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Review

Post-traumatic balance disorder

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ABSTRACT

The causes of balance disorder are many and various, and the subjective syndrome of cranial trauma patients is diagnosed by elimination. Progress in otoneurologic functional exploration and brain imaging, however, now generally allow this functional complaint to be given an objective basis. In recent years, new diagnoses have improved recognition of such pathologies in the appraisal of corporal injury for compensation purposes. The present article seeks to detail etiology and, by a review of the literature, to determine factors liable to influence management and appraisal in particular.

1. Introduction

Second to neck pain, balance disorder is the most frequent complaint following cranial or neck trauma; instability is reported in 23–81% of cases in the first days post-trauma [1]. Progression is often favorable, with symptom resolution within days or weeks. On the other hand, balance disorder may also persist, becoming disabling and sometimes preventing return to work. The balance system involves various sensory receptors, including the 2 balance organs, the eyes and somesthetic receptors. The balance organs comprise 3 semicircular canals and an otolithic system composed of utricle and saccule, the former coding for angular acceleration and the latter for linear acceleration. Afferent information from the various sensory receptors is integrated in the brain. If one receptor emits faulty information, there will be discordance and ensuing balance disorder. Interview and clinical and paraclinical examination are thus essential, to determine the origin of the balance disorder. A range of audiovestibular examinations explore the various parts of the inner ear [2]. Choice of examination and the performance of the clinical examination, however, require good knowledge of the etiologies underlying post-traumatic balance disorder. The ENT physician should first rule out labyrinthine origin, but may also refer the patient to other specialists such as a neurologist or ophthalmologist.

The present article seeks to detail the various etiologies, both labyrinthine and extra-labyrinthine, and, by means of a literature review, to determine factors liable to influence management and appraisal for compensation purposes in particular. We also present the means at the practitioner's disposal to identify malingerers.

2. Discussion

2.1. Vestibular etiologies

Benign paroxysmal positional vertigo (BPPV) is the principle causal factor to be considered in post-traumatic balance disorder. Classically, it is rotational, triggered by head movement. Patients with BPPV, and especially BPPV of the semicircular canal, may also complain of instability. Even minor cranial trauma may induce BPPV [3]. Time to symptom onset is usually a few days, but certain reports found onset after several weeks or even months. Post-traumatic forms frequently show recurrence and resistance to therapeutic maneuvers [4]. Involvement is classically bilateral, most frequently affecting the posterior semicircular canal [5]. Lateral semicircular canal involvement is also possible. Anterior canal involvement is very rare (3.2%) in idiopathic forms but classical (27.3%) in post-traumatic forms [6]; incidence is underestimated, as few physicians screen for it [7]. Videonystagmoscopic screening of the 6 canals is recommended, using the Dix-Hallpike or modified Dix-Hallpike maneuver; the Pagnini-McClure maneuver is used for the lateral canal, consisting in positioning the patient supine and imposing 90° head rotation on one side then the other; the Rose maneuver is used for the anterior canal, with the patient supine and

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the head in hyperextension. Videonystagmography (VNG) displays positional nystagmus graphically, providing a complete written record for the patient's file. If obesity, multiple fracture or neck stiffness prevent VNG, the patient should be referred to an otoneurology team equipped with a dynamic Thomas Richard-Vitton (TRV) chair [8]. Audiovestibular exploration is justifiable only for recurrent BPPV or resistance to therapeutic maneuver. In post-traumatic BPPV, however, audiometry seems indicated in case of any atypic presentation: normal results argue for BPPV. Vestibular work-up may be abnormal due to BPPV on the day of examination, and results should be interpreted accordingly.

Temporal bone fracture should be suspected in case of high-energy trauma and/or otorrhagia and/or Battle sign (retroauricular ecchymosis). Auditory impairment and facial palsy should be screened for systematically at interview and clinical examination. Temporal CT distinguishes between 2 types of fracture. Extralabyrinthine or "longitudinal" fracture accounts for 80% of cases and is caused by often lateral trauma. It often induces transmission hearing loss, due to hemotympanum and/or ossicle injury. Vestibular work-up includes VNG, to detect any nystagmus (spontaneous, positional or revealed by Head Shaking Test [HST] or nystagmus induced by bone vibration); the kinetic tests enable VNG to quantify directional preponderance, which indicates imperfect compensation, the importance of which in appraisal is well-known. Subjective vertical visual deviation is a further argument for poor compensation. The caloric reflex test and otolithic evoked potentials (OEP) screen for vestibular injury and determine laterality, and should be performed if otoscopy is normal (impedancemetry sometimes being contributive), or sufficiently late so as not to be artifacted by hemotympanum. "Transverse" translabyrinthine fracture, by often anterior or posterior impact, accounts for 20% of temporal bone fractures; it induces sensorineural hearing loss or complete deafness and/or facial palsy and/or cerebrospinal rhinorrhea. Given the seriousness of these conditions, vestibular function should be assessed only after they have been treated.

