

Evaluation of the apogeotropic posterior canal benign paroxysmal positional vertigo: Case studies from a tertiary clinic perspective

Alex Zxi Jian Ho, MBBS(Hons)¹, Saiful Adli Jamaluddin, PhD², Yahia F Hussein, MMed (ORL-HNS)³, Iylia Ajmal Othman, MSurg ORL-HNS³, Ismah Syazana Zainudin, MBBS¹

¹Department of Otorhinolaryngology, Head and Neck, Sultan Ahmad Shah Medical Centre @IIUM, ²Kulliyah of Allied Health Sciences, International Islamic University Malaysia, ³Kulliyah of Medicine, International Islamic University Malaysia

SUMMARY

Benign paroxysmal positional vertigo (BPPV) is a prevalent vestibular disorder characterised by intermittent vertiginous episodes due to dislodged otoconia predominantly affecting the posterior canal. Atypical variants like apogeotropic posterior canal BPPV (aPC-BPPV) present diagnostic challenges and are likely underreported, particularly in Malaysia. We present two cases of aPC-BPPV. The first involves a 57-year-old female with persistent vertigo, initially misdiagnosed as right anterior canal BPPV. Video head impulse testing (VHIT) revealed reduced VOR gain in the right posterior canal, and the Demi-Semont manoeuvre successfully resolved her symptoms. The second case is a 62-year-old female, also misdiagnosed with left anterior canal BPPV. VHIT confirmed left posterior canal involvement, and repeated Demi-Semont manoeuvres led to symptom resolution. These cases highlight the intricate diagnostic challenges associated with aPC-BPPV, especially in differentiating it from contralateral anterior canal BPPV. Conventional diagnostic methods may yield incorrect results, emphasising the importance of incorporating advanced tools like VHIT as an ancillary assessment for precise canal identification. Atypical nystagmus and persistent symptoms after standard treatment warrant re-evaluation, with unresolved anterior canal (AC-BPPV) cases referred to a tertiary centre, as they may represent aPC-BPPV. Effective management often requires repeated, canal-specific manoeuvres such as the Demi-Semont, which have shown greater success in relieving symptoms in aPC-BPPV cases. Accurate diagnosis and targeted treatment are crucial for managing atypical BPPV. Improved awareness and advanced diagnostic methods, such as VHIT, can enhance outcomes, especially in cases where atypical presentations are not well-documented.

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is a common vestibular disorder resulting from otoconia displacement in the semicircular canals, resulting in vertigo. The otoconial particles may either float (canalolithiasis) or adhere to the cupula (cupulolithiasis).¹ BPPV classification is based on oculomotor responses during changes in head positioning. The Dix-Hallpike manoeuvre evaluates vertical canal involvement while the supine head roll test assesses

horizontal canal involvement. Nystagmus patterns help identify the affected semicircular canal.²

Nonetheless, clinical presentations may not always align with established diagnostic criteria. Although BPPV typically affects the anterior, posterior, and lateral canals, atypical variants such as apogeotropic posterior canal BPPV (aPC-BPPV) and others have been observed. aPC-BPPV is the most documented atypical variant but is infrequently reported in Malaysia, where common BPPV cases prevail.^{2,3}

This article offers an in-depth description of our experiences with aPC-BPPV and provides a thorough review of relevant literature. To our knowledge, there are no published cases of aPC-BPPV in Malaysia. Through this, we aim to enhance diagnostic accuracy and treatment efficacy for such cases.

CASE PRESENTATION

Patient 1

A 57-year-old woman was under clinical observation for two years due to recurrent episodes of BPPV. She was diagnosed with bilateral mild to severe sensorineural hearing impairment approximately ten years prior. The initial evaluation revealed the presence of down-beating torsional nystagmus during the Dix-Hallpike manoeuvre towards the right, indicative of right anterior canal BPPV (AC-BPPV). The MRI of the brain was unremarkable. Consequently, a deep head hanging manoeuvre was executed. A week later, the patient returned with analogous complaints of recurrent vertigo. Upon re-evaluation using the Dix-Hallpike manoeuvre, a down-beating torsional nystagmus was again observed, corroborating the diagnosis of right-sided AC-BPPV. She was treated with the deep head hanging manoeuvre; however, her symptoms did not resolve. A repeated Dix-Hallpike manoeuvre indicated the presence of apogeotropic nystagmus upon positioning to the right.

