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Vertigo and migraine

‘How can it be migraine if I don’t have a headache?’

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Vestibular migraine is a common, treatable cause of vertigo that should be considered whenever vertigo cannot be clearly explained by an alternative condition. In clinical practice, the patient’s response to treatment with a migraine preventive agent can determine the diagnosis.

Key points

- **Vestibular migraine is one of the most common causes of vertigo but the diagnosis is still controversial in some circles.**
- **Vestibular migraine is a protean disorder and should be considered whenever the clinical features of vertigo are not absolutely typical of an alternative diagnosis.**
- **Although there can be strong clinical clues pointing to it, vestibular migraine is a diagnosis of exclusion.**
- **If the patient presents with unilateral or asymmetrical hearing loss or tinnitus then a diagnosis of vestibular migraine should be questioned.**
- **Treatment with a migraine preventive agent is usually effective and can determine the diagnosis.**

Headache and dizziness are two of mankind’s great afflictions, so common that they can almost be considered part of normal human experience. How often does a patient who is asked about headache reply: ‘I just get normal headaches’? Likewise, dizziness (even vertigo) can be viewed as ‘normal’ dizziness when experienced on quickly standing, with exhaustion or other illness, after taking drugs or alcohol or at heights.

Migraine occurs in about 12% of the population[1] and, according to a recent population survey, vertigo occurs in about 8% of people each year, often of sufficient severity to provoke medical assessment.[2] Thus, it is not unexpected that migraine and vertigo should coexist in the one patient, but many studies have shown that the association is considerably more than would be expected by chance.[3] It is proposed that this discrepancy is due to vertigo occurring as a result of migrainous pathophysiology, a condition most commonly referred to as vestibular migraine.

Vestibular migraine is thought to be among the most common causes of vertigo seen in patients in daily clinical practice.[4,5] With experience, clinicians can recognise vestibular migraine as a relatively distinctive and recognisable syndrome, and not only a coexistence of two common symptoms. Diagnostic criteria for vestibular migraine have been proposed (see the box on page 37), and shown to

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have good long-term specificity.[6] However, vestibular migraine is still not a diagnosis accepted by all experts and is very much a clinical syndrome in evolution. For example, the current International Classification of Headache Disorders[7] does not include vestibular migraine, although it does include two rare disorders in which migraine is the accepted cause of vertigo – namely basilar migraine and benign paroxysmal positioning vertigo (BPPV) of childhood. It is the view of the author that these two rare disorders represent the very small tip of a very large vestibular migraine iceberg.

PATHOPHYSIOLOGY OF VESTIBULAR MIGRAINE

Not long ago migraine was seen as a neurovascular disorder. Now it is generally considered to be a brain disorder with a strong genetic component, although even in the case of migraine headache the precise pathophysiology is uncertain.[8]

The typical migraine aura is considered to occur because of a cortical process known as 'spreading depression', distinct from ischaemia. The aura usually precedes the headache, raising the possibility that the primary abnormality in migraine is cortical.

A link between vestibular migraine and migraine aura is a tempting hypothesis, but is challenged by the dissimilarity in temporal pattern between the two. Unlike typical migraine aura, vestibular migraine shows a highly inconstant relationship to headache and a highly variable duration, with episodes lasting seconds to days, or possibly even far longer.[9]

The vestibular system spans the inner ear, brainstem and cortex (parieto–temporal junction) and dysfunction at any of these levels could cause vestibular migraine. A study of patients during severe attacks of vestibular migraine found patterns of nystagmus that suggested a central disorder in most individuals, but also indicated a peripheral disorder in a significant minority of others.[10] Functional imaging studies such as PET in migraineurs have shown abnormalities in brainstem structures such as locus coeruleus.[11] The brainstem is the region where structures relevant to head pain and vertigo are closest.

The possibility that migraine and vestibular migraine are caused by a diffuse dysfunction of neuronal excitability is favoured by the observation that disorders of ion channel function can manifest as migraine or vertigo.[12] Indeed, mutation of the same calcium channel gene can cause either familial hemiplegic migraine (also with more usual migraine) or episodic ataxia (also with vertigo and often migraine headaches). Other observations supporting widespread neuronal dysfunction as a cause of migraine and vestibular migraine include the broad clinical spectrum of migraine (e.g. hypersensitivity to sensory stimuli) and the wide range of medications found to be effective in prophylaxis.

