Communication

Residual Dizziness after Liberatory Maneuver for BPPV: Two Types of Labyrintholithiasis?

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Abstract: Residual dizziness after a liberating maneuver is often referred to by patients as a more disabling set of symptoms than the positional vertigo itself. This situation seems to involve more than half of subjects with labyrintholithiasis. The authors examine the hypothesis according to which residual dizziness involves subjects with labyrintholithiasis on the basis of otoconia.

Keywords. residual dizziness; labyrintholithiasis; cupololithiasis; otocones; BPPV; liberating maneuvers; utricle; VEMPs objective vertical visual VVS; bucket test

1. Introduction.

Cupulo-canalolithiasis (benign paroxysmal positional vertigo – BPPV – or labyrintholithiasis) is the most frequently observed form of vertigo. The high prevalence of both BPPV and vestibular migraine (VM) and the extent to which these are accompanied by anxiety are documented (1). Labyrintholithiasis typically causes positional vertigo, as it is triggered by head movements and is characterized by observation of a typical positional nystagmus. In an epidemiological analysis, Benign Positional Paroxysmal Vertigo and vestibular migraine (Vestibular Migrane VM) appear to be less frequent than they really are. In contrast, Menière's disease, which is about 10 times less frequent than BPPV, appears to be overestimated (1).

BPPV is also present in children and is therefore also a pediatric pathology. An Italian study conducted in 423 children evaluated for balance disorders found this pathology in 10.2% of cases (2)

BPPV is mostly seen as having its pathogenesis in detachment of otocones (otoconia) from the macules of the utricle, which then slide into the ear canals. There follows sensitization of the semicircular canals themselves to the gravitational field (3,4). However, it must be said that, albeit in a smaller percentage of cases, other particles such as blood clots, leukocyte agglomerates or autoimmune complexes (6), can equally obstruct the lumen of the semicircular canal, causing the same symptoms. In this case the pathogenesis is always in the canals but macular utricular structures are not involved in the pathological process.

In both cases, the therapy is mainly rehabilitative and makes use of specific movement exercises ("maneuvers") depending on the location of the canal complex involved.

In labyrintholithiasis, even after resolution of objective vertiginous symptoms through a liberating maneuver, phenomena of constant postural imbalance (residual dizziness after benign paroxysmal positional vertigo treatment) are frequently observed (5)

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These symptoms are often referred to by patients as more disabling than positional vertigo itself, since, unlike the latter, they cannot be controlled with positional avoidance behaviors (7).

J. I. Seok et al (8) describe two types of residual dizziness: continuous giddiness and short-term instability, which occurs during movements of the head, standing or walking. According to the same authors, the dizziness persists 16.4 +/- 17.6 days. A longer duration of BPPV before treatment was significantly associated with residual dizziness.

Numerous hypotheses have been proposed to explain the residual dizziness.

T. Seo et al (9) attribute it to persistent utricular dysfunction, but this does not explain why residual dizziness is not uniformly observed. According to Seo at al. (9), residual post-maneuver dizziness is present in two-thirds of patients with BPPV.

With this in mind, the same researchers considered it necessary to study utricular Vestibular Evoked Myogenic Potentials ocular (oVEMPs) in patients complaining of residual dizziness. The test was performed before rehabilitation treatment and one week after it. Residual dizziness was significantly associated with the pathological outcomes of oVEMPs.

From these premises we wish to propose a new hypothesis.

1.1. Hypothesis

As stated in the introduction, in relation to labyrintholithiasis it is believed that the canal lumen is obstructed by utricular otocones which, reaching the semicircular canal, either adhere to its dome, making it sensitive to gravity (cupulolithiasis), or remain free in the canal, generating a piston effect on the cupola itself (canalolithiasis).

In both cases at the origin of the picture there is initially a pathology of the utricle with loss of otocones.

It has been said that in reality the same symptomatic picture can be obtained, as well as by the aforementioned movement of utricular otocones, by the anomalous presence in the semicircular canal of any substance, provided it has a critical mass value capable of obstructing the outflow of liquids into the canal lumen.

