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CLINICAL CASE

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Inferior Vestibular Neuronitis: Case Report

Neuronitis Vestibular inferior: Reporte de Caso

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Abstract

Vestibular neuronitis is a cause of acute vestibular symptoms which occurs commonly and affect primarily the superior branch of the vestibular nerve, giving rise to distinctive clinical features. Occasionally, cases of Inferior Vestibular Neuronitis (IVN) presenting with vertigo, unsteadiness and spontaneous downward torsional nystagmus, has been proposed in literature. This nystagmus has received even less attention with regard to pathophysiology. Due to the low frequency of Inferior Vestibular Neuronitis and its unusual presentation as a peripheral vestibulopathy, we should be aware for signs and symptoms with the aim of performing an accurate diagnostic workup and offer appropriate treatment.

Keywords: Acute Vestibular Syndrome, Spontaneous Torsional Nystagmus, Video head impulse test (v-HIT), Cervical Vestibular evoked myogenic potential (c-VEMP), Inferior Vestibular Neuronitis

Resumen

La neuronitis vestibular es causa de síntomas vestibulares agudos que se presentan con frecuencia y afectan principalmente a la rama superior del nervio vestibular, dando lugar a características clínicas distintivas. En ocasiones, se han descrito casos de neuronitis vestibular inferior (IVN) que se presentan con vértigo, inestabilidad y nistagmo torsional descendente espontáneo. Este nistagmo ha recibido aún menos atención en cuanto a su fisiopatología. Debido a la baja frecuencia de la neuronitis vestibular inferior y su presentación inusual como vestibulopatía periférica, debemos estar atentos a los signos y síntomas para realizar un diagnóstico preciso y ofrecer el tratamiento adecuado.

Palabras clave: *Síndrome vestibular agudo, Nistagmo torsional espontáneo, Prueba de impulso cefálico asistido por video (v-HIT), Potencial evocado miogénico vestibular cervical (c-VEMP), Neuronitis vestibular inferior.*

Introduction

Spontaneous, sudden, and unilateral vestibular loss with intact hearing and absence of brainstem signs is commonly associated with a viral infection and is diagnosed as vestibular neuronitis (VN)[1,2]. This clinical entity represents a frequent cause of peripheral vestibular vertigo and might cause 7% of the outpatients evaluated in vertigo centers, as found in a study[1]. Anatomically, the superior branch of the vestibular nerve innervates the utricle, the superior and horizontal semicircular canals. Its longer course and more tortuous traversing in a narrow bony canal make it more likely to be trapped and result in possible ischemic labyrinthine changes, thus substantiating the concept of partial VN[3]. The saccule and posterior semicircular canal are innervated by the inferior division of the vestibular nerve. After unilateral vestibular deafferentation there is hypofunction on both the caloric test (CT) and the v-HIT of the horizontal semicircular canal on the ipsilesional side showing both qualitative and quantitative changes.

Although less frequently reported in the literature, the value of the diagnostic of the vestibular evoked myogenic potentials

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(VEMP) and specifically cervical response (c-VEMP) have been studied, since the saccule that is tested in this examination reflects the functioning of the IVN with altered homolateral vestibulocollic reflexes[5,6]. In IVN, CTand horizontal v-HIT is generally normal, but vertical canal function, displayed in the right anterior/left posterior (RALP) and left anterior/right posterior (LARP) planes, is affected[7,8]. The spontaneous nystagmus in IVN combines horizontal, torsional, and downward components and reflects functional impairment of the posterior semicircular canal (PSC), which may be a useful clinical sign in the diagnosis[9,10]. Nevertheless, this nystagmus pattern can be seen in central vestibular disorders as well, and that is why some authors have referred to the phenomenon as "atypical"[11] torsional spontaneous nystagmus. Sometimes, the aggravation of the symptoms with position change leads to misdiagnosis with benign paroxysmal positional vertigo (BPPV). According to the above, we report a patient with a clinical diagnosis of IVN, diagnostic work up and treatment.

Clinical Case

A 34-year-old woman free from a previous vestibular history presented with a sudden episode of vertigo, imbalance and vomiting, without auditory symptoms, 1 month before consulting at our institution. She was evaluated in Canada by a specialist in this condition who ordered a physical therapy consult for potential BPPV. But symptoms remained after several repositioning maneuvers (RM). After returning to Chile, she had been evaluated by other professionals who re-tried RM unsuccessfully. She joined our institution with anxiety and extreme fear of changing her head position. Upon admission, a counterclockwise (rightward) torsional nystagmus was identified, which was suppressed with fixation and decreased in intensity on leftward gaze. An otoneurological examination demonstrated normal hearing and impedance. The v-HIT with the EyeSee Cam (EyeSeeCam v.1.1.1) for the horizontal canals and the RALP plane was normal.

