Helen Turner, Claire Lavender and Peter Rea

Sudden-onset dizziness and vertigo symptoms:

assessment and management of vestibular causes

H Turner, BA, MRCS, DO-HNS, specialist registrar in ENT, Department of ENT, Nottingham University Hospitals, Queen's Medical Centre, Nottingham. **C Lavender**, BSc, MRCGP, DRCOG, GP, Wareham Surgery, Wareham, Dorset. **P Rea**, MA, FRCS (ORL-HNS), honorary professor of balance medicine, De Montfort University, Leicester, and consultant ENT surgeon, University Hospitals Leicester, Leicester.

Address for correspondence

Helen Turner, Department of ENT, Nottingham University Hospitals, Queens Medical Centre, Derby Road, Nottingham NG7 2UH, UK.

Email: helen.turner@doctors.org.uk

Submitted: 19 December 2019; **Editor's response:** 30 December 2019; **final acceptance:** 10 January 2020.

DOI: https://doi.org/10.3399/bjgp20X710369

INTRODUCTION

Approximately 2% of adults seek medical attention annually for symptoms of moderate to severe dizziness, with a third of consultations attributable to a vestibular cause.[1] Aside from the healthcare burden, there are significant socioeconomic implications for patients presenting with these symptoms. The diagnosis can be challenging given the wide range of possible causes — both vestibular and non-vestibular (see this graphically online at www.balancedisorderspectrum.info).[2]

The four most common vestibular diagnoses causing sudden-onset dizziness will be reviewed in detail. These are vestibular neuritis (or neuronitis) (VN), benign paroxysmal positional vertigo (BPPV), vestibular migraine (VM), and Ménière's disease.

DIFFERENTIAL DIAGNOSIS

Both vestibular and non-vestibular causes of dizziness are listed (Supplementary Table 1) but this is not exhaustive.

CLINICAL ASSESSMENT

A detailed history (Supplementary Table 1) can diagnose the majority of both vestibular and non-vestibular causes.

A detailed neuro-otological examination should be undertaken including otoscopy, cranial nerve examination, HINTS examination (Supplementary Table 2), Romberg's (test of proprioception and dorsal column function) and Unterberger's test (as per Romberg's but with the patient marching on the spot, rotating towards side of vestibular hypofunction), and a lying and standing blood pressure reading. Dix–Hallpike testing can be used to diagnose BPPV. Presence of geotropic (beating towards the ground), torsional nystagmus on Dix–Hallpike testing supports a diagnosis of BPPV and can be followed by a therapeutic Epley manoeuvre.

The main concern here is not missing a cerebellar vascular event involving the anterior or posterior inferior cerebellar arteries (AICA or PICA respectively), which can mimic VN. AICA strokes in particular can also cause hearing loss. The HINTS examination is a useful tool to distinguish VN from cerebellar stroke (Supplementary Table 2). This examination has been shown to have 100% sensitivity and 96% specificity for stoke,[3] but does need practice and training, and only relates to acute presentations of dizziness.

DIAGNOSES AND TREATMENTS

Vestibular migraine

VM is a variant of migraine and has a prevalence of approximately 1% of the population.[2] It is emerging as a clinical entity with publication of diagnostic criteria in 2012 inclusive of a category for probable VM.[4] The diagnostic criteria comprise vestibular symptoms for up to 72 hours, known or current history of migraine (not essential for diagnosis), presence of a characteristic headache, photo/phonophobia, or aura symptoms. Triggers include stress, tiredness, skipping meals, certain food groups, and menstruation. Avoidance of triggers and lifestyle changes improve many patients' symptoms. Cases persisting despite trigger avoidance and lifestyle changes can be commenced on prophylactic migraine medications.

Vestibular neuritis

This condition classically causes debilitating sudden vertigo often following a viral upper respiratory tract infection. It is thought to be due to viruses affecting the superior vestibular nerve in its narrow bony canal. VN is also sometimes incorrectly referred to as labyrinthitis — labyrinthitis by definition involves a loss of hearing as well.

The HINTS examination (Supplementary Table 2) can be used to differentiate this from acute cerebellar strokes.

Supportive treatments are offered including short-term anti-emetics (long-term vestibular sedatives delay

REFERENCES

1. Neuhauser HK, Radtke A, von Brevern M, *et al.* Burden of dizziness and vertigo in the community. *Arch Intern Med* 2008; **168(19):** 2118–2124
2. Hugill A, Rea P, Fu G. Interactive infographic for the identification of balance disorders. 2018. http://www.balancedisorderspectrum.info/circular.html (accessed 6 Apr 2020).
3. Kattah JC, Talkad AV, Wang DZ, *et al.* HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke* 2009; **40(11):** 3504–3510.
4. Lempert T, Olesen J, Furman J, *et al.* Vestibular migraine: diagnostic criteria. Consensus document of the Bárány Society and the International Headache Society. *J Vestib Res* 2012; **22(4):** 167–172.
5. Fishman JM, Burgess C, Waddell A. Corticosteroids for the treatment of idiopathic acute vestibular dysfunction (vestibular neuritis). *Cochrane Database Syst Rev* 2011; **(5):** CD008607.
6. Patel M, Agarwal K, Arshad Q, *et al.* Intratympanic methylprednisolone versus gentamicin in patients with unilateral Ménière's: a randomised, double-blind, comparative effectiveness trial. *Lancet* 2016; **338(10061):** 2753–2762.
7. Adrion C, Fischer C, Wagner J, *et al.* Efficacy and safety of betahistine treatment in patients with Meniere's disease: primary results of a long term, multicentre, double blind, randomised, placebo controlled, dose defining trial (BEMED trial). *BMJ* 2016; **352:** h6816.
8. Lopez-Escamez J, Carey J, Chung WH, *et al.* Diagnostic criteria for Menière's disease. *J Vestib Res* 2015; **25(1):** 1–7.

