Journal of Clinical Neuroscience xxx (2018) xxx–xxx

Contents lists available at ScienceDirect

Journal of Clinical Neuroscience

journal homepage: www.elsevier.com/locate/jocn

Review article

Clinical characteristics and treatment choice in vestibular migraine

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ARTICLE INFO

Article history:
Received 24 August 2017
Accepted 22 February 2018
Available online xxxx

Keywords:
Vestibular
Migraine
Headache
Vertigo

ABSTRACT

This retrospective review aims to survey the clinical characteristics and management of vestibular migraine (VM) patients seen in a tertiary hospital multi-disciplinary balance disorders clinic, and how this aligns with the evidence base in the literature. A single investigator reviewed the medical records of the patients who presented to a tertiary hospital balance disorders clinic over a four month period and identified 90 cases of VM. The mean age of patients with a diagnosis of VM was 50 years (range of 17–84) and 72 (80%) were female. Vertigo (96%) and headache (60%) were the predominate symptoms. Vestibular function testing abnormalities included six (5%) with a positive video head impulse test and seven (6%) with oculomotor abnormalities. Pizotifen (30%) and amitriptyline (21%) were the two most commonly used medications whilst only 14 (16%) received vestibular physiotherapy. This study suggests that VM is a very common presentation to a tertiary balance disorders clinic, but there is little consensus in choice of initial management and vestibular rehabilitation is underutilized. This data may be valuable in informing the practice of neuro-otology as well as in the planning of future service provision.

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1. Introduction

Vestibular Migraine (VM), was first described by Slater in 1979 as Benign Recurrent Vertigo [1] but it was not recognised as a formal diagnostic entity until as recently as 1999 [2]. VM is the second most common cause of vertigo after Benign Paroxysmal Positional Vertigo (BPPV) [3], with an incidence of 1.4%, 1 year prevalence of 4.9% and an estimated lifetime prevalence of 7.4% [4]. Previous studies have suggested that VM accounts for 6–7% of presentations to dizziness clinics [2,5]. Vertigo is two to three times more common in migraineurs than in headache free controls [6] and women are affected two to three times more often than men [7]. The pathophysiology of VM remains uncertain, but is likely of a central nervous system origin [8] and an association between migraine and vestibular dysfunction has been proposed and widely acknowledged [3].

VM is characterised by recurrent episodes of vertigo, disequilibrium or balance impairment lasting from minutes to greater than 72 h [9], except for chronic migrainous vertigo which may persist for six months or more [10]. It may be associated with or without headache, and may also be accompanied by a myriad of other symptoms including nausea, vomiting, photosensitivity, phonosensitivity, osmosensitivity, mild subjective hearing loss, tinnitus and aural fullness with or without visual aura [9]. This list is neither exhaustive nor requisite, and patients generally present with some, but not all of these symptoms and whilst some patients have fairly stereotyped episodes, others experience varying combinations of symptoms during different episodes.

The diagnostic criteria for VM has most recently been defined by a joint committee of the Barany Society and a subcommittee of the International Headache Society (IHS) [11]. In most patients, there is no vestibular deficit or hearing loss noted on vestibular function testing or audiometry, which may aid exclusion of other causes of vestibular symptoms such as vestibular neuronitis (VN) and Meniere's Disease (MD) [9]. It must be noted however that mild abnormalities on audio-vestibular testing in the interictal period have been documented, and are not uncommon [8]. Oculomotor abnormalities are common and can occur in both the active and symptom free periods. They can include spontaneous downbeat, upbeat, horizontal or pure torsional nystagmus with or without persistent central positioning nystagmus (CPN) (10–20%) [2,12,13] in the absence of any other cerebellar or brainstem signs [2]. Saccadic pursuit or gaze evoked nystagmus may

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https://doi.org/10.1016/j.jocn.2018.02.020
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Please cite this article in press as: Power L et al. Clinical characteristics and treatment choice in vestibular migraine. J Clin Neurosci (2018), https://doi.org/10.1016/j.jocn.2018.02.020

also be present in around 10–20% of patients [2,14]. Twenty percent of patients also display headshaking nystagmus which can be downbeating or horizontal [12].

Ten to twenty percent of patients with VM will have unilateral hypofunction on caloric stimulation [13,15–17], with up to 11% displaying bilateral caloric hyporesponsiveness [18,19]. Some patients (9–11%) may also display vestibular hypofunction on video head impulse (vHIT) examination [14,16,17]. Rotatory chair may reveal a reduced VOR gain [2] as a rare finding [8]. Cervical and ocular vestibular evoked myogenic potentials (cVEMPs and oVEMPs) may be atypical. A study by Kang et al. in 2016 showed that 11% of VM patients had abnormal cVEMP results and 27% revealed abnormal oVEMP results [16].Balance impairment is also common with around 15% displaying a positive Romberg's test [12] and up to 38% exhibiting abnormal sensory organisation test (SOT) results [16]. The average prevalence of unexplained hearing loss in VM is 7.5% [20], these losses are often mild, and not progressive over the course of the disease [7,19], which contrasts with the characteristic, fluctuating, progressive low frequency hearing loss which is seen in MD [20,21]. Whether these audiovestibular abnormalities are truly related to VM by cause and/or association remains a contentious issue.

