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CRANIO-CERVICAL TRAUMATOLOGY AND VERTIGO

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Abstract: *The traumatic pathology of the head and neck is often associated with peripheral or central vertiginous manifestations. These are due either to direct or indirect traumatic damage of the temporal bones - the labyrinth's place - (involving direct damage to the middle or inner ear, or by affecting the cervical vascularization or the osteo-muscular skeleton of the cervical area. The secondary psychopathological reactions associated with this type of traumatology may also be the secondary source of vertigo. This article reviews the clinical forms of vertigo that may occur as an immediate or delayed consequence of cervical and cranial traumatic pathology, with their particular diagnostic and modalities of therapy.*

Key words: *cranio-cervical traumatology, vertigo, diagnosis*

1. Introduction

The traumatic pathology of the head and neck is frequently associated with peripheral, central and / or mixed vertiginous manifestations, due to either direct injury to cerebral parenchyma and / or skull bones (the temporal bones are the labyrinth's center) or consequences of complex cervical lesions with the interest of regional cervical blood vessels.

The vertigo occurring in the context of cranio-cervical traumatology (CCT), and can be classified, by the onset, into two broad groups: one clinical forms with onset of immediate vertigo (less than 24 hours) and disorders causing vertigo with relatively late onset, (after at least 24 hours). In this second group, the vertiginous manifestations that occur late after the acute CCT episode, include some pathological processes whose signs settle slowly, few weeks after the trauma [15], [11], [22].

IMMEDIATE VERTIGO: Paroxysmal benign positional vertigo (VPPB), labyrinth contusion, Temporal bone fracture, Perilimphatic fistula /

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Semicircular channels dehiscence, and rupture of round window membrane. LATE VERTIGO: post-traumatic labyrinthitis, post-traumatic inner ear hydrops, cervicogenic "vertigo" and cervical vessel damage (after WHIPLASH), post-traumatic vestibular epilepsy, post-traumatic migraine, vertigo from posttraumatic intracranial hematoma and diffuse axonal lesions, post- CCT psychogenic vertigo (the old "sd.post-concussion") [14], [22].

2. Main Clinical Forms

In these cases, vertigo occurs in relationship and after CCT, in a patient who has not had any previous balance disorder of this type. In this context, the following pathological entities can be described in the order of frequency of occurrence:

2.1. Post-traumatic benign postural traumatic vertigo

These are the most common forms of post-traumatic vertigo (occurring in over 28% of CCT) [16]. It is due to the traumatic detachment of the macula otholites and their possible migration into the semicircular channels. It usually occurs in the first 24 hours after CCT, but onset may sometimes delayed 5-10 days. It is manifested by the appearance of vertigo with nystagmus at sudden changes of the head position. The access time is short (up to 30 seconds), stops at rest, repeats frequently during a day, for weeks, often accompanies by vegetative manifestations, but without associated auditory manifestations. The diagnosis resides through the reproduction of symptomatology by the classic Dix Halpike and Roll Test maneuvers, which indicate the typical and asymmetrical affection of several semicircular channels, sometimes bilateral involvement. They are resistant to treatment, require repeated classical otolithic repositioning maneuvers, depending on the affected channel (Epley, Semont, BBQ etc), and sometimes the reeducation of residual instability due to remanent secondary otolithic sd. (by vestibular kinetic therapy and cerebral compensatory medication), [2], [15], [18], [22].

2.2. Vertigo from traumas of temporal bones and its complications:

Labyrinth contusions: it is manifested by a reversible fluctuation of the ear-ear vestibular function. Common, even after minor CCT, is generated by spasm and microhaemorrhage in the labyrinth bone. Changes are only functional, auditory and vestibular, usually unilateral (non-persistent sensorineural and / or unilateral hearing loss), successive to a recent CCT. Treatment consists of instrumental surveillance, cochlear vasodilators, corticoids, non-ototoxic broad spectrum antibiotics (if there are also infected cranio-facial lesions). Classically, there are no detectable bone lesions, there are actually lesions of vestibular sensory neuroepithelial or cochlear (isolated or associated, in varying degrees and complexities). Labyrinth contusion may also be produced by cranial fractures, but not of

labyrinth, or other CCT, without line of fracture (Cranial transversal-oblique cranial fractures, which penetrate the temporal rock perpendicular, produce major inner ear lesions (hemorrhagic intralabirinthyc lesions, especially of the membranous labyrinth, communications with subarachnoid and lymphatic spaces and pneumolabirinth). Other CCT may interest the inner ear by accidental penetration (crossing the middle ear), which also leads to its destruction.

