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Clinical Manifestations of Benign Paroxysmal Positional Vertigo of the Horizontal Semicircular Canal: Pathophysiology of Atypical Manifestations

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Abstract

The presence of a direction changing positional nystagmus (DCPN) in a patient with vertigo is the characteristic sign to diagnose a benign paroxysmal positional vertigo of the horizontal semicircular canal (HSC-BPPV). If a persistent DCPN is observed when positional tests are performed, a light or heavy cupula phenomenon must be suspected. In order to differentiate between these clinical phenomena, a null plane test should be considered for diagnosis. On the other hand, when a paralytic or irritative fixed direction nystagmus is found, then a complete or incomplete jamming within the HSC may be involved. In this particular situation a head shaking nystagmus test may be clarifying. When persitent DCPN or paralytic-irritative nystagmus are found in positional testing, ENT clinicians must keep alert to search for atypical clinical forms of HSC-BPPV.

Keywords: Horizontal Semicircular Canal BPPV; Atypical Nystagmus BPPV; Direction Changing Positional Nystagmus

Abbreviations: DCPN: Direction Changing Positional Nystagmus; ICVD: International Classification of Vestibular Disorders; HSC-BPPV: Horizontal Semicircular Canal.

Introduction

The International Classification of Vestibular Disorders (ICVD) of the Barany Society describes benign paroxysmal positional vertigo of the horizontal semicircular canal (HSC-BPPV) as a typical disorder, including canalolithiasis and cupulolithiasis [1]. BPPV is the main cause of vertigo in the primary care consultation and represents up to 25% of the patients who consult for vertigo, of them up to 7% correspond to the HSC-BPPV [2].

Supine head roll test is the gold standard for diagnosing benign paroxysmal positional vertigo of the HSC-BPPV. A positive result of this test consists of a horizontal direction-changing positional nystagmus (DCPN), and it may be of two different types: geotropic or apogeotropic. When the nystagmus eye movement provoked by the roll test coincides with the side towards which the head is turned, then a geotropic HSC-BPPV is diagnosed. On the other hand, if the nystagmus is in the direction opposite that of the head rotation, then the apogeotropic type is considered. In regard to this, the geotropic clinical presentation of HSC-BPPV has

been related to the presence of particles that float freely inside the horizontal semicircular canal (canalithiasis) while the apogeotropic variant has been related to the adherence of the otoconia to the cupula of the HSC (cupulolithiasis) [2]. Lately, another variant of canalithiasis of the most anterior portion of the HSC has been reported, which has a presentation similar to cupulolithiasis but with an apogeotropic nystagmus lasting less than a minute and with the presence of latency [2]. Regarding this, patients with "persistent" geotropic direction changing positional nystagmus (duration of more than 1 minute) in the supine lateral head positions in the absence of central neurologic signs and with normal results in cerebral imaging have been described [2]. This type of nystagmus is neither compatible with mobile otoconia in a semicircular canal nor with dense otoconia attached to a cupula. It has been suggested that this phenomenon may be due to changes in the density of the cupula or the endolymph, but this remains speculative [2].

The pathophysiological mechanism of horizontal DCPN still remains a challenge, and it has been reported both in patients with disorders in the central nervous system such as migraine [3], and in the peripheral vestibular system such as HSC-BPPV, in this case in close relation to the presence of particles or changes in the density of the endolymph within the horizontal semicircular canal. HSC-BPPV has been described as the main etiology of DCPN but does not always manifest with a typical nystagmus; that is why ENT specialists must always be alert when they observe any of the variants described onwards. Regarding this, and depending on the disorders that may occur inside the canal, different clinical manifestations may be found. This brief review shows different variants of presentation of HCS-BPPV and its pathophysiology [4,5].

Pathophysiology of HSC

In the last years, causes other than the classic one described by ICVD of HSC-BPPV have been reported and may be observed in ordinary clinical examinations according to different eye movement presentations (nystagmus) by means of simple Frenzel glasses. These atypical presentations may be explained by pathophysiological disorders that affect the integrity (density or jamming) of the HSC lumen. These atypical clinical presentations may be classified as: heavy cupula, light cupula, partial jamming (plugging) and complete jamming of the HSC lumen [4-7]. In this section, we will make a clinical description of the observed nystagmus in the examination setting, followed by its pathophysiological explanation.

When the head roll test elicits a DCPN, characterized by no latency and lasting more than one minute, which is defined as persistent, the first step is to determine which ear has the disorder. To do this, we must perform the "bow and lean test", that consists on bringing the head forward and, after at least 3 minutes, bringing it back. The clinical interpretation is as follows: When the head roll test has shown a geotropic DCPN, the nystagmus in the bow and lean test beats in the direction of the affected ear when the patient keeps his head forward. Conversely, if the nystagmus has been apogeotropic, then the affected ear will be that of the direction of the nystagmus in the head-back position [4,5,8].