Although clinical characteristics and diagnostic testing have been examined for VM, its treatment is far less formalised. Current treatment for VM includes both non-pharmacological and pharmacological therapy. The mainstay of management for VM is prophylactic, and less so abortive medications (including anti-vertiginous and anti-emetic medications) [3].

Pharmacological treatment tends to be broadly categorised as either prophylaxis or management of an acute episode. Examples of the former are pizotifen [10], beta-blockers such as propranolol [22] or metoprolol [23], anti-convulsants such as topiramate [24], anti-depressants such as amitriptyline [23] or venlafaxine [22], or calcium antagonists such as verapamil, flunarizine or cinnarizine [3]. Management of acute episodes includes the use of high dose aspirin, non-steroidal anti-inflammatory agents and triptans [25].

Non-pharmacological treatment may include lifestyle factor modifications such as dietary alteration [26], sleep management, and avoidance of triggers. Rehabilitation in the form of vestibular physiotherapy has also demonstrated success in improving clinical outcomes on and subjective and objective metrics [27]. These numerous treatment options and the often idiosyncratic or even haphazard manner in which they are utilized, reflects the lack of a clear consensus on the treatment of VM. Amongst the many factors considered by the treating clinician are patient co-morbidities, frequency of attacks and potential medication side effects and interactions [3,28]. As such, this retrospective review seeks to describe the characteristics of VM, explicate the management of VM in a tertiary balance disorders clinic, and describe how this aligns with the evidence base in the scientific literature.

2. Methods

A single investigator reviewed the medical records of patients who attended the Royal Victorian Eye and Ear Hospital's multidisciplinary Balance Disorder and Ataxia Service (BDAS), where patients are assessed by Neurologists, Neuro-otologists, Otolaryngologists and emergency department doctors or registrars who are closely supervised. All patients receive vHIT immediately prior to their initial consultation, and may receive comprehensive vestibular function tests (VFT) should the clinical diagnosis of VM remain uncertain. The records of patients who presented to the service in the months of February to May of 2015 (n = 220) were examined and all of those who had been diagnosed with VM were included in this study (n = 90). Patients were diagnosed by various clinicians with either definite or probable VM using the Barany Society and International Headache Society (IHS) diagnostic criteria for VM [11].

Data regarding age at diagnosis, gender, referral source, previous diagnoses, features elicited on history and examination, initial investigations, initial management and frequency of review appointments, patient reported outcome at initial follow-up and subsequent management plans were extracted from patient records. This included clinician notes and questionnaires filled out by patients prior to their initial appointment.

All patients gave informed consent with approval of the Royal Victorian Eye and Ear Hospital's Human Research Ethics Committee.

3. Results

3.1. Patient demographics

Fifty-two patients (58%) were diagnosed with definite VM, and 38 (42%) were diagnosed with probable VM based on IHS diagnostic criteria. The mean age of patients with a diagnosis of VM was 50 years with a range of 17–84 years. Of the 90 patients diagnosed with VM, 72 (80%) were female.

3.2. Referral sources

Sixty percent (n = 54) of the study cohort were referred to by primary care clinicians such as general practitioner doctors. The remaining patients were referred by Neurologists (n = 13, 14%), Otolaryngologists (n = 14, 16%) and Physiotherapists (n = 2, 2%) working externally to our hospital.

3.3. Initial consultation

3.3.1. Findings on history

The frequency of reported symptoms in our VM population is described in Table 1.

Sixty percent (n = 54) of patients had a concomitant history of headache-type migraine. Twenty (22%) had a family history of migraine in a first-degree relative. There were no differences in characteristics between those VM sufferers presenting with and without visual aura.

Twenty-one patients (23%) completed a Dizziness Handicap inventory (DHI) on their first visit. The mean DHI score was 57.7, reflecting moderate disability referrable to dizziness-type symptoms.

3.3.2. Examination

On presentation, 77 (86%) patients had a normal neuro-otological examination, which included a detailed oculomotor and cranial nerve examination as well as assessment of cerebellar function, manual muscle testing for power, tone and deep tendon reflexes, somatosensory examination, speech assessment and vHIT.

Table 1
The frequency of various reported symptoms in the study cohort.

Symptom	Frequency
Vertigo	96%
Headache	60%
Visual disturbance	51%
Nausea	49%
Tinnitus	44%
Aural fullness	30%
Phonophobia	26%
Hearing loss	23%
Vomiting	19%
Other neurology (e.g. sensory disturbance)	14%
Otalgia	2%

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