FEATURES OF VESTIBULAR MIGRAINE

Definite vestibular migraine

- Two or more attacks of vestibular vertigo (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance – i.e. sensation of imbalance or illusory self or object motion that is provoked by head motion).
- Migraine, as defined by the ICHD.[7]
- Concomitant migrainous symptoms during two or more vertigo attacks.
- No evidence of other central or otological causes of vertigo.

Possible vestibular migraine

- Two or more attacks of vestibular vertigo.
- At least one of the following:
  - migraine, as defined by the ICHD[7]
  - at least one migrainous symptom during two or more vertigo attacks
  - migrainous headaches
  - photophobia
  - phonophobia
  - visual or other auras.
- No evidence of other central or otological causes of vertigo.

ABBREVIATION: ICHD = International Classification of Headache Disorders.

CLINICAL FEATURES OF VESTIBULAR MIGRAINE

Diagnosis of vestibular migraine is based on three main factors. These are:

- adequate exclusion of other disorders
- clinical pattern
- response to migraine prophylactic therapy.

TABLE. EXAMPLES OF PROPHYLACTIC THERAPEUTIC OPTIONS FOR VESTIBULAR MIGRAINE

Medication	Usual dosage	Common side effects	Serious side effects	Comments
Pizotifen	0.5 mg to 2.5 mg nightly	Sedation, weight gain	Very safe	–
Propranolol	10 mg daily to 40 mg twice daily	Low blood pressure, fatigue, constipation	Exacerbation of asthma, bradyarrhythmia, depression	The most studied β-blocker
Metoprolol	25 mg daily to 50 mg twice daily	Low blood pressure, fatigue, constipation	Exacerbation of asthma, bradyarrhythmia, depression (but to a lesser extent than propranolol)	Better tolerated β-blocker (e.g. compared with propranolol)
Amitriptyline*	10 mg to 100 mg nightly	Sedation, dry mouth, blurred vision	Tachyarrhythmia, urinary retention	The most studied tricyclic antidepressant
Nortriptyline*	10 mg to 75 mg nightly	As for amitriptyline but to a lesser extent	As for amitriptyline but to a lesser extent	Better tolerated tricyclic antidepressant (e.g. compared with amitriptyline)
Dothiepin*	25 mg to 150 mg nightly	As for amitriptyline but to a lesser extent	As for amitriptyline but to a lesser extent	Better tolerated tricyclic antidepressant (e.g. compared with amitriptyline)
Sodium valproate*	100 mg nightly to 500 mg twice daily	Weight gain, sedation, dizziness, gastrointestinal upset, tremor	Hepatic damage (very rare)	Teratogenic
Topiramate	25 mg to 100 mg nightly	Paraesthesia, weight loss, speech problems and difficulty with memory	Kidney stones, acute glaucoma, depression or personality change	PBS authority required (streamlined)
Betahistine	8 mg daily to 32 mg twice daily	Gastrointestinal upset, headache	Very safe	Private script

* Off label use. However, these agents and the doses suggested are commonly used in clinical practice for the prophylactic treatment of migraine.

A diagnosis of exclusion

There is no finding on clinical examination or investigation that is diagnostic of vestibular migraine. Indeed, if unequivocal abnormalities are present then the diagnosis of vestibular migraine must be seriously questioned. Thus, the diagnosis of vestibular migraine is based on the combination of recognising a consistent clinical syndrome and adequate exclusion of other disorders. This is a diagnostic process common to many disorders in clinical medicine, including migraine headache itself.

Clinical pattern

Vestibular migraine is common and its clinical pattern of presentation is protean. The presence of vestibular migraine should be at least considered in the differential diagnosis for most cases of vertigo. The clinical features of vestibular migraine overlap with all other major vestibular syndromes (peripheral and central),[13] and both Menière's disease[14] and BPPV[15] are more common in migraine.

