Review

Post-Traumatic Benign Paroxysmal Positional Vertigo: Diagnostic Challenges and Contemporary Management.

Shanib Ali, Seema Monga , Nasreen Begum.

Background: Post-traumatic benign paroxysmal positional vertigo (PT-BPPV) is a common but underdiagnosed cause of dizziness after head injury. It is distinct from idiopathic BPPV in its higher rates of bilateral and multicanal canal involvement, lower single-session repositioning success, and greater recurrence risk.

Objectives: To synthesise published evidence on the epidemiology, pathophysiology, diagnosis, and management of PT-BPPV, and to provide a clinician-oriented, decision-focused practice framework.

Methods: Structured narrative clinical review of PubMed/MEDLINE, Embase, Cochrane Library, IndMED, and IMSEAR, with the search updated through February 2026, restricted to English-language publications from January 2000 onwards. Studies were included if they reported original data on PT-BPPV epidemiology, diagnosis, management, or recurrence. Only studies with verifiable bibliographic details were cited with numerical data.

Results: Compared with idiopathic BPPV, PT-BPPV carries significantly higher odds of bilateral disease (OR 4.72), multicanal involvement (OR 4.67), and lower single-manoeuvre resolution (OR 0.56)[1,]. Head trauma is an independent recurrence risk factor[2].Vitamin D deficiency is modifiable and supplementation reduces recurrence in deficient patients[3,4]. Five evidence-based clinical steps are presented to guide diagnosis and management.

Conclusions: Bilateral positional testing, multi-session canalith repositioning, 25-hydroxyvitamin D evaluation, vestibular rehabilitation, and psychological screening form the evidence-based framework for PT-BPPV management. Prospectively registered multicentre trials are needed to strengthen the evidence base.

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Introduction

Post-traumatic benign paroxysmal positional vertigo (PT-BPPV) is the most common identifiable vestibular cause of dizziness after head injury. It is caused by displacement of otoconia into the semicircular canals and shares the clinical hallmark of idiopathic BPPV -- brief, position-triggered vertigo -- but differs in its epidemiology, canal involvement pattern, treatment response, and recurrence risk.

Despite its treatability, PT-BPPV is underrecognized in trauma and emergency settings. This structured narrative clinical review addresses a focused practice question: how should PT-BPPV be diagnosed and managed in trauma patients? The most relevant primary studies, meta-analyses, and clinical practice guidelines are synthesised, and a decision-oriented framework is provided. Where Indian-context data are available from published indexed studies, they are noted in a dedicated practice implications section.

Methods

A structured narrative clinical review was conducted using PubMed/MEDLINE, Embase, Cochrane Library, IndMED, and IMSEAR. The initial search covered publications from January 2000 to December 2024 and was subsequently updated through February 2026 to capture recent publications. Only English-language publications were included. Search terms included: post-traumatic BPPV, traumatic benign paroxysmal positional vertigo, head injury dizziness, canalith repositioning, and mild traumatic brain injury vestibular. The 2017 AAO-HNS clinical practice guideline[5] and Barany Society consensus documents were included regardless of publication date.

Studies were included if they reported original data on PT-BPPV epidemiology, diagnosis, management, or

recurrence, or if they provided evidence-based guidance directly applicable to clinical practice. Studies were excluded if their numerical data could not be traced to a confirmable primary source. This review did not apply formal PRISMA methodology or risk-of-bias assessment; it is a narrative clinical review intended to support clinical decision-making rather than provide a quantitative synthesis.

Epidemiology

PT-BPPV develops in a substantial proportion of patients after head injury. International trauma cohort data estimate prevalence at 15-28% of patients with closed head injury.[6,7]. Road traffic accidents are the dominant mechanism in working-age adults globally; falls are the leading cause in the elderly; and sports-related concussion increasingly accounts for cases in younger populations.

In the elderly, unrecognised PT-BPPV independently worsens fall risk, creating a cycle of vestibular dysfunction and recurrent injury. In athletes, undiagnosed BPPV prolongs return-to-sport timelines. Systematic positional assessment is absent from most trauma and sports medicine assessment protocols worldwide.

