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Otoacoustic emissions evoked in Ménière's disease

Emissões otoacústicas evocadas na doença de Ménière

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ABSTRACT

Purpose: To verify the responses of Evoked Otoacoustic Emissions by transient stimulus and distortion product in individuals with Ménière's Disease.
Methods: Cross-sectional study with a sample composed of 60 individuals, aged 19 to 75 years, divided into two groups: study group, with 32 individuals with a medical diagnosis of Ménière's disease, without other risks and a control group formed by 28 individuals with cochlear loss without Meniere's disease, age and sex matched to the study group. Eligibility criteria: type A curve, without conductive or mixed loss or suspected retrocochlear alteration. The audiological evaluation consisted of anamnesis, inspection of the external acoustic meatus, pure tone audiometry, logoaudiometry, measures of acoustic immittance and transient evoked otoacoustic emissions and distortion product.
Results: Individuals with Ménière's disease had a higher occurrence of unilateral hearing loss, low pitch tinnitus, vertigo and ear fullness in relation to the control. In these individuals, there was greater incompatibility between the results of OAE and pure tone audiometry: in unilateral hearing loss, alterations in OAE were observed in ears with normal hearing thresholds on the contralateral side, characterizing cochlear dysfunctions. In the ears with cochlear loss, there was the presence of TEOAE and absence of DPOAE, in contrast to the control group, which showed the absence of TEOAE and DPOAE, as expected in cochlear losses of other etiologies.
Conclusion: The investigation of emissions in Ménière's disease identified cochlear dysfunction in the contralateral ear in unilateral cases and the presence of TOAE with absence of DPOAE in ears with hearing loss, differentiating from cochlear losses of other etiologies.

Keywords: Hearing tests; Ménière's disease; Hearing loss; Vertigo; Tinnitus; Endolymphatic hydrops; Hearing

RESUMO

Objetivo: Verificar as respostas das emissões otoacústicas (EOA) evocadas por estímulo transiente e produto de distorção em indivíduos com doença de Ménière. **Métodos:** Estudo transversal com casuística composta por 60 indivíduos de 19 a 75 anos de idade, distribuídos em dois grupos: grupo estudo, com 32 indivíduos com diagnóstico médico de doença de Ménière, sem outros riscos, e grupo controle formado por 28 indivíduos com perda coclear, sem doença de Ménière, pareado por idade e gênero ao grupo estudo. Critério de elegibilidade: curva tipo A, sem perda condutiva ou mista ou suspeita de alteração retrococlear. A avaliação audiológica foi composta por anamnese, inspeção do meato acústico externo, audiometria tonal limiar, logoaudiometria, medidas de imitância acústica e emissões otoacústicas evocadas por estímulo transiente e produto de distorção. **Resultados:** Os indivíduos com Ménière apresentaram maior ocorrência de perda unilateral, zumbido *pitch* grave, vertigem e plenitude auricular em relação ao controle. Nesses indivíduos, houve maior incompatibilidade entre os resultados das EOA e da audiometria tonal: nas perdas unilaterais, observaram-se alterações nas EOA nas orelhas com limiares auditivos normais do lado contralateral, caracterizando disfunções cocleares. Nas orelhas com perda coclear, houve presença de EOAT (por estímulo transiente) e ausência de EOAPD (produto de distorção), contrapondo-se ao grupo controle, que apresentou ausência de EOAT e de EOAPD, como o esperado em perdas cocleares de outras etiologias. **Conclusão:** A pesquisa das emissões na doença de Ménière identificou disfunção coclear na orelha contralateral nos casos unilaterais e presença de EOAT com ausência de EOAPD nas orelhas com perda auditiva, diferenciando-se das perdas cocleares de outras etiologias.

Palavras-chave: Testes auditivos; Doença de Ménière; Perda auditiva; Vertigem; Zumbido; Hidropsia endolinfática; Audição

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INTRODUCTION

Ménière's disease (DM) was first described in 1861 by French physiologist Prosper Ménière as a symptomatic triad characterized by tinnitus, hearing loss and episodes of vertigo in paroxysmal attacks. It is the most frequent vestibulopathies in adults, especially over 40 years old[1]. Its incidence varies greatly: 157 per 100,000 in the United Kingdom, 46 per 100,000 in Sweden, 7.5 per 100,000 in France, and 15 per 100,000 in the United States[2], or 34-190 per 100,000[3], for example. In Brazil, the lack of data regarding the incidence of Ménière's disease points to little epidemiological studies in the area.

