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Contralateral delayed endolymphatic hydrops: Clinical features and long term outcome

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ABSTRACT

Background: Contralateral delayed endolymphatic hydrops (CDEH) is a clinical entity characterized by fluctuating low frequency hearing loss and/or vertigo, mimicking Ménière's disease (MD), that manifests after the appearance of severe non-hydropic hearing loss (NHHL) at the other ear.
Objectives: to describe the clinical features and the course of 57 patients affected by CDEH.
Method: this is a retrospective study; 57 patients affected by CDEH, out of 1065 patients seen in the same period and affected by MD, were subjected to otoscopy, PTA threshold evaluation, impedance testing, ABR, research of positioning nystagmus, vestibular function evaluated by means of bithermal caloric test under video-oculographic, and MRI with gadolinium.
Results: the CDEH was definite in 24 cases (42%), probable in 2 (4%) and possible in 31 (54%). The mean PTA threshold at the hydropic ear was 41 dB. At the last follow-up, 40 patients (70%) did not report vertigo or fluctuating hearing loss. Among the 17 patients who still reported symptomatology, 11 (64%) were affected by fluctuating hearing loss alone, 4 (23%) reported a subjective worsening of hearing loss and 2 (12%) an acute vertigo crisis.
Conclusions: contralateral delayed endolymphatic hydrops is a relatively rare form of Ménière disease that manifests more frequently as a definite form or with fluctuating low-frequency hearing loss. The prognosis at a long term follow-up is relatively good in terms of vertigo resolution. Contralateral delayed endolymphatic hydrops rarely determines a severe hearing loss in the better ear.

1. Introduction

Delayed endolymphatic hydrops (DEH) is a clinical entity characterized by fluctuating low frequency hearing loss and/or vertigo, mimicking Ménière's disease (MD), that manifests after the appearance of severe non-hydropic hearing loss (NHHL) in one ear (Kamei T et al., 1971; Wolfson RJ et al., 1975; Nadol JB et al., 1975; Shuknecht HF et al., 1978; Giannoni B et al., 1998; Albera R et al., 2016; Shuknecht HF et al., 1990). DEH is classified in two forms, respectively defined as ipsilateral delayed endolymphatic hydrops (IDEH) and controlateral delayed endolymphatic hydrops (CDEH) (Giannoni B et al., 1998). IDEH identifies a condition in which vertigo alone manifests after the appearance of a NHHL without involvement at the other ear. CDEH, instead, is a clinical condition characterized by a typical MD affecting the ear contralateral to the ear affected by NHHL (Kamei T et al., 1971; Wolfson RJ et al., 1975; Nadol JB et al., 1975; Shuknecht HF et al., 1978). The incidence of IDEH and CDEH are reported to be quite the same (Albera R et al., 2014; Reynard P et al., 2018; Albera R et al., 2019). Vertigo in DEH has the same characteristics of MD (Albera R et al., 2014); in the ear that causes vertigo, histopathological and MRI studies in CDEH showed modifications resembling those found in idiopathic MD (Shuknecht HF et al., 1978; Fukushima M et al., 2016).

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NHHL in CDEH has been referred to congenital and acquired forms and, in the latter group, the hearing suffering con be due to sudden hearing loss, head or acoustic trauma, ear surgery, viral infection, otitis media, streptomicin, meningitis, inner ear abnormality or idiopathic (Nadol JB et al., 1975; Casani A et al., 1993; Shojaku H et al., 2010). The age of appearance of NHHL is between birth and 70 and the interval between the two events ranges from 1 to 70 years (Wolfson RJ et al., 1975; Shuknecht HF et al., 1978; Giannoni B et al., 1998; Shuknecht HF et al., 1990; Casani A et al., 1993; Shojaku H et al., 2010).

The cause of the suffering of the second ear in CDEH has not been explained as yet. It has been suggested that it can be due to genetic mutation, inner ear malformations, viral infections or autoimmune disease, even if no markers have been found (Casani A et al., 1993; Berrettini S et al., 2016). The heterogeneity of the origin could explain the very wide range of symptoms attributed to DEH.

The development of hearing loss in the only hearing ear is a dramatic event, leading patients to be anxious. This condition must therefore be considered as an audiologic emergency (Berrettini S et al., 2016; Albera R et al., 2013).

