Neurological Sciences (2020) 41:263–269
https://doi.org/10.1007/s10072-019-04101-0

REVIEW ARTICLE

Central mimics of benign paroxysmal positional vertigo: an illustrative case series

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Received: 13 June 2019 / Accepted: 9 October 2019 / Published online: 6 November 2019

Abstract
Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disorder that is diagnosed based solely on clinical findings. Rarely, central lesions can present with positional vertigo and nystagmus, mimicking BPPV. Recognised red flags that may help distinguish central mimics from BPPV include the presence of additional neurological symptoms and signs, atypical nystagmus patterns, and the absence of a sustained response to repositioning manoeuvres. We present seven cases that illustrate how heuristic bias may affect the detection of these features in practice. Furthermore, our cases suggest that isolated downbeat positional nystagmus (simulating anterior canal BPPV) and apogeotropic horizontal nystagmus on the supine roll test (simulating horizontal canal BPPV) should be considered additional red flags.

Keywords Clinical neurology · Nystagmus · Vertigo

Introduction

Benign paroxysmal positional vertigo (BPPV) results from abnormal activation of the semicircular canals, due to detached otoconia moving in the canal endolymph, in response to changes in head position [1].

The clinical diagnosis of BPPV relies on a typical history and specific diagnostic manoeuvres. Neuroimaging with MRI is only indicated when additional neurological features are present or when positional vertigo and nystagmus have atypical features or fail to resolve with repeated therapeutic positional manoeuvres [2].

Certain cerebellar lesions can present with positional vertigo and nystagmus. These are usually associated with additional clinical features that point to a central cause. It is exceedingly rare, but possible, for positional vertigo and nystagmus to be the sole clinical finding in central paroxysmal positional vertigo (CPPV) [1].

We present seven cases in which the clinical features were initially considered to represent BPPV by a tertiary neurologist, neuro-otologist or otolaryngologist, but found to have CPPV due to cerebellar lesions. The features that help differentiate the two entities are reviewed.

Cases

Case 1

A 46-year-old woman presented with a 5-month history of anorexia, vomiting, and intermittent frontal and occipital headaches of increasing frequency. Bending was associated with vomiting, headache and vertigo.

On examination, there were no pyramidal or cerebellar signs. Tandem gait was normal. Eye movements were normal. When lying supine with the head inclined forward by 30°, head turning to either side (supine roll test) resulted in

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apogeotropic nystagmus (beating away from earth), without a latency, and decreased in the maintained posture. This was interpreted to be horizontal canal BPPV.

MRI scan of the brain was performed in view of the increasing headaches and revealed a midline posterior fossa ependymoma extending from the foramen magnum to the fourth ventricle (Fig. 1a). After resection, there was resolution of vomiting and headache, and rapid improvement in positional vertigo.

Case 2

A 50-year-old man awoke with acute vertigo and vomiting. Brief positional vertigo and vomiting, when lying down and turning to the left continued for 2 weeks and then improved. There was a single episode of vertigo 7 years previously, lasting several hours, with total resolution over 1 day. On examination, the patient moved like a wooden soldier, avoiding head movements.

Neurological examination showed normal eye movements, normal gait with eyes closed and normal tandem gait. On Dix-Hallpike manoeuvre to the right, there was immediate torsional nystagmus towards the right ear, which rapidly became obliquely downbeat and diminished over 20 s and ceased. Subsequent repeated vomiting precluded repeat testing to assess for habituation. The absence of a latent period and an oblique downbeat component to the nystagmus raised suspicion of central pathology, but all other factors suggested peripheral pathology. It was concluded that the patient was likely to have atypical BPPV. A brain MRI without gadolinium was reported as normal.

