Traumatic Labyrinthine Concussion in a Patient with Sensorineural Hearing Loss

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SUMMARY – *Blunt head trauma without any temporal bone fracture or longitudinal temporal bone fracture, with an associated fracture of the labyrinth may cause labyrinthine injury with sensor neural hearing loss and vertigo because of a concussive injury to the membranous labyrinth. Sudden sensory neural hearing loss is relatively frequent. In most cases, the etiology is not discovered. One of the possible causes for sudden deafness is inner labyrinth bleeding or concussion, which were difficult to diagnose before the advent of magnetic resonance imaging. Vertigo without a demonstrable fracture may also be the result of labyrinthine concussion, cupulolithiasis and perilymphatic fistula. We describe the clinical case of a patient with acute traumatic hearing loss and vertigo, without skull base fracture detected on computed tomography. Magnetic resonance study was also performed. We have integrated the discussion with features that allow the differential diagnosis from other similar conditions.*

Introduction

Annually, 5% of the population sustain head injuries. Temporal bone trauma is frequent even if it is difficult to assess due to the diversity of clinical presentations and complex anatomy^{1,2}.

A head injury can lead to an isolated damage to the otolith organs (vestibular labyrinthine concussion) or to injuries to the labyrinth of the inner ear without damage to the otic capsule or intralabyrinthine limiting membranes (cochlear labyrinthine concussion)^{3,4}. Sudden sensory neural hearing loss (SSHL) caused by intralabyrinthine hemorrhage is extremely rare, and there are few reports in the literature⁵. Our study ruled out a variety of possible etiologies for post-traumatic auditory and vestibular dysfunction through radiological resources.

Excessive stapes footplate excursion can also cause hearing loss and similar pathophysiologic consequence, so differentiating concussion from a posttraumatic perilymphatic fistula (PLF) can be difficult. The features differentiating PLF from labyrinthine concussion are

episodic vertigo, movement-triggered vertigo, persistent unsteadiness and often nausea. The vertigo in these patients can last from several minutes up to several hours⁶.

Repair processes following trauma and infective, inflammatory or destructive insults to the otic capsule can cause labyrinthitis ossificans (LO) (sclerosing labyrinthitis) that can develop as fibrosis or membranous labyrinth ossification⁷. Magnetic resonance imaging (MRI) is better than computed tomography (CT) in the depiction of acute and fibrotic LO stages⁷. It may be difficult to differentiate a labyrinthine “concussion” from an eighth nerve stretch injury, although other testing modalities may help (e.g. VEMP).

Vertigo can be due to vestibule, vestibular nerves, or vestibular aqueduct transection, usually as the result of a transverse fracture^{8,9}.

On CT even the normal endolymph in the inner ear structures appears isodense mimicking pneumolabyrinth. The advent MRI made it possible to diagnose this condition with greater accuracy⁶. Concussion of the labyrinth provokes pathological alterations including circu-

Figure 1 FSE T2 thin section at the level of cerebellar pontine angles showed no morphological and/or signal alterations of internal auditory canals.

Figure 2 MIP image of 3D-FIESTA at the level of internal auditory canals fails to display the anterior, posterior and lateral semicircular canals bilaterally.

Figure 3 SE T1 imaging at the same level showed a hyperintense signal of labyrinth and cochlea, more evident on the right side.

Figure 4 3D-T1 FSPGR after contrast administration revealed enhancement of labyrinth and cochlea, more marked on the right side.

latory disturbances, impaired production and composition of labyrinthine fluid and problems with its outflow leading finally to hydrops of the labyrinth.

Case Report

A patient reached our attention for rapid onset hearing loss and postural imbalance 24 hours after traumatic head injury. He was awake, well-oriented and behaving normally. The first brain CT scan, with particular attention to the temporal bones, was performed: the patient did not have apparent brain lesions and/or fractures of temporal bones.

We cannot rule out a component of true vertigo as damage to the vestibular end organs or to the vestibular nuclei in the brainstem. Our patient presented symptoms persisting 48h after the trauma and his nystagmus has worsened over time¹⁰.

A specialist examination for dizziness was performed with past pointing, Romberg sign, gait ataxia and moving platform posturography, helpful to quantify balance deficits. We searched for “nystagmus” through electronystagmography, related to head and/or neck position or to vibration of the neck.

Clinical evaluation revealed that the tympanic membrane and the external auditory canal were normal, there was no hemotympanum noted on otoscopy or CSF in the external or middle ear because there was no associated temporal bone fracture. There was no purulent discharge or stapediovestibular dislocation with pneumolabyrinth. His facial nerve examination was intact bilaterally.

Pure tone audiometry showed severe bilateral sensorineural hearing loss. Vestibular assessment showed that the patient had an ataxic gait and we found a vertical nystagmus appearing in all positions of the head. There were additional minor organic basic abnormalities of

rhythm and amplitude. Using threshold stimulation and electronystagmography, we found abnormal post-traumatic responses quite different from those arising from a damaged labyrinth, and similar to those found in vertebrobasilar insufficiency¹¹. The patient presented benign positional vertigo as the sequel of blunt head trauma and labyrinthine concussion.

MRI evaluation of brain and internal auditory canal (IAC) was performed on a 1.5 T system (GE Signa Excite HD) with SE T1-weighted, FSE T2-w, GE T2*, FLAIR, DWI, and 3D FIESTA sequences. After gadolinium administration we acquired thin section SE T1 images with and without fat suppression and 3D-FSPGR with MPR and MIP.