The Videooculonystagmography revealed spontaneous counterclockwise torsional nystagmus not suppressible by fixation, that was reproducible in all head roll and Dix-Hallpike positional tests, and exacerbated by neurovegetative symptoms, especially in the head-hanging position (**Figure 1A**). The considerable gain in the LARP canal plane also demonstrates a significant asymmetry an approximately 60% reduction in the right anterior canal (**Figure 1B**). The air cVEMP is asymmetrical with an asymmetry ratio of 44%, which demonstrates obviously hypofunction of the saccular response on the right side (**Figure 2A**).

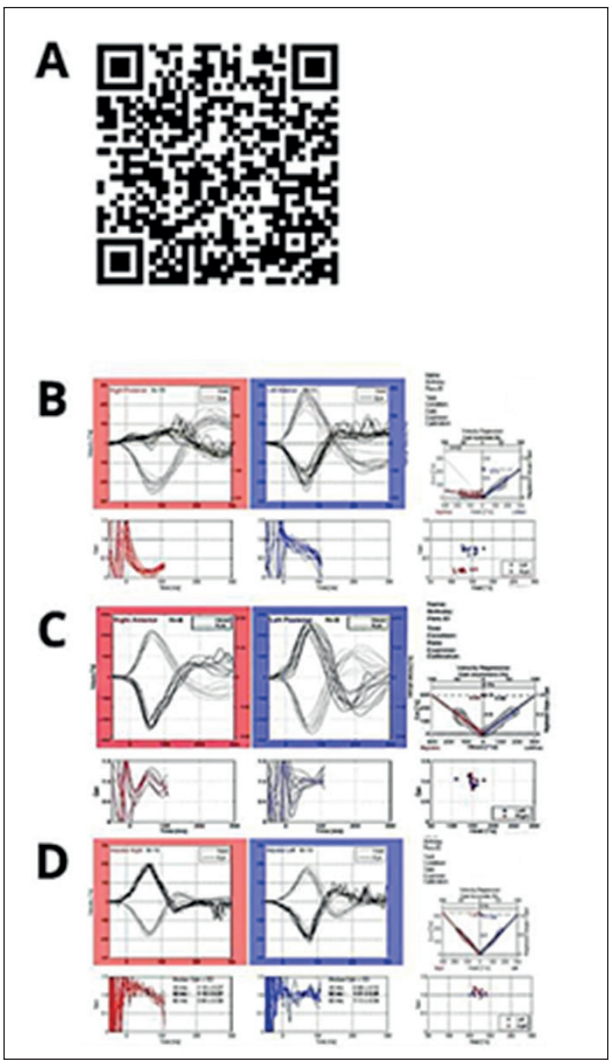

Figure 1. A (video). Demonstrates spontaneous nystagmus without fixation, with a rotatory march to the right, then downward, then counterclockwise. **B**. The v-HIT LARP plane reveals reduced gain on the right PSC. **C**. The v-HIT RALP plans are normal **D**. The v-HIT lateral semicircular canals are within normal limits.