Symptomatology: destructive peripheral vestibular syndrome (violent vertigo with nausea and vomiting, axial and segmental deviations, spontaneous horizon-rotating nistagmus to the healthy part) along with classical otoscopic signs (othoragia, cerebrospinal leakage, haemotympanum, neurosensoryal or mixed hearing loss, lesions of external auditory chanel) occurring in CCT context. Important: In a patient with CCT and encephalic damages, spontaneous nystagmus occurs only if its con-sciousness is partially preserved. In the superficial coma there is only the slow phase of nistagmatic beats, and in the case of a deep come, spontaneous nystagmus will be absent. Investigating the presence of nystagmus in a comatose is done by practicing the cold heat stimulation manoevra, with water at 4 C (Frazer technique). Water is totally contraindicated in the case of lesions of the eardrum or CAE, and using high-resolution temporal bone CT, cerebral MRI, and classical cochleovestibular investigations transcend the diagnosis. Evolution: It is dominated by the prognosis of neurological lesions and the possibility of microbial superinfection of the injured labyrinth (acute serous or bacterial labyrinthitis are common), but in a normal evolution the vertiginous manifestations tend to spontaneously regress. The ap-pearance of central cerebral compensation (manifested by the disappearance of spontaneous nystagmus) is all the more rapid since the destruction of the labyrinth was more complete, and as the individual is younger or his bed mobilization is earlier, and the central neurological lesions are less severe [2], [19]. Treatment is usually conservative. Prophylactic antibiotics are administered for about 4 weeks. Miringototomy and insertion of a eardrum ventilation tube may be indicated, especially for serous otitis, which persists after one month [2], [22].

Vestibular therapy: Spontaneous installation of the central compensation phenomenon is usually expected (in about 2-4 weeks the patient becomes asymptomatic). If the vertiginous manifestations persist, it shows that there are functional relicvates of the injured vestibule, which blocks the installation of the central compensation.

Labyrinthtotomy can be solution (which complements the incomplete destruction of the labyrinth) or unilateral vestibular neurotomy. Take into account of the surgical decision, the actual degree of dis-ability caused by vertigo and the state of the hearing function (a normal cochlea denied a surgical labyrinth destruction) [2], [15], [22].

Infection of the bone (acute labyrinthitis) can occur by otogenic origin or meningogenic. In the initial serous phase, can use corticoids and vasodilators, and the evolution towards

purulent form attracts the surgical decision under the cover of antibiotics [2], [10].

The chronicisation of the labyrinthytis (especially the otogenic variant) is often associated with the ossification of the membrane labyrinth with the subsequent appearance of the signs of the post CCT inner ear otic hydrops. The mechanism is by scar, typical after severe damage to the membrane labyrinth and appears late (after years). It seems to be predisposed to those, all with Sd of broad vestibular aqueduct - preexisting [3]. Treatment: it is common to the Meniere sd. [14]

2.3. Vertigo in post-traumatic perilimphatic fistula:

Perilimphatic Fistula (FP) is a posttraumatic dehiscence at the site of round or oval windows or a labyrinthic semicircular canal through which a communication between the perilimphatic fluid compartment of the inner ear and the middle ear is made. FP is a functional, transient and recurrent vestibular dysfunction.

FP may occur after a hyper or hypopressive bruise transmitted to the vestibule, either from the cerebrospinal space (“explosive type"), or from the outside, via the Eustache tube (“implosive type”) during a blast, scarlet or even cough or strong sneezing (“microtraumatism”).

Symptomatology: FP produces inner ear fluids disturbances and intra-labirinthic pressure, with abnormal stimulation of the vestibular macula and cupulas, resulting in either canallith or otolithic manifestations. Thus manifestations such as vertigo (rotary sensations) and linear motion sensations with instability are triggered by position changes, CAE pressure, Valsalva maneuver, or strong sounds (Tullio phenomenon). Considering the fluid communication with the cochlear compartment, there is also a fluctuating “positional” hypoacusias (ascertained by the Frazer positional audiometry test). It describes the positive sign of the fistula (when the digital CAE pressure closure on the same side as FP produces vertigo and nystagmus), which unfortunately is rarely found in daily practice. Diagnosis is difficult, requires high resolution CT and if FP is very symptomatic, a possible middle ear surgery will be followed by FP plugging.

Evolution: some FPs spontaneously cured in about 6-12 months, but only in the mucosa part, the subjacent bony remaines beant; some may become infected (evolution to suppurative labyrinthitis and meningitis in repetition). These require traceability and prophylactic antibiotic treatment in any possible new otitic or IACRS episode. Paroxysmal vertigo can continue months and years after CCT, while cochlear function also degrades. In this case the treatment is surgical by plugging the bone of FP.