Our hypothesis is that, consequently, at least two kinds of positional vertigo due to labyrintholithiasis can be identified: one with canal-utricular-otoconial pathogenesis and the other affecting the canal without utricular-otoconial involvement ("systemic"). Both would manifest themselves with identical vertiginous symptoms of obstruction of the lumen, but, according to our hypothesis, only the former would be accompanied by postural symptoms, as this is a consequence of pathology of the utricle.

The liberatory repositioning maneuver would therefore resolve the entire complex of symptoms in the non-utricular form. On the other hand, in the utricular-otoconial one, once the positional vertigo has been resolved, symptoms determined by loss of utricular otoliths would persist, and the situation certainly does not resolve itself with liberating maneuvers. In this case, not only postural disorders would persist but also, as demonstrated by Yates (10), those deriving from poor cardiovascular compensation. This condition would be even more frequent in subjects who take diuretic drugs or in any case in conditions that cause hypotension. In this connection, the inner ear is rich in sympathetic fibers while parasympathetic ones are less frequent (11), a condition that makes it very sensitive to hypotension and quite resistant to hypertension (12).

Hyun-Ah Kim and Hyung Lee (13) seem to agree that in BPPV residual dizziness after successful treatment may be associated with sympathetic dysfunction.

Therefore, in these cases the picture of residual dizziness could also be aggravated by concurrent pharmacological therapies, especially diuretic or antihypertensive ones (12). Pirodda et al. argue that there is the possibility of an influence of hemodynamic imbalance due to hypotensive modifications followed by vasomotor modifications affecting the microcirculation of the inner ear as a cause not only of vertigo but also of an increase in the prevalence of tinnitus (which in many cases can be considered a symptom of inner ear disorder). Tinnitus (14) has been found in subjects undergoing "aggressive"

antihypertensive therapy as well as in patients with severe heart failure, thus demonstrating a relationship between hemodynamic changes and inner ear dysfunction.

We now wish to highlight another important aspect of macular stimulation, relating to the relationships between the latter and blood pressure control.

We are well aware of the role played in the pressure regulation of the aortic and carotid sinuses. The increase in blood pressure stretches the walls of the arteries, exciting the baroreceptors present both in the carotid sinus and in the aortic arch. This leads to activation of the glossopharyngeal nerve and the parasympathetic nervous system, mediated by the efference reflected in the vagus nerve. The latter will increase release of acetylcholine, which will decrease the activity of the sympathetic nervous system. All this will lead to a reduction in heart rate and vasodilation, with a decrease in blood pressure.

We wonder what determines activation of the aortic and carotid baroreceptors when we change position and move, for example, from the orthostatic position from the clinostatic one.

The answer is provided by the previously mentioned BJ Yates et al. According to the authors, stimulation of the vestibular system determines changes in blood pressure and respiratory muscles. In particular, it would provoke a useful response to compensate for the orthostatic hypotension that we observe after rotations of the body upside down, as during the movements of a vertical climb. Experimentally, transection of vestibular nerves in anesthetized or waking cats impairs the ability to correct the decreases in blood pressure that result from tilting the body upward. The vestibular system also has influences on the respiratory muscles to help compensate for the respiratory needs that occur during changes in body position.

The reported data suggest that the influences of the vestibular system and in particular of its inertial receptors contained in the utricle and saccule, on the autonomic and respiratory systems serve to maintain homeostasis during movement.

Consequently, it is reasonable to assume that post-maneuver dizziness will be more frequent in subjects with "utricular" labyrintholithiasis than in those with "non-utricular" labyrintholithiasis

2. Personal Experience

We examined a homogeneous age group consisting of ten subjects of both sexes, mean age 64 years.

Asked what they remembered, their history was suggestive of positional vertigo due to labyrintholithiasis and therefore, before carrying out the diagnostic and rehabilitative maneuvers, we proposed to them, with the help of a colleague specializing in psychiatry, a psychometric analysis and a study of the subjective vertical. The psychometric tests aimed at evaluating any neuropsychological comorbidity and the study of subjective vertical utricular pathology.