Endolymphatic hydrops, a distension of the endolymphatic spaces in the inner ear, is the main histopathological finding of Ménière's disease[4].

Its etiology is related to viral or bacterial infectious processes, temporal bone development anomalies, genetic factors, trauma, otospongiosis, among others[1].

Diagnosis is made based on well-defined clinical criteria. According to the American Academy of Otorhinolaryngology-Head and Neck Surgery's criteria (AAO-HNS), Ménière's disease can be assigned to individuals who reported two or more episodes of vertigo lasting 20 minutes, hearing loss observed in at least one occasion and presence of tinnitus and/or ear fullness[3].

Definitive conformation of the pathophysiological alteration that characterizes Ménière's disease can only be proven by a pathological study of temporal bones, postmortem[5].

Audiological evaluation of individuals with Ménière's disease was initially conducted using pure-tone audiometry threshold, speech audiometry and acoustic immittance measures. Electrocochleography (ECOG) has also been used to identify hydrops and aid diagnosis[6]. More recently, otoacoustic emissions (OAE), defined as a sound generated by the cochlea that propagates from the inner ear to the ear canal[7], have been recommended to identify cochlear alterations in Ménière's disease[8].

Emission research has been useful in the topodiagnosis of hearing loss, identification of cochleopathy and cochlear dysfunctions, and monitoring of cochlear function, contributing to diagnose Ménière's disease. Evidence shows that small changes in cochlear functioning could be detected by otoacoustic emissions before audiogram alterations[9,10].

Overall, audiometry results in cochlear losses are compatible with otoacoustic emissions. However, some tests performed with individuals affected by Ménière's disease at UNESP's Audiology Clinic have shown transient evoked otoacoustic emissions (TEOAE) with thresholds higher than 30 dBHL, as in retrocochlear alterations. Such clinical findings prompted us to study otoacoustic emissions in Ménière's disease.

Given this context, our interest lied in studying TEOAE and DPOAE responses in individuals with Ménière's disease, hypothesizing the incompatibility between emissions and pure-tone audiometry as typical of this disease.

Hence, this study examined the symptomatic manifestations, auditory alterations, and compatibility between evoked otoacoustic emissions and pure-tone audiometry thresholds in individuals diagnosed with Ménière's disease.

METHODS

In this cross-sectional study, audiological evaluations were conducted with 32 individuals diagnosed with Ménière's disease by a medical team from the Department of Otorhinolaryngology, Federal University of São Paulo (UNIFESP), and compared to 28 individuals with sensorineural hearing loss without Ménière's disease, evaluated at the Department of Speech, Language and Hearing Sciences of the same university. This study was approved by UNIFESP's Research Ethics Committee, under no. 3.733.753. All participants signed the informed consent form.

Eligibility criteria for participating in the control and study groups included individuals without mixed conductive loss or clinical suspicion of retrocochlear alteration, evaluated by an ENT physician. The study group consisted of 32 individuals, aged 19-75 years, 21 (65.6%) of them women, diagnosed with Ménière's disease and type A tympanogram. Individuals exposed to occupational noise, with otosclerosis, undergoing chemotherapy or radiotherapy, or previous use of ototoxic drugs were excluded. The control group included 28 individuals, aged 19-74 years, 19 (67.8%) of them women, diagnosed with cochlear hearing loss by another etiology, without Ménière's disease, and type A tympanogram. The control and study groups were paired by age and gender. Mean age was 53.5 years and 55.8 years in the study group and control group, respectively.

All individuals underwent complete audiological evaluation in an acoustic booth, with anamnesis, inspection of the ear canal, pure-tone audiometry threshold (PTT), speech audiometry, acoustic immittance measures, and assessment of transient-evoked and distortion-product emissions. Ear canal inspection was performed using a TK otoscope to rule out the presence of foreign bodies or excess cerumen, which could compromise the evaluation. Pure-tone audiometry threshold was performed using Interacoustics AD-229 audiometer, TDH-39 headphones, duly calibrated[11]. Pure-tone air-conduction thresholds were measured at frequencies of 250 Hz to 8000 Hz by descending technique[12]. We evaluated pure-tone bone-conduction thresholds when air-conduction thresholds were over 25 dBHL, at frequencies of 0.5 to 4 kHz. Hearing thresholds equal to or below 25 dBHL were considered normal, whereas thresholds over 25 dBHL characterized hearing loss. The degree of hearing loss was classified according to the mean of the 500 Hz, 1000 Hz and 2000 Hz frequencies[13].