Practice implication (India): To our knowledge, one of the few indexed Indian studies reporting PT-BPPV incidence is a prospective cohort from a tertiary neurosurgery centre in Kerala, in which Haripriya et al. reported PT-BPPV in 17% of mild-to-moderate TBI patients[8].Systematic vestibular screening is not yet incorporated into standard post-trauma protocols at most Indian centres.

4. Pathophysiology

Otoconial Displacement

Traumatic shear forces detach calcium carbonate otoconia from the utricular macula. Displaced otoconia migrate into semicircular canal endolymph (canalolithiasis) or adhere to the cupula (cupulolithiasis), causing aberrant cupular deflection and positional vertigo. The greater mechanical forces involved in trauma explain the significantly higher rates of bilateral and multicanal involvement compared with idiopathic BPPV[1].

Vitamin D, Calcium Metabolism, and Otoconial Integrity

Vitamin D regulates calcium homeostasis and the structural integrity of otoconial matrix proteins. Meta-analytic data demonstrate a significant association between 25-hydroxyvitamin D deficiency (serum level <20 ng/mL) and BPPV recurrence, and show that supplementation in deficient patients reduces recurrence rates.[3,4].This is a modifiable risk factor and should be assessed in all patients with recurrent disease.

Coexisting Pathology

More severe injuries -- temporal bone fracture, perilymphatic fistula, intralabyrinthine haemorrhage -- produce distinct additional pathology requiring separate management. Cervicogenic dizziness, provoked by neck movement rather than head position, frequently coexists with PT-BPPV after whiplash and must be recognised as a separate entity.

5. Clinical Characteristics: PT-BPPV versus Idiopathic BPPV

PT-BPPV shares the core phenomenology of idiopathic BPPV but is distinguished by its epidemiology and treatment course. Table 1 summarises the key clinical differences based on the best available comparative evidence[1].

6. Diagnostic Evaluation

Diagnosis requires a structured approach covering clinical history, bedside positional testing, and targeted investigations. The diagnostic framework is summarised in Table 2.

Clinical History

The temporal relationship between head injury and dizziness onset (typically hours to days), the positional character of vertigo (rolling in bed, looking up, bending forward), episode duration under 60 seconds, nausea, and oscillopsia are cardinal features. Patients frequently do not volunteer positional triggers; direct questioning about specific head movements is essential.

Bedside Positional Testing

The Dix-Hallpike manoeuvre is the reference standard bedside test for posterior and anterior canal BPPV[1,5]. Cervical spine clearance must be confirmed before testing in the acute post-traumatic period. Bilateral testing is essential given the higher prevalence of bilateral disease in PT-BPPV[1]. The supine roll test is mandatory to detect horizontal canal BPPV.

Red Flags and Central Mimics

Central positional nystagmus -- caused by posterior fossa tumours, cerebellar degeneration, or demyelination -- lacks the latency and fatigability of peripheral BPPV and mandates neuroimaging before repositioning. Persistent postural-perceptual dizziness (PPPD) -- chronic non-spinning dizziness worsened by movement -- frequently develops after BPPV, particularly in anxious patients, and requires distinct management[9].Red flags mandating MRI are listed in Table 3.

7. Contemporary Management

The management algorithm for PT-BPPV is presented in Table 4. Multiple canalith repositioning procedure (CRP) sessions are the norm in PT-BPPV rather than the exception.

Table 1. Clinical comparison of idiopathic and post-traumatic BPPV.

