Page 301 to 305

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Downbeating Nystagmus in Benign Paroxysmal Positional Vertigo: an Apogeotropic Variant of Posterior Semicircular Canal

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Abstract

BPPV is the most frequent peripheral vestibular syndrome. In most cases, the interpretation of paroxysmal positional nystagmus presents no difficulty. We present a series of cases of patients with BPPV of posterior semicircular canal apogeotropic variant, atypical manifestation for this type of disorder. We included 20 patients who presenting symptoms and vestibular signs consistent with BPPV of the anterior canal and that, subsequently, were diagnosed as having PSC BPPV.

Keywords: *Down beating nystagmus; Apogeotropic variant; Benign paroxysmal positional vertigo; Episodic vestibular syndrome; BPPV*

Abbreviations: ASC: Anterior semicircular canal; BPPV: Benign paroxysmal positional vertigo; DB: Downbeating nystagmus; DH: Dix-Hallpike maneouver; LPCS: Left posterior semicircular canal; MRI: Magnetic resonance imaging; PPN: Paroxysmal positional nystagmus; PSC: Posterior semicircular canal; RPCS: Right posterior semicircular canal

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Introduction

Benign paroxysmal positional vertigo (BPPV) is the most frequent within peripheral vestibular syndromes [1]. The interpretation, in most cases, of paroxysmal positional nystagmus (PPN) does not cause problems. The nystagmus typical of posterior semicircular canal (PSC) involvement due to canalolithiasis is vertical upwards (upbeating) with a torsional component towards the ear that is downward, so it can be considered as geotropic, also having the typical latency and with a duration of less than 1 minute [2-4]

Some patients who consult for positional vertigo, find a vertically down beating paroxysmal nystagmus with or without a clear torsional component, suggestive of BPPV due to anterior semicircular canal involvement (ASC), but which do not resolve with the maneuvers

destined for that canal or that later develop a nystagmus with characteristics compatible with PSC BPPV, a behavior already described on another occasion by Vanucchi., *et al*. [1] We present a series of cases of patients with BPPV of posterior semicircular canal apogeotropic variant, atypical manifestation for this type of disease but that should be considered for the therapeutic and prognostic implications

Materials and Methods

We included 20 patients who consulted between January 2016 and April 2017, presenting symptoms and vestibular signs consistent with BPPV of the anterior canal and that did not resolve with replacement maneuvers for that canal. All the patients were evaluated by neurologists or otorhinolaryngologists with special dedication in neuro-otology. Neuroimaging was performed when it was considered necessary. These patients, subsequently, were diagnosed as having PSC BPPV, taking as a criterion the lack of response to the replacement maneuvers for ASC BPPV and the resolution of symptoms/signs with maneuvers for PSC (Semont and Epley).

Results

55% of the patients were women. The average age was 59.6 years. 17 patients consulted for vertigo/positional dizziness, and 8 patients complained of instability. 20% had a history of PSC BPPV, 2 patients with recurrent BPPV, and one patient with mild cranial trauma. 50% of the patients presented positional downbeat nystagmus without a torsional component. 55% of the patients underwent neuroimaging (MRI), which were normal.

All patients underwent replacement maneuvers for ASC BPPV, without obtaining successful results. 5 patients, in the control, develop signs of PSC BPPV. All the patients resolved their symptoms with maneuvers for PSC BPPV (Semont and Epley). (See Table 1) (See Video 1)

Video: Video 1 apogeotropic variant right PSC BPPV.wmv

No.	Sex	Age (years)	History	Reason for consultation	Physical examination	Neuroimaging	PSC BPPV maneuvers and results
1	M	55		Positional vertigo	DB positional nystagmus torsional left in right DH	None	Reverse Semont face down left, face up right, conversion to right posterior geotropic BPPV, resolve with right Semont
2	M	66		Positional Dizziness and instability	DB positional nystagmus, torsional left in right DH	Normal	Epley for right PSC BPPV with improvement
3	M	74	recurrent BPPV	Positional vertigo	DB positional nystagmus, torsional left in right DH	Normal	Epley for Right PSC BPPV with improvement
4	M	45		Positional vertigo	DB positional nystagmus torsional right in left DH	None	Reverse Semont face down right, face up left, conversion to left posterior geotropic BPPV, resolve with left Epley
5	M	34	Head minor trauma	Positional vertigo and instability.	DB positional nystagmus torsional right in left DH	None	Epley for left PSC BPPV with improvement
6	F	61		Positional vertigo	DB positional nystagmus, torsional left in right DH	None	Epley for Right PSC BPPV, doesn't resolve, Semont for right PSC BPPV with improvement
7	F	47	Previous PSC BPPV	Positional vertigo	DB positional nystagmus, torsional left in right DH	Normal	Epley for Right PSC BPPV with improvement