After 10 days, the positional dizziness gradually improved but vomiting persisted especially on positional change. There

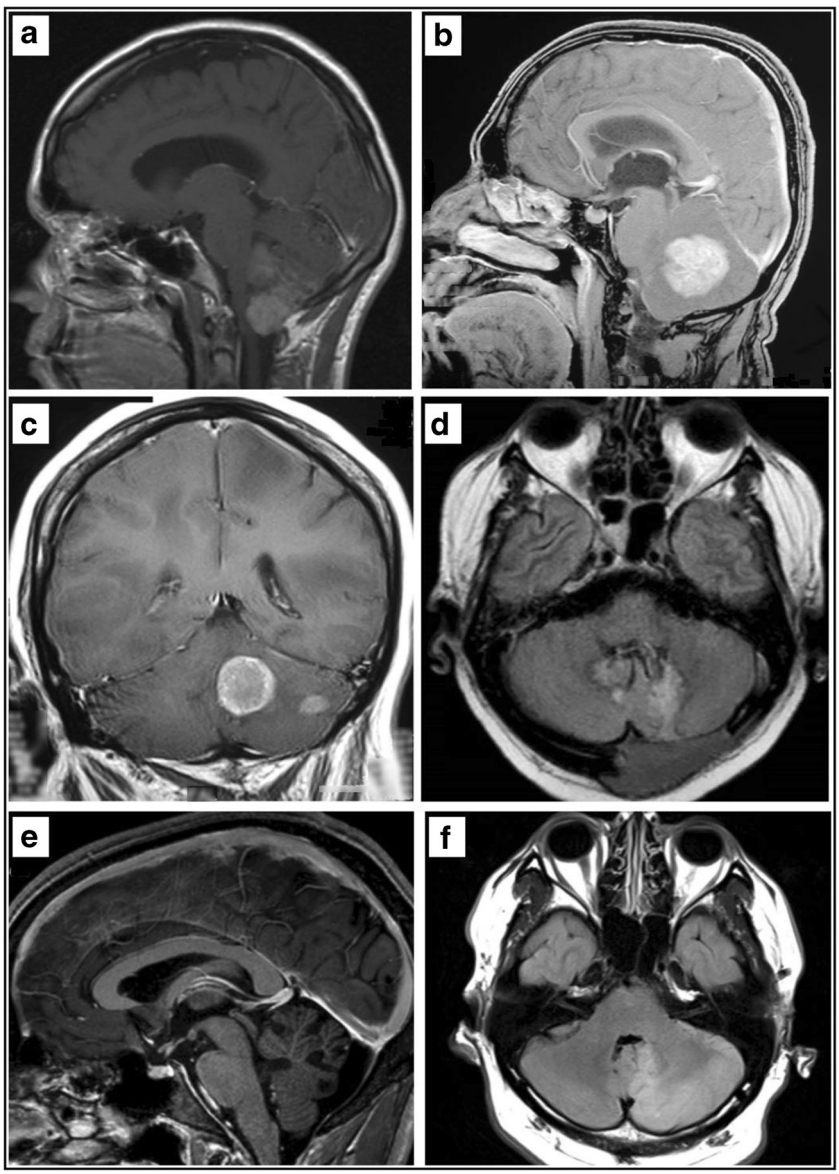

Fig. 1 MR images for patients 1–5 and 7 showing cerebellar lesions. **a** Patient 1 with posterior fossa ependymoma (T1 + gadolinium); **b** patient 2 with cerebellar lymphoma (T1 + gadolinium); **c** patient 3 with multiple enhancing cerebellar lesions (T1 + gadolinium); **d** patient 4 with metastatic lesion of cerebellar vermis and left hemisphere (FLAIR); **e** patient 5 with multiple cerebellar lesions due to glioneuronal tumour (TFE + gadolinium); **f** patient 7 with left PICA infarct (FLAIR)

were no new signs and Dix-Hallpike testing was negative. It was presumed that he still had atypical BPPV. By 7 weeks, the positional dizziness, nausea and vomiting had completely resolved. Repeat neurological examination was normal, although positional testing was not repeated.

At 9 weeks, the patient had become unsteady on tandem gait with severe pain at the top of his neck, worse on lying supine. Subjective dizziness on Dix-Hallpike testing was again noted; however, there was no nystagmus and no other abnormalities on neurological examination. A repeat MRI brain with gadolinium showed a lobulated midline cerebellar mass with hydrocephalus (Fig. 1b). Biopsy showed lymphoma and treatment with radiotherapy was commenced. In retrospect, the initial MRI was abnormal.

