Vestibular Migraine:

Recognising and Managing the Most Commonly Missed Vestibular Diagnosis

Vestibular Medicine for General Clinicians

Topic 5 of 14

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Version 2.0 | April 2026

How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 25–30 minute sitting, or used as a desktop reference. It is supported by an A4 cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

Callout Box Guide

□ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, emergencies, and critical safety points requiring immediate action.

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I. Why Vestibular Migraine Matters in General Practice

Vestibular migraine (VM) is the most common cause of recurrent spontaneous vertigo in adults and, in population surveys, has a lifetime prevalence of around 1 percent [1,2]. Despite this, it remains the single most commonly missed vestibular diagnosis in primary care. Tertiary clinic series show patients with VM have typically seen multiple clinicians and carried several incorrect labels — "anxiety", "labyrinthitis", "inner ear problem" — for an average of five to eight years before the diagnosis is made [3,4].

The diagnostic difficulty has a single root cause: headache is not required during an episode. More than 30 percent of patients with VM never have headache during their vertigo attacks, and a further 30 percent have headache only inconsistently [5]. Clinicians trained to equate migraine with headache will therefore miss VM reliably.

□ **Key Point:** If a patient has recurrent episodic spontaneous vertigo lasting minutes to hours, and a personal or family history of migraine, vestibular migraine is the most likely diagnosis — with or without headache during the episode. Recognition is the clinical win; treatment is generally highly effective.

VM also carries a substantial disease burden. Compared with age-matched controls, patients with VM have higher rates of anxiety, depression, sleep disturbance, occupational impairment, and reduced participation in social activities [6]. Once the diagnosis is made and a structured management plan is offered, the majority respond well: greater than 50 percent reduction in attack frequency is typical with adequate prophylaxis [7].

□ **Clinical Insight:** In any patient with recurrent episodic vertigo and no hearing change, assume vestibular migraine until proven otherwise. The absence of headache does not exclude the diagnosis.

II. Epidemiology and Pathophysiology

VM affects approximately 1 percent of the adult population at any given time, with a 1-year prevalence around 0.9 percent and a lifetime prevalence approaching 1.5 percent [2]. It is the second most common cause of episodic vertigo after BPPV and the leading cause of recurrent spontaneous (non-positional) vertigo. Female-to-male ratio is approximately 3:1, peak prevalence is in the fourth and fifth decades, and many women experience worsening attacks perimenopausally [8].

The pathophysiology is incompletely understood but follows the broader migraine model. Functional imaging studies show altered thalamic, brainstem vestibular nucleus, and cortical processing during and between attacks [9,10]. The condition is best conceptualised as a sensitised central nervous system that periodically generates dizziness in the same way it might generate headache, photophobia, or aura — a single neurophysiological trait expressed through different sensory channels.

Genetic and Hormonal Influences

Family history of migraine is present in approximately two-thirds of patients with VM. Female sex is a strong risk factor; attacks are frequently triggered by hormonal changes — menstruation, oral contraceptives, pregnancy, and perimenopause. The recognised migraine triggers — sleep disturbance, stress, dehydration, dietary triggers, weather change — apply equally to VM.

□ **Clinical Pearl:** Explaining to patients that vestibular migraine is a brain condition, not an ear condition, is often therapeutic in itself. Many patients have been told repeatedly that their "ears are fine" and have lost faith in the system. Naming the condition correctly restores legitimacy and engagement.

III. Diagnostic Criteria — Bárány Society and ICHD-3

VM is defined by clinical criteria endorsed jointly by the Bárány Society and the International Headache Society (ICHD-3, 2018; updated 2022) [5,12,13]. The criteria are designed to be applied at the bedside without imaging or specialised vestibular testing. Figure 1 summarises the diagnostic decision pathway.