To verify the inclusion criterion—tympanic-ossicular integrity—, we obtained acoustic immittance measurements using an Interacoustics middle ear analyzer, model AT 235, with a 226 Hz probe. Tympanograms were classified as A, B, C, A_d and A_r[14]. Individuals with middle ear alterations (type B and C tympanograms) were excluded. Transient evoked otoacoustic emissions (TEOAE) survey was performed using a Otodynamics ILOV6 equipment, in an acoustically treated booth, connected to a microcomputer. Nonlinear clicks were used as stimuli, with regular pulses of 80 milliseconds, presented in a series of 260 cycles per second, in a 20 ms window. TEOAE were considered present when there were emissions 3 dB above noise in the frequency bands from 1 to 4 kHz, with response reproducibility and probe stability greater than 70%[15].

Distortion product otoacoustic emissions (DPOAE) were evoked by two pure tones, presented simultaneously, with close sound frequencies (f1/f2=1.22). The response component considered was 2f1-f2, with F1 and F2 stimulus intensity level

of 65 dBSPL and 55 dBSPL, respectively. Response analysis considered the amplitude and signal-to-noise ratio at frequencies of 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz and 8 kHz. DPOAE was considered present for positive response with a signal-to-noise equal to or over 5 dB and negative noise[16]. When four or more frequencies were absent, we considered DPOAE to be absent.

Considering all the evaluations performed, final diagnosis was defined as: normal hearing sensitivity (hearing thresholds below or equal to 25 dBHL on audiometry, with the presence of transient-evoked and distortion-product otoacoustic emissions); cochlear hearing loss (hearing thresholds over 25 dBHL, with absence of otoacoustic emissions in the frequencies where loss occurred) and cochlear dysfunction (hearing thresholds below or equal to 25 dBHL, with absent or partial otoacoustic emissions). Thus, unilateral hearing loss were classified as cochlear loss with normal hearing or contralateral cochlear dysfunction.

Results were considered compatible when there was absence of TEOAE in hearing losses with thresholds over 25 dBHL and absence of DPOAE in hearing losses with thresholds over 40 dBHL.

Statistical analysis adopted a 5% significance value ($p \leq 0.05$). We used the SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). The Mann-Whitney U-test, univariate analysis of variance (ANOVA), Kruskal-Walli's test, Spearman's correlation test, McNemar's test, and Fisher's exact test were applied.

RESULTS

The study and control groups included, respectively, 32 individuals with hearing loss from Ménière's disease and 28 individuals with hearing loss by other etiologies. Statistical analysis (Mann-Whitney U test) showed no difference between the groups regarding gender ($p > 0.999$) and age ($p = 0.482$).

Table 1 presents the group distribution according to the affected side, tinnitus classification, presence of vertigo and ear fullness. Compared with the control group, the study group presented higher occurrence of unilateral loss, severe tinnitus, presence of vertigo and ear fullness.

In the TEOAEs and DPOAEs, comparative analysis showed no difference between the left and right ears in bilateral losses, both in the control and study groups.

Degree of hearing loss ranged from mild to profound in both groups. Mild and moderate losses accounted for most cases: 22.7% mild loss and 26.4% moderate loss in the Ménière's disease group; 15.1% mild loss and 30.2% moderate loss in the control group. Severe hearing loss was 7.5% in both groups, whereas profound loss accounted for 5.7% in the control group and 3.8% in the study group. Hearing loss occurred at isolated frequencies, without defined degree of loss, in 39.6% of the study group and 41.5% of the control group. As for audiometric configuration, both groups presented higher occurrence of flat curves: 30.2% in the Ménière's disease group and 41.5% in the control group. Rising curves were more frequent in the study group (30.2%), compared to control (7.6%). The sloping configuration occurred most often in the control group (39.6%), compared to the study group (26.4%). Reversed U-shape curves accounted for 9.4% in both groups, whereas anacusis was 3.8% in the study group and 1.9% in the control group.

For pure-tone audiometry, we compared the hearing thresholds over 25 dBHL obtained for each analyzed frequency between the groups (Figure 1). We identified 53 years with hearing loss in each group: 21 individuals with bilateral loss (42 years) and 11 with unilateral loss (11 ears) in the study group; 25 individuals with bilateral loss (50 ears) and 3 with unilateral loss (3 ears) in the control group. Three ears from the control group and 11 ears from the study group presented hearing thresholds within the normal range. We found a statistically significant difference between the groups only at the 500 Hz frequency, with the study group showing a higher proportion of ears with altered results ($p = 0.039$).