Feature	Idiopathic BPPV	Post-Traumatic BPPV
Age at onset	Peak 50-70 years	Broader range; younger patients predominate (TBI demographics)
Sex	Female > Male (~2:1)	Male predominance in road trauma cohorts
Canal involved	Posterior (~80-90%)	Posterior canal predominates; bilateral and multicanal involvement significantly more frequent[1]
Bilateral disease	~10% of cases	Significantly higher (OR 4.72; p=0.006 vs idiopathic)[1]
Multicanal disease	Uncommon (~12%)	Significantly higher (OR 4.67; p=0.018 vs idiopathic)[1]
Single-session CRP success	>80%	Lower (OR 0.56 vs idiopathic; p=0.013)[1]; multiple sessions expected
Recurrence	~15-20% at 1 year	Head trauma is a significant independent recurrence risk factor[3]
Vitamin D association	Deficiency linked to recurrence	Same; supplementation reduces recurrence in deficient patients[3,4]
Psychological comorbidity	Anxiety common	Anxiety, depression, and PPPD frequent after traumatic vestibular injury[9]

CRP = canalith repositioning procedure; OR = odds ratio; PPPD = persistent postural-perceptual dizziness. Superscript numbers refer to Vancouver-format references.

Table 2. Diagnostic approach to post-traumatic dizziness.

Clinical Step	Key Actions	Pitfalls / Red Flags
History	Confirm onset relative to injury; characterise positional triggers; record episode duration (<60 s = BPPV); note nausea, oscillopsia, auditory symptoms	Patients may not report positional triggers spontaneously; brief consultations miss the diagnosis
Cervical clearance	Confirm no unstable cervical fracture before positional testing in acute trauma	Omitting clearance in acute trauma is unsafe
Dix-Hallpike test	Bilateral testing essential; 45-degree head rotation, rapid head-hang; observe for upbeat-torsional nystagmus with latency and fatigability	Central nystagmus lacks latency and fatigability and does not suppress with fixation
Supine roll test	Head elevated 30 degrees, brisk 90-degree rotation each side; geotropic = canalolithiasis; apogeotropic = cupulolithiasis	Horizontal canal BPPV missed if roll test omitted
Red flag screen	Seek: pure vertical nystagmus, direction-changing nystagmus, gait ataxia, cranial nerve signs, diplopia, failure to respond to CRP, new vertigo in known malignancy	Any red flag requires MRI brain (posterior fossa sequences) before repositioning
Vitamin D	Measure serum 25-hydroxyvitamin D in recurrent BPPV; target >30 ng/mL	Deficiency is modifiable; missed opportunity in recurrent cases[3,4].
Imaging	MRI if atypical nystagmus, CRP failure, or neurological signs; CT temporal bone if fracture or perilymphatic fistula suspected	Imaging not required for classic presentation

CRP = canalith repositioning procedure; MRI = magnetic resonance imaging.

Table 3. Red flags mandating neuroimaging before canalith repositioning.

No.	Red Flag Feature
1	Nystagmus without latency or fatigability on repeat testing
2	Pure vertical nystagmus (downbeat or upbeat)
3	Direction-changing nystagmus with fixed head position
4	Nystagmus pattern inconsistent with a single semicircular canal
5	Severe gait ataxia disproportionate to vestibular complaint
6	Acute hearing loss with severe vertigo (consider AICA territory ischaemia)
7	Diplopia, dysarthria, dysphagia, or cranial nerve palsy
8	Failure to respond to 4-5 well-performed canalith repositioning procedures
9	New positional vertigo in a patient with known malignancy
10	Headache onset coinciding with onset of positional vertigo

Any of these features mandates MRI brain with posterior fossa sequences before repositioning manoeuvres are performed.

Table 4. Management algorithm for post-traumatic BPPV.

