



BPPV - A MAJOR DIAGNOSTIC DILEMMA. CASE STUDY OF POSTERIOR CANAL BPPV NOT RESPONDING TO CANAL REPOSITIONING MANOEUVRE, POSING A DIAGNOSTIC CHALLENGE IN MANAGEMENT.

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ABSTRACT

Posterior Canal Benign Paroxysmal Positional Vertigo (BPPV) is a common vestibular disorder characterized by recurrent brief episodes of vertigo triggered by specific head movements. While the majority of cases can be successfully resolved through repositioning manoeuvres, a subset of patients experiences unresolved or persistent symptoms despite appropriate treatment. This case report aims to discuss an unresolved case of Posterior canal BPPV after giving medical management and manoeuvres and provide a comprehensive conclusion based on current knowledge and research about management of such cases.

KEYWORDS

INTRODUCTION:

Benign paroxysmal positional vertigo (BPPV) is the prime cause of vertigo in about 17%(1) of cases. Researches have shown 90% cases the BPPV are of posterior canal, whereas lateral canal is 8% and of superior canal is of only 2%(2). This is a challenging case of posterior canal BPPV of a young female, who presented with multiple recurrent episodes of BPPV symptoms besides giving medical treatment and doing several canal repositioning manoeuvres.

CASE REPORT:

Case of 45 year old female, came to OPD with history of rotatory vertigo on getting up from bed which lasted for less than a minute, associated with nausea and vomiting. ENT and Otoneurological evaluation was normal. Dix Hallpike showed upbeatting torsional nystagmus with reversal suggestive of left posterior canal BPPV. Basic vestibular evaluation with video-nystagmography (VNG) confirmed the findings of left posterior canal BPPV. Epley's manoeuvre was given for correction, and patient was observed for resolution of symptoms.