Table 2 summarizes the results of the transient evoked emissions survey, which covered all ears with hearing loss (106 ears: 14 unilateral and 46 bilateral). In comparing the groups, we found a difference only in the 4000 Hz band. Compared with the control group, the study group had a lower proportion of ears with absent TEOAE at 4000 Hz .

The results of the distortion product otoacoustic emissions survey showed no differences between groups (Table 3).

Table 4 presents the joint analysis of TEOAE and DPOAE results, comparing the groups. Results show that individuals with Ménière's disease had a higher proportion of ears with a "present TOEAE, absent DPOAE" result, compared with the control group.

Group comparison of the compatibility or incompatibility between the results of pure-tone audiometry threshold and

Table 1. Comparison between control and study groups regarding the affected ear, tinnitus classification and presence of vertigo and ear fullness (N=60)

Variable	Category	Group				Total		p-value
		Study		Control				
		n	%	n	%	n	%	
Type of loss	Unilateral	11	34.38	3	10.71	14	23.33	**0.037***
	Bilateral	21	65.63	25	89.29	46	76.67	
Tinnitus classification	Severe	28	87.50	15	53.57	43	71.67	**0.005***
	Acute	4	12.50	13	46.43	17	28.33	
Vertigo	Present	26	81.25	11	39.29	37	61.67	**0.001***
	Absent	6	18.75	17	60.71	23	38.33	
Ear fullness	Present	16	50.00	3	10.71	19	31.67	**0.002***
	Absent	16	50.00	25	89.29	41	68.33	

Fisher's exact test; *Statistically significant at 5% level ($p \leq 0.05$)
Subtitle: n = number of individuals; % = percentage

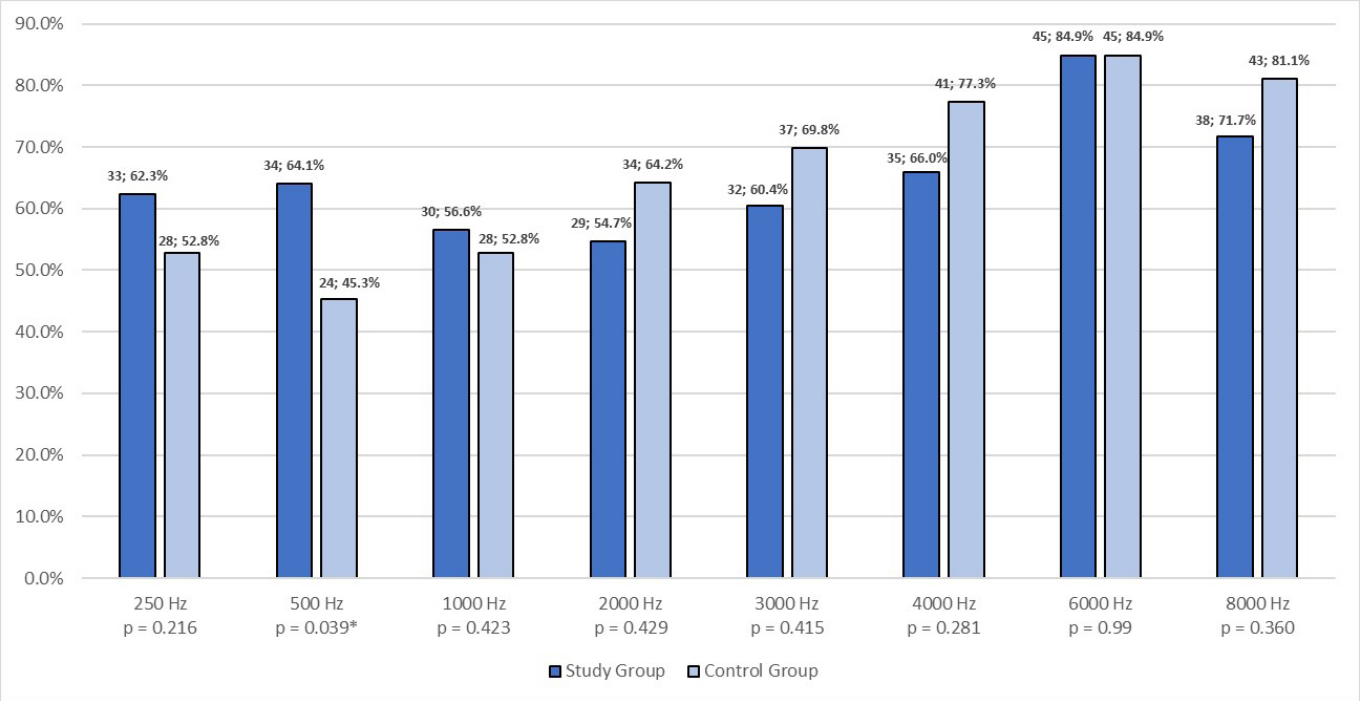

Figure 1. Occurrence of altered thresholds (over 25 dBHL) in the frequencies evaluated in the study and control groups
Subtitle: p = p-value; % = percentage; *Statistically significant at 5% level (p ≤ 0.05)

