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► To cite this version:

Arnaud Attyé, Michael Eliezer. Endolymph magnetic resonance imaging: Contribution of saccule and utricle analysis in the management of patients with sensorineural ear disorders. *European Annals of Otorhinolaryngology, Head and Neck Diseases*, 2020, 137 (1), pp.47 - 51. 10.1016/j.anorl.2019.11.001 . hal-03490051

HAL Id: hal-03490051

<https://hal.science/hal-03490051>

Submitted on 21 Jul 2022

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Endolymph magnetic resonance imaging: contribution of saccule and utricle analysis in the management of patients with sensorineural ear disorders

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Funding information

The study was supported by Guerbet SA for MRI in healthy subjects.

Abstract

Endolymphatic hydrops features excess endolymph in the membranous labyrinth, and is a marker of Menière's disease.

Between the early 1980s and late 2000s, MRI in Menière's disease aimed purely to rule out tumor or malformation as differential diagnoses for the pressure disorder. Progress in high-resolution MRI, however, now enables excess endolymph to be visualized in the membranous labyrinth, differentiating saccule and utricle in Menière's disease and in other clinical presentations such as cochleovestibular schwannoma. More recently, non-visibility of the saccule was demonstrated in a subgroup of Menière's disease patients, and utricle atelectasis in case of uni- or bi-lateral vestibular areflexia.

Endolymph quantification remains highly controversial in terms of grading approach, but a simple semiology based on excess or deficient visualization of endolymph according to the compartment sheds light on the pathophysiological mechanisms of cochleovestibular disorder and may in future allow effective monitoring of medical and surgical treatment.

Key-words

MRI; endolymphatic hydrops; saccule; utricle; Menière's disease

Introduction

In 1938, Hallpike and Cairns reported membranous labyrinth distension on histology in Menière's disease [1]. Altmann and Fowler examined 29 temporal bones and reported systematic distension of the endolymphatic compartment [2]. A recent meta-analysis of histologic studies of temporal bones in Menière's disease found systematic dilation of the cochlear duct, dilation of the saccule in 80% of cases, of the utricle in 55% and of the ampulla in 13% [3].

The condition begins in the cochlear duct and progressively extends to the saccule via the ductus reuniens. The dilated saccule comes in contact with the stapes footplate in 60% of cases.

Only in 1999, however, did Counter et al. [4] apply 4.7 T gadolinium-enhanced MRI to the guinea pig endolymphatic compartment [5]. The contrast medium crossed the blood-perilymph barrier to spread preferentially toward the perilymph. This barrier resembles the blood-brain barrier, allowing passage of low-molecular-weight molecules: taurine, mannitol, steroidal hormones, and gadolinium [6,7]. The blood-endolymph and perilymph-endolymph barriers, in contrast, are impermeable for gadolinium. The preferential passage of gadolinium enhances contrast between the impregnated perilymph, in hypersignal on MRI inversion recovery pulse sequences, and the endolymph, in hyposignal. In 2006, Naganawa et al. performed the first-in-man study, showing optimal enhancement of perilymphatic structures on an inversion recovery pulse sequence 4 hours after intravenous gadolinium injection [6]; it was not, however, possible to distinguish the perilymphatic and endolymphatic compartments. Subsequently, the same team were able to make this distinction, by intratympanic injection of diluted gadolinium [8,9], which diffused progressively through the

round and oval windows into the perilymph, peaking at 24 hours. This technique has the advantage of providing contrast between structures, but the drawback of requiring 24 hours between injection and scan. Moreover, labyrinth inflammation has been reported at high intratympanic doses in animals [10].

With improvements in imaging, endolymphatic structures can now be visualized on 3D-FLAIR sequences (commonly used for brain studies with T2 signal, canceling the cerebrospinal fluid signal) after double-dose intravenous injection or, more recently, with a single gadolinium dose [11,12]. For endolymph grading, the Japanese team advocated semi-quantitative assessment to distinguish between endolymph on the one hand in the cochlea, with no hydrops if the Reissner membrane is not displaced, moderate hydrops in case of displacement leading to a cochlear duct area not exceeding that of the vestibular ramp, and severe hydrops when the endolymphatic cochlear duct area exceeds that of the cochlear vestibular ramp, and on the other hand in the vestibule, with no hydrops if <33% of the endolymph is in the vestibule, moderate hydrops for 33-50% and severe hydrops for >50% [13]. This classification was modified in an article in the American Journal of Neuroradiology, replacing Reissner membrane displacement in the case of the cochlear endolymph by partial vestibular ramp obstruction, and defining vestibular hydrops as grade 1 for >50% of the endolymph within the vestibule and grade 2 as total loss of peripheral perilymph visibility [14].