MR examination did not show brain signal alterations. On FSE T2 thin section sequences (2 mm) no signal alterations of IACs were noted (Figure 1). On 3D-FIESTA sequence the anterior, lateral and posterior semicircular canals were not displayed in their entirety (Figure 2). On SE T1 a moderate hyperintense signal of labyrinth and cochlea were noted, especially on the right side (Figure 3). The 3D T1 FSPGR sequence, performed after contrast administration, revealed enhancement of labyrinth and cochlea, more marked on the right side (Figure 4).

Discussion

Auditory and vestibular symptoms are common after a closed head injury and have been attributed to labyrinthine concussion as well as damage to other central and peripheral vestibular pathways. The term "labyrinthine concussion" usually excludes temporal bone fracture, even if usage of the term sometimes includes these conditions¹².

When the patient reached our attention 24h after a head trauma, he presented a post-traumatic auditory and vestibular dysfunction, including benign paroxysmal positional vertigo precipitated by labyrinthine concussion or traumatic shearing of otoconia from otolith organs. Post-traumatic positional vertigo is more common after severe head injuries, with cranial vault or temporal bone fractures, in particular with longitudinal temporal bone fracture. The vestibular complaint is usually transient and it tends to subside, with spontaneous resolution occurring over a few days due to the regression of anatomic lesions and especially the establishment of central balance phenomena. In the case of labyrinthine commotion even hearing

loss can regress, at least partially. The literature suggests that recovery occurs in three to nine months in most individuals, but symptoms can persist for more than a year in ten to 15%⁹. Even if they are variable and not well-characterized, labyrinthine concussion symptoms generally include vertigo, tinnitus and sensorineural hearing loss particularly affecting high frequencies with recruitment. When present, the most common pattern of hearing loss is similar to that of a noise-induced hearing loss, with a loss that is most apparent at 4 kHz^{10,13}. A high frequency hearing loss, caused by cochlear concussion, is due to basilar membrane shearing, hair cell degeneration and eventual auditory nerve fiber avulsion⁶.

Examination of the patient with active symptoms may demonstrate a vestibular harmonic peripheral irritative or debilitative syndrome, i.e. a nystagmus directed towards the side of the lesion appearing acute for a few hours, followed by contralateral beating nystagmus with past pointing and falling in the direction of the nystagmus slow phase. Vestibular testing may demonstrate a reduction in the caloric response, vestibulo-ocular reflex gain abnormality and phase after rotational testing¹⁰.

No specific dizziness or vertigo is also a feature of the post-concussion syndrome that develops in up to 50% of people with minor head injury.

The prognosis for hearing function is worse than for vestibular symptoms⁸. In some cases, onset may be delayed by several days. These symptoms typically are short-lived and gradually subside over a period of days to weeks. In particular dizziness and nausea symptoms accompanying "concussion" usually resolve over six weeks, cognitive symptoms and headaches persist longer¹⁴. In many cases, chronic symptoms are psychological in origin. Balance symptoms generally resolve in ten days after concussion^{9,12}. If bleeding occurs by concussion without fracture, MRI may be indicated to confirm the diagnosis. The correct MR study should be performed 36-48 hours after the injury to allow the transformation of deoxyhemoglobin into methemoglobin, which has a bright signal in both T1 and T2 images. In addition, T1-weighted MRI is useful in the diagnosis of intralabyrinthine hemorrhage (ILH). In normal subjects, the perilymph and the endolymph do not have a T1 signal compared to the cerebrospinal fluid. ILH is more commonly found in the basal gyrus of the cochlea and next to the oval window. We ruled out a perilymphatic fistula

because the patient did not have oval or round window rupture or shock damage to hair cells. We did not find fracture of the otic capsule or central auditory pathway injury. We assumed that the patient had bleeding within the lumen of the inner ear structures after trauma, because labyrinth and cochlea appeared moderately hyperintense on SE T1 with enhancement after contrast administration. The disappearance of semicircular canals can be attributed to the arrest of lymphatic flow.

Bleeding occurred by concussion without actual fracture. ILH has also been described in patients with aplastic anemia, sickle-cell disease, leukemia, following surgery for the treatment of vestibular schwannoma. Blood in the endolymph and the perilymph changes the hydrostatic pressure, which alters cochlear function and nerve stimulation⁵.

CT and MRI techniques allow more detailed anatomic studies of the inner ear but in radiologic evaluation of temporal bone trauma, high-resolution CT and MRI are complementary. CT visualizes the bony labyrinth, fractures through the temporal bone, ossification of the bony labyrinth, and pneumolabyrinth. CT is still the best

technique to study patients with fractures, congenital malformations and otodystrophies involving the inner ear. During recent years MR imaging has emerged as an excellent method to detect pathologies in the internal auditory canal and membranous labyrinth and to characterize petrous apex lesions. MRI, especially T2-weighted images, visualizes the membranous labyrinth and vestibulocochlear nerve complex. MR has even proved its value in patients with fractures and congenital malformations, for instance, making possible the diagnosis of labyrinthine concussion and absence of the vestibulocochlear nerve. However, MR and CT are often complementary, as is the case for patients with mixed hearing loss, congenital malformations and petrous apex lesions.

In the case we examined, in spite of a significant clinical presentation observed after head trauma, with abrupt onset of hearing loss associated with vertigo, initial CT studies were negative. However a hypersignal on T1-weighted images of the labyrinth structures strongly suggested acute intralabyrinthine hemorrhage associated with the disappearance of semicircular canals due to blocked lymph flow.

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