The Tullio phenomenon may occur in posttraumatic circumstances that lead to the appearance of a FP, or which lead to an abnormal anatomical continuity between the middle ear and the otolithic macula. It is manifested by vertigo triggered by intense low-frequency sounds (otolithic stimuli). Patients accuse

are vertigo, body instability, and oscilopsies when passing trucks, compressors, etc. (when low-frequency sounds are transmitted directly to otolithic zones).

Treatment is again the cause of the Tullio phenomenon (ie FP plugging or restoration of the modified bone chain of middle ear) [2], [8], [14].

2.4. Vertigo in post-traumatic intracranial hematoma and diffuse axonal lesions

The cerebral traumatic context is not always obvious, especially since it may take several weeks or months to install the intracranial hematoma. After a CCT, the intracranial hematoma can develop either on the brain surface, extracerebral (with extradural variants - directly under the cranial axis with rapid or subdural evolution - in contact with the brain, after a few weeks) or intracerebral.

The subdural hematoma can thus be installed by performing the shape of the chronically subdural hematoma. Its favorable factors are represented by the presence of coagulation changes (chronic anticoagulant therapy, chronic consumption of aspirin or other NSAIDs, alcoholism, chronic hepatopathy, hematopoiesis, etc.) and minor skull injuries [6].

Symptomatology: Elderly patients with the above-mentioned pathology, vertigo, and chronic late-onset headache with a tendency to fall backwards in the Romberg axial manoevra-can predict cerebellum haemorrhage (or hematom), and with lateral fall (hematoma in contact with cerebral hemispheres).

Diagnosis requires skull TC, MRI and neurosurgery exam, the treatment consisting of emergency evacuation of the hematoma, especially if the patient also has consciousness disorders. In the absence of these, temporary treatment with cortisone may favor spontaneous resorption of the hematoma [2], [14], [22].

Pure deceleration forces can cause brain diffuse axonal lesions [14]. Mechanism: small areas of bleeding (petechial haemorrhage) and disruption of neuronal circuits (axonal lesions). Significant effects of these diffuse microlesions do not occur in patients with CCT who have reported no loss of consciousness. A loss of consciousness of at least 30 minutes seems to be necessary for a significant effect associated with this type of cerebral lesion [1]. Diagnosis necessarily requires angio- MRI.

2.5. Cervical vertigo (most frequent form after "WHIPLASH" mechanism):

Vertigo in “cervical whiplash”:

The notion of "cervical vertigo" is quite confusing. It is now accepted that only a clear and strong cervical spine injury at the level of cervical proprioceptors may be a possible cause of vertigo. CCT must produce a real "sensory conflict" by producing an abnormality of cervical posture, which later -through cervical proprioceptors -will send new sensory information that “contradicts” normal vestibular or visual information, generating the “vertigo” sensation. In

fact, the sensation is not vertigo, but the "imbalance" That occurs always after major cervical trauma, never minor one! [7, 8].

The notion of "cervical whiplash" describes the damage to the neck that followed a brutal "back" hit/collision. Mechanisms involved in the generation of post-Whiplash lesions: damage caused by severe neck hyperextension - rupture of the anterior longitudinal ligament, muscle haemorrhage and disc rupture and bulge, and occasionally even brain damage [14].

Visual disturbances, as well as internal ear disturbances, are classically attributed to vertebrobasilar artery injury. The vertebral arteries can be damaged by vertebral movement or elongation of the vertebral arteries. The origin of these sensations is probable, knowing the role of postural reflexes with origin in cervical muscles, which are transmitted through spinovestibular paths and contribute to stabilizing the eye. It is therefore admitted that vertebral osteoarticular lesions or cervical muscles can trigger abnormal proprioceptive inflows that arrive in the cerebral trunk are the origin of a genuine sensory conflict, producing pseudo-vertigo sensations. It has been shown that nuchal lesions can produce true visual illusions of movement [7], [12]. So Clinical Whiplash is similar to the old "postconccusive syndrome of CCT" but to the neck. The clinical diagnostic criteria required to associate vertigo with cervical post-traumatic affections, and include [14], [22]:

- vertigo should be associated with clinical signs of suffering of the high cervical spine C1 - C4 (with pain in the palpation of spiny apophyses, painful limitation of cervical movements, etc.)
- the existence of strong cervical pathology: fracture or dilation of the cervical column, muscle elongation, recent local surgery, recent port of cervical minerva.
- positive cervical imaging balance for osteoarticular lesion in the area (CT, cervical column MRI, etc.)

Prognosis: Cervical stiffness can persist in 20-45% of patients. Dizziness associated with whiplash occurs at 20-60% and can persist for years, 75% of patients are recovered for up to 1 year [12], [17].

