Case Report

Apogeotropic Posterior Semicircular Canal BPPV—A Case Series from South Rajasthan

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Abstract

Apogeotropic variant of posterior semicircular canal benign paroxysmal positional vertigo (*apo*-PSC-BPPV) is a rare peripheral vestibular disorder, characterized by paroxysms of positionally triggered dizzy spells associated with non-positional disequilibrium. It is diagnosed by observing characteristic diagnostic oculomotor responses (torsional downbeating positional nystagmus) during positional testing (Dix-Hallpike and enhanced straight head hanging tests), in conjunction with a response to physical therapy. Much rarer anterior semicircular canal benign paroxysmal positional vertigo (ASC-BPPV) elicits identical oculomotor responses during positional testing. *Propter hoc,* response to physical therapy at short-term follow-up is crucial in distinguishing the *apo*-PSC-BPPV from ASC-BPPV. We are presenting a case series of seven patients of *apo*-PSC-BPPV (of which three were bilaterally affected), who attended our otoneurology center, between February 1, 2023, and July 31, 2023. Demographic profile, clinical course, and physical therapy with responses at short-term follow-up at 1 hour and after 24 hours are discussed.

Keywords: Apogeotropic posterior semicircular canal BPPV, Dix-Hallpike test, downbeating nystagmus, quick liberatory rotation maneuver (QLRM)

Introduction

In the years 1995 and 2012, Clinical Vestibulogists across two different regions of Italy reported an unusual phenomenon amongst patients with torsional peripheral positional downbeating nystagmus (*p*DBN) treated by some form of physical therapy for an ostensible anterior semicircular canal benign paroxysmal positional vertigo (ASC-BPPV). Within a few days of treatment, these patients returned with a recurrence of positionally triggered dizzy spells and elicited upbeating torsional nystagmus localizing to the long ampullary arm of the posterior semicircular canal contralateral to the initially treated anterior semicircular canal.[1,2] The occurrence of typical PSC-BPPV on the opposite side right after recovering from ASC-BPPV with physical therapy was judged implausible. By the year 2015, Vestibulogists in Italy figured out that canalolithiasis in the non-ampullated portion of the posterior semicircular canal near the crus commune, results in apogeotropic posterior semicircular canal benign paroxysmal positional vertigo (*apo*-PSC-BPPV).[3,4] It was also unveiled why, during positional tests, an ipsilateral (say right) *apo*-PSC-BPPV generates oculomotor responses identical to a contralateral (left) ASC-BPPV [Figure 1], and three different maneuvers for therapeutic repositioning of *apo*-PSC-BPPV were evolved.[3,4]

The *apo*-PSC-BPPV is still a rare variant among peripheral positional vestibular syndromes, and the reported frequency in patients with peripheral positional vertigo, attending a dedicated *Balance and Dizziness Center* in Italy has been recently reported to be 3.97 percent (25 of 630 patients).[5] It is diagnosed in any patient with symptoms of positionally triggered vertigo attacks in whom oculomotor responses to the provocative positioning elicit downbeating torsional positional nystagmus. The torsional component of the positional nystagmus is directed to the non-involved (healthy) side, as per Ewald's third law [Figure 1].[6] Otoconial clot within the non-ampullated region of the posterior canal displaces ampullopetal during Dix-Hallpike test (DHT) and the enhanced straight head hanging test (ESHHT). The inhibitory neural circuitry of posterior canal projects to the ipsilateral inferior oblique and contralateral superior rectus, generating slow-phase vestibulo-ocular reflex (VOR)[7,8] which is ipsitorsional and upbeating; thereon, the fast-phase VOR, which is a refixation saccade and clinically recognized as positional nystagmus, is