The CDEH diagnostic criteria, proposed by the Japan Committee for Equilibrium Research in 1987 (Komatsuzaki A et al., 1987), state that it must be characterized by the delayed development of fluctuating hearing loss in the opposite ear after a NHHL and that it may be associated with episodic attacks of vertigo; central nervous pathologies and acoustic nerve lesions must be excluded. CDEH presents a slight prevalence in females (Albera R et al., 2014; Shojaku H et al., 2010) and 18–50% of cases manifests with fluctuating low frequency hearing loss only (Giannoni B et al., 1998; Shojaku H et al., 2010).

Vestibular tests are often normal, at least in the early stage, while in prolonged CDEH they can show vestibular suffering, like in MD (Lin MC et al., 2012).

Therapy proposed in treating CDEH is based on steroids (systemic or intratympanic), immunosuppressants, vasodilators, diuretics (Giannoni B et al., 1998; Berrettini S et al., 2016; Albera R et al., 2018) and Meniett device (Shojaku H et al., 2011) but results are uncertain and there is no evidence of efficacy. Sac surgery has been suggested to solve vertigo and, in case of bilateral hearing loss, hearing aids or cochlear implants must be considered (Berrettini S et al., 2016), while vestibular deafferentative surgery is rarely considered in order to avoid the risk of determining hearing worsening at the better ear (Canale A et al., 2018).

The aim of the paper was to describe the clinical features and the course of CDEH over time in a large sample of cases out of more than 1000 patients affected by MD with a long follow-up.

2. Materials and methods

2.1. Ethical concerns

All procedures performed in this study were in accordance with the ethical standards of our institutional research committee and with the 1964 Helsinki declaration and its later amendments. Informed consent was obtained from all participants included in this study.

2.2. Data collection

This is a retrospective study based on 57 patients affected by CDEH according to the Japanese criteria (Shojaku H et al., 2010), partly modified on the basis of the more recent guidelines in defining the characteristics of hearing loss (Chandrasekhar SS et al., 2019; Lopez-Escamez JA et al., 2015), consecutively seen at our department in the period 2005–2016. This sample represents 5.3% of 1065 patients seen in the same period and affected by MD according to AAO-HNS 1995 (Monsell EM et al., 1995), therefore comprising definite, probable and possible forms. In the overall sample, 37 patients (3.4%) were affected by IDEH.

Inclusion criteria were the presence of a unilateral preexisting moderate or severe sensorineural hearing loss associated with the appearance, at the opposite ear, of a low frequency fluctuating hearing loss without or with vertigo crisis resembling MD (Monsell EM et al., 1995; Lopez-Escamez JA et al., 2015). Patients affected by peripheral and central auditory/vestibular pathologies other than NHHL and CDEH were excluded from the study. Patients affected by bilateral MD were also excluded by the study. Patients affected by menieric vertigo alone in the presence of contralateral NHHL were also excluded since these forms are considered IDEH.

Clinical parameters evaluated were age, cause of hearing loss, disease duration, interval between hearing loss in the first ear and CDEH, type of symptoms (hearing loss alone or hearing loss and vertigo), contralateral ear condition, pure tone audiometry (PTA) threshold and evolution of the disease at follow-up, therapy and outcome. Data were collected at the time of the first visit to our Department and at each control.

Each patient was submitted to otoscopy, PTA threshold evaluation, impedance testing, ABR, research of positioning nystagmus, vestibular function evaluated by means of bithermal caloric test under video-oculography, MRI with gadolinium. MRI was performed in order to exclude cerebellar pontine angle pathologies. According to AAO-HNS 1995 (20), the mean PTA threshold was referred to the average value at 0,5-1-2-3 kHz.

At each control, the PTA threshold and caloric tests were repeated.

Treatment was based on oral steroid (prednisone 1 mg/kg/day) for 1 week at each acute episode of hearing loss and/or vertigo; intercritical treatment was based on a permanent hyposodic diet and cycles of oral diuretics at low dosage (clortalidone 12,5 mg/day).

Statistical analysis was carried out by means of SPSS package. The significance level was set at 0.05.

3. Results

Mean duration of the disease at the moment of diagnosis was 29 months, with a standard deviation (SD) of 67 and a range of 1–480 months. Mean age of the group was 59 years (range 23–85). 27 patients (47%) were male and 30 (53%) female. Mean follow-up was 87 months (range 24–180). The side affected by CDEH was the right one in 35 cases (61%) and the left in 22 (39%).

