

Endolymphatic Hydrops Revealed by Magnetic Resonance Imaging in Patients With Acute Low-Tone Sensorineural Hearing Loss

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Objective: Acute low-tone sensorineural hearing loss (ALHL) has been reported to be associated with endolymphatic hydrops (EHs). However, evaluation of the size of the endolymphatic space has not been reported. We attempted to visualize EH in ALHL using magnetic resonance imaging (MRI).

Study Design: Prospective diagnostic study.

Setting: University hospital.

Methods: We evaluated 25 ears of 25 unilateral ALHL patients. Three-tesla MRI was obtained 24 hours after intratympanic injection of gadolinium (Gd) (n = 5) or 4 hours after intravenous injection of Gd (n = 20). A radiologist blinded to the patients' clinical data classified the degree of EH in the vestibule and cochlea into 3 groups: none, mild, and significant.

Results: On the affected sides, cochlear EH was recognized in 23 ears (92%) and was classified as significant EH (n = 15) or mild EH (n = 8); vestibular EH was detected in 22 ears (88%), classified as significant EH (n = 16) or mild EH (n = 6). Cochlear EH was more frequently observed in the affected ear than in the contralateral ear (90% versus 40%, $p < 0.05$).

Conclusion: In ALHL, EH was observed not only in the cochlea but also in the vestibule as in Ménière's disease. **Key Words:** Acute low-tone sensorineural hearing loss—Cochlear Ménière's disease—Endolymphatic hydrops—Magnetic resonance imaging—Parvovirus.

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Acute low-tone sensorineural hearing loss (ALHL) is acute sensorineural hearing loss confined to low-tone without vertigo and may include various pathophysiologic processes. In 1982, Abe reported the clinical characteristics of 39 patients with ALHL without fluctuation or recurrence and suggested that ALHL may be a mild type of sudden hearing loss and that the pathophysiologic features of ALHL may differ from those of Ménière's disease (1). By contrast, it is well known that ALHL frequently occurs in the early stage of Ménière's disease (2). In 1994, Yamasoba et al. (3) reported that ALHL patients frequently showed abnormal results in functional tests such as the glycerol test or electrocochleography (ECoG)

and that some patients developed cochlear or classic Ménière's disease in the long term. They concluded that ALHL may be caused by endolymphatic hydrops (EH) confined to the cochlea. Many reports have supported this idea with similar clinical features or abnormal functional test results in ALHL patients (4,5). Autoimmunity is also considered a possible cause of ALHL (6). However, the pathophysiology of ALHL remains controversial.

Recently, we visualized the endolymphatic space in Ménière's disease using 3T MRI after intratympanic or intravenous injection of gadolinium (Gd) (7–9). MRI after intratympanic injection of Gd is a promising alternative to the histologic confirmation of EH. In this study, we show the relationship between EH, as revealed using 3T MRI, and the clinical characteristics of ALHL.

MATERIALS AND METHODS

Twenty-five ears of 25 patients with ALHL were included. Cases were diagnosed in our hospital between 2009 and 2012,

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TABLE 1. *Diagnostic criteria for acute low-tone sensorineural hearing loss without vertigo*

Main symptoms	
1. Acute or sudden onset of cochlear symptoms including ear fullness, tinnitus, and hearing loss	
2. Low-tone hearing loss	
3. Without vertigo	
4. Unknown cause	
For reference	
1. Audiometric criteria of low-tone hearing loss.	
(1) The sum of hearing levels at low frequencies of 0.125, 0.25, and 0.5 kHz is 70 dB or more.	
(2) The sum of hearing levels at high frequencies of 2, 4, and 8 kHz is 60 dB or less.	
2. Cochlear symptoms may be recurrent.	
3. May progress to Ménière's disease.	
4. May be accompanied with light dizzy sensation.	
5. May be bilateral.	
Definite: All of the main symptoms. Audiometric criteria (1) and (2).	
Probable: All of the main symptoms. Audiometric criteria (1) and the same hearing levels at high frequencies of 2, 4, and 8 kHz as the contralateral ear.	
This criteria is suggested by the Acute Severe Hearing Loss Study Group, the Ministry of Health, Labor and Welfare of Japan in 2011.	

according to the criteria determined by the Ministry of Health, Labor, and Welfare of Japan in 2011 (Table 1). Acoustic tumor or ALHL with vertigo were excluded from the study.