Patients signed an informed consent.

The psychometric tests (15) included the following:

- The Dizziness Handicap Inventory (16) which with its 25 items was developed to assess the condition of handicap resulting from disease of the vestibular system;
- DASS-21 Depression Anxiety Stress Scales (17), a psychometric test consisting of 21 questions that can provide an indication of the levels of anxiety and depression;
- PSWQ The Penn State Worry Questionnaire (18), a 16-item questionnaire that aims to measure the trait of concern about one's symptoms.
- With regard to the study of the Visual Vertical subjective (VVS) or Subjective Visual Vertical (SVV) (19), wanting to use almost a screening method and not to upset the patient who, being in an acute vertiginous phase, does not appear available to perform movements of the head, The Bucket Method was chosen (20).

Similarly, before the diagnostic-rehabilitative sessions, the same subjects underwent a study of the Visual Vertical Subjective (VVS), by means of "bucket tests".

Variations greater than 4° from zero were considered pathological for this test.

The presence of residual dizziness was assessed with a DHI psychometric test (21)

At the first subjective vertical test, 4 out of 10 subjects were found to be pathological. The presence of labyrintholithiasis was subsequently confirmed in the whole group examined. Residual dizziness was observed in 6 of the ten cases examined.

The altered visual vertical did not therefore appear to be exhaustive in predicting residual dizziness. Indeed, not all subjects with residual dizziness present VVS with pathological values.

If the data of the altered VVS are plotted against those obtained from the DASS21 analysis (anxiety), it is highlighted that the coexistence of utricular pathology and anxiety develops residual dizziness with greater frequency. We do not obtain the same result plotting the unaltered VVS data against the DASS 21 ones.

For the sake of completeness, we report that none of the 10 subjects presented changes in the PSWQ test and therefore there does not seem to be an obvious concern of the patients for their pathological state.

In the follow-up sixty days after the initial symptoms none of the subjects presented residual dizziness or VVS alterations(tab.1).

Table 1.

Initials	age	gender	Bucket test	Res, dizz I	DASS 21	PSWQ	Res Dizz II	Bucket test
F.L.	46	female	normal	no	normal	normal	no	normal
B.S.	50	female	pathological	yes	positive	normal	no	normal
A.M.	67	female	normal	yes	positive	normal	no	normal
L.F.	65	male	normal	yes	normal	normal	no	normal
A.G.	68	female	pathological	yes	positive	normal	no	normal
P.S.	70	female	pathological	yes	positive	normal	no	normal
N.I.	68	male	normal	no	normal	normal	no	normal
A.F.	69	female	pathological	yes	positive	normal	no	normal
G.L.	70	male	normal	no	positive	normal	no	normal
P.Q.	70	male	normal	no	normal	normal	no	normal

3. Conclusions

The first studies lead us to hypothesize that there are two different types of labyrintholithiasis: one utricular-otoconial and the other not. The residual dizziness seems to be the prerogative of subjects with primitive utricular pathology and that, among these, the symptoms will be more evident in subjects with high levels of anxiety.

Conversely, the mere presence of anxious symptoms alone cannot determine the clinical picture.

The present one is intended to represent a starting point for research also with the aim of involving other researchers in research on residual dizziness