Case 3

A 65-year-old woman presented with 1 month of positional vertigo. Her symptoms had started with a severe headache lasting 2 h, associated with nausea and vomiting. On two occasions, she felt a bursting sensation in her right ear. She had developed a tendency to veer to the right when walking.

On examination, she was unsteady standing with her feet together, but tandem gait was normal. Eye movements were normal. Finger-nose testing on the left was mildly clumsy. There was one short episode of downbeat nystagmus on the Dix-Hallpike manoeuvre to the right. Supine roll test to either side showed apogeotropic nystagmus which started after a short latency and decreased with time. Repeated repositioning treatment for horizontal canal BPPV performed for 2 weeks was unsuccessful.

A MRI scan showed multiple lesions in the cerebellar hemispheres with the largest one near the vermis, suggestive of metastatic disease (Fig. 1c). Symptoms remained refractory despite treatment with steroids and radiotherapy. The patient died 2 months after her initial evaluation.

Case 4

A 65-year-old man with previous bowel cancer presented with a 2-week history of severe rotational vertigo, precipitated by angular and vertical head movements. He had unsteadiness, nausea and as subsequently advised by his family, occasional slurred speech over the previous 3 weeks.

On examination, the patient had a narrow based gait but was unable to walk in tandem. Ankle jerks were absent. Light touch was diminished in the soles of feet (unchanged since previous chemotherapy). There were hypermetric saccades on lateral eye movements, with overshoot more pronounced on re-centering.

On lying supine from sitting, there was a small amount of down beat nystagmus followed by 25 s of left beating nystagmus. On moving from supine to right ear dependent, there was a small amount of apogeotropic nystagmus which decreased and resolved with maintained position but then reoccurred after an interval on repeat clinical examination. The remaining neurological examination was normal.

A MRI head scan showed a lesion involving the left cerebellar hemisphere and vermis, consistent with metastases (Fig. 1d). The left cerebellar lesion was excised and he was referred to hospice for ongoing management. He died 4 months after the surgery.

Case 5

A 32-year-old man had a 6-year history of occasional attacks of vertigo and nausea lasting less than 30 s, when changing head position. Examination revealed no spontaneous nystagmus and normal head impulse test. A supine head-hanging manoeuvre elicited, after a short latency, downbeat nystagmus lasting 10 s. A liberatory manoeuvre to treat anterior semicircular canal BPPV was performed, moving the patient's head forward (chin to chest) and bringing the head and body into the sitting position. A subsequent head-hanging manoeuvre did not evoke nystagmus. The patient was asymptomatic when released from the Emergency Department.

Forty-eight hours later, the patient was symptomatic again and the clinical examination was identical to the first consultation. A MRI with gadolinium revealed non-enhancing lesions in the midline cerebellum due to glioneuronal tumour of the fourth ventricle (Fig. 1e).

Case 6

A 40-year-old male presented with 6 months of brief episodes of vertigo and nausea provoked by rolling over in bed and bending the head forward. Neurological examination was normal, with no nystagmus at rest and normal head impulse test. A supine head hanging manoeuvre and bilateral Dix-Hallpike manoeuvres resulted in brief positional downbeat nystagmus with nausea and vertigo. A reversion of the nystagmus direction to upbeat was occasionally observed when he sat up from the supine position. A repositioning manoeuvre intended to treat anterior canal BPPV was repeatedly ineffective.

Brain MRI showed a cerebellar cystic lesion located posterolateral to the fourth ventricle, suggestive of neurohydatosis. Serum IgG antibody for *Echinococcus granulosus* was positive and treatment with albendazole was started. He declined surgery, and further follow-up showed no clinical or radiological lesion progression.

Case 7

A 28-year-old man developed vertigo when he hit his head on a windowsill in the process of lying down. There was a preceding 3-week history of cough-induced neck pain radiating

to the occiput and across the top of the head, not associated with any injury.

He had vertigo on movement, with associated severe vomiting. He was reluctant to stand. At times there was third degree nystagmus to the left, but this was not persistent. After moving around the bed, the patient would develop recurrent vomiting. There was normal pursuit, normal saccades, no vertical skew, no finger-nose ataxia, and no clear heel-shin ataxia, although he was of large build and performed the test poorly.