Five patients were evaluated by 3T MRI 24 hours after intratympanic administration of gadopentetate dimeglumine (Magnevist; Bayer Healthcare, Wayne, NJ, USA) diluted 8-fold with saline (v/v 1:7), as described previously (9). The other 20 patients were evaluated by 3T MRI 4 hours after intravenous injection of a standard dose (0.2 ml/kg body weight, i.e., 0.1 mmol/kg body weight) of gadodiamide hydrate (Omniscan; GE Healthcare).

All scans were performed using a 3T MRI scanner (Magnetom Verio; Siemens, Erlangen, Germany) equipped with a receive-only, 32-channel, phased-array coil. Three-dimensional fluid-attenuated inversion recovery (3D-FLAIR) and 3D inversion-recovery sequence with real reconstruction were used for the patients after intratympanic Gd administration for evaluation of EH (7). Heavily T2-weighted 3D-FLAIR MRI was performed in the patients after intravenous Gd administration. HYDROPS (hybrid of reversed image of positive endolymph signal and native image of positive perilymph signal) was also used to detect EH; it has recently been reported to be useful for

visualizing the endolymphatic space (8). A radiologist who did not know the corresponding clinical information classified the degree of EH in the vestibule and cochlea into 3 grades according to the criteria described previously: none, mild, and significant (10).

The data were analyzed with SPSS 20.0 for Windows (SPSS, Chicago, IL, USA). Differences between the groups in the grades of EH were assessed using a χ^2 test, and other variables were assessed using Student's *t* test and a Mann-Whitney *U* test. The level of significance was set at $p < 0.05$.

RESULTS

The clinical characteristics of the 25 ALHL patients are shown in Table 2. Their average age was 47.3 ± 3.0 years, and the male-to-female ratio was 8:17. All patients had unilateral ALHL. Recurrent hearing loss was observed in 9 ears (36%), and average time from onset to MRI was 20.9 months, with a range of 0.3 to 150 months. The worst mean hearing level in the diseased ear for low frequency (125, 250, and 500 Hz) was 45.4 ± 2.5 dB, and that for high frequency (2, 4, and 8 kHz) was 16.1 ± 1.9 dB. Sixteen patients experienced no vestibular symptoms, and the other 9 patients experienced subtle dizziness but no Ménière-type vertigo attacks. Mean age and high-tone hearing threshold were significantly higher in probable ALHL patients compared with definite ALHL patients ($p < 0.05$), but no significant differences between the 2 groups were observed in other characteristics. Table 3 shows the clinical course and imaging data for each case. The first symptom was ear fullness in 9 cases, hearing loss in 7 cases, tinnitus in 12 cases, and hyperacusis in 1 case. The onset of the cochlear symptoms was acute in all cases. The period from onset to the first audiometric confirmation of hearing loss was 1 day to 1 month. There were cases that meet the diagnostic criteria for cochlear Ménière's disease (11), possible Ménière's disease (12), or sudden deafness (SD) (13) as well as ALHL: 9 cases can be diagnosed as cochlear Ménière's disease; 9 cases as possible Ménière's disease; and 12 cases as SD.

Table 4 shows the frequency of EH in the affected ears of the ALHL patients. Cochlear EH was observed in