Following this, a video head impulse test (VHIT) was conducted to further assess the vestibulo-ocular reflex (VOR) function across all six semicircular canals, which disclosed a reduced VOR gain in the right posterior semicircular canal (Figure 1). In response to these findings, she was treated with the Demi-Semont manoeuvre. During her subsequent consultations, the patient exhibited persistent symptoms

This article was accepted: 11 June 2025

Corresponding Author: Alex Zxi Jian Ho

Email: alexhozxiian@gmail.com

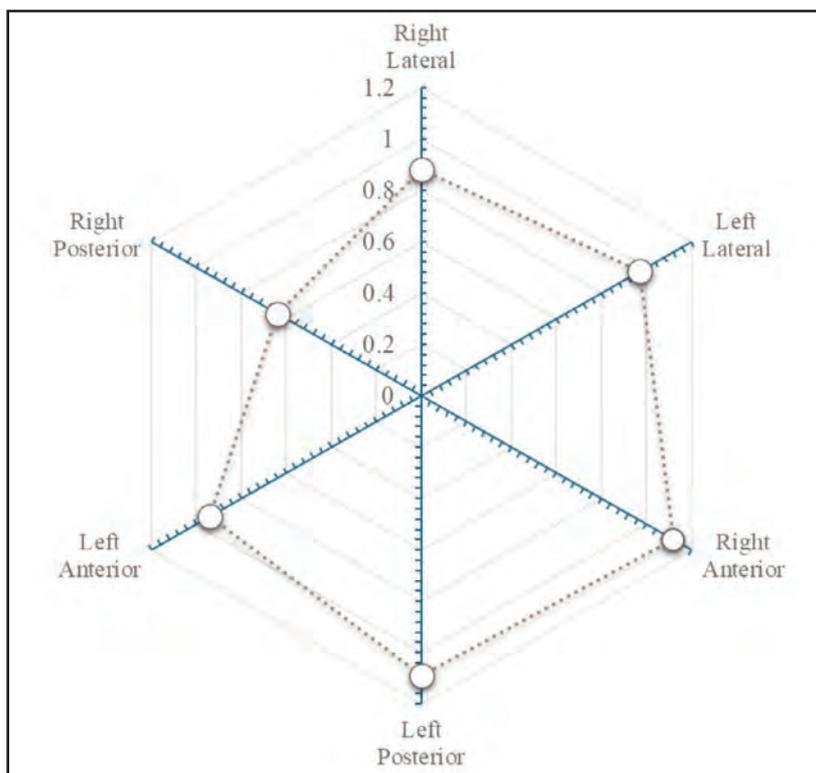


Fig. 1: VHIT results showing average instantaneous velocity gain at 60 milliseconds for the lateral canals and the velocity regression gain for the vertical semicircular canals (anterior & posterior) for Patient 1. There was a clinically significant reduction of VOR gain in the right posterior semicircular canal with VOR gain of 0.64 ± 0.25 as compared to the average VOR gain of other canals of 1.0 ± 0.11 .

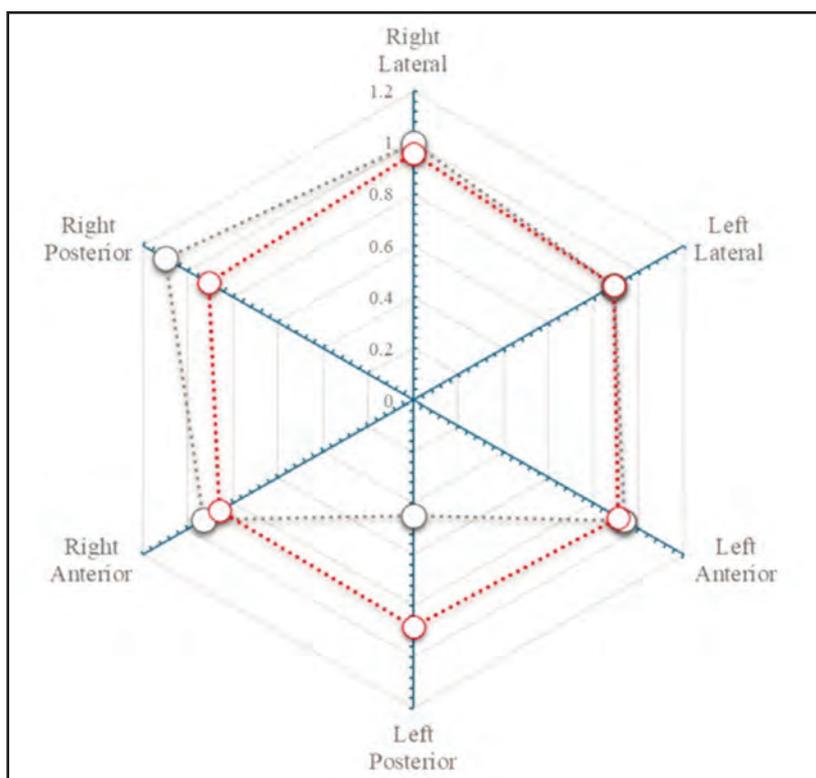


Fig. 2: VHIT results showing average instantaneous velocity gain at 60 milliseconds for the lateral canals and the velocity regression gain for the vertical semicircular canals (anterior & posterior) for Patient 2. The dotted red and grey-lined markers indicate the VOR gain of the first and second VHIT evaluations, respectively. The second evaluation showed a clinically significant reduction of VOR gain in the left posterior semicircular canal at 0.45 ± 0.35 .

despite adherence to a structured vestibular rehabilitation program. Nevertheless, she maintained an active lifestyle and reported a significant reduction in the incidence of dizziness.