Table 2. Comparison between control and study groups regarding transient evoked otoacoustic emissions in ears with hearing loss (N=106 ears)

TEOAE	Category	Group				Total		p-value
		Study		Control				
		n	%	n	%	n	%	
1000 Hz	Present	28	52.83	24	45.28	52	49.06	0.560
	Absent	25	47.17	29	54.72	54	50.94	
2000 Hz	Present	28	52.83	21	39.62	49	46.23	0.242
	Absent	25	47.17	32	60.38	57	53.77	
3000 Hz	Present	27	50.94	18	33.96	45	42.45	0.115
	Absent	26	49.06	35	66.04	61	57.55	
4000 Hz	Present	25	47.17	13	24.53	38	35.85	**0.025***
	Absent	28	52.83	40	75.47	68	64.15	

Fishers exact test
Subtitle: TEOAE = transient evoked otoacoustic emissions; n = 106 ears; % = percentage

Table 3. Comparison between control and study groups regarding distortion product otoacoustic emissions in ears with hearing loss (N=106 ears)

DPOAE	Category	Group				Total		p-value
		Study		Control				
		n	%	n	%	n	%	
1000 Hz	**Present**	12	22.64	18	33.96	30	28.30	0.140
	Absent	41	77.36	35	66.04	76	71.70	
2000 Hz	**Present**	11	20.75	10	18.87	21	19.81	0.500
	Absent	42	79.25	43	81.13	85	80.19	
3000 Hz	**Present**	13	24.53	8	15.09	21	19.81	0.165
	Absent	40	75.47	45	84.91	85	80.19	
4000 Hz	**Present**	7	13.21	6	11.32	13	12.26	0.500
	Absent	46	86.79	47	88.68	93	87.74	
6000 Hz	**Present**	4	7.55	4	7.55	8	7.55	0.642
	Absent	49	92.45	49	92.45	98	92.45	
8000 Hz	**Present**	3	5.66	2	3.77	5	4.72	0.500
	Absent	50	94.34	51	96.23	101	95.28	

Fishers exact test
Subtitle: DPOAE = distortion product otoacoustic emissions; n = 106 ears; % = percentage

Table 4. Joint result of transient evoked otoacoustic emissions and distortion product otoacoustic emissions considering ears with and without hearing loss (N=120 ears)

Variable	Category	Group				Total		p-value
		Study		Control				
		n	%	n	%	n	%	
Joint result	absent TEOAE and DPOAE	30	46.88[a]	42	75.00[b]	72	60.00	**0.022***
	present TEOAE, absent DPOAE	28	43.75[a]	11	19.64[b]	39	32.50	
	present TEOAE and DPOAE	6	9.38[a]	3	5.36[a]	9	7.50	

Fisher's exact test; *Statistically significant at 5% level ($p \leq 0.05$). Letters ([a]) indicate subsets of the "group" variable whose column proportions are not significantly different from each other at 5% significance level ($p \leq 0.05$)
Subtitle: TEOAE = transient evoked otoacoustic emissions; DPOAE = distortion product otoacoustic emissions; n = 120 ears; % = percentage

Table 5. Group comparison regarding compatibility between pure-tone audiometry threshold and otoacoustic emissions by ear and individual (N=60)

Compatibility	Category	Group				Total		p-value
		Study		Control				
		n	%	n	%	n	%	
RE	Compatible	17	53.13	21	75.00	38	63.33	0.109
	Incompatible	15	46.88	7	25.00	22	36.67	
LE	Compatible	16	50.00	18	64.29	34	56.67	0.350
	Incompatible	16	50.00	10	35.71	26	43.33	
Individual	Compatible	9	28.13	17	60.71	26	43.33	**0.018***
	Incompatible	23	71.88	11	39.29	34	56.67	

Fisher's exact test; *Statistically significant at 5% level ($p \leq 0.05$)
Subtitle: RE = right ear; LE = left ear; n = 60; % = percentage

otoacoustic emissions (Table 5) showed that individuals with Ménière's disease had a higher occurrence of incompatibility compared with individuals without the disease.