Competing interests

Professor Rea is a collaborator on a novel artificial intelligence-led diagnostic system for balance disorders (BalanceAID), which is currently fundraising. Professor Rea's work for autonomy, a US biotech company studying intra-tympanic therapies for Ménière's disease generates income for his hospital's charitable funds.

Discuss this article

Contribute and read comments about this article: **bjgp.org/letters**

compensation). Physical activity is encouraged where possible to aid vestibular compensation and expedite recovery times. There is a potential role for systemic steroids, although a Cochrane review in 2011 determined there was insufficient evidence for usage.[5]

Most patients have an uneventful recovery within weeks, although a small proportion develop secondary BPPV, chronic imbalance caused by the resulting vestibulopathy, over-reliance on visual stimuli, or neuropsychological sequelae such as persistent postural-perceptual dizziness (PPPD).

Benign paroxysmal positional vertigo

BPPV has a prevalence of approximately 1–2% of the population.[2] It is caused by displacement of otoconia from the utricle, most commonly into the posterior semi-circular canal (canalithiasis). The history is typically short spins exacerbated by head movements, particularly rolling over in bed or looking up. There may be a history of previous head trauma or recent VN. BPPV is supported by a positive Dix–Hallpike test or reproducible symptoms in the Dix–Hallpike position if no nystagmus is seen. This can be followed by an Epley manoeuvre with advice to sleep propped up for a few days thereafter. Brandt–Daroff exercise can be performed by the patient if symptoms recur.

Resistant cases may be due to cupulolithiasis (otoconia adherent to the cupula) and the Semont manoeuvre can be helpful. Acute worsening of dizziness immediately after an Epley may be caused by octoconia diverted into the lateral semi-circular canal. A log roll manoeuvre ('BBQ roll') is adopted in these patients.

Surgery to the posterior semi-circular canal is performed in a tiny cohort of patients with intractable recurrent symptoms despite repositioning manoeuvres.

Ménière's disease

The prevalence of Ménière's disease is approximately 1 in 1000 of the population.[2] Vertigo typically lasts from 20 minutes to 12 hours with patients also reporting tinnitus, aural fullness, and fluctuating hearing loss. The hearing loss often recovers between attacks in the early stages of the disease but eventually becomes permanent. Drop attacks occur infrequently, late in the disease.

The underlying histopathological marker is endolymphatic hydrops.

Treatments can include dietary and lifestyle changes (salt and caffeine reduction), betahistine, bendroflumethiazide, topical agents delivered via the middle ear (such as steroids and gentamicin), and very rarely surgery on the labyrinth. Intra-tympanic steroids have been shown to lead to an average 90% reduction in vertigo over 2 years[6] and are now a mainstay of therapy. Dietary advice and betahistine are commonly commenced in general practice with further treatments recommended by an otolaryngologist. However, it should be noted that a *BMJ* publication in 2016[7] showed no reduction in vertigo over placebo with betahistine, and this should be discussed with patients before prescription. It has some vestibular sedative action.

There is diagnostic overlap with vestibular migraine. Diagnostic criteria for Ménière's were published in 2015.[8] Ménière's is classified into definite and probable categories; the latter patients do not demonstrate audiometric evidence of hearing loss. Other diagnostic criteria include two or more vertiginous episodes lasting up to 12 hours and fluctuating aural symptoms (tinnitus, aural fullness, and deafness). Patients can be asymptomatic between episodes early in the disease.

ADVICE

Ask patients to keep a diary of recurrent episodes, especially linked to food and menstruation.

Exercise is generally encouraged to expedite vestibular compensation.

Patients should be made aware they should abstain from driving during acute episodes and of their legal obligations regarding driving. They should be referred to the DVLA website and, if acute incapacitating attacks recur, may have their licence suspended.

WHEN TO REFER TO SECONDARY CARE

Patients who become dehydrated because of vomiting during acute attacks are admitted for intravenous fluids, often under on-call medical teams.

Patients can be referred to their local ENT service or audio-vestibular medicine for recurrent or chronic vestibular symptoms. Many trusts now have dedicated balance clinics. Where neurology is suspected, the stroke service or neurology department are more appropriate. Where syncopal symptoms exist, the local medically led syncope clinic is preferred.

Funding

None.

Provenance

Freely submitted; externally peer reviewed.