Hearing loss in the NHHL ear was more frequently idiopathic or due to sudden hearing loss (75% of cases); less frequently it was due to acoustic neuroma (1 case in follow-up, 1 case submitted to gamma-knife and 1 case operated on), to hearing loss that appeared in early childhood (ECHL), to chronic otitis (in 2 out of 3 cases after surgery), to trauma and to meningitis. In the adult idiopathic forms hearing loss evolved progressively over time, but in no case a diagnosis was done at the time of appearance and we have not found any certain explanation that could help us in the diagnosis.

Vestibular function at the NHHL resulted impaired in 5 patients affected by sudden hearing loss (20%), in 3 affected by acoustic neuroma (100%) and in 2 whose hearing loss was due to trauma (100%).

The mean PTA threshold at the NHHL ear was 80 dB (range 50–117) in the 35 patients where the threshold could be detected (value higher than 120 dB) at the frequencies considered (0.5–3 kHz). PTA threshold differences are not significant when comparing subjects affected by idiopathic, ECHL and sudden

hearing loss, the only groups with a relevant number of cases ($p > 0.05$ at the ANOVA test). In the other 22 cases (38%) it was not possible to evaluate the threshold with sufficient precision since the maximum level of the sound test was not heard in at least one of the four frequencies considered. In the group with a more severe hearing loss the cause was idiopathic in 8 cases, sudden hearing loss in 7, acoustic neuroma in 3, ECHL in 2, traumatic in 1 and chronic otitis operated on in 1.

Mean age of appearance of CDEH was 56 years (SD 14); the age differences in relation to the cause of NHHL are not significant ($p > 0.05$ at the ANOVA test applied to the more frequent forms) except in case of trauma in which the age was lower. However, the number of patients is too low to be representative (Table 1). Mean interval between NHHL and CDEH was 217 months (SD 216, range 82–876) and it is longer in ECHL than in the other conditions (Table 1).

At the ear affected by CDEH we have never found pathological modifications with otoscopy, impedance testing, functional vestibular tests, ABR and MRI.

According to AAO-HNS 1995 classification of MD (20), the CDEH was definite in 24 cases (42%), probable in 2 (4%) and possible in 31 (54%). In all the 31 possible forms (100%) the symptom was hearing loss. In the 24 definite forms vertigo and hearing loss appeared together in 17 cases (65%), hearing loss appeared as first symptom in 4 (24%) and vertigo in 2 (11%). In these 6 patients the mean interval between the appearance of the two symptoms was 16 months (SD 21, range 1–55).

As for the cause of NHHL ear the distribution of the definite, probable and possible forms appears similar in idiopathic hearing loss and ECHL while after sudden hearing loss we have found the prevalence of possible forms ($p < 0.05$ at the chi square test); in the other cases the number of patients is too low to obtain valuable information with the statistical analysis (Table 2).

The mean PTA threshold at the hydropic ear was 41 dB (SD14). Fig. 1 shows the mean PTA thresholds in relation to the cause of NHHL and compared with PTA in NHHL. Idiopathic and sudden hearing loss determined, at the hydropic ear, a similar degree of hearing loss ($p > 0.05$ at the Student's t-test) while in the other groups the degree of hearing loss was 5–10 dB higher, except in the case of meningitis, where the threshold was lower. However, the number in those cases in not sufficient to obtain any reliable statistical information.

At the last follow-up (24–180 months, average 87) 40 patients (70%) did not report vertigo or fluctuating hearing loss. Among the 17 patients who still reported symptomatology, 11 (64%) were affected by fluctuating hearing loss alone, 4 (23%) reported subjective worsening of hearing loss and 2 (12%) an acute vertigo crisis.

Outcome was not related to the cause of NHHL ($p > 0.05$ at the chi square test), to the diagnostic level, age, disease duration and PTA threshold at diagnosis ($p > 0.05$ at the Student's t-test).

The mean PTA threshold at the last control was 40 dB (SD 20), 1 dB less than the control carried out at diagnosis, and the difference is not significant at the Student's t-test for paired data ($p > 0.05$). In the 9 cases in which we have found the PTA threshold worsening more than 10 dB (Monsell EM et al., 1995; Lopez-Escamez JA et al., 2015) (16% of the entire sample), the mean value of worsening was 25 dB.