Once a supine head roll test elicits a "persistent" geotropic DCPN and the affected ear is determined by the bow and lean test, a light cupula phenomenon must be suspected, and the null plane (point test) in supine position is useful to diagnose it. This test consists of slowly turning the head 20-30º to the unaffected side, in which the persistent DCPN stops. The light cupula phenomenon has been classically described in positional alcohol nystagmus and, it has recently been postulated that the presence of proteoglycans adhered to the cupula generate a "float effect" that makes the cupula lighter, the sulfated proteoglycans are synthesized and secreted in the own cupula. Moreover, it has been proposed on the assumption that the specific gravity of the endolymph may increase due to a hemorrhage, inner ear hypoperfusion or inflammation, and in general by an over-compensation of the endolymphatic homeostasis [4, 5].

On the other hand, when a persistent apogeotropic nystagmus is observed in the head roll test, and the null plane is achieved by turning 20-30º towards the side of the affected ear, then a heavy cupula phenomenon must be suspected. Heavy cupula theory is controversial, and has classically been related to ingestion of heavy water. The apogeotropic DCPN is thought to be caused by detached otoconia rather than by an increased specific gravity of the endolymph or even a canalithiasis that fills the interior of the HSC. This theory may explain why particle repositioning maneuvers are more successful in heavy cupula than in light cupula phenomenon. Furthermore, the transition of apogeotropic to a geotropic nystagmus has been described in heavy cupula phenomenon; this clinical finding may be explained more likely by detached otoconia than specific gravity of the endolymph [4,5] (Table 1).

Another atypical form of presentation of HSC-BPPV, frequently as a complication of performing particle repositioning maneuvers to treat any sort of BPPV, is a persistent vertigo associated with spontaneous horizontal fixed-direction nystagmus. It is usually accompanied by a hypoexcitability of the horizontal semicircular canal in caloric test, and a head impulse test showing a saccadic eye movement to the opposite side of the direction of horizontal fixed-direction nystagmus is generally observed in the setting of examination. In this case, a complete obstruction

of the HSC must be suspected, and performing a head-shaking maneuver may resolve the blockage and transform the atypical positional spontaneous paralytic nystagmus into a typical geotropic DCPN of HSC-BPPV due to canalithiasis. The pathophysiological mechanism is a complete plugging of the HSC that may generate a negative pressure inside the HSC in the same way a plunger would when positional tests are performed.

Table 1: Pathophysiology, clinical manifestations findings and diagnostic tests of HSC-BPPV.

Pathophysiology	Physical examination findings	Diagnostic tests
Heavy cupula (Kim MB, 2018; Tang X, 2019)	Persistent apogeotropic DCPN	Null plane (Same-side supine position)
Light cupula (Kim MB, 2018)	Persistent geotropic DCPN	Null plane (Opposite-side supine position)
Complete plugging of HSC (MartelluccíS,2020; Schwartz FK, 2022)	Paralytic fixed-direction hSN	Positive HIT, Reversed after HSN
Incomplete plugging of HCS (Castellucci A, 2019)	Irritative fixed direction hSN	Negative HIT Reversed after HSN

HSC-BPPV: Benign Paroxysmal Positional Vertigo of the horizontal semicircular canal
DCPN: Direction Changing Positional Nystagmus
HIT: Head Impulse Test
HSN: Head Shaking Nystagmus
hSN: horizontal Spontaneous Nystagmus

On the other hand, when particle repositioning maneuvers were previously performed, and a fixed irritative horizontal positional nystagmus is elicited after positional tests, then the plugging of the canal may be not complete, and a positive pressure is generated on the cupula, which generates a constant stimulation of the affected side cupula. As in the previous clinical situation, repeated head shaking maneuver may also clinically transform the irritative fixed-nystagmus into a geotropic DCPN. In this regard, incomplete HSC jamming in association with vertical cupulolithiasis of the posterior semicircular canal on the opposite side has also been reported. In this particular clinical situation, a torsional component of the nystagmus may be observed at the same time on examination [9]. Therefore, both pathophysiologic disorders presented above can be considered another atypical form of HSC-BPPV eye movement presentation not previously described in ICVD [6,7,9] (Table 1).

Conclusion

In conclusion, the diagnosis of HSC-BPPV may be difficult because of the presence of a "persistent" DCPN that suggests a light or heavy cupula phenomenon, in these particular cases, the null plane can be useful in its diagnosis. Moreover, the presence of a fixed-direction paralytic or irritative nystagmus when we perform positional tests (head roll test) after a particle repositioning maneuver was previously performed, may indicate the presence of partial or complete HSC jamming that may be unveiled by means of head shaking nystagmus maneuver.

Disclosure

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