

Increased intracranial tension and cochleovestibular symptoms: an observational clinical study

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Objectives

Meniere's disease is thought to be pathophysiologically due to increased pressure in the endolymphatic spaces leading to distortion of the sensory elements. As the inner ear fluids are in direct and indirect contact with cerebrospinal fluid (CSF), it was hypothesized that changes in CSF hydrodynamics could affect inner ear fluid pressures.

Patients and methods

This study was conducted on 150 patients presenting with benign increased intracranial tension diagnosed by Dandy's criteria and by radiological data. All patients were subjected to a detailed vertigo questionnaire and underwent comprehensive clinical, audiological, and vestibular testing to detect any vestibular abnormalities.

Results

Of the studied patients, 38 (25.3%) (34 females and four males) presented with audiovestibular symptoms: ear fullness and/or hearing loss, tinnitus, attacks of vertigo, and instability. Some patients reported atypical positional or motion-induced vertigo. A total of 13 patients presented with sensorineural hearing loss (SNHL). Clinical head impulse test (HIT) was positive in 30 patients, abnormal caloric test results in 24 patients, and 12 had positive Dix–Hallpike test results. All patients with audiovestibular symptoms were previously treated with betahistine (24 mg, three times a day) with poor response. Patients with abnormal test results were shifted to acetazolamide+betahistine. Thirty patients had a dramatic response on both audiological and vestibular manifestations.

Conclusion

Changes in CSF pressure significantly affect inner ear fluids in some patients. Symptoms and tests may mimic Meniere's disease, and we recommend evaluating patients with atypical Meniere's disease (MD), especially early bilateral affection and/or poor response to conventional therapy, for increased intracranial tension (ICT). Further testing of other cochlea-vestibular functions in these patients is under way.

Keywords:

cerebrospinal fluid, intracranial tension, Meniere's disease, vertigo

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Introduction

Proper function of the inner ear depends on the delicate balance between inner ear fluids. It is thought that symptoms can arise from the pressure differential between the endolymph and perilymph leading to distortion of the neural bearing membranes and thus dysfunction of the system. Abnormal endolymph accumulation is called endolymphatic hydrops and leads to the syndrome tagged Meniere's disease or variants thereof [1]. It can also lead to changes in middle ear compliance, cochlear microphonics, and DPOAE [2–7]. Perilymphatic pressure is directly transmitted to the endolymph via the thin Reissner's membrane; the pressure difference between endolymph and perilymph is less than 0.5 mmHg, and both perilymphatic and endolymphatic pressures can be considered equivalent to the inner ear pressure [8,9]. Intracranial pressure (ICP) has been shown to influence the perilymphatic pressure [10–12],

because the cranial cerebrospinal fluid (CSF) and inner ear intralabyrinthine fluid communicate through three routes: the vestibular aqueduct, the cochlear aqueduct, and the internal auditory canal [13,14]. Consequently, inner ear pressure can mirror ICP, and this may play a key role in inner ear pressure regulation and fluid homeostasis and may be related to inner ear diseases [15,16].

The hypothesis was whether differential changes in pressure of the inner ear fluids due to transmission of ICP via naturally occurring channels can mimic symptoms of endolymphatic hydrops. It may be then possible that in a subset of patients with clinically

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diagnosed Meniere's disease (MD), raised ICP can be transmitted to the inner ear and cause their symptoms [17,18].

Patients and methods

This study was conducted on 150 patients presenting with benign increased intracranial tension diagnosed by Dandy's criteria and by radiological data (clinical, fundus and MRI). All patients were subjected to a detailed audiovestibular questionnaire. In addition, they underwent a pure tone and speech audiometry, impedencemetry, and a comprehensive clinical and instrumental vestibular testing to detect any vestibular abnormalities.

Ethical approval

The study was approved by the Ain Shams University Faculty of Medicine IRB (Approval number ENT-7124/2014). The approval includes an informed consent, approved and regulated by the IRB.

Results

There were 92 females and 58 males (65% females) aged 6–67 (mean age 43) years. Of the studied patients, 38 (25.3%) (34 females and four males) presented with audiovestibular symptoms: ear fullness and/or hearing loss, tinnitus, attacks of vertigo, and instability. Some patients reported atypical positional or motion-induced vertigo. Some comorbidities were also recorded: hypertension (17), diabetes (9), and hyperlipidemia (6). A total of 80 (53.3%) patients had papilledema. However, in the group with audiovestibular symptoms, only five (13.1%) had papilledema (grade I/II).