TABLE 2. *Characteristics of acute low-tone sensorineural hearing loss patients*

	Acute low-tone sensorineural hearing loss patients (n = 25)	Definite (n = 18)	Probable (n = 7)
Mean age (yr)	47.3 ± 3.0	42 ± 2.8	63.6 ± 1.4
Male-to-female ratio	8 : 17	6 : 12	2 : 5
Symptoms			
Hearing loss	14 (56.0%)	11 (61.1%)	3 (42.8%)
Ear fullness	20 (80.0%)	14 (77.8%)	6 (85.7%)
Tinnitus	21 (84.0%)	14 (77.8%)	7 (100.0%)
Dizziness	9 (36.0%)	6 (33.3%)	3 (42.8%)
Recurrence of hearing loss	9 (36.0%)	4 (22.2%)	5 (71.4%)
Duration from onset to MRI (mo)			
Mean	20.9 ± 7.6	8.5 ± 3.5	52.7 ± 22.4
Range	0.3–150.0	0.3–29.0	3.0–150.0
Hearing level (dB)			
Average of 125, 250, and 500 Hz	45.4 ± 2.5	43.1 ± 3.4	53.3 ± 4.4
Average of 2, 4, and 8 kHz	16.1 ± 1.9	11.3 ± 1.2	28.3 ± 2.9

TABLE 3. Clinical course and imaging data for acute low-tone sensorineural hearing loss patients

Case no.	Affected side	IT/IV	First symptoms	Duration from onset to first audiogram	Duration from onset to MRI (mo)	Hearing level (dB)		Recurrence	Dizziness	Diagnostic criteria [✱]					Cochlear EH	Vestibular EH
						Average of 250, 500 Hz	Average of 125, 4, and 8 kHz			Cochlear MD	Possible MD	SD				
1	Right	IT	Ear fullness	1 d	6	46.7	3.3	No	No	No	o		2	2		
2	Left	IT	Hearing loss	1 mo	63	53.3	8.3	Yes	No	No	o		1	2		
3	Left	IT	Hearing loss, tinnitus	2 d	3	68.3	13.3	No	No	No	o		2	2		
4	Left	IV	Hearing loss	2 wk	2	58.3	10.0	No	No	No	o		0	1		
5	Right	IV	Hearing loss, tinnitus	2 d	3	50.0	16.7	No	No	No	o		2	2		
6	Left	IV	Hearing loss	2 d	2	25.0	13.3	No	Yes	Yes	o		1	2		
7	Left	IV	Tinnitus	4 d	0.3	31.7	16.7	No	No	No	o		2	2		
8	Left	IV	Ear fullness, tinnitus	1 d	4	25.0	8.3	No	No	Yes	o		1	2		
9	Left	IV	Ear fullness	1 d	5	46.7	20.0	No	No	Yes	o	o	2	1		
10	Right	IV	Ear fullness	6 d	29	55.0	11.7	Yes	Yes	Yes	o		2	0		
11	Right	IT	Hearing loss, tinnitus	1 d	5	26.7	5.0	No	No	No			1	1		
12	Left	IV	Ear fullness	1 d	4	60.0	16.7	No	No	No	o	o	2	2		
13	Right	IV	Hyperacusis	2 d	6	43.3	25.0	No	Yes	Yes	o		2	2		
14	Right	IV	Tinnitus	1 wk	103	51.7	28.3	Yes	No	No	o		1	0		
15	Right	IV	Ear fullness	2 wk	2	41.7	5.0	No	No	No	o	o	1	0		
16	Left	IV	Hearing loss	1 wk	84	61.7	28.3	Yes	Yes	Yes	o		2	1		
17	Left	IV	Tinnitus	2 d	3	46.7	25.0	No	No	No	o	o	2	1		
18	Left	IT	Tinnitus	1 d	150	60.0	21.7	Yes	No	No	o		1	1		
19	Left	IV	Tinnitus	1 wk	14	71.7	45.0	Yes	No	No	o		2	2		
20	Right	IV	Tinnitus	1 mo	9	23.3	10.0	Yes	No	No	o		2	2		
21	Right	IV	Ear fullness	4 d	9	38.3	25.0	Yes	Yes	Yes	o		2	2		
22	Right	IV	Tinnitus	3–5 d	5	43.3	11.7	No	Yes	Yes	o	o	1	2		
23	Left	IV	Tinnitus	2 d	3	58.3	11.7	No	Yes	Yes	o	o	2	2		
24	Left	IV	Ear fullness	2 wk	2	38.3	16.7	No	No	No	o	o	2	2		
25	Left	IV	Ear fullness	3–5 d	8	25.0	5.0	Yes	No	No	o		0	2		

