Post-Concussion Endolymphatic Hydrops: The Case of the Falling Vent Cover

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Dr. Prosper Meniere, a celebrated physician, and surgeon was born in France in 1799 and lived until 1862. During his lifetime, he identified several otologic conditions. As the Physician-in-Chief of the famous Paris School for the Deaf, he was the first to suggest vertigo came from the ear and not from the cerebellum as classically accepted. His presentation at the French Academy of Science in 1861 was the earliest to describe patients with both hearing loss and vertigo as an ear disease. Although his colleagues accepted his ideas, they were not mainstream. It was not until 1937 when researchers discovered the pathology of what Prosper Meniere first described in 1861. Eventually, his writings became the basis for what is known as Meniere's disease (MD) and the more contemporary endolymphatic hydrops (ELH).

THE MECHANISM BEHIND MD AND ELH

Gurkov and colleagues describe the mechanism of ELH from studies using high-resolution magnetic resonance as a …" distension of the endolymphatic space of the inner ear into areas that are normally occupied by the perilymphatic space."[1] These changes result in biomechanical and biochemical actions disrupting the transfer function, resulting in modifications to the cochlear amplifier and the development of hearing loss. This distension can also occur in the cochlear duct/sacculus and the utricle and semicircular canals. In trauma cases, dysfunction of the endolymphatic passage, introduction of cellular debris disrupting endolymphatic drainage, changes to cell production, or the absorption of endolymph are implicated.[2]

Displacement of the apical portion of the cochlea due to its width and elasticity properties are most affected.[1] This appears to be the reason behind the development of low-frequency hearing loss. The greater the severity of basilar membrane dislocation, the more severe the disease and symptoms. In some cases of MD, hearing loss can become severe to profound, eventually resulting in cochlear implantation. Modern implant studies have shown hearing can be significantly improved without labyrinthectomy while reducing tinnitus and suppressing vertigo.[3]

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THE DIAGNOSIS

Contemporary studies identify MD as affecting 50-200 per 100,000 individuals, most prominent in people aged 40-60 years.[4] There is no single cause for MD, which in many cases remains idiopathic, taking months to years to diagnose. The diagnosis of MD is made clinically after ruling out other conditions producing vertigo, such as labyrinthitis, labyrinthine concussion, migraine, and BPPV, amongst others. Although vertigo is present in all these conditions, the treatments can be different. The American Academy of Otolaryngology - Head and Neck Surgery considers the MD diagnosis when symptoms include "spontaneous vertigo attacks lasting 20 minutes to 12 hours, with low to mid-frequency sensorineural hearing loss in the affected ear before, during, or after one of the episodes of vertigo."[4] The observation of fluctuating tinnitus or aural fullness may accompany these symptoms.

ELH is a disorder caused by an adverse event such as head trauma, ear infection, allergies, inner ear tumors, dehydration, ear surgery, or systemic disorders.[5] Although it is difficult to identify the causation in many cases, ear surgery and head trauma are relevant because of the event and timeline of the response. In head trauma, causations such as BPPV, labyrinthine concussion, vestibular nerve injury, damage to the otolithic mechanisms, and perilymphatic fistula also occur.[6] It is much less common to observe post-traumatic ELH.

ELH is characterized as cochlear hydrops, vestibular hydrops, or both. This is different than MD, which is well established as including an ELH component, but with a unique set of symptoms and disease course. For example, MD patients have bouts of severe vertigo with a time restriction, while ELH patients with hearing loss may or may not have vertigo. When present, it does not occur in discrete episodes but is persistent and at times characterized as dizziness. Further, in cases of ELH associated with trauma or ear surgery, symptoms can improve over one to two years post-event.[5]

Complicating factors include delayed ELH, which develops months to years following the observance of a moderate to severe unilateral non-hydropic hearing loss. In rare instances, the unaffected contralateral ear develops ELH with classic MD symptoms. The cause of the second ear has not been identified, although genetic mutation, inner ear malformations, viral or autoimmune disease are implicated.[7] In cases of cochlear hydrops with low-frequency fluctuating hearing loss, 33% of the patients develop MD.[8]

The primary difference between post-concussion ELH and MD is how they develop and the course of the disease. ELH may be primary or secondary. Primary idiopathic ELH is known as MD, and secondary ELH occurs due to an event such as trauma, inner ear disorders, or systemic diseases.[5]

Figure 1. HVAC Ceiling Vent Cover

Because both categories can present with findings of low-frequency fluctuating hearing loss, tinnitus, and dizziness or vertigo, distinguishing factors can be challenging to assess.

TREATMENTS

A variety of treatments have been proposed and utilized for MD over the years. Without exception, intratympanic interventions to prevent attacks and delay disease progression are reported as likely to be beneficial compared to other interventions, such as salt and caffeine restriction and betahistine.[9,10] Unfortunately, treatment efficacy is limited in many cases. However, symptom treatment, support, vestibular rehabilitation in stable individuals, and for some, CBT is crucial.

According to the Vestibular Disorders Association, goals for ELH patients include stabilizing the body's electrolyte levels, treating underlying conditions, medications to stabilize balance, improving general health and quality of life, or, if more persistent, endolymphatic decompression surgery.[5]

In medical-legal cases involving head and neck injuries, it is rare to observe post-concussion ELH. Frequently, damages to the audiovestibular mechanisms exist from injury but do not mimic MD or ELH. However, when ELH is identified early in the medical record with a negative history of hearing loss, tinnitus, ear fullness, or vertigo, the findings codify the nexus between the accident and the clinical findings.