Table 2
Distribution of definite, probable and possible forms in relationship to the cause of hearing loss at the NHHL.

Cause of hearing loss at the NHHL	Definite	Probable	Possible
Hydiopathic (23 cases)	13 (56%)	0	10 (44%)
Sudden hearing loss (20 cases)	5 (25%)	2 (10%)	13 (65%)
ECHL (4 cases)	2 (50%)	0	2 (50%)
Acoustic neuroma (3 cases)	0	0	3 (100%)
Chronic otitis (3 cases)	3 (100%)	0	0
Trauma (2 cases)	0	0	2 (100%)
Meningitis (1 case)	0	0	1 (100%)

NHHL: non-hydropic hearing loss; ECHL: early childhood hearing loss.

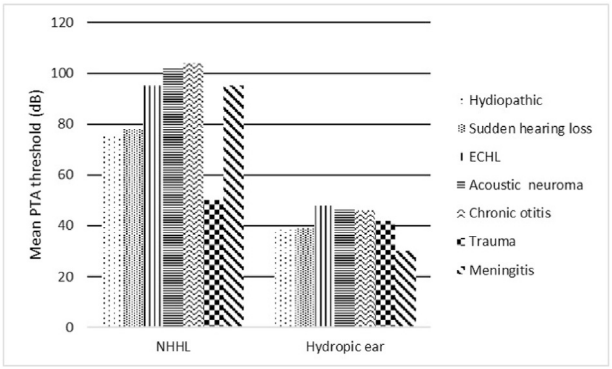

Fig. 1. Mean PTA threshold in NHHL and Hydropic ear in relation to the cause of HL in non-hydropic ear.
NHHL: non-hydropic hearing loss; ECHL: early childhood hearing loss.

4. Discussion

CDEH is an atypical form of MD that can be found in about 5% of the overall cases. This rate is the same we had described in a previous paper about the clinical features of MD (Albera R et al., 2014). In line with our previous report (Albera R et al., 2014) and with Shojaku (Shojaku H et al., 2010) CDEH is a little more frequent than IDEH.

In this study we have followed the Japanese guidelines (Komatsuzaki A et al., 1987), the only ones that until now have described CDEH as a clinical entity. As for the more recent guidelines of MD (Chandrasekhar SS et al., 2019; Lopez-Escamez JA et al., 2015) we have mainly adopted the oldest one (Monsell EM et al., 1995) since it better describes the monosymptomatic form of MD (possible MD), that is presented more frequently in CDEH.

Table 1
Clinical parameters regarding the ear affected by CDEH in relation to the cause of hearing loss at the NHHL.

Cause of hearing loss at the NHHL	Number of cases	Age of appearance of CDEH (years)	Interval between hearing loss at the first ear and CDEH (months)
Hydiopathic	23 (40%)	59 ± 9	248 ± 135
Sudden hearing loss	20 (35%)	56 ± 17	105 ± 119
ECHL	4 (8%)	56 ± 12	711 ± 116
Acoustic neuroma	3 (6%)	53 ± 11	120 ± 50
Chronic otitis	3 (6%)	57 ± 13	300 ± 94
Trauma	2 (3%)	33 ± 9	82 ± 3
Meningitis	1 (2%)	25	240

Plus-minus values are the mean ± standard deviation.
NHHL: non-hydropic hearing loss; CDEH: contralateral delayed endolymphatic hydrops; ECHL: early childhood hearing loss.

In the larger casuistry of CDEH (Shojaku H et al., 2010) until now published, the idiopathic hearing loss was the most frequent cause of deafness at the NHHL (63%), followed by sudden hearing loss (9%); among the idiopathic forms, hearing loss appeared principally in early childhood and similar data were reported by Casani (Casani A et al., 1993). In general, our results agree with these data but we have found a lower rate of ECHL, 15% of cases versus 85% of cases where hearing loss appeared in adulthood. The other causes of NHHL are similar in all the casuistries and are represented by otitis media, with or without surgery, meningitis and trauma. We have also found 3 cases in which the first ear was affected by acoustic neuroma, a pathology never reported until now. The lesion was left untreated in one case, submitted to gamma-knife therapy in one case and operated in one case.