Aspects of vertigo in post-traumatic dissection of cervical vessels:

Dissection of a cervical vessel is not an exceptional event in cervical traumatology. Following this phenomenon, a cavity containing blood and thrombi is formed inside the wall of a damaged cervical artery. Coagulations can either produce a complete obstruction of the respective cervical artery, or they can be trained as emboli, to the cerebral hemispheres (through the internal carotid artery) or to the cerebral or cerebral trunk (through the vertebral artery). In both cases, these processes cause strokes. Any preexisting vascular comorbidities are an unfavorable prognostic factor [6].

Symptomatology: The traumatized patient has a laterocervical pain, on the side of the injured artery, Claudius Bernard Horner's omolateral syndrome, along with the neurological signs of the brain damaged by the cerebral accident

(including vertigo). Diagnosis is determined by echographic examinations, Doppler of the neck, arteriography, cervical and cranial CT. Emergency treatment is medical and consists of control of coagulation, rest in bad, and treatment of stroke.

Evolution of arterial lesions is followed by periodic Doppler examinations; total reparations occurs between 3 and 6 months [12], [14], [21].

2.6. Post- CCT psychogenic vertigo in post-traumatic intracranial hematoma and diffuse axonal lesions

They include several forms of clinical treatment that the psychiatrist is responsible for:

"Factitious vertigo": or the adoption of the "role" of the ill persone (occurs by "vertigo" accusations after a CCT, in fact, people need to be in the spotlight - the respondents have an unconscious need for increased affection).

Psychogenic vertigo: it is directly related to psychological causes, such as depression, anxiety. Anxiety and depression can result from traumatic brain injuries that create a self-perpetuating psychological reaction. It is a frequent consequence of CCT, even benign, manifested by a subjective syndrome that includes: instability, capricious headache, and various neurovegetative signs. The characteristic of this syndrome is that the clinical and paraclinically examinations show the absence of cerebral or vestibular organic abnormalities. The causes of this syndrome appear to be found in minimal, hard or even undetectable intracerebral lesions through current investigation methods. Patient symptomatology associates unsystematic equilibrium disorders associated with emotional disturbances occurring in post-traumatic context. The treatment addresses the emotional, anxious and depressive context, joining a typical anti-migraine treatment for headache, psychotherapy, antidepressants, sedatives, and cervical physiotherapeutic procedures [1].

Post-traumatic stress disorder (PTSD): can determine the re-examination of CCT and vertigo [9], [14].

"Malingering" vertigo: the so-called "money issue" was noted, - especially for people with head trauma, - when "disability" is partially correlated with the financial incentive targeted as harm, it was found that applicants / compensation / damages recipients report the incidence and severity of the symptoms being "significantly higher" than those who were not seeking or not receiving financial compensation [4], [19].

2.7. Post-traumatic Migraine

It occurs in about 41% of CCTs. Associates dizziness (non-vertigo) with headache, difficult to distinguish from anxiety or tension headache [5]. Sometimes a true MAV (vertigo-migraine) is formed. Respond to classic migraine (triptans) and BETAHISTINUM cochlear-vestibular vasodilators if signs of migraine vertigo (European Consensus 2016 ROMA) [12-14].

2.8. Post CCT epileptic vertigo

It is due to temporal lobe cerebral lesions (processing the vestibular signals), vertigo is accompanied by altered consciousness [23]. Typical symptom is "rapid rotation," followed by alteration of consciousness, but this symptom has other causes potentially more frequent potentials (requires differential diagnosis with VPPB). Treatment is neurological: anticonvulsant [23, 24].

3. Conclusions

This brief review confirms the existence of a close association between head and neck traumatic pathology and the appearance of vertiginous phenomena. Although CCT is present in the history of many people with vertigo, they are not always their singular cause. In most cases, the etiology of vertigo in the post-traumatic context is multiple, complicated and complex. Other secondary sources of vertigo / dizziness should not be neglected: the combination of labyrinth in overinfection, vestibulotoxic antibiotic treatment (at the moment motivated by their vital indication in some CCTs), vestibuloplegic-sedative, secondary traumatic haemorrhage, advanced age of the patient, mobilization late, comorbidities and cardiovascular, metabolic and neuro-psychopathological conditions pre-existing for CCT.

In the case of cranio-cervical traumatic pathology the vestibular, neurological, ophthalmic and cervical spinal anatomical structures are affected; in the possible case of non-participation of the patient (coma), the anatomical and functional evaluation of the vestibular acoustic organ is necessary for the purpose of early therapy, differential diagnosis and for the aim of medico-legal evaluation. Therefore, the emergency care of these cases requires modern paraclinical examinations: auditory evoked potential, stapedial reflex, computerized electronystagmography (ENG test), videonystagmography (VNG test) and other vestibular function tests, possible only in over-specialized medical centers.

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