Audiological evaluation

Pure tone audiometry

Pure tone and speech discrimination tests were performed. All results were normalized for age and comorbidities. There were 20 patients with normal hearing, and 13 with sensorineural hearing loss (SNHL) (four bilateral). The hearing loss was predominantly low tone in 8 [mean 32 dB (bilateral in 3)] and flat in 5 [mean 40 dB (1 bilateral)]. In five patients there was a mean conductive hearing loss of 23.5 dB (with no other causes).

Vestibular test

Clinical positive head impulse test (HIT) result was seen in 30 [video head impulse test (vHIT) was unavailable at the time] patients, abnormal caloric test results were seen in 24 [canal paresis (CP) 16 unilateral and eight bilaterally reduced] patients, and

12 patients had positive Dix–Hallpike test result, with four typical of posterior canal benign paroxysmal positional vertigo (BPPV) and eight geotropic variable. Other oculographic tests and CDP were all normal.

Management plan

The patients with cochleovestibular symptoms were referred to one of the authors (H.A.S.) who was blinded to the primary diagnosis. The clinical picture is suggestive of an atypical peripheral cochleovestibular lesion. The assumption was that it was some form of endolymphatic hydrops or Meniere's disease. The patients were started on betahistine (24 mg, three times a day) for a trial period of 4 weeks and asked to report back. All patients had a partial albeit unsatisfactory response with persistence of vestibular symptoms and ear fullness.

After the run-in period, acetazolamide was added as 250 mg, three times a day, for 2 weeks. In 30 patients, there was complete disappearance of the sense of fullness and of any vestibular symptoms. In one patient, the low tone loss was normalized. In the remaining eight patients, the sense of fullness disappeared with a subjective improvement of hearing (but no change on the audiological pattern). Vestibular symptoms were reported to be greatly diminished without any interference with daily activities.

The medications were continued for 8 weeks after which dosage reduction was tried (betahistine and acetazolamide twice daily). In 17 (44.7%) patients, symptoms recurred and we reverted back to the high dose. Twelve patients regained their favorable symptomatic response, whereas the remaining five had a persistence of their symptoms. In those patients, other symptoms and signs of uncontrolled IICP persisted, and they were planned for further neurosurgical management. We plan to review them after their drainage procedures.

Discussion

Inner ear pressure differentials can cause symptoms related to functional neurosensory disruption [19]. Induced changes in perilymphatic pressure can cause changes in measurable inner ear parameters such as DPOAE SP, CM, or AP/SP ratios by postural changes or by changes in middle ear pressure [2–7,20]. Due to the direct pressure transmission between endolymphatic and perilymphatic spaces, pressure changes in either system directly affects the whole inner ear. Endolymphatic hydrops (clinically manifested as MD) and perilymphatic hypertension

can thus potentially cause the same clinical manifestations. Due to the natural connections between the CSF spaces and the perilymph [endolymphatic duct, cochlear aqueduct and internal auditory meatus (IAM)], it is theoretically possible that in some patients changes in CSF pressure can be directly transmitted to the perilymph and cause EH-like disease [21–24]. In this cohort of 150 patients with BIH, there were 38 (25.3%) patients with audiovestibular symptoms. Testing suggested a peripheral cochleovestibular dysfunction. Thirty (78.9%) patients responded favorably to measures to lower intracranial fluid pressures using acetazolamide. There was a recurrence of symptoms on dosage reduction, suggesting a true positive effect of the drug in this subset of patients. Of notice is that the proportion of patients with audiovestibular symptoms with concomitant papilledema is much lower than the studied population suggesting a differential effect on the inner ear and eye due to hitherto unspecified causes.

Conclusion

Some patients with a clinical history suggestive of endolymphatic hydrops may have secondary perilymphatic hydrops due to increased ICP. This may in part explain the response of some patients with diagnosed MD to diuretics which may act by modulating ICP rather than acting directly on the inner ear [25–27]. Further diagnostic measures should then be warranted (MRI, fundus examination and perimetry, and possibly measurement of ICP) to check whether they can be liable to visual loss, CSF leaks, or other central neurological dysfunction [23].

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Conflicts of interest

There are no conflicts of interest.

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