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A high jugular bulb and poor development of perivestibular aqueductal air cells are not the cause of endolymphatic hydrops in patients with Ménière's disease

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ABSTRACT

Objective: The presence of endolymphatic hydrops in the inner ear, which can be detected with gadolinium-enhanced magnetic resonance imaging (Gd-MRI), is widely recognized as the main pathological cause of Ménière's disease (MD). However, the precise mechanisms underlying the development of endolymphatic hydrops remains unclear. One hypothesis proposes a relationship between the presence of a high jugular bulb (HJB) and MD, which disrupts the vestibular aqueduct leading to the development of endolymphatic hydrops. This study sought to identify anatomical features in MD patients using computed tomography (CT) images of the temporal bone.
Methods: Fifty-nine MD patients meeting the AAO-HNS diagnostic criteria and exhibiting endolymphatic hydrops in Gd-MRI were enrolled between July 2009 and December 2015. We only included MD patients who showed unilateral endolymphatic hydrops in Gd-MRI. Sixty-six patients with otosclerosis or facial palsy were also enrolled as control participants. In both groups, patients with other pathologies (e.g., chronic otitis media or cholesteatoma) and patients <16 years old were excluded. HJB was defined as a JB that was observable in the axial CT image at the level where the round window could be visualized. JB surface area was measured on the axial image at the level where the foramen spinosum could be visualized. Finally, to investigate the relationship between the pneumatization of perivestibular aqueductal air cells and the existence of endolymphatic hydrops, the development of the air cells was rated using a three-grade evaluation system and the distance between the posterior semicircular canal (PSCC) and the posterior fossa dura was measured.
Results: The presence of HJB was observed in 22 of 59 affected sides of MD patients and in 17 healthy sides. The likelihood that HJB was detected on an affected side (22/39) was not significantly above chance (50%). The HJB detection rate did not significantly differ between the three groups (MD affected side, MD healthy side, and control patients). Furthermore, there were no significant group differences in JB surface area, distance between the PSCC and posterior fossa dura, or the development of perivestibular aqueductal air cells.

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Conclusion: We did not find any relationship between the anatomy of the temporal bones and the existence of endolymphatic hydrops. Moreover, we found no evidence suggesting that HJB or poor development of perivestibular aqueductal air cells were the cause of endolymphatic hydrops in MD patients.

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1. Introduction

The presence of endolymphatic hydrops in the inner ear is widely recognized as a pathological cause of Ménière's disease (MD), as suggested by histopathological examinations of the temporal bone [1]. According to the diagnosis guidelines for MD, a true diagnosis requires the identification of vestibular and cochlear hydrops by cadaveric investigation [2]. However, confirmation via cadaveric investigation is not performed in most cases of MD diagnosis. Therefore, several attempts have been made to identify in vivo biomarkers of endolymphatic hydrops in the inner ear. These include the use of an electrocochleogram (EcochG) to obtain the dominant negative summating potential/action potential ratio (−SP/AP), the evaluation of the hearing improvement level using the glycerol test, and the measure of the vestibulo-ocular reflex (VOR) gain increase after the administration furosemide (i.e., furosemide test) [3]. Furthermore, it was recently reported that hydrops can also be detected by inner ear magnetic resonance imaging (MRI) following the intratympanic or intravenous administration of gadolinium (Gd-MRI) [4–6]. Current evidence suggests that Gd-MRI, compared with alternative methods, has the highest efficacy rate (about 90%) for the detection of endolymphatic hydrops [7]. Gd-MRI allows for the visualization of an enlarged endolymphatic space, which appears as low-intensity signal areas in MD patients. Although endolymphatic hydrops can be observed in MD patients, the precise causes of the phenomenon remain unclear and controversial.

MD typically develops unilaterally (i.e., in a single ear) suggesting that specific lateralized anatomical factors may trigger the emergence of hydrops. Indeed, several reports have suggested that the anatomy surrounding the temporal bone in MD patients plays a specific role. For instance, the morphology of the dural venous sinuses, such as the presence of a high jugular bulb (HJB) or a jugular bulb (JB) diverticulum, is highly variable across individuals. The anatomy of the dural venous sinuses is shaped by both the embryologic and postnatal development of the brain and by lifelong cerebral circulation. Anatomical abnormalities of the dural venous sinuses can extend into the middle and inner ear, and are associated with a variety of symptoms, including tonal or pulsatile tinnitus, vertigo, and hearing loss [8]. Wadin et al. reported that HJBs could be reliably identified with a high probability in MD patients using computed tomography (CT) to image the temporal bone [9] and suggested that HJB might induce hydrops by disturbing the vestibular aqueduct. Furthermore, Jarhrsdoerfer et al. surgically demonstrated that HJB could obstruct the vestibular aqueduct [10]. Other studies have previously reported poor development of perivestibular aqueductal air cells in MD patients, possibly caused by the presence of HJB [11–13].

The present study sought to identify anatomical features of the temporal bone that potentially underlie the emergence of endolymphatic hydrops in MD patients, by analyzing the CT images of MD patients in whom Gd-MRI had previously revealed unilateral endolymphatic hydrops.

2. Materials and methods

Patients diagnosed with unilateral definite MD (AAO-HNS) [2] participated in this study. All participants developed unilateral hearing impairment and had frequent episodes of definitive vertigo spells (at least once a month for the past 6 months on average, with a maximal interval of 2 months between episodes), as defined by the AAO-HNS diagnostic criteria [2]. All MD patients also exhibited endolymphatic hydrops in the inner ear, as evidenced by Gd-MRI scans obtained between July 2009 and December 2015 [5,6]. Patients with otosclerosis or facial palsy (without a diagnosis of MD) also participated in the present study as control participants. For both groups, cases with poor pneumatization in the mastoid cavity due to chronic otitis media, with cholesteatoma or other pathologies, and patients <16 years old were excluded from the present study. Furthermore, a single bilateral MD case and any MD patients who did not show endolymphatic hydrops in enhanced MRI were also excluded. This resulted in a final sample of 59 patients with MD (28 men, 31 women; median age 60 years; age range 33–79 years) and 66 control patients (132 ears) with either otosclerosis or facial palsy (33 men, 33 women; median age 44.5 years; age range 17–82 years). The present study was approved by the ethics committee at our hospital and was performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from all patients.

2.1. Gd-MRI

We used two types of gadolinium (Gd) administration: intratympanic and intravenous. The intratympanic method used Gadodiamide hydrate (Omniscan®) diluted eightfold in saline, injected into the affected tympanic cavity; MRI images were acquired 24 h after the injection [5]. The intravenous method used a double dose of gadoteridol (Prohance® 0.4 ml/kg), injected intravenously; MRI images were acquired 4 h after injection [6,14]. The intratympanic method was performed for three patients, and the intravenous method was performed for 56 patients. All MR images were obtained with a 3 T MRI unit (Sigma Excite HD 3T, GE Healthcare) using a two-dimensional fluid attenuated inversion recovery (2D-FLAIR) acquisition sequence for hydrops evaluation. 2 mm thick axial images were adjusted parallel to the anterior commissure-posterior commissure line in an identical manner to the standard routine for MR

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