EH was graded as follows: 2 = significant EH, 1 = mild EH, and 0 = no EH. ^{*} Cases marked with o indicate they meet the criteria for cochlear MD, possible MD and SD, respectively, as well as ALHL. IT indicates intratympanic injection of gadolinium; IV, intravenous injection of gadolinium; Cochlear MD, cochlear Ménière's disease; Possible MD, possible Ménière's disease; SD, sudden deafness; Cochlear EH, cochlear endolymphatic hydrops; Vestibular EH, vestibular endolymphatic hydrops;

TABLE 4. EH on the affected ears of the acute low-tone sensorineural hearing loss patients

	Acute low-tone sensorineural hearing loss patients (n = 25%)	Definite (n = 18%)	Probable (n = 7%)	p value, definite versus probable
Cochlear EH				
Significant	15 (60.0)	10 (55.6)	5 (71.4)	
Mild	8 (32.0)	6 (33.3)	2 (28.6)	
No	2 (8.0)	2 (11.1)	0 (0)	<i>p</i> = 0.599
Vestibular EH				
Significant	16 (64.0)	13 (72.2)	3 (42.8)	
Mild	6 (24.0)	3 (16.7)	3 (42.8)	
No	3 (12.0)	2 (11.1)	1 (14.3)	<i>p</i> = 0.339

EH indicates endolymphatic hydrops.

23 ears (92%) and vestibular EH in 22 ears (88%). There was no difference between the frequency of EH in definite ALHL and probable ALHL.

The frequency of EH in the affected ear was compared with that of the contralateral ear in 20 ALHL patients using intravenous Gd injection (Table 5). Cochlear EH was found to be more frequent on the affected side than the contralateral side (18 ears [90%] versus 8 ears [40%], respectively), and EH was more severe on the affected side than on the contralateral side. Vestibular EH was observed on both the affected and contralateral sides. There was no difference in frequency of cochlear or vestibular EH, regardless of the recurrence (Table 6).

Figure 1 shows a HYDROPS image of a 56-year-old woman taken 4 hours after intravenous Gd injection. She experienced right aural fullness and hearing loss, with slight dizziness. MRI revealed significant EH in the right cochlea and no EH in the contralateral cochlea. Vestibular EH was also recognized in both ears and was more significant in the affected ear.

DISCUSSION

Here, we summarize the imaging of the endolymphatic space in ALHL patients. Cochlear and vestibular EH are frequently revealed using 3T MRI obtained 24 hours after intratympanic injection or 4 hours after intravenous injection of Gd. Our results revealed that cochlear and vestibular EH were observed in 92% and 88%, respectively, in ALHL. In Ménière's disease, a previous study

showed that cochlear and vestibular hydrops were revealed in 64% and 100%, respectively (14). EH is frequently observed in ALHL as in Ménière's disease, which may imply that ALHL is associated with EH. In this study, patients with "definite" ALHL and "probable" ALHL showed differences in age and hearing levels but no differences in other characteristics or frequency of cochlear or vestibular EH.

Some reports have suggested that ALHL has similar functional abnormalities to those of Ménière's disease, which is commonly believed to be caused by EH. Positive glycerol test results were found in 36% to 74% of ALHL patients, and the negative summing potential/action potential (-SP/AP) ratio in ECoG was abnormally increased in 63% (3,5). By contrast, positive glycerol tests were found in about half of patients with Ménière's disease (15,16), and abnormally increased -SP/AP ratios were detected in 42% to 75% (17). These studies also suggested a strong connection between ALHL and EH.

In our study, vestibular EH was observed in 88% of patients, whereas only 36% of our subjects were aware of dizziness. The usefulness of the vestibular evoked myogenic potentials (VEMP) test for detecting asymptomatic saccular hydrops has been reported (18). However, the VEMP test has never been evaluated in a substantial number of ALHL patients. Asymptomatic EH of the vestibular may precede clinical symptoms or functional impairment in ALHL (19). It has been reported that approximately 30% of ALHL patients eventually developed classical Ménière's disease over a 3-year period (3).

