Migraine and vestibular dysfunction

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Summary

- Vestibular symptoms in migraine are disabling and may be the dominant feature leading to inaccurate diagnosis
- Recurrent episodic vertigo is common in migraineurs and most frequently due to the migraine process itself
- Co-morbid peripheral vestibular disorders occur at higher prevalence in migraineurs
- Vestibular dysfunction can be a trigger in migraine and needs due recognition
- Vestibular migraine is the chameleon to spot as the drive is migrainous but the course of vertigo is highly variable in duration, attack frequency and association with cephalagia- with the latter not infrequently attenuated. For pointers to identifying the vestibular dysfunction see Table 1.

The association of vestibular aura/dysfunction with migraine is often under-recognised leading to delayed treatment, adding further to the burden of disability migraineurs suffer. Migraine is one of the commonest neurological complaints with a lifetime prevalence of 16%, with headache representing one fifth of neurology outpatient referrals.[1] It is recognised by WHO to be ranked amongst the top 10 most disabling conditions with psychiatric morbidity at the top of the league table. Vertigo is also extremely common with a life-time prevalence in adulthood of 7%, having a significant psycho-social impact. Migraineurs are more likely to suffer from vertigo with 3.2% affected and this is in excess of the anticipated co-morbid prevalence of 1.1%.[2] This cohort of migraineurs with vestibular dysfunction, have significant associated psychiatric morbidity with the highest indices of anxiety reported in vertiginous cohorts with vestibular migraine.[3]

Whilst cephalgia is typically the most prominent complaint, vestibular disturbance can predominate as part of an aura presentation – brainstem aura – or through parallel dysfunction in vestibular pathways – vestibular migraine – or as co-morbid vestibular disorders such as BPPV and Meniere's. Discriminating between the centrally driven migrainous process and peripheral lesions is important to tailor treatments and direct management.

Common vestibular disorders in migraine

Recurrent vertigo is most common in migraineurs and although this is frequently migrainous in origin, the high prevalence of vestibular disorders raises the question of overlapping pathophysiology.

Benign Paroxysmal Positional Vertigo (BPPV):

Transient vertigo due to BPPV is common, with attacks typically lasting seconds and recurring over several weeks but usually self-limiting. It is triggered by specific changes in the position of the head and is caused by the movement of displaced otolith crystals within the semi-circular canals. In tertiary neuro-otology clinics BPPV is one of the most common underlying diagnoses; accounting for a fifth of cases in the 5000 cases reviewed at the Munich centre.[4] In those with idiopathic BPPV they are however twice as likely to have co-morbid migraine.[5] BPPV in this patient group needs to be independently treated with canalith repositioning manoeuvres, particularly given that the vestibular presentation may serve as a trigger for migraine.

Meniere's Disease:

The differentiation between Meniere's and migraine in a single acute vertiginous attack with emesis and headache is often difficult. However repeated attacks allow for discrimination of Meniere's: with audiometrically evident hearing loss (typically unilateral although can become bilateral over time, rarely alternating between ears) and horizontal nystagmus which may initially only be manifest during the attack. In addition to profound vertigo lasting typically in excess of 20 minutes, report of tinnitus or aural fullness together with progressive hearing loss is diagnostic.[4] The condition is driven by endolymphatic hydrops with paraclinical supporting evidence of significant audiological loss and peripheral vestibular dysfunction on oculographic calorics. Once again migraine is twice as common in this group[6] and with audiological loss fluctuating it may not be captured on pre-arranged testing in the early stages and testing at the time of the attack should be pursued if there is any doubt. Treatment for Meniere's includes a low salt diet, diuretics and high dose betahistine.

The relationship between migraine and Meniere's remains unclear however a causal relationship has been questioned following the observation that those with migraine develop Meniere's earlier and more frequently have bilateral hearing loss.[7]

Migraine in association with vestibular symptoms

Two migraine phenotypes are recognised in the international classification of headache disorders (ICHD version 3 beta:[8]) with prominent vestibular symptoms: Migraine with brainstem aura and Vestibular migraine.