A normal vestibulo-ocular reflex was confirmed by video-oculography. Supine roll test resulted in apogeotropic nystagmus which decreased with time, as well as vertigo and oscillopsia, in both left and right ear dependent positions. The nystagmus was slightly more prominent with left ear dependent. He was treated for horizontal canal BPPV affecting the right ear. A CT head scan organised by Emergency Department colleagues was normal.

The following morning, he was much improved. There was no spontaneous nystagmus, but variable and asymptomatic apogeotropic nystagmus with the left ear dependent alone. There was slight dysmetria on heel-shin testing on the left. He could stand and walk normally, but he was unsteady walking heel-toe. MRI/MRA confirmed a left posterior inferior cerebellar artery infarct (Fig. 1f) and dissection of the left vertebral artery.

Discussion

BPPV is a common peripheral vestibular disorder with a cumulative incidence of 10% over the lifetime [3]. It results from abnormal activation of the semicircular canals, due to detached otoconia moving in the canal endolymph, in response to changes in head position [1].

Posterior canal BPPV is the most common variant accounting for up to 90% of BPPV cases [3]. The diagnostic manoeuvre of Dix-Hallpike or a side-lying manoeuvre (Semont manoeuvre) elicits a combination of torsional nystagmus towards the lower ear combined with upbeat vertical nystagmus [2]. The less frequent horizontal canal BPPV is diagnosed with a supine roll test. This results in bilateral geotropic (fast phase towards the earth) nystagmus if the otoconia are in the posterior arm of the horizontal canal, and less commonly, bilateral apogeotropic (fast phase away from the earth) nystagmus if the otoconia are in the anterior arm of the horizontal canal, or attached to the cupula (cupulolithiasis). The least common is anterior canal BPPV (1–2%), in which a Dix-Hallpike in either direction or supine straight head hanging manoeuvre, elicits downbeat nystagmus with a small torsional component towards the affected ear [2].

Positional nystagmus in BPPV typically beats in the plane of the affected canal and in the expected direction for canal excitation [4]. Features believed to be characteristic of the nystagmus associated with BPPV (BPPN) are a short latency before onset, duration less than 1 min, and habituation, defined as decreased intensity with repeat testing. Resolution of positional nystagmus after liberatory manoeuvres strongly supports a diagnosis of BPPN [2].

The differential diagnosis of BPPV includes central paroxysmal positional vertigo and nystagmus (CPPV/CPPN) which can be seen in vestibular migraine, as well as in degenerative, inflammatory, or structural lesions of the cerebellum or brainstem [2]. Cerebellar lesions are believed to cause CPPN due to the loss of inhibitory influence on the vestibulo-ocular reflex and the cervico-ocular reflex [5–7]. Contrary to BPPV, the main direction of induced nystagmus in CPPN is typically aligned with rotational axes of the semicircular canal which is inhibited during the positioning manoeuvre [8].

The most common locations for structural lesions causing CPPV are the cerebellar nodulus followed by the lingula, although lesions of the cerebellar hemisphere, peduncle, lateral medulla and cerebello-pontine angle can also cause CPPV [9–15].

Analysis of cases

We have presented cases that were initially thought to represent BPPV, despite apparent incongruities with this diagnosis. This highlights the susceptibility of diagnosticians to availability heuristic, where more weight is placed on diagnoses that come to mind easily [16]. Clinicians are also prone to confirmatory bias and tend to search for and assign more significance to findings that fit with preconceived expectations. Our cases had some features which could be consistent with BPPV; however, there were certain incongruities which were not given high priority initially.

The history and examination features of our cases are summarised in Table 1.

Positional vertigo associated with nausea was present in all patients, and vomiting was a feature in 4/7. Only one patient (case 7) had non-sustained spontaneous nystagmus. Positional testing resulted in apogeotropic horizontal nystagmus in two cases (1 and 7), suggestive of horizontal canal BPPV, and brief downbeat nystagmus in two cases (5 and 6), suggesting anterior canal BPPV. Three patients had positional nystagmus in multiple planes (including apogeotropic horizontal in two cases (3 and 4) which would require multiple canal involvement, if due to BPPV. Table 2 summarises the direction of nystagmus and the suggested canal involvement.