Once a diagnosis of vestibular migraine is raised, it should be reviewed and reconsidered at every opportunity, especially if response to therapy is suboptimal or clinical features change.

Response to migraine prophylactic therapy

If the diagnosis of vestibular migraine seems possible at the end of a patient's clinical assessment then it is very reasonable to trial a migraine preventive agent (Table). Response to therapy may not absolutely prove vestibular migraine but often secures a comfortable clinical diagnosis.

A CONFIDENT APPROACH TO ASSESSING VERTIGO

Because vestibular migraine is a great mimic, it is difficult to diagnose without a confident general approach to vertigo. Clinical assessment of dizziness and vertigo can often be daunting and unsatisfactory, partly because an organised approach is lacking. Focusing on the following crucial questions when taking a history can lead to a more confident diagnosis of vertigo in general and vestibular migraine in particular.

- Is it vertigo?
- What are the associated symptoms?
- What is the temporal profile of the vertigo and does it fit into any of the typical temporal syndromes?
- Is there any relevant past medical or family history of migraine?

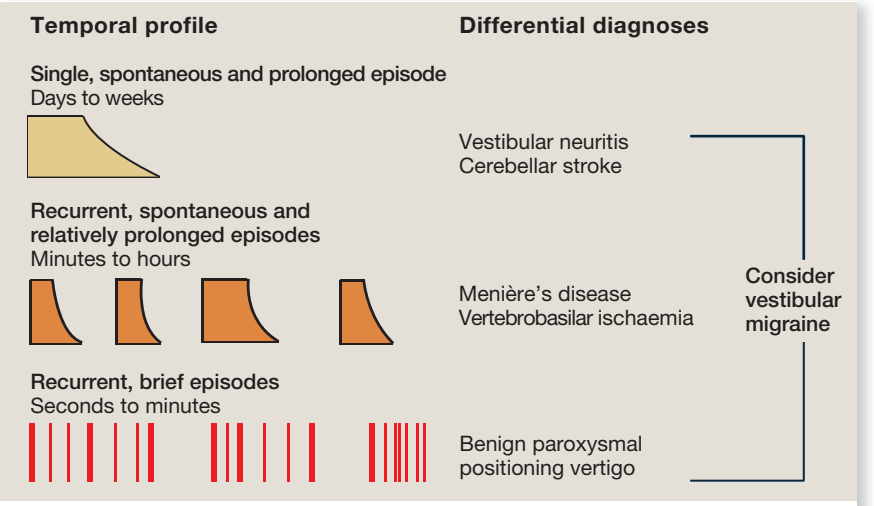

Figure. The left side of the figure indicates the three main temporal vertigo syndromes. The centre indicates the differential diagnosis for each. Vestibular migraine should be considered for each of the three syndromes. It most commonly presents as recurrent spontaneous vertigo of any duration and next most commonly as brief recurrent positional vertigo.

Is it vertigo?

Vertigo is often defined as an illusion of spinning of the environment. However, in practice it is quite common for vestibular disorders to present with dizziness that is not 'typical rotatory vertigo'. An emerging and less restrictive definition of vertigo encompasses not just spinning but any illusion of motion.[16] Even so, some patients with definite vestibular disorders (e.g. BPPV or Menière's disease) cannot easily describe their experience in words more specific than being simply 'dizzy'. However, the recognition of patients with vertigo can be aided by taking into account triggering factors, temporal pattern and associated symptoms.

It is worth remembering that vertigo is a symptom not a diagnosis, and placing too much emphasis on defining and recognising vertigo risks 'not seeing the forest for the trees'. In this article, vertigo is used in the broadest possible sense. It is also important to note that it is particularly common for vestibular migraine to present with atypical vertigo.

Associated symptoms with vertigo

Symptoms that support the presence of vertigo or vestibular dizziness include nausea, vomiting, hearing loss, tinnitus, aural fullness and unsteadiness. Nausea, vomiting and unsteadiness aid in the recognition of vertigo but are not specific to a particular diagnosis, although if these symptoms appear to be out of proportion to that of dizziness then a central cause of dizziness is favoured. Unilateral hearing loss, tinnitus and aural fullness also aid in the recognition of vertigo, but occur much more commonly with specific conditions, such as Menière's disease.