Step	Intervention	Evidence / Notes
1. Canal identification	Dix-Hallpike (posterior/anterior canal) + supine roll test (horizontal canal) at every visit; treat dominant canal first	Bilateral and multicanal disease significantly more prevalent in PT-BPPV[1]
2. CRP - posterior canal	Epley manoeuvre first-line; Semont manoeuvre if cervical extension is limited	Level A evidence (AAO-HNS 2017)[2]; OR 0.56 vs idiopathic for single-session success[1]
3. CRP - horizontal canal	Barbecue roll (Lempert) for canalolithiasis; Gufoni manoeuvre for cupulolithiasis	Multiple manoeuvre sessions expected in PT-BPPV
4. Reassess after each manoeuvre	Repeat positional testing to detect canal conversion before ending the session	Canal conversion well documented; missed if reassessment omitted
5. Number of sessions	Do not label refractory until 4-5 well-performed sessions completed	Lower single-session resolution is the rule in PT-BPPV[1]
6. Post-procedure restrictions	No post-procedure positional restrictions recommended	AAO-HNS 2017 guideline, Level A[2]
7. Vitamin D supplementation	Measure 25-hydroxyvitamin D; supplement if <20 ng/mL; target >30 ng/mL (oral cholecalciferol)	Meta-analysis supports recurrence reduction[4]; Indian pilot RCT corroborates[9]
8. Vestibular rehabilitation	Prescribe for residual imbalance; gaze stabilisation plus balance training	Evidence-based adjunct; task-shift to trained physiotherapists where specialists unavailable
9. Refractory BPPV	Re-characterise all canals; exclude multicanal disease, central pathology, vitamin D deficiency, vestibular hypofunction	Systematic reassessment before specialist referral
10. Psychological screen	PHQ-9 and GAD-7; refer for CBT if PPPD or significant anxiety/depression	PPPD requires distinct management from BPPV[6]

CRP = canalith repositioning procedure; CBT = cognitive-behavioural therapy; PPPD = persistent postural-perceptual dizziness.

7.1 Canalith Repositioning Procedures

The Epley manoeuvre is first-line for posterior canal BPPV[5]. The Semont manoeuvre offers equivalent efficacy and may be preferred when cervical extension is limited. The barbecue roll (Lempert) is preferred for horizontal canal canalolithiasis. Given

the high prevalence of bilateral and multicanal disease[1],all canals must be assessed bilaterally, the dominant canal treated first, and reassessment performed after each manoeuvre to detect canal conversion. Post-procedure positional restrictions are not recommended[5]. A case should not be designated refractory until at least four to five well-performed sessions have been completed.

7.2 Vitamin D Supplementation

Serum 25-hydroxyvitamin D should be measured in all patients with recurrent BPPV. Documented deficiency should be corrected with oral cholecalciferol to a target level above 30 ng/mL. Meta-analytic evidence supports a reduction in recurrence with supplementation in deficient patients[3,4].

7.3 Vestibular Rehabilitation

Supervised vestibular physical therapy -- gaze stabilisation exercises, habituation, and balance training -- is an evidence-based adjunct to CRPs for residual disequilibrium. Home exercise programmes with written and video-based instruction are appropriate where specialist physiotherapy is unavailable.

7.4 Psychological Comorbidity

Anxiety and depressive symptoms are common after traumatic vestibular dysfunction, and PPPD may develop in patients with high anxiety burden[9].Screening using PHQ-9 and GAD-7, and referral for cognitive-behavioural therapy, are appropriate components of comprehensive care.

8. Evidence Summary

The principal studies informing this review are summarised in Table 5.

Table 5. Evidence summary of included studies.

Study	Country	Design	n	Key Finding
Di Cesare T et al., 2021[1] Int J Audiol 60(5):393-7	Italy	Retrospective cohort	795	Bilateral OR 4.72; multicanal OR 4.67; single-manoeuvre success OR 0.56; all p<0.05 vs idiopathic BPPV
Bhattacharyya N et al., 2017[5] Otolaryngol HNS 156(3S):S1-47	USA	CPG (AAO-HNS)	N/A	Level A: Epley manoeuvre; Dix-Hallpike reference standard bedside test; no post-procedure restrictions
Li X et al., 2025[2] Eur J Med Res 30(1)	China	Meta-analysis	Pooled	Head trauma: independent recurrence predictor after CRP (SMD 0.48; 95% CI 0.34-0.63)
Jeong SH et al., 2022[3] J Neurol 269(2):619-26	Korea	Meta-analysis	Pooled	Vitamin D supplementation significantly reduces BPPV recurrence; most pronounced in deficient patients
Rhim G & Kim MJ, 2024[4] Nutrients 16(5):689	Korea	Literature review	Pooled	Recurrence 13-65% by follow-up duration; vitamin D deficiency significant risk factor
Staab JP et al., 2017[9] J Vestib Res 27(4):191-208	Int'l	Consensus criteria	N/A	Diagnostic criteria for PPPD; CBT and serotonergic therapy recommended
Hilton M & Pinder D, 2014[11] Cochrane Database Syst Rev	Int'l	Cochrane SR	Pooled	Epley superior to sham for posterior canal BPPV
Haripriya GR et al., 2018[8] Indian J Otolaryngol HNS 70(3):337-41 PMID 30211085	India	Prospective cohort	128	PT-BPPV reported in 17% of mild-to-moderate TBI patients at tertiary centre in Kerala
Sharma K et al., 2022[10] Indian J Otolaryngol HNS 74(Suppl 3):4405-8	India	Pilot RCT	40	Vitamin D3 plus CRP reduced recurrence vs CRP alone in posterior canal BPPV
Kong TH & Seo YJ, 2026[6] Front Neurol 17:1784282 DOI: 10.3389/fneur.2026.1784282	Korea	Background review	N/A	Structured clinical pathway proposed for PT-BPPV; heterogeneous canal involvement noted; repeated CRPs often required