Final diagnoses established in both groups were classified as: unilateral cochlear loss with normal hearing thresholds in the contralateral ear; unilateral cochlear hearing loss with contralateral dysfunction, and bilateral cochlear loss.

Occurrence of hearing thresholds within normal limits, cochlear loss, and cochlear dysfunctions showed no statistical difference between the groups ($p = 0.295$) (Figures 2 and 3).

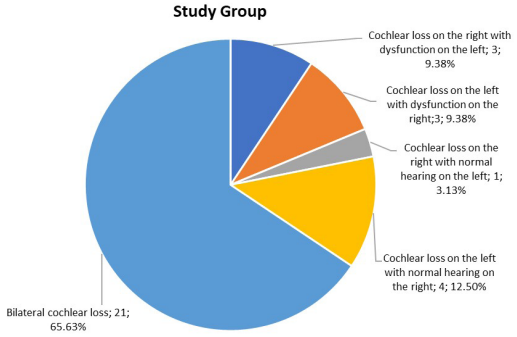

Figure 2. Final study group diagnosis
Subtitle: % = percentage

DISCUSSION

This study examined the symptomatic manifestations, hearing alterations, and compatibility between evoked otoacoustic emissions and pure-tone audiometry thresholds in individuals diagnosed with Ménière's disease (MD).

To verify whether the characteristics observed in the 32 individuals with Ménière's disease differed from those with cochlear loss by other etiologies, we included a control group consisting of 28 individuals with sensorineural hearing loss by various etiologies and without suspected MD. Importantly, this group included individuals diagnosed with cochlear losses due to metabolic diseases, kidney disease, sickle cell anemia, heart disease, chemotherapy, and noise exposure.

Participants in the study group (Ménière's disease) were aged 19-75 years, with most individuals between 31 and 60 years old. Mean age was 53.5 years, similar to other studies in the literature[1,17]. In fact, Ménière's disease often begins in the third or fourth decade of life[1,3,6]. Age and gender comparison between the groups showed no statistical difference, an expected finding since the groups were previously paired to avoid sample bias.

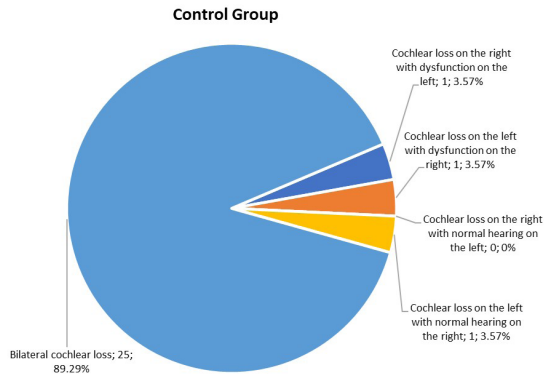

Figure 3. Final control group diagnosis
Subtitle: % = percentage

There was a predominance of women with Ménière's disease (65.6%), similar to other studies[1,3,5,8,17-19]. Fact already observed in a longitudinal study with 169 individuals with MD, in which 57% were women[18]. In a more recent study, 66.5% of the 200 individuals with MD were women[17]. Other research also found a predominance of women in the sample of individuals with MD[1,2,19]. These findings point to a predominance of women with Ménière's disease. In fact, the Multidisciplinary Diagnostic Committee for Ménière's Disease reports a small female predominance[3].

When comparing the study and control groups, we observed a higher occurrence of tinnitus, vertigo and ear fullness in the former. Symptom analysis showed a higher occurrence of vertigo in individuals with MD (81.2%) compared to individuals without MD (39.3%), and a higher occurrence of severe tinnitus (87.5%) compared to individuals with sensorineural loss by other etiologies (53.6%). Moreover, only 10.7% of the individuals without MD presented ear fullness versus half of the study group.

An expected finding, for since it was first described by Prosper Ménière, the symptomatic triad (hearing loss, tinnitus, and vertigo) was pointed out as characteristic of the disease. In fact, diagnosis is made based on well-defined criteria and include episodes of vertigo lasting at least 20 minutes, sensorineural hearing loss at low and medium frequencies, and the presence of tinnitus and/or ear fullness[3]. In the literature consulted, most studies on individuals with Ménière's disease observed the occurrence of tinnitus, vertigo, and ear fullness[1,3,5,8,17-19]. Moreover, the occurrence of severe tinnitus in individuals with MD has also been widely described in the literature[1,3,17].