Migraine with brainstem aura

Having previously been called basilar artery migraine in reference to the presumed unifying

Table 1: Summary of vestibular dysfunction seen in migraine

	Typical duration of attack	Pattern of presentation	Triggers	Tests to consider	Specific treatments
BPPV	secs to mins	recurrent over weeks; fatigable	Head position	Epley Vestibular	Canalith repositioning manoeuvres
Meniere's disease	>20mins -24hrs	episodes often with prolonged inter-ictal periods (mths)	?high salt diet ?migraine	Audio-vestibular	Diuretics Low salt diet Betahistine
Motion sickness	Prolonged >30mins - hrs	triggered	Motion Visual perception of movement	Nil	Anti-emetics
Migraine with brainstem aura	<60mins	episodic to chronic	Migraine triggers: – stress – menstruation – sleep architecture – diet & dehydration – alcohol – exercise – weather	MRI	Anti-migraine including anti-epileptics, pizotifen and flunarazine Role for customised vestibular rehabilitation once cephalgia is suitably controlled.
Vestibular migraine	Variable: secs, mins, hrs	episodic to chronic		Audio-vestibular	
	recurrent	Vestibular dissociation from cephalgia common			

arterial pathophysiology, our current understanding of migraine has taken us beyond a primary vascular aetiology (as outlined by Holland and Afridi, ACNR 2014;V13(7):19-21) and this is recognised by the ICHD in renaming this as migraine with brainstem aura. Whilst vestibular dysfunction may occur in this phenotype, in association with other brainstem aura phenomena, its presence is in keeping with aura and as such the duration of the vertiginous attack is consistent with that of Leao's cortical spreading depression lasting one hour. Two or more brainstem mediated phenomena evolving or occurring in succession over more than five minutes include: vertigo, tinnitus, hypoacusis, diplopia, bilateral visual symptoms, simultaneous bilateral sensory paraesthesia, ataxia, loss of consciousness. This is in conjunction with migrainous unilateral, throbbing, moderate to severe headache, starting during or within one hour of the aura and lasting more than four hours with hypersensitivity to external sensory stimuli (photophobia, phonophobia, osmophobia) and typical aggravation on routine physical activity. Please refer to Box 1 for summary of ICHD3beta diagnostic criteria for migraine with brainstem aura.

Migraine with brainstem aura is rare in both migraine and neuro-otology, occurring in 10% of those with migraine with aura.[9]

Vestibular migraine

Vestibular migraine with vestibular symptoms in isolation of other brainstem aura is much more common accounting for up to 10% of patients seen in tertiary neuro-otology dizzy clinics.[4] Please refer to box 2 which details the ICHD3beta diagnostic criteria for vestibular migraine. Importantly the duration of vertigo can be very variable and in up to 70% is not in keeping with aura duration: lasting seconds, hours or days. In addition the association with cephalgia is variable with only 48% reporting consistent cephalgia although migrainous associated symptoms of phonophobia and photophobia are more prevalent. Vertigo can

Box 1: Migraine with brainstem aura (1.2.2)

Former terms: Basilar artery migraine; basilar migraine; basilar-type migraine.

Migraine with aura symptoms clearly originating from the brainstem or with evidence of bi-hemispheric involvement, but no motor weakness.

Diagnostic criteria:

At least two attacks fulfilling the following criteria:

1. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but no motor or retinal symptoms
2. At least two of the following brainstem symptoms:
   a. dysarthria
   b. vertigo
   c. tinnitus
   d. hypacusis
   e. diplopia
   f. ataxia
   g. decreased level of consciousness
3. At least two of the following characteristics:
   a. at least one aura symptom spreads gradually over 5 minutes, and/or two or more symptoms occur in succession
   b. each individual aura symptom lasts 5-60 minutes
   c. at least one aura symptom is unilateral
   d. the aura is accompanied, or followed within 60 minutes, by headache

NB. transient ischaemic attack has been excluded.

Box 2: Vestibular Migraine (A1.6.5)

Former terms: Migraine-associated vertigo/dizziness; migraine-related vestibulopathy; migrainous vertigo

A current or past history of Migraine without aura or Migraine with aura with concurrent vestibular symptoms.