Labyrinthine concussion is defined as sensorineural hearing loss mainly affecting high frequencies, with or without vestibular symptoms, following cranial trauma without labyrinthine fracture. The concussion of the labyrinth causes micro-hemorrhage [9]. Labyrinthine concussion should be considered in case of vestibular impairment (e.g., hypovalence on caloric test) associated with mainly high-frequency hearing loss, without bone lesion or fracture on temporal CT. Clinical examination is, unfortunately, poorly contributive. There is usually no spontaneous nystagmus, although nystagmus may be revealed on HST. VNG may find unilateral hypovalence and/or directional preponderance. As noted above, audiometry often finds descending sensorineural hearing loss predominating at 4000–8000 Hz [10]. Temporal CT is normal. Cases of labyrinthine concussion have been reported with proven contralateral labyrinth fracture, by bone-conducted pressure transmission [11]. Progression is usually satisfactory within 5 days; symptoms resolve spontaneously in most cases within a few weeks to 2 months, but may persist or even worsen, in which case associated cerebral concussion should be suspected. Balance disorder associated to the auditory deficit is a factor of poor prognosis.

As a surgical treatment exists, perilymphatic fistula (PLF) should be suspected in priority in case of post-traumatic balance disorder associated with auditory involvement. However, the typical presentation of post-traumatic rapidly deteriorating sensorineural hearing loss associated with vertigo triggered by pressure maneuvers is rare. PLF inducers multiple audiovestibular symptoms. Diagnosis is based on a range of clinical and paraclinical findings, requiring meticulous interview, appropriate audiovestibular tests and high-quality imaging. At present there are, unfortunately, no precise diagnostic tests, so that diagnosis is uncertain, and possibly late [12], complicating management, especially for expert appraisal. A diagnostic scale, published in 2005, diagnoses PLF with 100% sensitivity and 70% specificity, based on clinical and paraclinical data [13]; results should be regularly updated in the light of other tests such as positional audiometry and electrocochleography, which have proved contributive [14,15]. PLF consists in active leakage of the labyrinthine fluid known as perilymph into the tympanum via the weak points of the oval and round windows [16]. Spontaneous PLF without evident trauma has been reported [17], but traumatic etiologies are more frequent [12,18]. Symptom onset is usually immediate, but the difficulty of diagnosis may cause delay, and surgery may be implemented only some months or years post-trauma. Audiometry may be normal, and in case of hearing loss there is no specific profile. Positional pure-tone audiometry completes auditory work-up, positioning the patient in left or right lateral decubitus (depending on the affected ear) with renewed auditory testing. It is considered positive in case of > 10 dB impairment on ≥ 3 frequencies on change in position. Specificity is good but sensitivity low; in some cases of pure vestibular involvement, positional pure-tone audiometry can identify the affected ear. On balance assessment, slightly fewer than half of patients with proven PLF seem to show vestibular asymmetry on caloric testing [19]. Indirect signs (Tullio's sign, instability or nystagmus triggered on tragal pressure or Valsalva maneuver) should be screened for systematically, but lack sensitivity; a record of the tests should be entered in the patient's file. Asymmetric results on OEP [20] and electrocochleography [21] contribute to diagnosis. Temporal CT and brain MRI are highly contributive, but normal imaging does not rule out PLF [22]. Apart from rare but pathognomic pneumolabyrinth, CT may show fluid filling one-third, two-thirds or all of the round window recess: this is significant only if the rest of the tympanum is well aerated. In the oval window, the position of the footplate (dislocation, disorientation or fuzzy aspect) and any adjacent fluid emission are screened for. Brain MRI is contributive to differential diagnosis, and should be prescribed ahead of any surgery. Absence of nystagmus or of intraoperative perilymph leakage in no way rules out diagnosis [18]. Depending on symptom severity, surgery may be considered, consisting in filling the windows with material. It is essential to inform the patient that surgical exploration of the middle ear in case of doubt as to diagnosis may not always succeed in providing proof of fistula, in which case filling is performed "blindly": i.e., systematically and preventively. Filling the labyrinth windows may induce 5–10 dB transmission hearing loss. Without pathognomic signs such as pneumolabyrinth or in-vivo visualization of perilymph leakage, diagnosis is difficult and controversial. Although prognosis seems correlated with early treatment [23], there does not seem to be any deadline, as surgery performed more than 10 months post-trauma in some cases provided functional benefit [24].

Otolithic disorder used to be undetectable, but new specific paraclinical examinations now enable identification. Previously, clinical examination failed to reveal otolithic disorder in these patients with their particular complaints, now grouped together as "otolithic syndrome", defined by particular disorders such as a sensation of sinking into the ground, walking on cotton wool, or inebriation; onset is classically immediate on trauma, with symptoms that may last 6 weeks [25]. Animal studies demonstrated otolith destruction following trauma, thus definitively proving the existence of otolithic disorder [26]. Screening requires a subjective visual vertical test and cervical or ocular OEPs. Asymmetric results are significant, 72% of patients with post-traumatic instability showing abnormal otolithic test results [27]. Vestibular rehabilitation in otolithic syndrome [28,29], such as working on an inclined plane or with otolithic stimulation, should be attempted, although some cases of otolithic syndrome prove refractory.

It is established that minor cranial trauma may lead to decompensation of previously asymptomatic inner ear deformity [30].

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