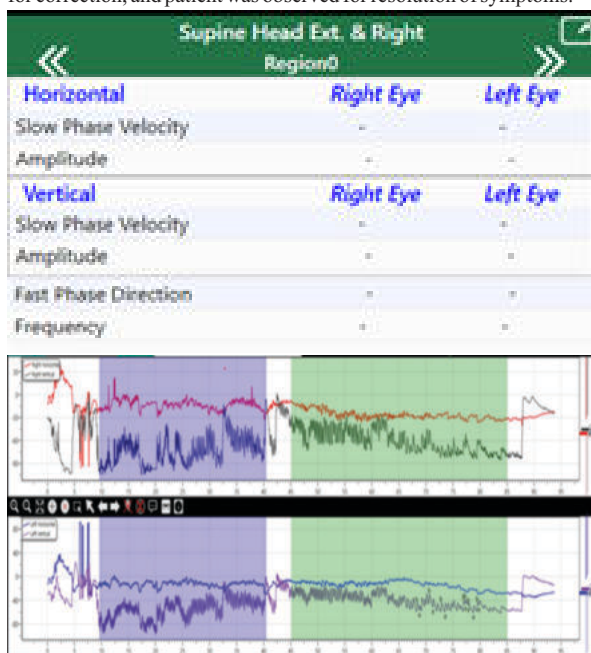


Figure 1: Dix Hallpike Right – Supine head extension right

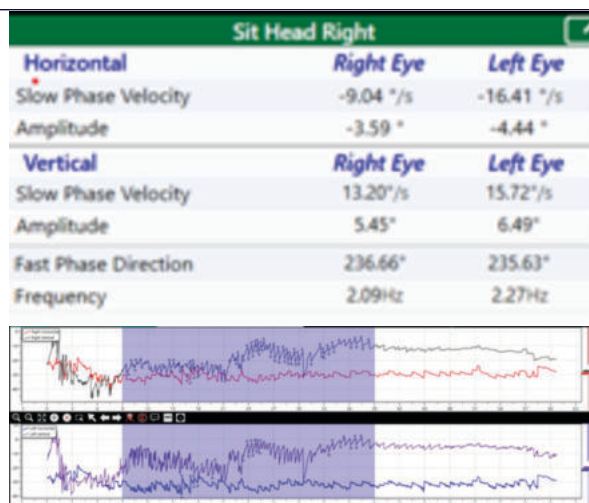
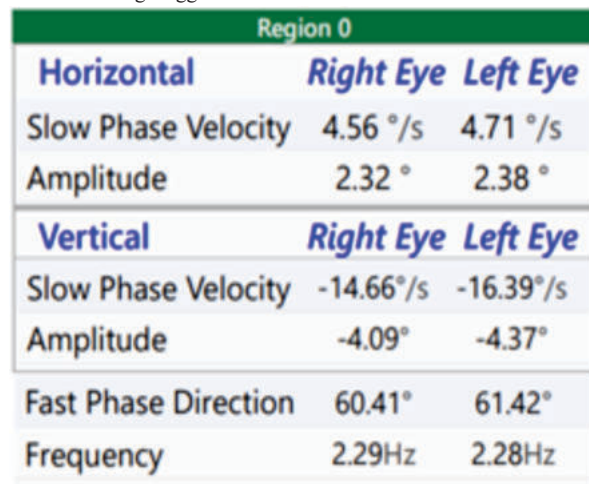


Figure 2: Dix Hallpike Right – Sit head right

She complained of similar symptoms after a day with rotatory spins persisting on getting up from bed. Dix Hallpike and VNG also showed the same findings suggestive of Left Posterior canal BPPV.



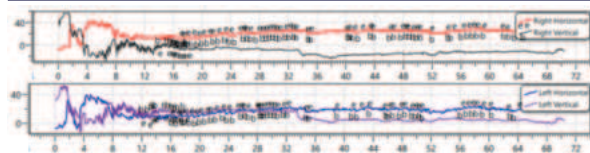


Figure 3: Dix Hallpike Left – Supine head extension left

This episode was corrected with Left Semont leberatory manoeuvre keeping a possibility of cupulolithiasis variant of BPPV. Patient still continued to experience further spinning episodes of vertigo on getting up from bed as seen on her repetitive of follow ups till the next one month , during which manoeuvres were repeated in each visit.

After treating with combination of manoeuvres and medical treatment, she remained asymptomatic for 3 months and again developed vertigo on turning position in bed and getting up from the bed. This episode was associated with headache. In this episode she performed self manoeuvres for correction and her symptoms got worse. VNG was performed and was diagnosed with left intense up beating posterior canal BPPV, corrected with left Epley's manoeuvre. Similar recurring episode she got after 10 days with similar symptoms and examination findings. This time we added Tab. Sibelium 10mg and with gave left Epley's and extended Semont's manoeuvre.



Figure 4: Dix Hallpike Left – Supine head extension left



Figure 5: Dix Hallpike Left – Sit head left

Similar recurring episode she got after 10 days with similar symptoms and examination findings. This time she was treated with Tab. Sibelium 10mg with left Epley's and extended Semont's manoeuvre. In view of her frequent and recurrent episodes even after giving manoeuvre corrections and medical treatment, she was advised MRI brain. MRI Brain showed type II vascular loop in the left internal

auditory canal with mildly attenuated flow related signal in supraclinoid segment of both internal carotid arteries. This unresolving case of posterior canal BPPV posed a significant challenge in management in clinical practice.

DISCUSSION:

Posterior canal BPPV is typically caused by the displacement of otoconial debris into the posterior semi-circular canal of the inner ear. This condition can usually be effectively managed with canalith repositioning procedures, such as the Epley or Semont manoeuvres, which aim to relocate the displaced particles and restore vestibular function. Some resolve without specific treatment, proving the self-limiting nature of the disease. However, a subset of patients continues to experience symptoms even after appropriate treatment, posing a challenge to clinicians and researchers.