More recently, endolymph imaging has been performed without contrast injection, using high-resolution T2 heavily weighted sequences [15,16] for direct study of the saccule walls in T2 hyposignal, so as to measure displacement.

The present review analyzed articles studying deficient or excess endolymphatic fluid on MRI, and clinical correlations. The PRISMA guidelines for systematic reviews and meta-

analyses were used, tracking definition of participants, type of intervention, main endpoint, and type of study.

We selected only articles including labyrinth MRI in control subjects and cochleovestibular patients, differentiating the utricle and saccule in the MRI methodology estimating deficit or excess of endolymph.

A structured PubMed search was performed March 3, 2019, using the search-terms “MRI” and “Endolymphatic Hydrops”, retrieving 415 articles. The 170 articles published since 2013 were selected. After reading titles and Abstracts, 56 articles appeared relevant. Those not differentiating utricle and saccule for endolymph visualization were excluded, leaving the 11 articles found in the present Discussion and Table 1. Other articles referred to in the present review are mentioned for explanatory reasons.

Discussion

Imaging excess endolymph: “primary” and “secondary” hydrops

Nagoya’s semi-quantitative grading, modified by Baráth et al., did not distinguish saccule and utricle in assessing hydrops [13,14]. Two later classifications made the distinction.

SURI (Saccule to Utricle Ratio Inversion) [17] defines saccular hydrops according to the utricle, for 2 reasons: biomechanical difference, the saccule wall being more compliant than the utricle; and variation between MRI scanners, with utricle and saccule areas varying concomitantly when the biophysical parameters of the 3D-FLAIR sequence are modified [18]. Saccular hydrops was therefore defined by size equal to or greater than the utricle on 2 reference slices. However, saccular hydrops was found in only 50% of Menière’s disease

patients, and depends on hearing threshold [19], being systematic in low-frequency sensorineural hearing loss exceeding 40 dB.

The main advantage of this classification was its perfect specificity, with no SURI in healthy subjects. Like in experimental animal models, it was assumed that when saccular compliance is exceeded cochlear lesions occur. However, endolymph can also be secreted by saccular dark cells, and cochlear spiral ligament cell lesions seemed to preexist hydrops in animal models of Menière's disease [20]. Nevertheless, the SURI score had the drawback of poor sensitivity in proven Menière's disease.

Bernaerts et al.'s classification [21] used the semi-quantitative cochlear grading proposed by Baráth et al., adding an extra grade based on SURI to the Japanese classification, increasing both specificity and sensitivity in Menière's disease patients. The authors also advocated assessing blood-labyrinth barrier permeability as an associated criterion, as patients with unilateral Menière's disease show contrast uptake in the basal turn of the cochlea associated with hydrops itself [21–23]. Interestingly, an Italian team studied anatomic variants in utricle and saccule position in healthy subjects, and found a minority variant of ovoid saccule [24].

Recently, high-resolution 3D-T2 rather than 3D-FLAIR sequences were tested, having the advantage of not requiring contrast injection [15,16]. Venkatasamy et al. reported that diagnosis of endolymphatic hydrops on 3D-T2 can be based on saccular height and width in the coronal plane, with 84% sensitivity and 100% specificity [16]. However, this method studies only the saccule and cannot assess contrast medium uptake. Moreover, several studies reported that, even if the utricle shows only slight dilation, it may protrude into the lateral semicircular canal in Menière's disease, which cannot be studied on T2-weighted sequences [25].

In vestibular schwannoma, elevated perilymphatic protein induces a fall in signal in the perilymphatic compartment, spontaneously contrasting with the endolymph, which still shows normal hyperintensity on T2-weighted sequences [26]. This signal reduction is visible only when the vestibular schwannoma completely obstructs the inner ear canal. A study using high-resolution T2-weighted sequences found a correlation between hearing loss and utricular hydrops [27]. 3D-FLAIR sequences without injection can also diagnose utricular hydrops in case of obstructive schwannoma [28].

Imaging endolymph deficit: intralabyrinth fistula and utricular atelectasis

A case-control study of Menière's disease according to the 2015 AAO-HNS criteria reported saccule non-visibility not only ipsilaterally but sometimes contralaterally to the clinically affected ear [17]; this abnormality had never been found in control subjects. The authors hypothesized an endolymphatic fistula allowing gadolinium to pass between perilymph and saccule, as suggested by Kimura and Schuknecht in 1975 [29]. In contrast to saccular hydrops, saccule non-visibility did not seem to correlate with the degree of hearing loss, 2 patients showing the abnormality on the contralateral side.