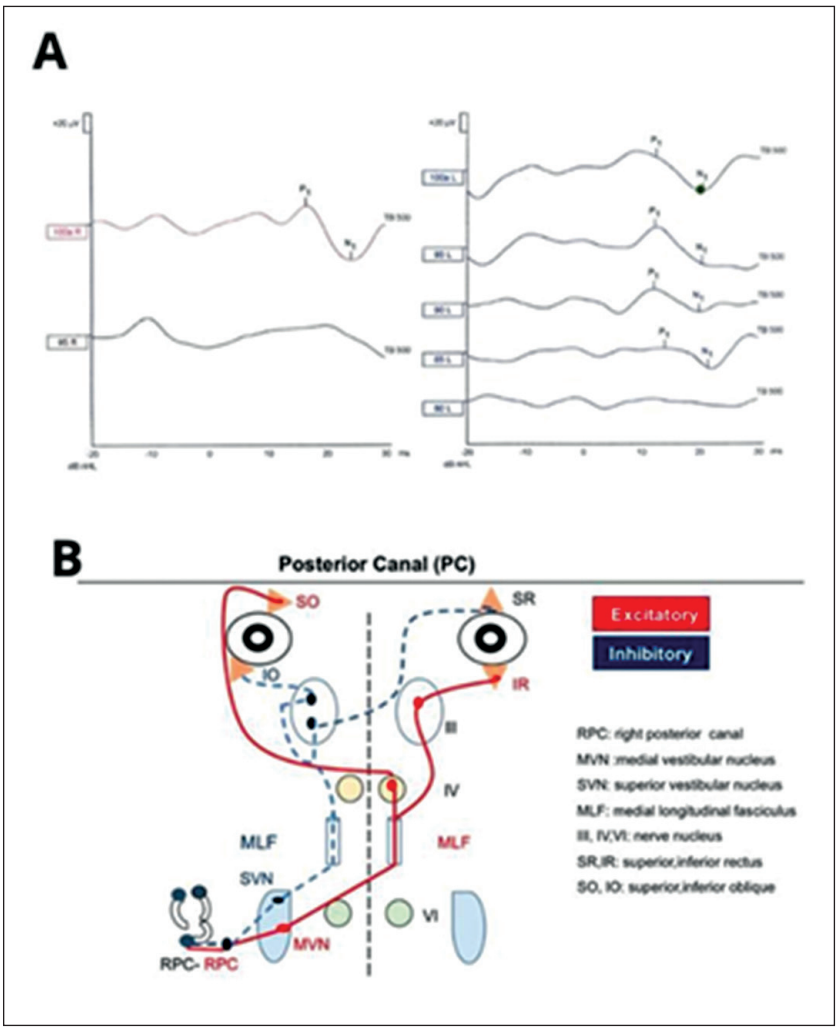

Figure 2 A. Cervical VEMP reveals diminution of responses at the right ear with 44% asymmetry. **B.** Adopted and redesigned from: Ito M, Nisimaru N, Yamamoto M. Excitatory pathways of the vestibulo-ocular reflex from rabbit semicircular canals. Exp Brain Res. 1976; 24:257–71. (Reprinted with permission from Springer Nature Group).

A brief Linthicum CT was performed (seated, the patient received 2 cc., of 2°C water for 20 seconds for the horizontal semicircular canal study with the head in position to receive ipsi PSC). Positive responses were observed in both lateral canals and the left PSC while no response was elicited in the right PSC. Due to clinical examination and otoneurological data a brain MRI with gadolinium contrast, specifically of the posterior fossa, was requested (General Electric Signa Excite HD 1.5 T –Waukesha, USA– with 8-channel head coil. HI res axial internal auditory canal images were acquired on a 3D FIESTA sequence with the following parameters: TR 4.4; TE 1.4; BW 62.5 Hz/px, slice thickness 0.8 mm, matrix 320×224, ETL 3, flip 65, FOV 18 cm). The MRI was read as unremarkable. On the basis of these findings, a diagnosis of IVN was considered and the patient was recommended for treatment with Vestibular Rehabilitation.

Discussion

The manifestation of IVN is likely due to impairment of the inferior division of the vestibular nerve. Hearing loss was not present, spontaneous torsional nystagmus in the clockwise plane, which showed a right-beating

component, was identified in Dix-Hallpike position, minimal CT (symmetrical) and normal horizontal v-HIT in addition to ipsilateral c-VEMP suggests the diagnosis of IVN[1,2,6] in this patient. The minimal Linthicum CT was adequately used for this case, it tests both horizontal and posterior semicircular canal simultaneously[8], it is very short, and it does not produce significant symptoms in a patient who is emotionally fragile.

Spontaneous torsional nystagmus raised the initial concern for central vestibulopathy until the MRI imaging dismissed it. Disorders of the vestibular system that disrupt tonic afferent input from all three semicircular canals cause a slow-phase nystagmus toward the side of the lesion that is broken by fast-phase eye movement toward the side on which that end-organ is intact. This is the generic position of spontaneous horizontal-torsional vestibular nystagmus defined as "typical"[9] (STN). The nystagmus in IVN has been descriptively reported to be "atypical" STN.

Lee SU et al.[10] investigated 16 patients with this nystagmus pattern, 11 of them with IVN, the others with Ménière's disease or central vestibulopathies. Kim JS et al.[11] reviewed 703 patients with VN as a diagnosis and found only 9 with isolated IVN, all of whom had similar "atypical" nystagmus characteristics. Unilateral IVN on the right results in denervation of the PSC resulting in decreased excitability of the contralateral inferior rectus (IR) and disinhibition of the contralateral superior rectus (SR). The primary action of the SR results in elevation, the secondary in adduction, and the tertiary in intorsion, thus producing a counterclockwise nystagmus of the left eye (it will rotate inward and downward). This is further compounded by hypofunction of the contralateral superior oblique (SO), and disinhibition of the contralateral inferior oblique (IO). The primary action of the IO is extortion, the secondary action abduction, and the tertiary action elevation, leading to nystagmus in a counterclockwise pattern on the right eye (down and out)[10-14] **(Figure 2 B)**.

The only treatment was vestibular rehabilitation, which allowed a fast clinical recovery. When follow-up evaluation was completed, the patient had no symptoms, results also mentioned in various published studies[15].

Conclusion

The presence of an "atypical" torsional nystagmus in the context of an acute vestibular syndrome should prompt a thorough otoneurological evaluation. The combination of a normal horizontal semicircular canal v-HIT and CT response, together with an altered c-VEMP and hypofunction in the LARP or RALP planes, should raise suspicion of a lesion involving the posterior semicircular canal pathway (inferior vestibular nerve). Brain MRI with contrast, with emphasis on the posterior fossa, is required to rule out a central lesion.

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