THE CASE OF THE FALLING VENT COVER

The plaintiff is a 52-year-old female standing 5 feet, 7 inches, weighing 180 lbs. Her medical history includes type 2 diabetes mellitus, hypercholesterolemia, hypertension, bilateral carpal tunnel syndrome, allergic rhinitis, obesity, and depression. In addition, she reports having intermittent mild tinnitus for two years before the accident. However, a review of the records reveals no history of hearing loss, aural fullness, vertigo, migraine, vision loss, peripheral neuropathy, numbness, or other disorders requiring medications or treatment.

The plaintiff is at a conference facility for a friend's birthday on the day of the accident. She was standing next to her chair when, without warning, a 15-20 lb., 30 inches round HVAC vent cover shown in Figure 1 came off the ceiling and fell 15 feet, striking the plaintiff.

She is injured on the left side of the head, neck, and shoulder. Immediately she falls to the ground, and the paramedics are called to the scene. The EMT report identifies neck and shoulder pain and numbness in her left thumb. At that time, she denies headaches, loss of consciousness, memory issues, nausea, vomiting, numbness, or seizures.

The patient is transported by ambulance to the emergency room of the local hospital. The physician's chart notes reveal a direct blow to the left temporoparietal region of the head, left ear, left face and jaw, and left neck accompanied by moderate pain. Radiology of the head, neck, and shoulder reveals a negative brain CT, no acute fractures, or subluxations of the spine, but moderate central disc herniation of the C5/6, a C6/7 central disc bulge, and moderate cervical osteoarthritis. Review of Systems (ROS) revealed negative findings for loss of consciousness, hearing loss, vertigo, and tinnitus. The diagnoses were closed head injury and neck pain. The patient is examined, prescribed medication for pain and inflammation, provided educational materials and sent home to return in three days for a follow-up evaluation. On the third day post-accident, the patient reports the onset of chronic tinnitus in the left ear with increased neck pain. Her primary care physician sees her after that.

One month after the accident, the patient starts physical therapy for her moderate cervical spine injuries and neck pain. Two months after the accident, she was evaluated by an audiologist and ENT physician because of worsening tinnitus and decreased hearing in the left ear. The ENT examination is unremarkable. However, the audiological test results reveal a different story. Pure tone air and bone conduction thresholds indicate hearing sensitivity within normal limits for the right ear with a moderate to severe reverse curve sensorineural hearing loss in the left ear, shown in Figure 2. Speech reception thresholds were consistent with the audiograms bilaterally. Speech discrimination scores were 100% in the right ear and 76% in the left ear. Immittance testing revealed type A

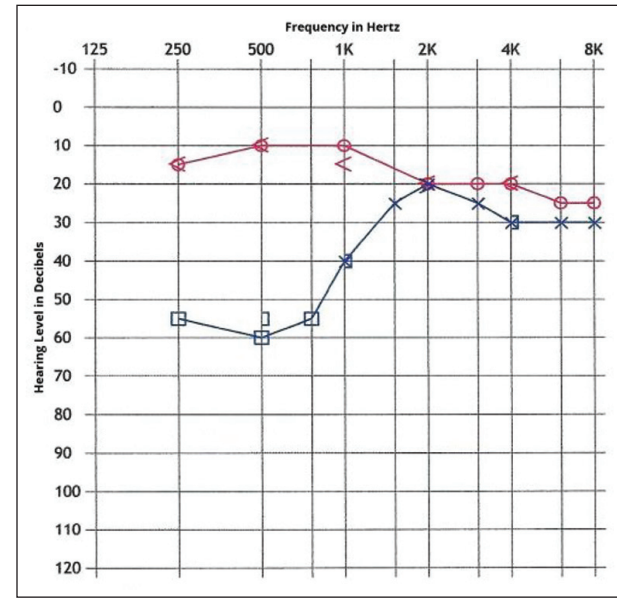

Figure 2. Post-Concussion Audiograms

tympanograms with ipsilateral acoustic reflexes present bilaterally. A tinnitus evaluation or LDLs were absent from the report. Subsequently, the attending audiologist fits the patient with mild amplification binaurally and starts tinnitus retraining therapy using the onboard white-noise generators. Two years after the accident, there were no further changes in hearing, and the tinnitus habituated. The patient continued to use her hearing aids in both ears thereafter.

In this case, the onset of unilateral chronic tinnitus started soon after the accident, but the plaintiff did not recognize left ear hearing loss until two months later. A delay in symptoms is not uncommon after head or neck trauma. Therefore, the delayed onset does not alter the connection between the injuries and the clinical findings. Nevertheless, the nexus between the accident, tinnitus, and hearing loss was evident. The final diagnosis of post-concussion ELH was determined based on a constellation of findings; 1. negative pre-accident history, 2. left side head, neck, and ear injuries, 3. chronic tinnitus localized to the left ear, 4. timeline of symptoms, and 5. reverse curve sensorineural hearing loss. Regardless of the injuries, the impairment, disability, and handicap, future treatments, rehabilitation, projected outcomes, and costs are essential to adjudication and settlement. The plaintiff was awarded damages for her hearing loss and tinnitus.