It is also possible that saccule non-visibility is due to pars inferior collapse, as seen on histology in various malformation syndromes and secondary degeneration phenomena [4].

Prospective studies will be needed to clarify the relation between saccule visibility and cochleovestibular symptoms.

In vestibular areflexia, isolated atelectasis of the pars superior (utricle and ampullae) was recently shown on MRI [30]. Functional exploration confirmed imaging, with no response of utricle or ampullae. In 1988, Merchant and Schuknecht found in a histologic study that 8 out

of 213 subjects showed utricle and ampulla atelectasis, with two clinical presentations [31]: paroxysmal, with vertigo episodes resembling vestibular “neuritis” without auditory or neurologic signs, and insidious, with indeterminate symptom onset. In both cases, patients reported chronic imbalance with instability or transient dizziness.

Contribution of MRI to understanding the pathophysiology of pressure disorders

As radiologic studies show, endolymphatic hydrops mainly involves the pars inferior (sacculae and cochlear duct), which have a different drainage from the pars superior (utricle and ampullae) [32]. The pars superior seems to be less concerned, thanks to the utricle-endolymph Bast’s valve [33]. The endolymphatic sac drains the endolymph through the vestibular aqueduct, as seen in the experimental model of endolymphatic hydrops developed by Kimura and Schuknecht, consisting in occluding the aqueduct [34]. In Menière’s disease there is, moreover, a sharp reduction in aqueduct wall visibility on 3D-FLAIR sequences, reinforcing this theory of abnormal longitudinal flow [35]. Their study is especially interesting as the aqueduct wall lesion, presumed to involve calcification of chondroblasts as shown histologically [36] and on cone-beam CT [37], was bilateral in patients with clinically unilateral disease.

Perilymph secretion may have two different sources: 1) plasma, via the blood-perilymph barrier, or 2) cerebrospinal fluid, via the cochlear aqueduct when it is permeable. The cochlear aqueduct provides rapid adaptation of perilymphatic pressure to sudden changes in cerebrospinal fluid pressure. When it is obstructed, as in 70% of cases, pressure equilibrium is impaired and delayed, as change in cerebrospinal fluid pressure is first transmitted to the endolymphatic sac, then to the endolymph, and on to the perilymphatic fluid [32].

In case of low intracranial pressure, there may be low perilymphatic pressure, and hence ex-vacuo endolymphatic hydrops [38], although this association between intracranial hypotension and MRI evidence of endolymphatic hydrops was not demonstrated by Attyé et al. [39]. The same is true for transiently elevated intracranial pressure (as during sneezing or nose-blowing), inducing round window rupture and thus perilymph leakage within the middle ear and consequent endolymphatic hydrops by lowered perilymphatic pressure [40]; to date, however, no radiology studies have reported any such association.

Conclusion

MRI studies provide precise anatomic information differentiating saccule and utricle involvement in several clinical presentations (Figure 1). Good radio-anatomic knowledge by otologists and improved reliability of MRI sequences should improve overall management of patients with labyrinth disorder.

Conflicts of interest

The authors received funding from Guerbet and Bayer, invested solely in MRI examination of healthy subjects. They have no other interests to disclose.

Table 1: MRI studies distinguishing utricle and saccule in their analytic criteria

1 st author	Publication date	ref.	Clinical presentation
Simon	2017	[13]	Menière's disease
Venkatasamy	2017	[14]	Menière's disease
Attyé	2016	[16]	Menière's disease
Eliezer	2018	[17]	Menière's disease
Attyé	2018	[18]	Menière's disease
Bernaerts	2019	[20]	Menière's disease
Eliezer	2018	[22]	Menière's disease
Conte	2018	[23]	Asymptomatic
Eliezer	2019	[26]	Vestibular schwannoma
Eliezer	2018	[29]	Vestibular areflexia
Attyé	2018	[34]	Menière's disease

Figure 1: Interpretation of utricle and saccule volume variations: a guide for otologists.

Upper line (A,B,C): diagrammatic 3D representation of labyrinth. E-J: 3D-FLAIR slices in healthy subjects (D and G), patients with endolymphatic hydrops (E and H) and patients with utricle atelectasis (F) and saccule fistula (I).

The utricle is shown by a white arrow, in normal position in image D and protruding into the non-ampullar part of the lateral semicircular canal in image E, resulting in canal hydrops. In image F, the utricle is only just visible, due to the atelectasis. The saccule is shown by a dotted white arrow, with normal volume (G), with positive SURI resulting in saccular hydrops (H), and non-visible (I). For information, posterior semicircular canal ampullae are shown by dotted gray arrows.

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