We observed that the affected ear has cochlear EH more frequently than the contralateral side (90% versus

TABLE 5. Comparison of endolymphatic hydrops Between the affected and contralateral ears

	Affected ear (n = 20%)	Contralateral ear (n=20%)	p value
Cochlear EH			
Significant	13 (65.0)	7 (35.0)	
Mild	5 (25.0)	1 (5.0)	
No	2 (10.0)	12 (60.0)	<i>p</i> = 0.003
Vestibular EH			
Significant	13 (65.0)	9 (45.0)	
Mild	4 (20.0)	8 (40.0)	
No	3 (15.0)	3 (15.0)	<i>p</i> = 0.357

TABLE 6. Comparison of endolymphatic hydrops between the ears with or without recurrent hearing loss

	Recurrent (n = 9%)	Nonrecurrent (n = 16%)	p value
Cochlear EH			
Significant	5 (55.6)	10 (62.5)	
Mild	3 (33.3)	5 (31.3)	
No	1 (11.1)	1 (6.3)	<i>p</i> = 0.894
Vestibular EH			
Significant	5 (55.6)	11 (68.8)	
Mild	2 (22.2)	4 (25.0)	
No	2 (22.2)	1 (6.3)	<i>p</i> = 0.407

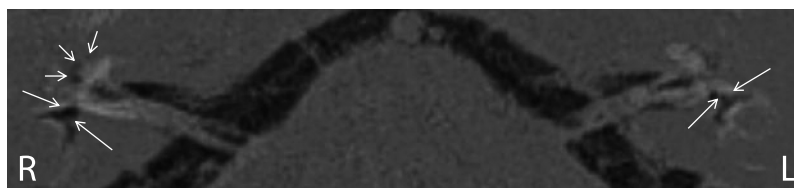


FIG. 1. MRI findings after 4 hours of intravenous injection of gadolinium. A 56-year-old woman with ALHL in the right ear. HYDROPS (the hybrid of reversed image of positive endolymph signal and native image of positive perilymph signal) image was obtained 4 hours after intravenous gadolinium injection (8). Significant EH was observed in the right cochlea (*short arrows*), and no EH was observed in the cochlea of contralateral ear. Vestibular EH was also recognized in both of the ear (*long arrows*).

40%). This finding suggests that ALHL is significantly associated with EH. Histopathologic examinations of the temporal bone in unilateral Ménière's disease revealed that more advanced EH involving the cochlea and the otolithic organs is seen in symptomatic ears than in asymptomatic ears (20,21), which is similar to the laterality of cochlear EH revealed by MRI in the ALHL cases in our study.

Fluctuating hearing loss without vertigo is frequently termed *cochlear Ménière's disease*. In 1972, the American Academy of Ophthalmology and Otolaryngology subcommittee on equilibrium and its measurement defined atypical Ménière's disease involving fluctuating hearing loss without vertigo as cochlear Ménière's disease (11). Recurrent ALHL can be regarded as "cochlear Ménière's disease." Of 9 cases that met the criteria, 8 cases had cochlear EH, and 7 cases had vestibular EH. Some reports have shown that cochlear Ménière's disease is associated with EH (22–24). Transtympanic ECoG revealed that 67% of ears with cochlear Ménière's disease demonstrated values consistent with EH (23). Recently, in 56 ears affected by cochlear Ménière's disease, 38 showed EH in the cochlea, and 44 showed EH in the vestibule (24). According to the most recent (1995) definition by the American Academy of Otolaryngology–Head and Neck Surgery (12), sensorineural hearing loss, fluctuating or fixed, with dysequilibrium but without definitive episodes is classified as possible Ménière's disease. Cases that met the criteria of possible Ménière's disease had both cochlear and vestibular EH except for 1 case that had only cochlear EH.