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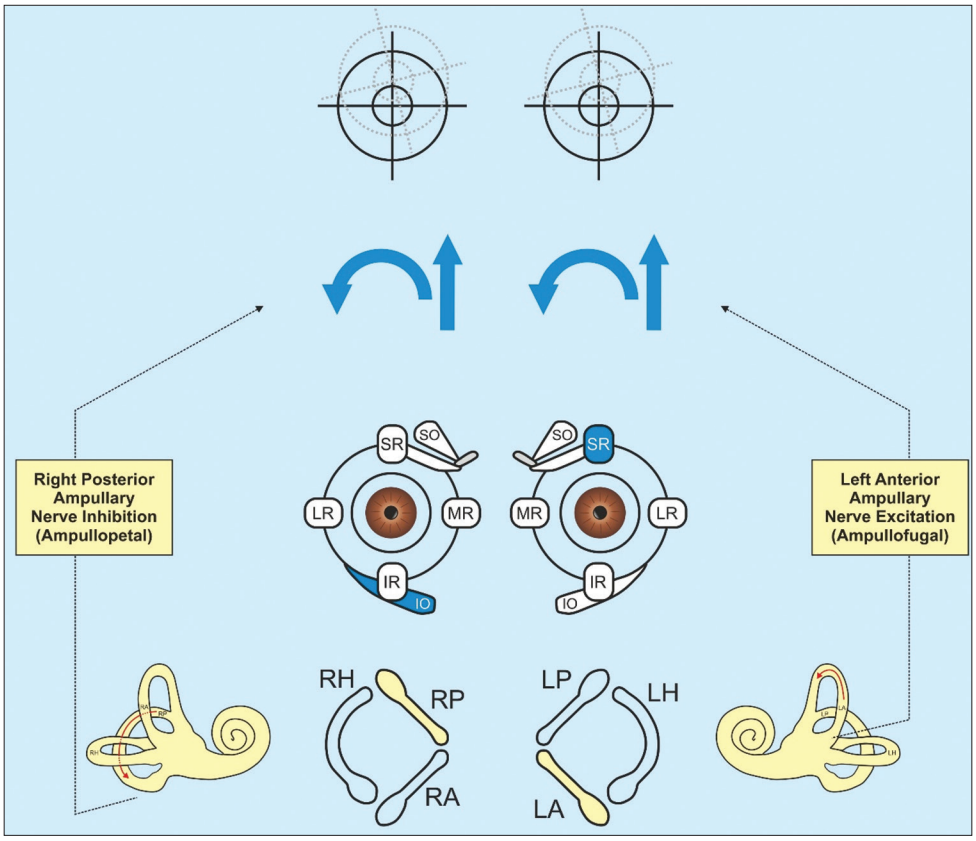

Figure 1: Inhibitory and excitatory projections of right PSC and left ASC. Inhibitory projections of right posterior semicircular canal and excitatory projections of left anterior semicircular canal are to right inferior oblique and left superior rectus resulting in slow-phase VOR which is upbeating and right torsional. *Propter hoc,* positional nystagmus (= fast-phase VOR) is downbeating and left torsional

contratorsional and downbeating [Figure 1]. Peripheral pDBN with torsional component could be either due to *apo*-PSC-BPPV or ASC-BPPV, and the oculomotor responses observed during positional tests in either are identical [Figure 1].[3] Hereat, Califano *et al*.[3,5] have proposed a classification of diagnostic certainty, based on response to treatment with physical therapy. In Califano's classification of *apo*-PSC-BPPV *Definite* implies intracanalar transformation to typical PSC-BPPV within 2 days of physical therapy, ***Probable*** when it resolves directly without transforming to typical PSC-BPPV after physical therapy, and *Possible* when symptoms persist despite five cycles of physical therapy/maneuvers and a normal MRI or when the patient is lost to follow up despite ongoing symptoms.

Material, Methods, and Observations

From February 1, 2023, to July 31, 2023, our Otoneurology Center in South Rajasthan identified and treated seven patients with *apo*-PSC-BPPV, of which three had bilateral disease. The demographic profile, symptomatology, oculomotor responses during positional tests, physical treatment, YouTube links of videos, and results of short-term follow-up at 1–4 hours, and after 24-hours are summarized in Table 1.