A completely normal neurological examination, including heel to toe walking, was recorded in only one patient. Each case was investigated for a central cause due to certain features contradicting BPPV: headache (sometimes positional or valsalva-related) and vomiting in cases 1, 2, 3 and 7; failure to respond to re-positioning therapy in cases 2, 3, 5 and 6; gait and/or limb ataxia in cases 3, 4 and 7. Only one patient had

Table 1 Clinical features of cases

Case	Atypical features suggestive of central cause	Positioning manoeuvre and direction of resultant nystagmus	Cerebellar pathology
1	Positional headache, and vomiting	SR: apogeotropic	Ependymoma
2	Vomiting and subsequent neck ache	DH-R: torsional then down beat	Lymphoma
3	Brief headache, vomiting, and mild finger nose ataxia	DH-R: down beat, SR: apogeotropic	Metastases
4	Imbalance, hypermetric saccades	LS: down beat & left horizontal, SR: apogeotropic	Metastases
5	Recurrent vertigo and downbeat nystagmus following an apparently effective liberatory manoeuvre	SHH: down beat	Glioneuronal tumour
6	Failed repositioning manoeuvre for downbeat nystagmus	SHH & bilateral DH: down beat	Abscess due to Echinococcus
7	Valsalva-related headache, vomiting, heel-shin ataxia and difficulty with tandem gait	SR: apogeotropic	Infarct

SR supine roll, *DH* Dix-Hallpike, *SHH* straight head hanging, *LS* lying supine from sitting

abnormal eye movements other than with positional testing, with hypermetric saccades in case 4. All patients were found to have lesions in the cerebellum.

Differentiating CPPV from BPPV

The features that may help differentiate CPPV from BPPV are listed in Table 3. However, these are not foolproof and the nuances associated with each are discussed below.

As in our cases, most patients with CPPV have additional neurological symptoms and signs, which may be subtle and therefore missed unless they are specifically looked for. Headache was common in our CPPV cases (4/7), though sometimes a minor or non-persistent feature. Headache may also occur in BPPV [18]. Prominent vomiting was common in our CPPV cases (4/7), though vomiting may uncommonly occur in BPPV.

In the small number of cases where positional vertigo and nystagmus are the sole clinical features of CPPV, the diagnosis relies on careful analysis of the positional nystagmus [17]. The features that may help distinguish CPPN from BPPN include the pattern and time-course of nystagmus, effect of visual suppression (not assessed in our cases), habituation with lessened nystagmus or resolution on repeat testing, and effect of repositioning manoeuvres [8].

Table 2 Direction of nystagmus and proposed SCC involvement

Case	Direction of positioning nystagmus	Proposed semicircular canal involvement
1	Apogeotropic	Horizontal
2	Torsional Downbeat	Posterior ? Anterior
3	Apogeotropic Downbeat	Horizontal Anterior
4	Apogeotropic Downbeat	Horizontal Anterior
5	Downbeat	Anterior
6	Downbeat	Anterior
7	Apogeotropic	Horizontal

Patterns of nystagmus suggestive of CPPV

The most common pattern of CPPN is a downbeat nystagmus provoked by Dix-Hallpike or straight head hanging manoeuvre, which can mimic anterior canal BPPV [1]. Reversal of downbeat to upbeat nystagmus during uprighting does not distinguish anterior canal BPPV from central lesions.

The next most common central pattern is horizontal direction-changing apogeotropic nystagmus provoked by supine roll or head turning. This may appear identical to horizontal canal BPPV [1] as seen in four of our patients, and might be regarded as the most easily misinterpreted form of positional nystagmus. Geotropic horizontal nystagmus is less likely to represent CPPN, but has been reported [11].

The typical pattern of nystagmus seen in posterior canal BPPV is considered very unlikely to be CPPN. In our series, case 2 appeared to have brief torsional nystagmus in response to Dix-Hallpike testing, followed by a downbeat component which decreased in intensity with time. This was thought to represent atypical BPPV. Downbeat nystagmus has been described in posterior canal BPPV, attributed to its apogeotropic variant [19]. Nystagmus occurring in more than one plane such as that seen in cases 3 and 4 is considered a central feature, although this can be seen in canalithiasis of multiple canals [2].