Associated symptoms with vestibular migraine

In vestibular migraine, auditory symptoms such as bilateral tinnitus or aural fullness are usually minor and nonspecific. The presence of marked unilateral auditory symptoms, however, argues strongly against vestibular migraine as the sole diagnosis.

Vestibular migraine and (lack of) headache

Headache is a cardinal feature of migraine, but it is important to remember that it is not always present in patients with vestibular migraine. Although vertigo occurring with headache is part of the diagnostic criteria for definite vestibular migraine, many patients with the condition never experience headache with their attacks, and some patients with otherwise typical vestibular migraine have never had clear migraine headache.[17] Furthermore, headache is not an uncommon accompaniment of vertigo that manifests as a result of other causes, such as Menière's disease[13] and, in the setting of acute severe vertigo, the presence of headache should raise the possibility of cerebellar stroke (infarction or haemorrhage).

Temporal pattern

If dizziness is triggered or aggravated by head movement (and not just on first standing) it is more likely to be vertigo as opposed to nonvestibular dizziness. The main clinical significance of investigating a temporal pattern is to further classify the patient's vertigo – hopefully leading to a specific diagnosis, such as BPPV, Menière's disease or, indeed, vestibular migraine.

The main temporal parameters include whether the patient's vertigo is:

- single or recurrent
- prolonged or brief
- spontaneous or triggered

These three parameters combine to give three common syndromes of vertigo, classified by:

- single, spontaneous and prolonged episodes, lasting a duration of hours to days
- recurrent, spontaneous and relatively prolonged episodes, lasting a duration of minutes to hours
- recurrent, brief episodes that are triggered by head movement (positional or positioning), lasting a duration of seconds to minutes (Figure).

Single, spontaneous and prolonged episodes

Single, spontaneous and prolonged episodes of vertigo are often referred to as 'acute vestibular syndrome'. The main differential diagnosis is vestibular neuritis (labyrinthitis and neurolabyrinthitis are synonyms) and cerebellar stroke.

Vestibular migraine is almost always recurrent, but because there must always be a first episode, a diagnosis of vestibular migraine should also be considered when there has been a single prolonged episode – once the other possibilities have been adequately excluded. It is always necessary to consider stroke in this clinical setting, especially when there is headache, even if there are no other neurological symptoms and signs.

Recurrent, spontaneous and relatively prolonged episodes

Menière's disease and vestibular migraine are the main diagnostic possibilities associated with recurrent, spontaneous and relatively prolonged episodes of vertigo. Unilateral auditory symptoms and/or aural fullness are usually present in Menière's disease but rarely in vestibular migraine.[13] More serious disorders such as vertebrobasilar ischaemia (VBI) and acoustic neuroma can rarely present this way, but there are usually other strong clues to suggest their diagnosis. Other posterior circulation symptoms such as diplopia, dysarthria and paraesthesia can be seen in basilar migraine, but mandate adequate exclusion of VBI. Acoustic neuroma is usually associated with unilateral tinnitus and hearing loss with asymmetrical high-frequency loss on audiometry.

Recurrent, brief episodes triggered by head movement

BPPV is markedly the most common disorder causing the pattern of recurrent, brief episodes of vertigo that are triggered by head movement, but it is not uncommon for vestibular migraine to present in this way also. Usually, BPPV is a straightforward diagnosis with the patient giving a clear history of brief rotatory vertigo triggered by specific head movement. Vertical head movements are most affected in BPPV and most characteristic is vertigo triggered by rolling to the affected side in bed. In vestibular migraine, the presentation can be similar but is rarely so clear-cut. Often the triggering head movements are less specific and the patient is sensitive to cumulative head movement or intolerant of head motion without experiencing distinct vertigo (head motion intolerance) and motion sickness. On examination the Dix-Hallpike test is usually positive in BPPV, while in vestibular migraine the patient might well feel dizzy during this test but often this will be to both sides of the head and without the typical nystagmus of BPPV.