All seven patients had positionally triggered dizzy spells and a constant non-positional disequilibrium. Patients 3, 6, and 7 with bilateral disease had vomiting as well. The duration of symptoms ranged from 3 to 14 days. Six out of seven patients elicited downbeat nystagmus with a torsional component during DHT to either side and in the ESHHT. In left DHT, patient 5 after a latency of 5 seconds elicited a downbeating left torsional non-crescendo-decrescendo positional nystagmus of about 60s duration. In the right DHT, patient 5, after a latency of 1 second, initially elicited a right torsional upbeating crescendo-decrescendo positional nystagmus of 17 seconds, followed by a downbeating left torsional non-crescendo-decrescendo nystagmus of 55 seconds. The biphasic direction-reversing positional nystagmus during right DHT is explained by hypothesizing the initial otoconial clot location in the partially jammed non-ampullated arm of the posterior semicircular canal near the crus commune [Figure 2a]. Ampullopetal displacement of the otoconial clot during the initial left DHT moved it to the periampullary region [Figure 2b and c], and during the sequent right, DHT resulted in its stimulatory ampullofugal displacement causing an upbeating right torsional nystagmus initially. Thereupon, possibly because of a partial canal jam in the non-ampullated portion of right posterior canal, the otoconial clot ricocheted back in the ampullopetal direction with the accompanying second phase of inhibitory left torsional downbeating nystagmus [Figure 2d]. Oculomotor responses observed in patient 5 during right DHT are exceptional; only five cases of direction-reversing positional nystagmus (during

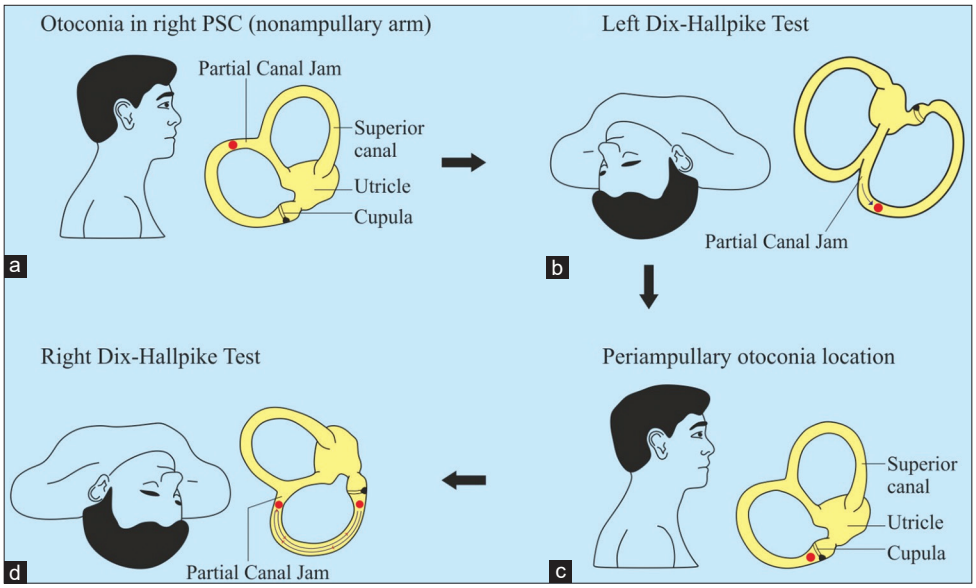

Figure 2: Otoconia during sitting, left and right DHT in patient 5. During left DHT, otoconial clot displaces ampullopetal in the partially jammed non-ampullated portion of right posterior semicircular canal (PSC) generating downbeating left torsional nystagmus (a and b). On uprighting debris moves to the periampullary location (c), and during right DHT debris first moves ampullofugal resulting in upbeating right torsional nystagmus, and while scuffling the jammed segment of canal it ricochets ampullopetal resulting in downbeating left torsional nystagmus (d)

DHT) in the posterior canal variant of BPPV are previously reported.[9,10] It is noteworthy that Yetiser's model[9] of biphasic positional nystagmus during DHT does not explain why the vertical component of BPPV remained upbeating but the torsional component reversed, and the case series of four patients by Jeong *et al.*,[10] of direction-reversing positional nystagmus during DHT only reported this phenomenon without elucidating its mechanism. In contrast, we hypothesize a partial reversible canal jam in the non-ampullated portion of the right posterior semicircular canal as the cause of biphasic direction-reversing positional nystagmus [Figure 2]. All except patient 7 were treated with QLRM. For QLRM patient was shifted from upright long-sitting on the examination table to the lateralized Dix-Hallpike position and maintained for 1 minute. Thereupon a quick rotation of head and body (in less than a second) to nose-down position on the non-involved side is carried out and maintained for 3 minutes, after which the patient is uprighted to short sitting.[3,5] A total of five QLRM were done in patients 1, 2, 4, 5, and patients were followed up by verifying positional tests at 1 hour and after 24 hours. These patients (1, 2, 4, and 5) had complete resolution of their initial symptoms with the disappearance of oculomotor responses on positional testing at short-term follow-up of 1 and 24 hours. Patient numbers 3 and 6 were treated with QLRM for right and left (five each for a side) initially and after 1 hour. Patient number 6 was asymptomatic at 24 hours with the resolution of oculomotor responses during positional test. The patient number 3 was symptomatic at 24 hours and elicited oculomotor response during positional test identical to initial examination. She was instructed to perform home-based Brandt-Daroff exercises[11] for a month but she was lost to follow-up. Patients with bilateral disease (patients 3 and 6) had multiple vomiting during QLRM. Patient 7 was instructed forced prolonged positioning (FPP) of the type Paolo Vannucchi reported in 2015,[2] to the left side on the first night and to the right on the subsequent night but the patient was lost to follow-up.

