia by 69%

(9). There are studies reporting sound induced nystagmus by more than 75% (13). In a study by Hillman (4) with 27 patients, chronic imbalance was found by 63%, vertigo induced with sound by 41%, and vertigo induced with pressure by 44%. In our study, we found sensation of imbalance in 63%, vertigo upon tragal compression by 4%, vertigo with high sound by 15%, and vertigo during lifting heavy things and pushing in 33% of the patients. In addition, nystagmus was found in 16 patients (59%) during physical examination. Another findings in these patients is hearing loss. Definitions about hearing loss related to the third window are confusing. Definition of air-bone gap is more correct than false CTHL. Because hearing loss is not related to a pathology resulted from external or middle ear. In addition, this loss is not caused by a pathology in the cochlea. Air-bone gap exists in audiometry and this is resulted from mechanical problems in the cochlea (15). Brandberg et al., found CTHL by 50% in their patients, and reported that the losses were more prominent at low frequencies (6). In a case series of 13 patients, Modugno found this rate as 54%, and also found the bone conduction thresholds as negative (16). In our study also we found marked CTHL at low frequencies by 37%. Similarly to the literature, we found bone conduction thresholds negative between 5 and 15 at least at one frequency.

Some patients may have clinically marked hearing loss without vestibular complaints. Air-bone gap is present on audiogram. This clinical condition may mimic otosclerosis, sometimes causing unnecessary exploration of the middle ear, and sometimes stapedectomy (17). Identification of SSCD in these conditions where clinical improvement can not be achieved may prevent unnecessary surgery (12). In this respect, ordering high-resolution CT before the exploration is of importance in order to not miss the diagnosis.

Air-bone gap has been reported to be closed with defect repair, thus the gap has been proven to be due to dehiscence (18). In addition, degree of hearing loss has been demonstrated to increase with the diameter of dehiscence (19).

Watson showed that, the threshold of vestibular evoked myogenic potentials (VEMP) is lower than 85 dB nHL in patients with SSCD, in the case of conduction type hearing loss, whereas these thresholds are never received in air-bone gap occurring in middle ear diseases (20). As is seen in our study, acoustic reflexes are received in pathologies of the 3rd window (8, 12, 17). Acoustic reflex and VEMP can be applied in patients with air-bone gap. Hıgh resolution temporal CT can be taken considering inner ear pathology, if acoustic reflexes are normal, and VEMP values have been decreased (21).

Sensitivity of TEOAE and DPOAE has provided them to become valuable diagnostic tools in the conditions affecting the cochlea such as ototoxicity and acoustic trauma. In addition, these values can show effects of ototoxic agents such as salicylates, gentamicin and cisplatin in an early period. Occasionally, this early diagnosis may be observed before audiological findings manifest. Thus it is possible to establish diagnosis and follow-up in the patients requiring intake of ototoxic drugs without losing time and probably before development of the loss audiologically.

DPOAEs are affected later and more hardly than TEOAEs in the conditions damaging the inner ear such as ototoxic drugs, and acoustic trauma (22). However, measuring DPOAE is an effective screening test especially in detection of ototoxicity in adults and children. DPOAE is more sensitive in monitoring cochlear functions compared to classical audiometry (23). However, a point which should be know is that TEOAE is more sensitive than DPOAE in monitoring the cochlea (22). In addition, it has been reported that TEOAE could not be detected in the losses higher than 30 dB (24). TEOAE is the most commonly observed within the frequency range of 700-4000 Hz. With the 3rd window theory, it has been observed with standard TEOAE that, cochlear pressure and inner ear function wasn't influenced, but significant shortening was found in the durations with a non-standard program, which is called MTWA (moving time window analysis) and measures emission durations (25). Whereas DPOAEs are detected in 90% of people with normal hearing (26), and unlike TEOAE these emissions may be found in patients with sensorineural hearing loss higher than 40 dB (27). However, these emissions are more practical than TEOAE in the measurements above 4 kHz

(28). Since DPOAEs are frequency specific, these emissions find a direct clinical area of application. Furthermore, the degree of hearing loss and audiometric configuration can be estimated with DPOAE (22).

In our study, evaluation of 7 separate frequencies between 1 and 8 kHz, led us to prefer DPOAE which is frequency specific and more sensitive than TEOAE in the measurements above 4 Hz. While OAE responses are found in the 3rd window lesions (8, 12, 16, 29), although the membrane is firm and definitely there is an energy to return back, there are opinions stating that OAE responses can not be received from mechanical problems in the cochlea (29). OAE values were found as normal in one patient with bilateral dehiscence and negative bone threshold and air-bone gap were detected (30). Merchant reported that OAE might be helpful (29). However, studies about DPOAE in patients with SSCD are insufficient.

In our study, we found a significant decrease especially in DP and SNR values only at one frequency in patients with unilateral dehiscence, and found no significant difference at the other frequencies. Significant decrease in DP and SNR values of the patients with bilateral dehiscence indicates that outer hair cells were negatively affected. In addition, DP values were found to be more sensitive than SNR values. Clinically, these different values were thought to be misleading.

As a result of our comparison between patients with bilateral dehiscence and the control subjects, it can be said that OAE indicated affected cochlea because of the significant decrease observed especially in DP values . However, OAE is not helpful for the diagnosis, because deterioration of OAE values were only at two frequencies in unilateral dehiscence group.

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