Meanwhile, SD is defined as the acute onset of hearing loss of 30 dB in 3 contiguous frequencies, generally without recurrence of hearing loss (13). Twelve cases of 25 in the present case series met the diagnostic criteria of SD as well, and they are considered as SD confined to low tone. Cochlear and vestibular EH were observed in 11 of 12 cases. It was reported that cochlear and vestibular EH were seen in 4 of 11 ears with SD in the temporal bone study (25). A MRI study also reported that EH was recognized in the cochlea and/or the vestibule in 4 of 7 ears with SD accompanying vertigo (26). In the present study, cochlear and vestibular EH were frequently observed in low-tone SD without vertigo. We evaluated our subjects by dividing them into 2 groups according to whether they experienced recurrence (Table 6). However,

MRI findings revealed no significant differences between the 2 groups, including in terms of frequency of cochlear and vestibular EH. This may imply that ALHL is associated with EH regardless of presence or absence of recurrence.

One interesting case in the current study was in a man with human parvovirus B19 (HP-B19) infection (Case 7 in Table 3). His only symptom was left ear fullness without vertigo or dizziness, and it occurred 10 days after his daughter had been diagnosed with erythema infectiosum. His average low-tone hearing level was 31.7 dB, and his HP-B19 IgM level was elevated. His symptoms were completely relieved 10 days later; on that day, 3T MRI 4 hours after intravenous Gd injection revealed significant cochlear EH on the affected side and bilateral vestibular EH. A previous study reported hearing loss with elevated HP-B19 IgG or IgM level (27). It has been reported that the immune complex produced locally may also block the focal capillary and cause endolymphatic sac dysfunction and EH formation after viral infection (28). In our patient, an immunologic reaction may have induced EH and hearing loss. Furthermore, we evaluated the state of the endolymphatic space shortly after the onset of hearing loss in this patient and found it to be similar to that in the other 24 subjects: cochlear EH on the affected side and bilateral vestibular EH. These findings indicate that EH may play an important role in the early stage of ALHL; however, more cases must be evaluated to confirm this.

ALHL is caused by EH, at least partly, and is distinguished from typical Ménière's disease in that ALHL patients do not experience vertigo. However, some have persistent tinnitus or ear fullness, and ALHL can recur. Data on clinical and pathophysiologic ALHL are limited, compared with data from patients with Ménière's disease, and further studies are required to elucidate the pathophysiology of ALHL more fully.

CONCLUSION

Cochlear and vestibular EH are frequently observed in patients with ALHL by 3T MRI after intratympanic or intravenous Gd injection. Cochlear EH was revealed more frequently on the affected side than on the contralateral side, which suggests that ALHL is closely associated with EH. MRI is a powerful tool with which to investigate the relationship between ALHL and EH.