Past history of migraine

Seeking evidence of migraine in the patient's past medical or family history is of crucial importance for reaching a diagnosis. A positive history does not prove that the patient's current vertigo is migrainous, but it provides an important clue. Always ask the patient about intermittent visual symptoms because some patients with vestibular migraine may have visual aura without headache. Probe patients for details of their headache as far as possible because migraine headache in vestibular migraine can often be relatively mild and might be discounted by patients as a 'normal' headache.

WHEN TO SUSPECT VESTIBULAR MIGRAINE

Specific scenarios that raise suspicion of vestibular migraine include:

- a mix of spontaneous and positional vertigo (i.e. vertigo that does not fit neatly into the above temporal syndromes) – Menière's disease usually only causes spontaneous vertigo and BPPV only positional vertigo
- a very long history of vertigo without any definite diagnosis being reached – causes of vertigo other than vestibular migraine are uncommon in childhood and usually declare themselves in years, if not months
- a past diagnosis of Menière's disease but with no clear presence of unilateral auditory features – in practice, Menière's disease can often be overdiagnosed and vestibular migraine underdiagnosed
- a past diagnosis of prolonged or recurrent BPPV but no definitively positive Dix-Hallpike test and no clear response to particle repositioning – BPPV is usually an easy diagnosis to make with the Dix-Hallpike test and patients usually respond very well to standard treatment
- recurrent vertigo that is not typical rotatory vertigo.

MIGRAINE AND ANXIETY

Chronic vertigo (particularly if unpredictable) is often complicated by anxiety. To confuse matters, anxiety may also cause dizziness and vertigo. Careful and sympathetic history-taking is often required to explore whether anxiety is the cause or the effect of a patient's vertigo, and even then it is not always possible to be sure. It is sometimes practical to

think of vestibular migraine and vertigo that are due to anxiety as 'migraine–anxiety related vertigo'[18] because both can be inextricably linked and have overlapping treatment strategies.

INVESTIGATIONS

Vestibular migraine is a protean and controversial disorder that is a diagnosis of exclusion. As such, some investigation is usually required. Where practical, it is generally prudent to perform a basic inner ear assessment (audiometry and vestibular function tests where available) and some form of brain/ear imaging (usually MRI, but a high quality CT is acceptable in more straightforward cases).

For a diagnosis of vestibular migraine, all standard clinical investigations should be normal or show incidental or age-related abnormalities, such as symmetrical high-frequency hearing loss. Nonspecific white matter lesions are more common in migraine sufferers generally and can cause diagnostic confusion and patient concern. Such MRI findings require careful consideration, usually by a neurologist, and often require a follow-up scan for reassurance. In most cases such findings should not rule out a diagnosis of vestibular migraine or prevent a trial of migraine therapy.

MANAGEMENT

Good management of vestibular migraine rests unequivocally on good communication. The patient needs to understand the basis on which the diagnosis is made and also that it remains a provisional diagnosis, and one that might be disputed by other doctors. The treating doctor must be able to tell the patient that he or she has done enough to rule out the 'sinister' disorders that so many patients with vertigo worry about, such as tumour and stroke. In some patients with milder vestibular migraine, providing a diagnosis and such reassurance is all that is required. Other patients, generally with more severe or frequent attacks, will require further treatment.

Currently, there is almost no high quality scientific evidence to guide therapy in vestibular migraine. However, clinical experience, and the limited scientific evidence that is available, indicate the skills acquired in managing migraine headache can be transferred to managing vestibular migraine, and with good results.[19] Thus, the main aspects of treatment are lifestyle modification (including avoiding specific triggers), treatment of acute episodes and preventive treatment.

Lifestyle modification

There are numerous lifestyle factors that can influence the occurrence of any type of migraine, including vestibular migraine. Stress, sleep, diet and alcohol are among the almost endless factors that can be incriminated in triggering migraine. A patient symptom diary can be a useful mechanism for increasing awareness of such individual triggering factors. For some patients, however, there are no obvious triggering factors and blanket lifestyle restrictions do not necessarily further the aim of improving patient quality of life. In the specific case of vestibular migraine, motion is a common trigger that can only be modified through unacceptably limiting the patient's normal daily activities.