Patient 2

A 62-year-old woman presented with a three-month history of episodic vertigo. The pertinent medical history included a prior diagnosis of acute labyrinthitis, which had been treated following an upper respiratory tract infection that occurred two months earlier. A baseline audiogram indicated a mild to moderate sensorineural hearing impairment in the right ear, alongside a profound sensorineural hearing loss in the left ear.

Initial assessment revealed a slight rotational down-beating nystagmus upon Dix-Hallpike manoeuvre to the left, indicating left anterior semicircular canal involvement. The results of the initial VHIT were deemed unremarkable (Figure 2). Subsequently, she underwent treatment involving the deep head hanging manoeuvre. Upon follow-up two weeks later, the patient reported ongoing episodes of persistent paroxysmal vertigo. The Dix-Hallpike manoeuvre performed towards the left indicated an up-beating torsional nystagmus. The Epley manoeuvre was executed as part of the treatment.

During her next appointment, she encountered another episode of positional vertigo, with the Dix-Hallpike manoeuvre exhibiting torsional up-beating nystagmus upon left positioning. A VHIT was conducted, which demonstrated a reduced VOR gain in the left posterior semicircular canal (Figure 2). She was then treated with the Demi-Semont manoeuvre. This episode persisted for approximately six weeks, during which each consultation necessitated the execution of the Demi-Semont manoeuvre as the assessment confirmed a diagnosis of left aPC-BPPV. Throughout this timeframe, a vestibular evoked myogenic potential (VEMP) test showed normal functionality of the saccule and inferior vestibular nerve in both auditory canals. Patient 2 reported symptomatic relief following each Demi-Semont manoeuvre and ultimately achieved symptom resolution after three treatment sessions. Furthermore, her brain MRI did not reveal any abnormalities that could be linked to her condition.

DISCUSSION

Posterior canal BPPV is the most common form of BPPV. In its cupulolithiasis variant, nystagmus is characterised as vertical, torsional, and up-beating, with a more prolonged but less intense presentation than in the canalolithiasis variant. At the same time, patients with bilateral SNHL may have concurrent vestibular degeneration, affecting hair cells, nerves, or labyrinthine structures, leading to impaired balance, as seen in Patient 1.

aPC-BPPV is marked by nystagmus occurring in any head-hanging position (right, left Dix-Hallpike, or head-hanging).⁴ This nystagmus has no latency, follows a crescendo-decrescendo course, lasts longer, and is less intense. It is primarily vertical-down-beating with a torsional component (clockwise for the right ear, anticlockwise for the left) and

does not fatigue with repeated positioning.⁴ Reduced labyrinthine impedance (as in superior canal dehiscence) and otolithic membrane damage can facilitate otolith mobilisation and displacement, increasing the risk of aPC-BPPV.⁵

Canalith jam occurs due to endolymphatic flow blockage by otoconial particles, leading to a transient reduction in the vestibular-ocular reflex. The condition may arise due to innate semicircular canal stenosis or the formation of a plug by otoconial debris, with these theories interrelated as canal stenosis can increase the risk of debris jamming at the stenosed site.⁶ This jamming alters intracanal hydrostatic pressure, causing persistent deflection of the cupula and spontaneous nystagmus with all head position changes.⁶

Diagnosing aPC-BPPV is challenging due to the difficulty in distinguishing it from contralateral AC-BPPV, as both conditions can be provoked by head-hanging positions (Dix-Hallpike test, straight head-hanging position). Differentiating these conditions is crucial as their treatments differ. The suspicion of atypical nystagmus should prompt further positional testing to determine the debris location within the canal. Helminski observed that in anterior canal BPPV, nystagmus typically has a slight or absent torsional component towards the affected ear, whereas the torsion is directed away from the involved ear in aPC-BPPV.⁷

aPC-BPPV can be treated with the Quick Liberatory Rotation manoeuvre. Other effective manoeuvres include the Demi-Semont and the 45-degree forced prolonged position technique.⁸ These manoeuvres confirm the diagnosis of aPC-BPPV, as AC-BPPV does not respond favourably to these treatments. Vannucchi et al. reported a 68.7% success rate with these manoeuvres.⁴ They also reported complete resolution in 5 of 11 patients (45%) with aPC-BPPV three days after a single Demi-Semont manoeuvre.⁴ While the frequency of manoeuvres was not specified, repeated treatments are common in BPPV and likely applicable. In our case, symptoms are resolved after multiple Demi-Semont sessions.