CBT = cognitive-behavioural therapy; CPG = clinical practice guideline; CRP = canalith repositioning procedure; OR = odds ratio; PPPD = persistent postural-perceptual dizziness; RCT = randomised controlled trial; SMD = standardised mean difference; SR = systematic review. Only studies with confirmable bibliographic details are included.

9. Practice Implications

The following recommendations apply across clinical settings.

- Bilateral Dix-Hallpike and supine roll testing should be performed in all patients presenting with post-traumatic dizziness. The assessment requires no specialist equipment and takes under three minutes.
- Cervical spine clearance must be confirmed before positional testing in the acute post-traumatic period.
- Bilateral and multicanal BPPV are significantly more prevalent in PT-BPPV[1].Assessment of all canals bilaterally is required at each visit.
- Multiple CRP sessions should be anticipated. A case should not be designated refractory until at least four to five well-performed sessions have been completed.
- Serum 25-hydroxyvitamin D should be measured in recurrent BPPV. Documented deficiency should be corrected targeting a level above 30 ng/mL.[3,4].
- Vestibular rehabilitation should be prescribed for residual imbalance.
- Psychological comorbidity (anxiety, depression, PPPD) should be screened and addressed[9].
- Red flags for central pathology (Table 3) should be sought at every assessment; their presence mandates neuroimaging before repositioning.
- **India-specific note:** Task-shifting to trained physiotherapists using standardised protocols is appropriate given limited specialist availability outside metropolitan centres. India's high background prevalence of vitamin D deficiency makes routine 25-hydroxyvitamin D assessment particularly relevant in recurrent PT-BPPV.

10. Limitations

- This is a structured narrative clinical review without a pre-registered protocol, formal risk-of-bias assessment, or duplicate independent reviewer selection. Publication bias cannot be excluded.
- Indian-specific epidemiological data are limited. To our knowledge, the Haripriya et al. cohort is one of the few indexed Indian studies reporting PT-BPPV incidence with a traceable primary source[8].Multicentre Indian studies with pre-specified outcomes are needed.
- Several numerical claims from earlier manuscript versions were removed because they could not be traced to indexed primary sources. Only figures from verifiable publications are reported here.
- Evidence on emerging technologies (smartphone nystagmus analysis, telemedicine-guided repositioning) is descriptive; prospective validation is awaited.

11. Conclusion

PT-BPPV is a common, treatable, and underdiagnosed condition in trauma care settings worldwide. It is clinically and prognostically distinct from idiopathic BPPV: bilateral and multicanal canal involvement are more prevalent, single-session repositioning success is lower, and recurrence risk is higher. Vitamin D deficiency is a modifiable contributor to recurrence.

Five evidence-based take-home points for clinicians: (1) perform routine bilateral positional testing in all post-trauma dizziness; (2) plan for multiple CRP sessions; (3) measure and correct 25-hydroxyvitamin D deficiency in recurrent disease; (4) prescribe vestibular rehabilitation for residual imbalance; and (5) screen for PPPD and psychological comorbidity. Prospectively registered multicentre trials are needed to strengthen the evidence base.

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