Regarding the degree of loss, we observed mild and moderate loss in individuals with Ménière's disease, similar to data in the literature[8,17,18]. Importantly, in 39.6% of the study group and 41.5% of the control group, we could not obtain the degree of loss by averaging from 500 Hz to 2000 Hz, for the losses occurred in isolated frequencies, for example in the low frequencies (250 Hz and 500 Hz). Conversely, we found flat and ascending configurations in one third of the Ménière's disease audiograms, as described in the literature[3,8,18,20,21].

Although 77.3% of the control group presented high threshold at 4000 Hz, 66% of the study group showed alteration at the same frequency, without statistical difference between the groups.

Early audiological assessments in individuals with Ménière's disease included pure-tone audiometry threshold, speech audiometry and immittance mesurements[20]. Electrocochleography was included in audiological evaluation due to its effectiveness in identifying endolymphatic hydrops[6]. More recently, otoacoustic emissions have been added to the audiological evaluation of individuals with Ménière's disease[8,10,21-23]. Some studies have performed TEOAE[21] and others, DPOAE[8,10,22-24]. Thus, the present study could be considered a pioneer for comparing TEOAE and DPOAE in individuals with Ménière's disease.

Bilateral hearing loss appeared in 65.6% of individuals with MD and in 89.3% of the control group. Unilateral loss occurred in 34.4% of the individuals with MD and in 10.7% of the control group, with a statistically significant difference between the groups. Therefore, individuals with Ménière's disease showed a predominance of unilateral losses when compared to individuals with hearing loss due to other etiologies, a finding similar to that reported by other studies[3,21,25]. The literature points to the presence of unilateral losses in Ménière's disease. A study with 39 individuals and mean age of 42.9 years found 66.6% of unilateral losses, a result much higher than our findings[1]. Such disagreement could be explained by the older mean age of the individuals in our study (53.5 years), since the literature reports evolution of unilateral to bilateral losses over time[18-20]. Studies have revealed that bilateral losses are associated with disease progression. Conversely, we observed a high occurrence of bilateral losses (65.6%) in individuals with Ménière's disease, also reported in the literature[1,18].

In bilateral losses, TEOAE and DPOAE results were similar regarding ear side and, thus, were grouped together. DPOAE responses were similar between groups. When comparing the TEOAE results, we found a difference in the 4000 Hz band, with more responses in the study group. This result was expected, since Ménière's disease primarily affects low frequencies. In fact, when comparing pure-tone audiometry threshold results, we observed a statistically significant difference between the groups at 500 Hz, similar to other studies[8,20]. Animal study by inducing hydrops in guinea pigs found hearing loss at low frequencies[22]. Studies have shown that low-frequency losses occur early in the disease, progressing to a flat hearing loss over time[18-20]. This was confirmed by a monitoring study with 161 individuals with Ménière's disease, which identified rising curves in 20% of the individuals in the early phase, dropping to 12.1% after 13-16 years[18]. Moreover, studies have shown that hearing fluctuation occurs mainly during the first year of Ménière's disease and at low frequencies[3,20,26]. By means of serial OAE evaluations and audiometry, an auditory monitoring of 30 individuals with Ménière's disease verified the presence of fluctuation as a characteristic of the disease[26]; data we could not confirm in our study, as we did not perform audiological monitoring of the individuals with Ménière's disease.

When comparing the OAE and pure-tone audiometry results, we observed absent TEOAE and DPOAE, present TEOAE and DPOAE, and present TEOAE with absent DPOAE in both groups. In the study group, 43.7% of the participants had present TEOAE with absent DPOAE. In the control group, most cases (75%) had absent TEOAT and DPOAE, with only

19.6% showing present and absent DPOAE. This difference was statistically significant between the groups, finding that had already been described in the literature, with occurrence of TEOAE in ears with hearing loss. As the hearing impairment in individuals with Ménière's disease does not involve the outer hair cells, TEOAE could be detected even in cases of moderate hearing loss[9]. A previous study involving 31 individuals with Ménières disease had already described the presence of TEOAE with thresholds over 25 dBHL[21]. Similarly, another research identified that five out of 15 individuals with Ménière's disease and thresholds over 40 dB had TEOAE[15].

When comparing TEOAE and DPOAE together, present TEOAE with absent DPOAE could be interpreted as a characteristic of endolymphatic hydrops in Ménière's disease, given its low prevalence in cochlear losses due to other etiologies. Several hypotheses may explain this finding. One refers to the different mechanisms that generate TEOAE and DPOAE[27]. In TEOAE, the generating mechanism occurs by linear reflection, which evokes responses from outer hair cells throughout the cochlea. In DPOAE, responses are generated by cochlear nonlinearity—distortion products—at specific points of the cochlea (F1, F2 and 2F1-F2). Moreover, since the click is broadband, this could provoke responses by interfering with the best audiogram threshold[9]. Another hypothesis suggests that the hearing alterations in endolymphatic hydrops do not involve the outer hair cells (OHC) but are attributed to the hydrodynamic and biomechanical micro-mechanism of the cochlea[21,23,28].