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References

1. H K Neuhauser The epidemiology of dizziness and vertigo, Handb Clinic Neurol, 2016;137:67-82.

- 2. C. Balzanelli , D. Spatar , L. O. Redaelli de Zinis Benign Positional Paroxysmal Vertigo in Children Audiol Res. 2021 Feb 1;11(1):47-54.
- 3. WT Kao,LS Parnes, RA chole . Otoconia and otolithic Membrane Fragments Within the Posterior Semicircircular Canal n benign Paroxysmal positional vertigo . Laryngoscope, 2017, 127:709-714
- 4. Schuknecht HF. Cupulolitiasi. Arco otorinolaringoiatra. 1969; 90:765–778
- 5. JL Zhang, L Hui Study on the characteristics of benign paroxysmal positional vertigo and the influencing factors of short-term residual symptoms after reposition treatment,
- 6. G. Papi, G. Guidetti, S. M. Corsello, C. Di Donato, A. Pontecorvi Correlazione tra VPPB e tiroidite autoimmune:il ruolo del danno vestibolare da immunocomplessi, Otoneurologia 2000 Maggio 2011 / n. 35 pag 3-5
- 7. AP, Navari E, Albera R, Agus G, Asprella Libonati G, Chiarella G, Lombardo N, Marcelli V, Ralli G, Scotto di Santillo L, Teggi R, Viola P, Califano L. Approach to residual dizziness after successfully treated benign paroxysmal positional vertigo: effect of a polyphenol compound supplementation Clin Pharmacol. 2019 Aug 1;11:117-125
- 8. J. I. Seok H. M. Lee, J. H Yoo, D. K. Lee Residual dizziness after successful repositioning treatment in patients with benign paroxysmal positional vertigo J Clin Neurol. 2008 Sep;4(3):107-10.)
- 9. T.Seo, K. Shiraishi, T. Kobayashi, K. Mutsukazu, T. Fujita, K. Saito, H. Watanabe, K. Doi, Residual dizziness after successful treatment of idiopathic benign paroxysmal positional vertigo originates from persistent utricular dysfunction, Acta Otolaryngol. 2017 Nov;137(11):1149-1152.
- 10. BJ Yates AD Miller Physiological evidence that the vestibular system partecipates in autonomic and respiratory control. J. Vestib. Res 1998, 8(1):17-25
- 11. C. Tian, D. Zha Chaoyong Tian Sympathetic Nervous System Regulation of Auditory Function Audiol Neurootol. 2022;27(2):93-103
- 12. A. Pirodda , C. Brandolini , G. C. Modugno Hypotension associated with autonomic dysfunction: a possible cause of vertigo? Med Hypotheses, 2004;63(6):108
- 13. H. Ah Kim, H. Lee Autonomic dysfunction as a possible cause of residual dizziness after successful treatment in benign paroxysmal positional vertigo Clin Neurophysiol. 2014 Mar;125(3):608-14
- 14. A. Messina, A. Corvaia, C. Marino Definition of Tinnitus. Audiol Res, 2022 May 23;12(3):281-289
- 15. A. Messina , , M. Epifanio M. Lo Voi , C. Raucea , G. Nuccio Uso dei questionari di otoneurologia, otoneurologia 2000 Maggio 2019 / n.50, pag 15-22)
- 16. Gary P. Jacobson, PhD; Craig W. Newman, PhD The Development of the Dizziness Handicap Inventory Arch Otolaryngol Head Neck Surg. 1990;116(4):424-427
- 17. Brown, T. A., Chorpita, B. F., Korotitsch, W., & Barlow, D. H. (1997). Psychometric properties of the Depression Anxiety Stress Scales (DASS) in clinical samples. Behaviour Research and Therapy, 35(1), 79-89)
- 18. Brown, T. A., Antony, M. M., & Barlow, D. H. (1992). Psychometric properties of the penn state worry questionnaire in a clinical anxiety disorders sample. Behaviour Research and Therapy, 30(1), 33-37
- 19. A. Messina study about subjective visual vertical in bed side examination of acute vertigo a cura di G. Asprella Libonati , top graf, 2016 pag 261- 2939
- 20. A Zwergal, N Rettinger, C Frenzel, M Dieterich, T Brandt, M Strupp(A bucket of static vestibular function, Neurology. 2009 May 12;72(19):1689-92)
- 21. Nola, G., Mostardini, C., Salvi, C., Ercolani, A. P., & Ralli, G. (2010). Validity of Italian adaptation of the Dizziness Handicap Inventory (DHI) and evaluation of the quality of life in patients with acute dizziness. Acta Otorhinolaryngol Ital.; 30(4): 190.