Acute therapy

Standard therapy for acute vertigo and nausea, such as prochlorperazine and antihistamines such as promethazine, is usually applied to patients with vestibular migraine. The use of rectal prochlorperazine for severe attacks with vomiting can avoid the need for parenteral therapy. Zolmitriptan has shown some benefit in a small randomised trial, but in current clinical practice specific acute migraine therapy is not commonly used.[20]

Prophylactic therapy

Many patients require some form of migraine prophylaxis to reduce the frequency and severity of attacks. There is no agent that works every time (success rate is about 60% for most agents) or that is consistently well tolerated. A process of 'trial and error' should be adopted with a good level of communication, so that the patient has a role in choosing a particular agent as well as a reasonable understanding of what to expect. The approach is usually a slow and cautious introduction of any agent, starting with a low dose and waiting about two to four weeks before adding further small incremental increases in dose until one of three results is achieved: good symptomatic response; troubling side effects; or maximum dose reached.

There are many preventive agents to choose from and GPs should be familiar with a few of these, some of which are used off label (Table). Some of the agents that are considered first-line in Australia include pizotifen, β-blockers (often propranolol or metoprolol) and tricyclic antidepressants (especially amitryptiline, dothiepin and nortriptyline). Other agents that are reasonably commonly used include anticonvulsants (especially sodium valproate and topirimate) and calcium antagonists (especially verapamil).[21] The duration of therapy depends on many factors once a response is achieved, but generally, if the treatment response is particularly good then often a dose reduction can be trialled after three to six months.

Betahistine is not considered a migraine preventive and is most often used as a symptomatic treatment for vertigo or for Menière's disease. Treatment with betahistine is simple and safe, and clinical experience suggests it can also help patients with vestibular migraine, with dosage ranging between 8 mg daily and (rarely) as high as 32 mg twice daily.

Nonprescription therapy

Many patients with vestibular migraine are young women who are particularly averse to weight gain as a side effect of treatment (which can be common, especially when taking pizotifen, antidepressants and sodium valproate). Such women generally view prescription

medication as a last resort. There are a number of nonprescription agents that have shown some efficacy in patients with migraine headache in randomised controlled trials. These include riboflavin (about 200 mg, twice daily), magnesium aspartate (500 mg, daily), the medicinal herb feverfew (*Tanacetum parthenium*) and the plant extract butterbur (*Petasites hybridus*). Riboflavin, magnesium aspartate and feverfew have been used often in the author's practice and have obtained some gratifying responses, occasionally in patients who have failed a number of prescription preventive agents. There is also a definite role for vestibular rehabilitation and psychological therapy in patients with chronic vertigo, particularly in whom anxiety and loss of confidence supervene.

ROLE OF THE GP IN MANAGING VESTIBULAR MIGRAINE

Most patients with suspected vertigo should be seen by an appropriate specialist at least once, although the role of the GP is crucial at every stage of management. The GP can also start the process of excluding other causes of vertigo with judicious testing and can 'plant the seed' that migraine can be a cause of vertigo. Initiating therapy in general practice is reasonable and can facilitate an early diagnosis as well as potentially helping the patient to recover sooner.

Perhaps most important for managing vestibular migraine is implementing the 'trial and error' approach to therapy. This includes monitoring for side effects and benefit, switching treatment to a new agent if the past therapy has failed and ensuring that an active approach to management is maintained.

Urgent referral to hospital is required when the patient is too unwell to manage at home or in the surgery, or when there is a significant risk that the patient has experienced a stroke – this includes many patient with a single prolonged episode of spontaneous vertigo.

CONCLUSION

If vestibular migraine is looked for in general practice, it will be found. Generally, in cases where the clinical features are suggestive of vestibular migraine, the investigations will be normal and the patient's response to migraine therapy will be good. By recognising vestibular migraine when it is present, clinicians can now provide a significant group of patients with receive a clear diagnosis and effective treatment. In the past, such patients often had neither and were instead left sitting in the clinical 'too hard basket' and all too often given a psychiatric diagnosis. MT

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