Despite the still unconfirmed hypotheses discussed by the literature, the presence of TEOAE in ears with loss over 30 dBHL in individuals with MD is widely known and may be a specific characteristic of endolymphatic hydrops[9,15].

Such results, therefore, could be explained by the difference in the mechanisms generating TEOAE and DPOAE, by fact that the click is broadband and suffers interference from the best audiogram thresholds, and by the possibility that hydrops does not involve a specific lesion of the outer hair cell, with changes in the cochlea's hydrodynamic and biomechanical mechanism[9,21,23,27,28]. Differences in TEOAE and DPOAE technology have already been discussed, indicating that DPOAE reject all frequencies, excepting the 2F1-F2[7]. TEOAE, in turn, record all frequency bands, and the cochlear response is observed between stimulation and the relaxation phase, which is relevant for low intensities[7]. Thus, the two techniques observe the cochlea under different conditions. In TEOAE, the time spent would be considered highly effective in separating the stimulus from the delayed response (reflection). In DPOAE, nonlinearity would be the main factor separating the stimulus from the response. Moreover, TEOAE cease to be recorded at losses between 25 and 30 dBHL, while DPOAE, at losses between 35 and 45 dBHL.

Group comparison regarding compatibility of the results obtained revealed a statistically significant difference, with the study group showing a higher occurrence of incompatibility (71.9%) compared to the control group (39.3%). The incompatibility found in the control group could be explained by the cases of hearing loss due to metabolic etiology, noise exposure, and chemotherapy, which could also present cochlear dysfunctions before alterations in the audiogram.

TEOAE present in ears with hearing loss and absence of distortion product appears to be a characteristic feature of individuals with MD and endolymphatic hydrops, confirming our initial hypothesis. This fact had already been observed in the clinical routine of the institution's Department of Audiology, which motivated this research.

In the present study, the incompatibility observed in ears with hearing loss was the presence of TEOAE in losses over 30 dBHL, which could give a false retrocochlear diagnosis. During DPOAE, however, the absence of response characterized the loss as cochlear. Hence the importance of performing TEOAE and DPOAE surveys in individuals with Ménière's disease.

Another incompatibility found in MD cases with unilateral loss was the absence of TEOAE and DPOAE in ears with hearing thresholds within normal range, characterizing cochlear dysfunctions in the contralateral ears. Such dysfunction suggests possible progression of loss and is therefore of clinical relevance.

Final diagnosis was established as: unilateral loss with contralateral dysfunction, unilateral loss with contralateral normal hearing, and bilateral cochlear loss. In the study group, six cases had hearing loss with cochlear dysfunction in the opposite ears and five cases had unilateral cochlear loss with contralateral normal hearing. The control group reported two cases of unilateral loss with contralateral dysfunction and one case of unilateral loss with contralateral normal hearing. Bilateral cochlear loss appeared in 21 individuals (65%) in the study group and 26 subjects (89%) in the control group. Results showed no significant statistical difference between the groups. This could be due to the presence of metabolic alterations, noise exposure, and chemotherapy in some individuals in the control group.

In summary, by comparing the study and control groups, we verified a prevalence of unilateral losses in individuals with MD, greater occurrence of vertigo, severe tinnitus, and ear fullness. Isolated diagnosis of each examination, pure-tone audiometry, TEOAE and DPOAE showed no statistical difference between the groups, but we confirmed incompatibility between the results. Individuals with MD presented cochlear dysfunctions with normal hearing thresholds and altered OAE in the contralateral ears. Cochlear losses showed presence of TEOAE and absence of DPOAE. Such incompatibilities could be considered as characteristic findings of Ménière's disease, which differ from cochlear loss due to other etiologies. Hence, TEOAE and DPOAE would be recommended in cases of Ménière's disease for a more accurate audiological diagnosis.

A limitation in our study was the absence of audiological monitoring, which could better indicate whether the contralateral ears showing OAE changes would evolve to loss over time. Further studies could clarify this issue.

CONCLUSION

Evaluation of otoacoustic emissions in Ménière's disease allowed us to identify cochlear dysfunction in the contralateral ear, in unilateral cases, and the presence of TEOAEs with absence of DPOAEs in ears with hearing loss, differing from cochlear losses by other etiologies.

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