VHIT may help to confirm the involved canal by detecting impairments in the vestibulo-ocular reflex (VOR) of the affected semicircular canal. Overall, VHIT sensitivity in identifying the affected semicircular canal was reported to be 72.9%. Castellucci et al. stated that VHIT differentiates aPC-BPPV from contralateral anterior canal BPPV in cases of positional downbeat nystagmus, thereby guiding appropriate corrective manoeuvres.⁹ A reduction in VOR gain for the involved canal, which normalises upon symptom resolution, is seen on VHIT.⁹ Follow-up post-treatment is crucial, as treatment response helps distinguish aPC-BPPV from the rarer AC-BPPV.

While referral to tertiary centres is ideal, aPC-BPPV can be diagnosed clinically without VHIT through careful history and positional testing. The Dix-Hallpike may show down-beating torsional nystagmus, with the torsional component beating away from the affected ear. Comparing both sides aids lateralisation. The Demi-Semont manoeuvre can assist in both diagnosis and treatment, especially in experienced hands. We recommend attempting it in patients with

persistent, prolonged, non-latent torsional down-beating nystagmus.

The cases exemplify the diagnostic and therapeutic complexities of aPC-BPPV, consistent with current literature. Both patients experienced persistent vertigo despite standard repositioning manoeuvres, necessitating the need for careful differentiation from atypical BPPV forms. The reduction in VOR gain in the implicated canals underscores the importance of using VHIT as an ancillary tool for accurate canal identification. In line with current recommendations, persistent and unresolved AC-BPPV cases should be reviewed or referred to a tertiary vertigo centre, as they may be aPC-BPPV, necessitating targeted management for better patient outcomes.

CONCLUSION

This research highlights the diagnostic and therapeutic challenges inherent in aPC-BPPV. Persistent or refractory AC-BPPV should be further investigated for possible canalith jam of the posterior semicircular canal. Accurate identification, facilitated by the VHIT is vital for effective intervention. The efficacy of specialised manoeuvres like the Demi-Semont highlights the significance of individualised approaches. These findings underscore the need for greater awareness and precise management to improve outcomes in atypical BPPV cases.

CONFLICT OF INTEREST

The author(s) declare no conflicts of interest regarding the research, authorship, and publication of this article.

REFERENCES

1. von Brevern M, Bertholon P, Brandt T, Fife T, Imai T, Nuti D, et al. Benign paroxysmal positional vertigo: Diagnostic criteria. *J Vestib Res* 2015; 25(3-4): 105-17.
2. Califano L, Mazzone S, Salafia F, Melillo MG, Manna G. Less common forms of posterior canal benign paroxysmal positional vertigo. *Acta Otorhinolaryngol Ital* 2021; 41(3): 255-62.
3. Richard W, Zainal H, Jamaluddin S, Rasheed s, Al-Hadeethi Y. Balance Disorders: A review of patients at a specialized vestibular clinic. *IUM Medical Journal Malaysia* 2022; 21: 121-7.
4. Vannucchi P, Pecci R, Giannoni B, Di Giustino F, Santimone R, Mengucci A. Apogeotropic posterior semicircular canal benign paroxysmal positional vertigo: some clinical and therapeutic considerations. *Audiol Res* 2015; 5(1): 130.
5. Castellucci A, Malara P, Martellucci S, Delmonte S, Ghidini A. Fluctuating posterior canal function in benign paroxysmal positional vertigo depending on how and where otoconia are disposed. *Otol Neurotol* 2021; 42(2): e193-e8.
6. Martellucci S, Castellucci A, Malara P, Pagliuca G, Clemenzi V, Stolfa A, et al. Spontaneous jamming of horizontal semicircular canal combined with canalolithiasis of contralateral posterior semicircular canal. *J Audiol Otol* 2022; 26(1): 55-60.
7. Helminski JO. Peripheral downbeat positional nystagmus: Apogeotropic posterior canal or anterior canal BPPV. *J Neurol Phys Ther* 2019; 43 Suppl 2: S8-S13.8.
8. Vannucchi P, Pecci R, Giannoni B. Posterior semicircular canal benign paroxysmal positional vertigo presenting with torsional downbeating nystagmus: an apogeotropic variant. *Int J Otolaryngol* 2012; 2012: 413603.9.
9. Castellucci A, Malara P, Martellucci S, Botti C, Delmonte S, Quagliari S, et al. Feasibility of Using the Video-Head Impulse Test to Detect the Involved Canal in Benign Paroxysmal Positional Vertigo Presenting With Positional Downbeat Nystagmus. *Front Neurol* 2020; 11: 578588.