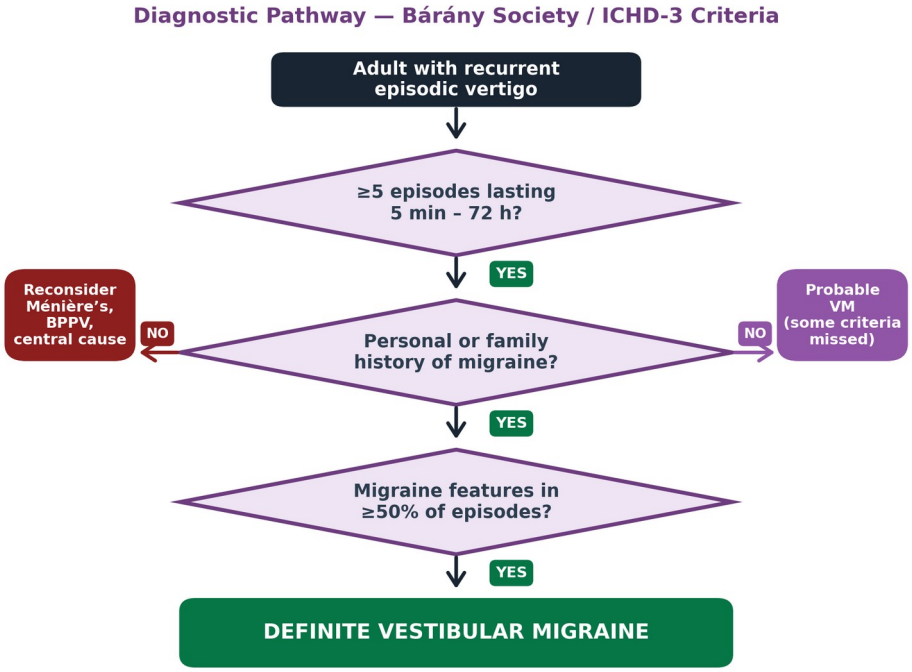

Figure 1. Diagnostic pathway for vestibular migraine — sequential application of the Bárány Society / ICHD-3 criteria

Source: Australian Dizziness Clinics — clinical algorithm.

Bárány / ICHD-3 Criteria — Summary

The criteria for definite vestibular migraine, summarised in Table 1, require all four conditions to be met. "Probable VM" is reserved for clinical pictures that meet some but not all criteria — in practice probable VM is treated identically to definite VM.

#	Bárány / ICHD-3 criterion	How it is satisfied at the bedside
A	≥5 episodes of vestibular symptoms	Moderate–severe intensity, lasting 5 minutes to 72 hours.
B	Current or previous migraine	Personal history (with or without aura) per ICHD-3. Family history is supportive but not sufficient alone.
C	Migraine feature in ≥50% of episodes	Headache (≥2 of unilateral, pulsating, moderate–severe, aggravated by activity); OR photophobia + phonophobia; OR visual aura.
D	Not better accounted for by another diagnosis	Audiometry, examination, and targeted imaging exclude Ménière's, BPPV, central pathology.

□ **Important:** Do not require headache during the attack to make the diagnosis. Over a third of patients with definite vestibular migraine never have headache during vertigo episodes. The migraine-feature criterion can be satisfied by photophobia and phonophobia alone.

IV. Clinical Features — What the Episodes Look Like

The clinical phenotype is heterogeneous, but several features recur and should prompt the diagnosis.

Episode Duration

Wide range — from 5 minutes to 72 hours — but most fall within 30 minutes to 24 hours. Brief seconds-long episodes argue against VM (consider BPPV); continuous symptoms over weeks argue against discrete VM (consider PPPD).

Type of Vertigo

Spontaneous internal vertigo, positional vertigo, head-motion-induced unsteadiness, or visually-induced vertigo. The positional vertigo of VM is not gravity-stereotyped, lasts minutes rather than seconds, and the nystagmus pattern (when present) is variable.

Associated Symptoms

- Photophobia and phonophobia — common, often the most useful migraine feature in patients without headache.
- Visual aura — scintillations, fortifications, blurred vision — present in a minority but highly specific.
- Nausea and vomiting — frequent.
- Cognitive fog and fatigue — often persists for hours or days after vertigo resolves (the "VM hangover").
- Visual vertigo — supermarket aisles, scrolling screens, busy patterns — common between attacks [14].

Triggers

Sleep deprivation, stress (or release of stress on weekends), hormonal change (menstruation, perimenopause), dietary triggers (red wine, aged cheese, MSG, chocolate, caffeine excess or withdrawal), dehydration, skipped meals, weather change, and visual stimulation. Multiple triggers operating in combination is the rule rather than the exception.

□ **Key Point:** Multiple migraine triggers, episodes lasting 5 minutes to 72 hours, photophobia and phonophobia during episodes, normal hearing, and normal examination between episodes — this pattern is vestibular migraine even if the patient has never had a headache in their life.