8	F	53		Positional vertigo and instability	DB positional nystagmus torsional right in left DH	Normal	Epley for left PSC BPPV no improvement, resolve with Semont for left PSC BPPV
9	F	57		Positional vertigo	DB positional nystagmus torsional right in left DH	Normal	Epley for left PSC BPPV with improvement
10	F	81	Recurrent BPPV	Positional vertigo and dizziness	DB positional nystagmus, torsional left in right DH	Normal	Epley plus Semont for Right PSC BPPV with improvement
11	M	70	RPSC BPPV	Positional vertigo	DB nystagmus with no response to ASC repositioning maneuver	None	Epley for RPSC BPPV with improvement
12	F	57		Dizziness and instability.	DB nystagmus, maximum in DH to the right, with no response to ASC BPPV repositioning maneuver	None	Epley for LPSC BPPV with improvement
13	F	91		Positional dizziness and instability.	DB nystagmus, maximum in DH to the left, with no response to ASC BPPV repositioning maneuver	None	Epley for RPSC BPPV with improvement
14	M	53		Positional vertigo	DB nystagmus with no response to ASC BPPV repositioning maneuver; later on the patient develops positional nystagmus compatible with LPSC BPPV.	Normal	Epley for LPSC BPPV with symtoms resolution
15	M	70		Positional vertigo	DB nystagmus with no response to ASC BPPV repositioning maneuver; later on the patient develops nystagmus compatible with RPSC BPPV.	Normal	Epley for RPSC BPPV with improvement
16	F	57		Positional vertigo	DB nystagmus, maximum in DH to the left, with no response to ASC BPPV repositioning maneuver	Normal	Epley for RPSC with symptoms resolution
17	F	52		Positional vertigo and instability.	DB nystagmus	None	Epley and Semont for RPSC BPPV with symptoms resolution
18	F	43	LPSC BPPV	Positional vertigo and instability.	DB nystagmus	None	Epley for LPSC BPPV with symptoms resolution
19	F	55		Positional vertigo	DB nystagmus, maximum with DH to the right, with no response to ASC BPPV repositioning maneuver; later on the patient develops nystagmus compatible with LPSC BPPV.	Normal	Epley for LPSC with symptoms resolution
20	M	72	RPSC BPPV	Instability	DB nystagmus with no response to ASC BPPV repositioning maneuver; later on the patient develops nystagmus compatible with RPSC BPPV.	Normal	Epley and Semont for RSC BPPV with symtoms improvement
Pat.: patient; BPPV: vértigo posicional paroxístico benigno; ACS: anterior semicircular canal; PCS: posterior semicircular canal; RPSC: right posterior semicircular canal; DB: downbeat; DH: Dix-Hallpike maneouver; LPSC: left posterior semicircular canal.							
All the above patients were no responsive to ASC BPPV repositioning maneuvers							

Table 1

Discussion

Recently the existence of ASC BPPV has been questioned and more specifically by the possible existence of apogeotropic PSC BPPV [4-6]. We have presented a series of patients who support this last point.

The explanation to justify the presence of downbeat nystagmus in BPPV of the posterior canal is that the otoconia would be in the non-ampullary distal arm of the posterior canal, near the common crus [3,4]

The Dix-Hallpike test, when the otoconias are close to the common crus, would cause the otoconia to move towards the dome, inhibiting the posterior channel, thus producing a downward vertical nystagmus with a torsional component of inhibitory type (Figure 1). This type of nystagmus could be triggered by both the Dix Hallpike maneuver (to the right or left), the Head-Hanging position, and even the lateral position. There is usually no latency, but it may have a crescendo-decrescendo course, and the nystagmus is not exhausted in the same way as the typical PSC BPPV. Rising to the upright position does not reverse the nystagmus direction, and it does not fatigue on repeated positional testing [3]. This can be explained by arguing that the excitation of the anterior canal on one side produces the same type of nystagmus as the inhibition of the posterior canal on the other side [3-6]

One of the findings that is difficult to justify is the absence in a large proportion of patients of the torsional component, a phenomenon already described by Jacopo Campi., *et al*. [6] A plausible explanation, is that the vector of the torsional component is smaller than the one that comes from the posterior channel [6].

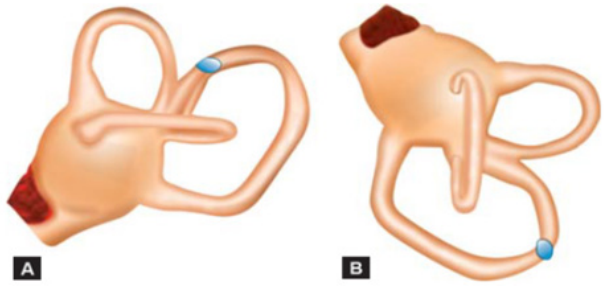

Figure 1A and B: *Apogeotropic variant of PSC BPPV, the debris inside the PSC close to the common crus, gravitate toward the PSC ampullary arm.*

Conclusion

While it cannot be stated that all BPPV with ASC compromise characteristics are an apogeotropic variant of PSC BPPV, the latter being more frequent than the previous one, it is expected that many positional down beating nystagmus are due to apogeotropic variant of the posterior semicircular canal. This last variant should be kept in mind when the patients did not respond to the typical replacement maneuvers for the ASC BPPV.

Conflict of interest

None.

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