REFERENCES

1. Abe T. Acute sensorineural hearing loss in low frequencies. *Otolaryngol Tokyo* 1982;54:385–92.
2. Enander A, Stahle J. Hearing in Meniere's disease: a study of pure-tone and audiograms in 334 patients. *Acta Otolaryngol* 1967;64:543–56.
3. Yamasoba T, Kikuchi S, Sugawara M, Yagi M, Harada T. Acute low-tone sensorineural hearing loss without vertigo. *Arch Otolaryngol Head Neck Surg* 1994;120:532–5.
4. Imamura S, Nozawa I, Imamura M, Murakami Y. Clinical observations on acute low-tone sensorineural hearing loss. Survey and analysis of 137 patients. *Ann Otol Rhinol Laryngol* 1997;106:746–50.
5. Nozawa I, Imamura S, Mizukoshi A, Honda H, Okamoto Y. Clinical study of acute low-tone sensorineural hearing loss: survey and analysis of glycerol test and orthostatic test. *Ann Otol Rhinol Laryngol* 2002;111:160–4.
6. Gottschlich S, Billings PB, Keithley EM, Weisman MH, Harris JP. Assessment of serum antibodies in patients with rapidly progressive sensorineural hearing loss and Ménière's disease. *Laryngoscope* 1995;105:1347–52.
7. Naganawa S, Satake H, Kawamura M, et al. Separate visualization of endolymphatic space, perilymphatic space and bone by a single pulse sequence; 3D-inversion recovery imaging utilizing real reconstruction after intratympanic Gd-DTPA administration at 3 Tesla. *Eur Radiol* 2008;18:920–4.
8. Naganawa S, Yamazaki M, Kawai H, et al. Imaging of Ménière's disease after intravenous administration of single-dose gadodiamide: utility of subtraction images with different inversion time. *Magn Reson Med* 2012;11:213–9.
9. Nakashima T, Naganawa S, Sugiura M, et al. Visualization of endolymphatic hydrops in patients with Meniere's disease. *Laryngoscope* 2007;117:415–20.
10. Nakashima T, Naganawa S, Pykkö I, et al. Grading of endolymphatic hydrops using magnetic resonance imaging. *Acta Otolaryngol* 2009;129:5–8.
11. Alford BR. Report of subcommittee on equilibrium and its measurement. Meniere's disease: criteria for diagnosis and evaluation of therapy for reporting. *Trans Am Acad Ophthalmol Otolaryngol* 1972;76:1462–4.
12. Committee on Hearing and Equilibrium. Committee on Hearing and Equilibrium guideline for the diagnosis and evaluation of therapy in Ménière's disease. *Otolaryngol Head Neck Surg* 1995;113:181–5.
13. Whitaker S. Idiopathic sudden hearing loss. *Am J Otol* 1980;1:180–3.
14. Tagaya M, Yamazaki M, Teranishi M, et al. Endolymphatic hydrops and blood-labyrinth barrier in Ménière's disease. *Acta Otolaryngol* 2011;131:474–9.
15. Ban JH, Lee JK, Jin SM, Lee KC. Glycerol pure tone audiometry and glycerol vestibular evoked myogenic potential: representing specific status of endolymphatic hydrops in the inner ear. *Eur Arch Otorhinolaryngol* 2007;264:1275–81.
16. Mori N, Asai A, Suizu Y, Ohta K, Matsunaga T. Comparison between electrocochleography and glycerol test in the diagnosis of Meniere's disease. *Scand Audiol* 1985;14:209–13.
17. Goin DW, Staller SJ, Asher DL, Mischke RE. Summating potential in Meniere's disease. *Laryngoscope* 1982;92:1383–9.
18. Lin MY, Timmer FC, Oriel BS, et al. Vestibular evoked myogenic potentials (VEMP) can detect asymptomatic saccular hydrops. *Laryngoscope* 2006;116:987–92.
19. Nakashima T, Sone M, Teranishi M, et al. A perspective from magnetic resonance imaging findings of the inner ear: relationships among cerebrospinal, ocular and inner ear fluids. *Auris Nasus Larynx* 2012;39:345–55.
20. Sperling NM, Paparella MM, Yoon TH, Zelterman D. Symptomatic versus asymptomatic endolymphatic hydrops: a histopathologic comparison. *Laryngoscope* 1993;103:277–85.
21. Kariya S, Cureoglu S, Fukushima H, et al. Histopathologic changes of contralateral human temporal bone in unilateral Ménière's disease. *Otol Neurotol* 2007;28:1063–8.
22. Teranishi M, Naganawa S, Katayama N, Sugiura M, Nakashima T. Image evaluation of endolymphatic space in fluctuating hearing loss without vertigo. *Eur Arch Otorhinolaryngol* 2009;266:1871–7.
23. Dornhoffer JL. Diagnosis of cochlear Ménière's disease with electrocochleography. *ORL J Otorhinolaryngol Relat Spec* 1998;60:301–5.
24. Kato M, Sugiura M, Shimono M, et al. Endolymphatic hydrops revealed by magnetic resonance imaging in patients with atypical Meniere's disease. *Acta Otolaryngol* 2013;133:123–9.
25. Yoon TH, Paparella MM, Schachern PA, Alleva M. Histopathology of sudden hearing loss. *Laryngoscope* 1990;100:707–15.
26. Chen X, Zhang XD, Gu X, Fang ZM, Zhang R. Endolymphatic space imaging in idiopathic sudden sensorineural hearing loss with vertigo. *Laryngoscope* 2012;122:2265–68.
27. Cotter CS, Singleton GT, Corman LC. Immune-mediated inner ear disease and parvovirus B19. *Laryngoscope* 1994;104:1235–9.
28. Pykkö I, Zou J. Do viruses cause inner ear disturbances? *ORL J Otorhinolaryngol Relat Spec* 2008;70:32–40.