V. Examination Findings and Differential Diagnosis

Between attacks, examination is typically normal. During an attack, findings are non-specific — central or peripheral patterns of nystagmus may both be seen, head impulse testing is variable, and the test of skew is usually negative. Bedside examination is therefore principally a tool for excluding alternatives.

Bedside Examination Priorities

- Smooth pursuit, saccades, gaze-evoked nystagmus — exclude central pathology (vestibular schwannoma, brainstem-cerebellar lesion).
- Spontaneous nystagmus, head-shake nystagmus, head impulse — look for unilateral peripheral vestibulopathy.
- Dix-Hallpike and supine roll test — exclude BPPV in patients reporting positional triggers.
- Otoscopy, tuning fork tests — screen for hearing asymmetry suggesting Ménière's or schwannoma.
- Stance and gait — Romberg, tandem gait — to exclude cerebellar ataxia.

Figure 2 summarises the differential triage by dominant clinical feature.

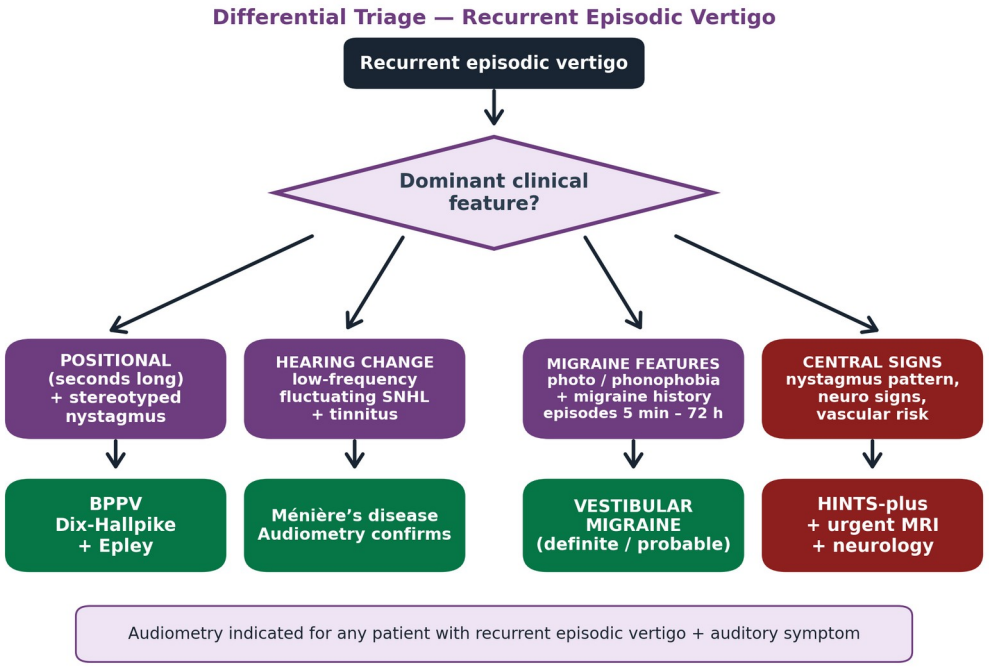

Figure 2. Differential triage for recurrent episodic vertigo — feature-led pathway through the four common causes
Source: Australian Dizziness Clinics — clinical algorithm.

Key Differentials

Table 2 summarises the differential diagnoses with the single feature that resolves each one in primary care.

Differential	Distinguishing feature	Decisive test
Ménière's disease	Episodic vertigo with documented fluctuating low-frequency SNHL, aural fullness, tinnitus.	Audiometry
BPPV	Seconds-long positional vertigo with stereotyped nystagmus.	Dix-Hallpike / supine roll
Vestibular paroxysmia	Very brief (seconds) episodes; sometimes triggered by head position.	Therapeutic trial of carbamazepine / oxcarbazepine
Posterior circulation TIA	Sudden onset with neurological symptoms or vascular risk.	HINTS-plus, urgent MRI
PPPD	Persistent (≥3 mo) non-vertiginous unsteadiness ↑ upright posture and complex visual environments.	Clinical pattern + DSM/Bárány criteria
Anxiety / panic disorder	Coexists frequently with VM; not "instead of" VM.	Treat both — anxiety scales (GAD-7)
□ **Clinical Insight:** Ménière's and vestibular migraine are frequently confused because both cause episodic vertigo lasting hours. The decisive distinguisher is audiometry. Fluctuating low-frequency sensorineural hearing loss over months points to Ménière's; normal audiometry with migraine features points to VM.		