Discussion

apo-PSC-BPPV presents with positionally triggered vertigo paroxysms, non-positional disequilibrium, and vomiting. Diagnosis is challenging because oculomotor responses during positional test (DHT, ESHHT) elicit downbeating positional nystagmus with a torsional component directed to the non-involved (healthy) side. For this reason, an ipsilateral *apo*-PSC-BPPV is almost impossible to distinguish from the contralateral ASC-BPPV. Califano's classification, based on response to treatment with physical therapy, while following up the patient for 2 consecutive days grades the *apo*-PSC-BPPV as definite, possible, and probable.[3,5] Justifiably after physical therapy/repositioning maneuvers, it is pertinent to closely follow up with the patient with a putative diagnosis of *apo*-PSC-BPPV for next 2 days, specifically looking for the extirpation of *p*DBN and co-occurring vertigo. A reasonable corollary would be to treat every patient of positional vertigo with peripheral *p*DBN as *apo*-PSC-BPPV because it is more common compared to ASC-BPPV.[3,5] Short-term follow-up with verifying positional test helps in establishing its diagnosis and distinguishing it from the rarer ASC-BPPV. Patients with bilateral *apo*-PSC-BPPV are more symptomatic and require a greater number of QLRMs, and a rational approach is to treat one side in one sitting on day 1, with the other side treatment on day 2. FPP innovated in 2015 by Paolo Vannucchi[2] in a sequential manner may be useful in patients like number 7 who cannot tolerate QLRM.

Table 1: Clinical and demographic profile of patients

Age and Sex	Symptoms			Duration of Symptoms	Positional Nystagmus		YouTube Link	Treatment	Short-Term Follow-Up
	Positional Vertigo	Non-Positional Disequilibrium	Vomiting		Vertical Component	Torsional component			
38/M	+	+	-	6 days	Downbeat	L	https://youtu.be/PTF-wVVpSuw	QLRM	Asymptomatic
52/F	+	+	-	8 days	Downbeat	L	https://youtu.be/nMvLsm1Khnk	QLRM	Asymptomatic
29/F	+	+	+	11 days	Downbeat	L, R	https://youtu.be/Y59wf-OirN8	QLRM	Symptomatic
46/F	+	+	-	7 days	Downbeat	L	https://youtu.be/ochbVIaSJbE	QLRM	Asymptomatic
37/M	+	+	-	14 days	Biphasic/Downbeat	L	https://youtu.be/UnSLJp3QAA	QLRM	Asymptomatic
52/M	+	+	+	4 days	Downbeat	L, R	https://youtu.be/_6x6D-DES_I	QLRM	Asymptomatic
62/F	+	+	+	3 days	Downbeat	L, R	https://youtu.be/h86Fyltejjs	FPP	Follow-up lost

+, Present; -, Absent; L, Left; R, Right; M, Male, F; Female, QLRM; Quick Liberatory Rotation Maneuver, FPP; Forced Prolonged Positioning

Conclusion

Although *apo*-PSC-BPPV as a disease entity is neither included in the Barany Society's classification of benign paroxysmal positional vertigo[12] nor in the recommendations of the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS),[13] it is an important emerging peripheral positional vertigo syndrome. Physical therapy remains the cornerstone of treatment provided that the diagnosis is established with characteristic oculomotor responses during positional tests, coupled with the response to treatment during close follow-up over ensuing two days.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

The authors whose name are listed above certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

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