In NHHL, the mean PTA threshold is situated in a moderate to severe range since it was within 50 and 117 dB in 35 patients and worse in 22. In these, it was not possible to accurately evaluate the threshold since it was higher than 120 dB at one frequency at least. We have not found any correlation between the PTA threshold and the cause of hearing loss in the idiopathic form and in case of sudden hearing loss. In the other forms of NHHL the deafness was more frequently severe.

The mean age of appearance of CDEH was 56 years without differences in relation to the cause of NHHL, at least in the more frequent pathologies. Shojaku (Shojaku H et al., 2010) reported similar data only in case of sudden hearing loss while in idiopathic forms the age of appearance of CDEH was much younger (28 years). A possible explanation could be that in our cases the ECHL forms are only 4, while in Shojuaku's casuistry (Shojaku H et al., 2010) they represented 44% of the overall sample. The delay between NHHL and CDEH is shorter in our casuistry if compared with Shojaku's data (Shojaku H et al., 2010) - 18 versus 27 years - while our data are similar to the values described by Giannoni (Giannoni B et al., 1998) and Casani (Casani A et al., 1993). A possible explanation of the shorter delay found in our sample when compared to Shojaku's observations (Shojaku H et al., 2010) may be the low number of ECHL that has a longer delay.

As for the clinical manifestation of CDEH, Casani (Casani A et al., 1993) described cases with the association of hearing loss ad vertigo only (definite forms according to Chandrasekhar SS et al., 2019; Lopez-Escamez JA et al., 2015), while Shojaku (Shojaku H et al., 2010) reported that the two symptoms are associated in 83% of cases and Giannoni (Giannoni B et al., 1998) stated that in literature vertigo could be occasionally associated with hearing loss, which should be the main symptom in CDEH. In our sample, at the moment of diagnosis at our Department 1–480 months after the appearance of symptomatology (mean 29 months) - the association of the two symptoms is present in 46% of cases (definite form Chandrasekhar SS et al., 2019; Lopez-Escamez JA et al., 2015) while in the remaining 54% of cases hearing loss was the only symptom reported by patients (possible form according to 1995 guidelines (Monsell EM et al., 1995). We therefore think that the clinical pattern could be better described by the 1995 guideline (Monsell EM et al., 1995).

As regards the evolution of the disease, we had previously demonstrated that the shift from a possible to a definite MD, according to the 1995 guidelines (Monsell EM et al., 1995), may occur if we wait enough time, but this is not the rule. Five years after the appearance of the first symptom, the appearance of the second symptom is a rare event (Albera R et al., 2014). Since the mean follow-up in this study was 7 years and we have observed no case who presented the evolution from the possible to the definite form, we can hypothesize that in about half of the cases the only symptom of CDEH is and remains hearing loss. We have only 6 patients who reported, before diagnosis, the beginning of the disease with one symptom, while the second symptom emerged later, with a mean delay of 16 months. Therefore, in accordance with our previous observation, the delay between the appearance of the first and the second symptom is shorter than in the idiopathic MD and the first symptom is more frequently hearing loss. It is interesting to note that the 2 patients affected by vertigo alone would have been diagnosed as IDEH if seen before the appearance of hearing loss; therefore, it is necessary to pay attention and wait enough time before suggesting a deafferentative therapy in case of DEH (Shuknecht HF et al., 1978).

Hearing loss in CDEH in our sample never reaches a severe degree, being situated in a middle range of hearing loss; this is important information since patients already have a severe hearing loss in the other ear and the appearance of an issue at the safe ear always determines an anxious response (Albera R et al., 2013).

The long term control was encouraging since both hearing decline and hearing fluctuation were found in a low rate of cases and even vertigo attacks only happened in a few cases. In conclusion, our data suggest that CDEH rarely determines a severe hearing loss in the better ear and a disabling vertigo; this is new information because previous reports did not evaluate the evolution of the disease over time.

As for therapy we have treated patients according to the more accepted treatment of MD suggested by literature (Giannoni B et al., 1998; Berrettini S et al., 2016; Albera R et al., 2018). However, until now there is no strong evidence about the efficacy of medical therapy in MD. In no case we have suggested intratympanic gentamicin and/or vestibular neurectomy because vertigo is not the major problem in CDEH and in order to avoid the risk of increasing hearing loss in the better ear (Canale A et al., 2018).

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Declaration of competing interest

None.

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