VI. Investigations — When and Why

VM is a clinical diagnosis. Investigations are used selectively, principally to exclude alternative diagnoses rather than to confirm VM.

Audiometry

Pure-tone audiometry should be obtained in any patient with recurrent episodic vertigo and any auditory symptom — tinnitus, hearing change, fullness — to screen for Ménière's, vestibular schwannoma, or sudden sensorineural hearing loss. In typical VM, audiometry is normal or shows symmetric age-appropriate thresholds.

MRI

Indicated where central pathology is plausible: progressive neurological signs, downbeat or persistent nystagmus, asymmetric hearing loss, late-onset (>60 years) first presentation, vascular risk profile with first attack, or persistent unilateral symptoms. MRI in straightforward recurrent VM is not required and not cost-effective [15].

Vestibular Function Testing

VNG, vHIT, VEMP, and rotational chair testing are not required for diagnosis but can be informative where the clinical picture is mixed. Findings in VM are non-specific — variable canal paresis, abnormal pursuit, or visual-vertical asymmetry have all been reported.

Bloods

Routine bloods are not required. Targeted tests (TFTs, B12, vitamin D, electrolytes, ECG) are useful when an alternative diagnosis is considered or when comorbid contributors to dizziness are suspected.

□ **Clinical Pearl:** Over-investigation delays treatment. In a typical patient with recurrent episodic vertigo, migraine history, and normal interictal examination, commence a trigger diary and prophylactic treatment rather than ordering a panel of tests. Investigations should be targeted to exclude specific alternatives, not to confirm VM.

VII. Management — Trigger Modification and Lifestyle

Management has two phases: identification and reduction of triggers (lifestyle), and pharmacological prophylaxis where lifestyle measures are insufficient. Patient education and clear framing of expectations is the foundation of both.

Trigger Diary

Ask the patient to keep a structured 6-week diary recording: attacks (date, duration, severity), sleep (hours, quality), menstrual cycle, meals (especially missed or delayed), caffeine and alcohol intake, stress, and weather. The diary objectifies subjective patterns and identifies the highest-yield modifiable triggers for that individual. Most patients identify two to four dominant triggers.

Universal Lifestyle Measures

Table 3 summarises the lifestyle measures that should be advised for every patient with VM, regardless of severity, before pharmacological prophylaxis is considered.

Lifestyle measure	Target	Comment
Sleep	Fixed bedtime and wake time, including weekends.	Both sleep deprivation AND over-sleep are common triggers.
Meals	3 regular meals daily; do not skip breakfast.	Meal-skipping is one of the most-cited triggers.
Hydration	1.5–2 L water daily.	Replace caffeine- and exercise-related losses.
Caffeine	≤200 mg/day (≈ one strong coffee).	Do not stop abruptly — taper over weeks. Withdrawal is a potent trigger.
Alcohol	Limit; particularly red wine, fortified wines, beer.	Often the single highest-yield exclusion.
Aerobic exercise	30 min moderate intensity, 3–5×/week.	Both prophylactic and stress-reducing.
Stress management	CBT, mindfulness, or counselling where indicated.	Anxiety treatment specifically reduces attack frequency.

Targeted Dietary Trial

Common dietary triggers include red wine, aged cheese, chocolate, citrus, MSG, nitrates (cured meats), aspartame, and high-tyramine foods. A structured 6-week trial of avoidance, followed by sequential reintroduction, is more useful than blanket exclusion. Specialist dietetic input is rarely necessary.

□ **Clinical Insight:** Many patients describe "I've tried everything" dietary exclusions. Ask specifically what they tried, for how long, and whether they kept a diary. Most have done unstructured short-term exclusions without a diary — the equivalent of an inadequate trial. A properly structured 6-week diary plus targeted elimination is usually more informative.

VIII. Pharmacological Prophylaxis

When attacks are frequent (≥2 per month), prolonged, or significantly disabling, prophylactic medication is indicated. Evidence is limited but consistent — agents proven in classical migraine are also effective in VM at similar doses [17,19]. Table 4 summarises the agents.