Given the rarity of anterior and horizontal canal BPPV, MRI with gadolinium should be considered for all patients with positional apogeotropic horizontal or downbeat nystagmus, as well as all other atypical BPPV.

Table 3 Differentiating CPPV from BPPV

Feature	BPPV	CPPV
Cerebellar/brainstem symptoms or signs	Absent	Usually Present
Direction and character of positional nystagmus	Upbeat and torsional towards lower ear (posterior canal), horizontal geotropic or apogeotropic (horizontal canal), downbeat (anterior canal)	Usually downbeat or horizontal apogeotropic
Latency of nystagmus after position change	Present with exception of cupulolithiasis of horizontal canal	Usually Absent
Time course	Crescendo-decrescendo	Decrescendo or persistent
Response to repeated position testing	Decreased intensity	Usually no improvement but decreased in < 20% [17]
Effect of visual fixation	Decreased intensity	No change
Response to repositioning treatment	Resolution	No improvement

Time course of nystagmus

BPPN typically starts after a latency of a few seconds and has a duration of less than 1 min. The absence of latency and duration longer than 1 min should therefore raise the suspicion for a central cause. However, these features do not necessarily exclude BPPN, since in horizontal canal cupulolithiasis the nystagmus starts without a latency and persists [2].

The profile of the nystagmus slow phase velocity (SPV) has been shown to be dissimilar between CPPN and BPPN. In BPPN, SPV of the nystagmus gradually increases, reaches a peak and then decays (crescendo-decrescendo pattern). In CPPN, the SPV is at its peak initially and decays exponentially (decrescendo pattern) [8]. The SPV profile of migraine-associated positional nystagmus is typically flat [20]. These features may be difficult assess without the use of video-nystagmography.

Visual suppression

Nystagmus of peripheral origin is typically suppressed by visual fixation [8]. A study comparing visual suppression of caloric nystagmus in patients with SCA-6 (a cause of central positional vertigo) and BPPV found that visual suppression was relatively impaired in SCA-6 patients compared with BPPV patients [21]. Again, this may be difficult to appreciate clinically or with Frenzel glasses, and electronystagmography should be considered.

Habituation and response to re-positioning manoeuvres

Reduction in the intensity of positional nystagmus after repeated positional testing is a feature of BPPN, although it does not absolutely exclude CPPN, and was seen in 2 of our 7 patients. Its absence should raise the suspicion for CPPN. Repositioning therapy is highly effective in BPPN, and lack of response to repeated treatment should raise the suspicion for a central cause. However, this does not exclude BPPN completely, since there are refractory cases. Similarly, an apparently positive response to repositioning therapy may not exclude CPPN. A temporary response was observed in cases 2 and 5 in our series, highlighting the importance of patient re-evaluation. Intermittent positional nystagmus, and spontaneous resolution of symptoms have been reported in early stages of diseases causing CPPN [13].

Conclusion

BPPV is the most common cause of positional vertigo. However, clinicians must be aware that positional vertigo due to vestibular migraine or rarer central lesions may mimic BPPV. In assessing a patient with positional vertigo, a careful clinical assessment without any preconceived notions of availability heuristic or confirmatory bias, is important to be able to identify the rare cases of CPPV. Features that we found most helpful in distinguishing CPPV and BPPV were the presence of additional neurological symptoms (including head ache and vomiting) or signs (limb or gait ataxia), and a failed sustained response to repositioning manoeuvres. Furthermore, apogeotropic horizontal nystagmus on supine roll test and isolated positional downbeat nystagmus should also be considered red flags for CPPV. In view of the multiple potential diagnostic pitfalls, an appropriate policy would be to give the patient written instructions that they should return for re-evaluation if not symptom free.

Compliance with ethical standards

Ethical standards statement National Ethics Advisory Committee's ethical guidelines for health and disability research in New Zealand were followed.

Conflict of interest The authors declare that they have no conflict of interest.

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