A lack of response to at least 3 consecutive sessions of CRM within 2 weeks, or frequent recurrence of symptoms despite partial or total response to CRM, should prompt the physician to investigate the possibility of other conditions—for instance, an intracranial space-occupying lesion especially of posterior fossa. Rarely, patients with tumors of the fourth ventricle or with vestibular schwannomas present with persistent positional vertigo(3). The exact causal relationship between episodic BPPV and an intracranial space-occupying lesion is debated. One of the mechanisms suggested is that there is vascular compromise of the affected labyrinth as the tumor causes ischemia of the anterior vestibular artery, which leads to posterior canal BPPV(4). Other treatable conditions that should be excluded include local aural conditions like chronic otitis media, Meniere's disease, or perilymph fistula(5). Perez et al(6) describe patients with Meniere's disease in whom attacks of BPPV may precede, accompany, or follow the attacks of Meniere's disease. Careful monitoring of patients who have had CRMs is therefore essential.

Once it is clear that BPPV is not improved by a particular CRM and no other treatable cause for posterior canal BPPV has been identified, an attempt should be made to try an alternative CRM or habituation procedure. It may be that cupulolithiasis, and not canalithiasis, is causing BPPV; hence, the appropriate CRM is required. Some authors describe differentiating between cupulolithiasis and canalithiasis by means of the Dix-Hallpike maneuver(7). In cupulolithiasis, when the head is placed in the Dix-Hallpike position, there is no latency in the onset of vertigo and nystagmus. Further, nystagmus and vertigo last as long as the head is maintained in the provoking position. In canalithiasis, however, there is usually a latency period of about 1 to 40 seconds before the onset of vertigo and nystagmus when the head is placed in the Dix-Hallpike position, and vertigo and nystagmus disappear within 60 seconds if the head's position is maintained. When the patients of Harvey and colleagues(8) responded neither to the PRM nor to a modified liberatory manoeuvre, the authors prescribed the Brandt-Daroff exercises. Occasionally, vestibular habituation therapy is more effective than CRMs. Steenerson and Cronin(9) found that the 3 patients who had persistent BPPV following CRMs had complete relief of symptoms following institution of vestibular habituation exercises.

Some patients with BPPV have either horizontal canal canalithiasis or a combination of horizontal canal and posterior canal canalithiasis. Such patients do not attain complete relief with PRMs such as the Epley or Semont manoeuvre alone. Indeed, Herdman and Tusa(10) suggest that a few patients who have been treated for posterior semi-circular canal BPPV may be found to have horizontal canal BPPV after treatment. The conversion of posterior semi-circular canal BPPV to lateral semi-circular canal BPPV is believed to be due to the shift of otoconial debris from the posterior canal to the horizontal canal via the utricle.

Finally, a certain proportion of patients who have persistent BPPV may benefit from surgical procedures like posterior ampullary nerve section or posterior semi-circular canal occlusion. Included in this group are those with vestibular atelectasis. This entity was first described by Merchant and Shucknecht(11) in a post-mortem diagnosis made in individuals whose temporal bones had collapsed walls of the ampullae of the semi-circular canal and utricle, causing restriction of movement of the cupula and otolithic membrane. These patients had experienced positional vertigo and persistent unsteadiness. In such patients PRMs are unlikely to be effective. The surgical procedures for BPPV are associated with a small but significant risk of hearing loss, which may be acceptable to patients with severe symptoms.

Increase frequency of recurrence of BPPV may be due to underlying comorbid conditions (12) and also along with central causes. Multiple factors can contribute to the persistence or recurrence of symptoms, including multiple canal involvement, underlying vestibular pathologies, migraine overlay and treatment-related factors. Clinicians should be aware of these factors and consider a comprehensive approach, including a thorough evaluation of the patient's clinical history, physical examination, and the possibility of underlying comorbidities. Collaboration between otolaryngologists, neurologists, and vestibular rehabilitation specialists may be necessary in managing unresolved cases.

CONCLUSION:

Even though posterior canal is the commonest and its very challenging to treat cases of posterior canal BPPV of cupulolithiasis causes. In this case we are presenting a case who came with signs and symptoms of posterior canal BPPV of (cupulolithiasis cause) along with central component. Even though comparing to peripheral causes of vertigo, central vertigo is less common, but its essential to distinguish the cause of vertigo for the better outcome of the patient. Vertigo management is always in a systematic and multidisciplinary way.

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