At least five episodes fulfilling the following criteria:

1. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours, including (as defined by the Bárány Society):
   a. spontaneous vertigo:
      i. internal vertigo (false sensation of self-motion);
      ii. external vertigo (false sensation that the visual surround is spinning or flowing);
   b. positional vertigo, occurring after a change of head position;
   c. visually induced vertigo, triggered by a complex or large moving visual stimulus;
   d. head motion-induced vertigo;
   e. head motion-induced dizziness with nausea (dizziness is characterised by a sensation of disturbed spatial orientation; other forms of dizziness are currently not included in the classification of vestibular migraine).
2. At least 50% of episodes are associated with at least one of the following migrainous features:
   a. headache with at least two of the following four characteristics:
      - unilateral location
      - pulsating quality
      - moderate or severe intensity
      - aggravation by routine physical activity
   b. photophobia and phonophobia
   c. visual aura

occur at any time point during the migrainous episode, with the attack frequency being highly variable with days to years between events. The average age of onset of vertiginous attacks post-dates the onset of migraine by years and migraine may have become quiescent, with the headache during the vertiginous attacks often attenuated- emphasising the utility of a directed interrogation of former migraine in those presenting with recurrent episodic vertigo.[10] Trigger factors precipitating attacks may also prove useful as the episodes are often triggered by well-established migraine triggers as detailed in the summary table. Ultimately further diagnostic clarity can be derived from the good response to treatment with anti-migraine preventatives. In clinical practise the full array of migraine preventatives are used including beta blockers, tricyclic antidepressants, anti-epileptics (topiramate, sodium valproate etc.), serotonergic (pizotifen) drugs and calcium channel blockers (flunarazine) –the latter with anecdotal significant utility. Unfortunately clinical drug trials in vestibular migraine remain either underpowered or compounded by lack of rigour in diagnostic inclusion.

Distinct from Meniere's disease, significant progressive hearing loss or vestibular deficit are not a persistent feature in vestibular migraine. The diagnosis of vestibular migraine should be based on the history as there are no diagnostic findings on examination or audio-vestibular assessment. Significant nystagmus in the migraineur should be treated with caution and a peripheral cause considered in those with otherwise normal examination and no clinical pointers to central disorders. Unilateral hypo-excitability on calorics without associated pathological nystagmus however should not raise concern as this simply indicates prior centrally compensated peripheral vestibular deficit. In both BBPV and vestibular migraine head motion precipitated vertigo occurs: in BBPV this is fatigable and self-limiting, with an abnormal examination whilst symptomatic; whilst the vertigo of vestibular migraine is recurrent, non-fatigable and with a typically normal examination when symptomatic.

Vestibular trigger to migraine

Discerning the underlying cause of vertigo in migraine is paramount as it will enable both tailored treatment and in the case of co-morbid peripheral vestibular disorders will also serve to reduce a potential migraine trigger. In a prospective study of the incidence of migraine in the 24 hour period following vestibular testing (including calorics) almost a half of migraineurs studied recorded migraine and this frequently occurred during the induced vertigo.[11] This underlies the need to carefully interrogate presentations where vertigo just precedes the onset of migraine and not assume that this is vestibular migraine.

Motion sickness

Motion sickness can be defined as prolonged symptoms of dysequilibrium in conjunction with nausea provoked by movement or the illusory perception of movement. In migraineurs, contrary to other peripheral disorders, there is a high prevalence of motion sickness with approximately two thirds affected either with an antecedent or concurrent history.[12,13] Current theories in migraine associated phenomena focus on sensory dysmodulation. It is proposed that motion sickness highlights failure of normal integration of vestibular, visual and proprioceptive cues with disruption of normal inhibitory gating mechanisms in the brainstem responsible for hypersensitivity of the emetic centre, sharing a similar underlying pathophysiology to migraine (for comprehensive review: 14). In movement induced motion sickness the principal mediator is the vestibular apparatus with experiments showing complete attenuation of this phenomenon in those without a functional vestibular apparatus. However visual induced motion sickness is not directly mediated by the vestibular apparatus but rather reflects dysmodulation of converging sensory inputs, and is more frequently seen in migraineurs compared to controls.[15] ◆

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