Agent	Dose range	Choose when	Avoid in
Propranolol	40–160 mg/day	Anxiety; hypertension	Asthma; bradycardia
Amitriptyline	10–50 mg nocte	Insomnia; chronic pain	Cardiac conduction disease
Topiramate	25–100 mg/day	Weight loss desirable	Cognitive side-effects; renal stones
Venlafaxine	37.5–150 mg/day	Anxiety; depression; perimenopause	Uncontrolled hypertension
Flunarizine	5–10 mg/day	European 1st-line	Not PBS-listed in Australia
Sodium valproate	500–1500 mg/day	Refractory; older male patient	Women of reproductive age (teratogenic)
Pizotifen	0.5–3 mg/day	Weight gain acceptable	Obesity
Candesartan	8–16 mg/day	Comorbid hypertension	Pregnancy
CGRP mAbs (erenumab, fremanezumab, galcanezumab)	Monthly or quarterly	Refractory cases	Specialist initiation only — PBS criteria apply

Choose the agent whose side-effect profile best matches the patient's comorbidities. Trial each agent at adequate dose for at least 6–8 weeks before declaring failure.

Acute Attack Treatment

For acute attacks, triptans have moderate evidence in VM, particularly rizatriptan and zolmitriptan [21]. Antiemetics (metoclopramide, ondansetron) and simple analgesia are useful adjuncts. Benzodiazepines can be used sparingly for severe vertigo but should not be regular.

Vestibular Rehabilitation

Customised vestibular rehabilitation is effective in VM, particularly in patients with head-motion intolerance, visual vertigo, and chronic interictal dizziness [22]. Rehabilitation works best when combined with prophylactic pharmacotherapy.

□ **Important:** Vestibular suppressants (prochlorperazine, promethazine, betahistine) are not prophylactic agents for vestibular migraine. They may be used briefly for severe acute attacks but prolonged daily use delays recovery, impairs central compensation, and does not address the underlying migraine biology.

IX. Clinical Decision Pathway

Figure 3 summarises the stepwise primary-care management of suspected vestibular migraine — from initial education and trigger diary through prophylaxis to specialist referral.

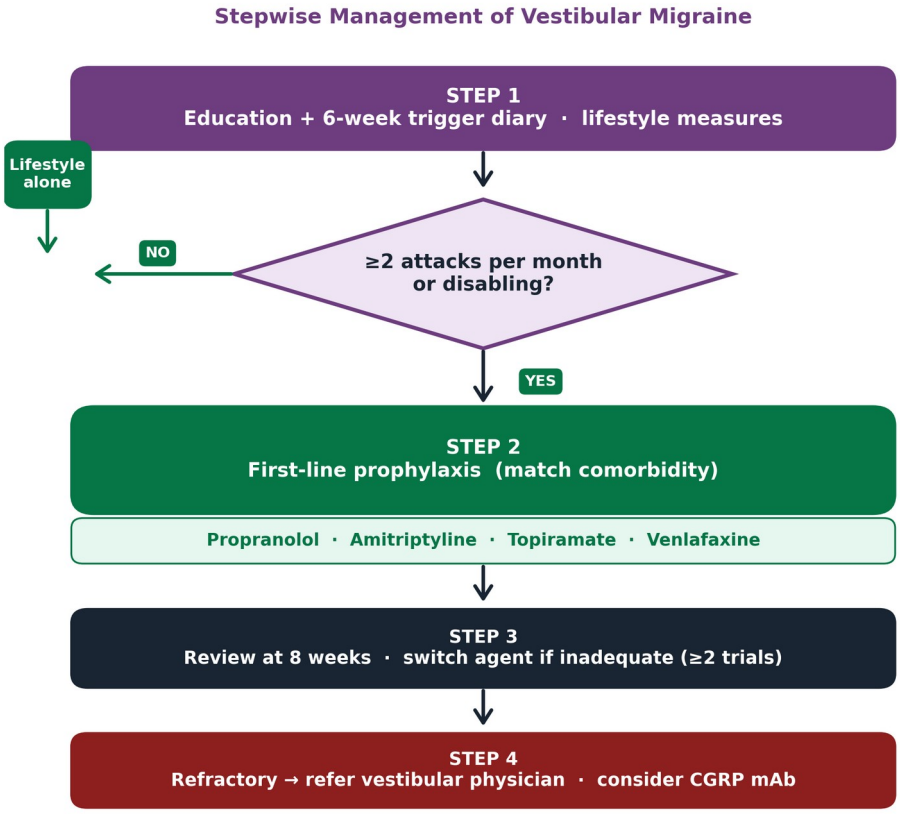

Figure 3. Stepwise management algorithm for vestibular migraine — four-step pathway from lifestyle to specialist referral

Source: Australian Dizziness Clinics — clinical algorithm.

□ **Key Point:** Most patients diagnosed in primary care achieve substantial improvement with lifestyle modification plus a single well-chosen prophylactic. Escalation beyond first-line prophylaxis warrants referral to a vestibular physician or dedicated service.

X. Referral and Long-Term Outlook

Most patients with straightforward VM can be managed entirely in primary care. Figure 4 summarises the indications for referral to a vestibular physician or dedicated service.

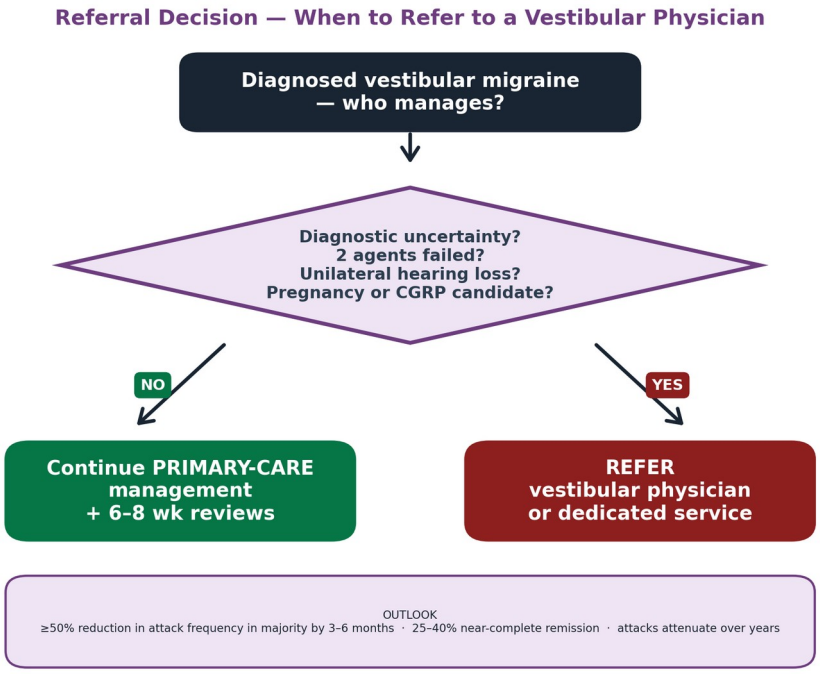

Figure 4. Referral decision pathway — criteria for primary-care management vs vestibular physician referral
Source: Australian Dizziness Clinics — clinical algorithm.

Indications for Referral

- Diagnostic uncertainty persists — particularly where Ménière's, PPPD, or a central cause cannot be confidently excluded.
- Attacks are refractory to adequate trials of two first-line prophylactic agents.
- Unilateral hearing loss, progressive neurological signs, or any red-flag feature.
- Persistent interictal dizziness or visually-induced dizziness is disabling — customised vestibular rehabilitation indicated.
- CGRP monoclonal antibody therapy is being considered (PBS prescribing criteria apply).
- Pregnancy or planning pregnancy — prophylactic drug choices are constrained.

□ **Clinical Insight:** A patient with vestibular migraine plus coexisting PPPD or coexisting BPPV is common, not unusual. Each condition requires its own targeted treatment. Dedicated vestibular assessment is particularly valuable where multiple diagnoses coexist.

Prognosis

With appropriate management, the majority of patients experience a reduction in attack frequency of greater than 50 percent within three to six months, and 25–40 percent achieve near-complete remission [7]. Long-term follow-up studies show that attacks often attenuate over years, particularly in women after menopause [11]. A proportion of patients develop coexistent PPPD, which requires specific recognition and treatment.

□ **Clinical Pearl:** Set expectations explicitly: the goal of prophylaxis is reduction of attack frequency and severity, not elimination. A successful response is usually visible within 6–8 weeks. Persist with a well-tolerated agent at adequate dose before switching. Frame the condition as a long-term neurological trait that can be well controlled rather than a disease that will be cured.

□ **Important:** Do not tell patients with vestibular migraine that they have "just anxiety", "an ear problem that will pass", or "nothing wrong". These messages drive disengagement and delay effective treatment by years. Name the condition, explain the mechanism, and commit to a structured management plan.

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