Vestibular Migraine: How to suspect and to treat it?

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Introduction

- It is a newly defined entity considered as a migraine variant
- First presentation in 1984 by Kayan: association migraine and vertigo
- *In 2012 Barany Society and International Headache Society* released a consensus document on the diagnostic criteria which are been included in the third edition of the *International Classification of headache disorders: ICHD-3.*
- 90% of Pubmed listed articles surged in the last two decades
Epidemiology

- In the study «The Epidemiology of vestibular migraine: A population-based survey study» Formeister EJ et al. de 2018, on 21780 patients in USA

- 11.9% have vertigo whose **23.4% respond to the Vestibular Migraine criteria**

- Only 10% of persons with the criteria of MV VM are diagnosed. There is an important misdiagnosis

- The diagnosis can be challenging in cases with hearing loss or Positionnal vertigo or strong headache associated
Diagnosis criteria

VESTIBULAR MIGRAINE

A. At least 5 episodes with vestibular symptoms of moderate or severe intensity lasting 5h min to 72 hours

B. Current or previous history of Migraine with or without aura according to the ICHD;

C. On or more migraine features with at least 50% of the vestibular episodes:
   - Headache with at least two of the following characteristics: one side location, pulsating quality, moderate or severe pain intensity, aggravation routine physical activity
   - Photophobia and phono phobia,
   - Visual aura;

D. Not better accounted for by another vestibular or ICHD.
PROBABLE VESTIBULAR MIGRAINE

A. At least 5 episodes with vestibular symptoms of moderate or severe intensity lasting 5h min to 72 hours

B. Only one of the criteria B et C for vestibular migraine is fulfilled (migraine history or migraine features during the episode)

C. Not better accounted for by another vestibular or ICHD.

https://content.iospress.com/download/journal-of-vestibular-research/ves00453?id=journal-of-vestibular-research%2Fves00453
Vestibular symptoms

Eligible for diagnosis of VM

- Spontaneous vertigo, with self motion or visual surrounding as spinning or flowing
- Positional vertigo, after a change of head position
- Visually induced vertigo, triggered by large moving visual stimulus
- Head motion induced vertigo (during head mvt)
- Head motion induced-dizziness with nausea. (Dizziness is characterized by sensation of disturbed spatial orientation.)
Vestibular migraine: suggestive patterns

• Combinations of spontaneous, positional, head motion-induced and visual vertigo

• Recurrent vertigo lasting days

• Positional vertigo with short episodes (hours to days) and frequent recurrences

• Menstrual vertigo

• Recurrent vertigo replacing recurrent headaches around menopause
Vestibular symptoms

Can happen:
- Before, during or after headache
- In 30% of cases: absence of headache during the crisis

Sometimes vertigo and headache are never concomitant → look systematically for the associated symptoms of migraine (nausea, vomiting, osmophobia, phono or photophobia).
Neuhauser et al. 2001
VM Characteristics - Adulte

- Prevalence: W > M (1 to 5 times more)
- **Middle age of onset is late:**
  - Women: 37.7 years
  - Men: 42.4 years
- **Family ground (with an autosomal dominant pattern)**

- The onset of crisis of headache or vertigo is often delayed by several years.
- Sometimes the migraine is stopped for several years when the vertigo attacks begin.

*Journal of Neurology 1999 – Dietrich et Brandt*
Triggers of Vestibular Migraine

- Are the same than Migraine attacks
- Lack of sleep, stress, anxiety
  - Hormonal changes and menstrual vertigo
  - Excess food
  - Visual stimulations
- Specificity: Vestibular Testing as caloric or rotatory

Vertigo as a migraine trigger.

Murdin L, Davies RA, Bronstein AM.
Visual abnormalities

Visual vertigo as common symptom
(Cass et al. 1997)

Imbalance after exposure to visual motion
(Imaizumi et al. 2015)

Visually-induced motion sickness
(Drummond 2005)
Duration

- Variable
- 10%: few seconds
- 30%: few minutes
- 30%: few heures
- 30%: few jours

→ « Barany society Criteria » : 5 min to 72 h
Associated symptoms

- **Auditory symptoms:**
  - 38%: Hearing loss, tinnitus, aural fullness,
  - Aggravation over the time but still remain moderate or transient
  - 18%: deafness on low frequency


- **Lower quality of life:**
  - 40-60% missed work or school
  - 23% visited hospital or emergency
  - Anxiety and depression are significantly associated with VM. Lack of timely diagnosis can also contribute to development of vestibular symptoms

Deafness and Vestibular Migraine

Radtke et al. 2012
**Examination: Physical findings**

- **Between attacks:** ex is often normal; the most commonly abnormality is positional nystagmus even patient is asymptomatic.

- **During attacks:**
  - Positional or spontaneous nystagmus in 70% cases resolved on the follow-up.
  - **Oculomotor findings with central look (8.6-66%):** (Dietrich and Brandt)
    1/ Gaze nystagmus = direction changing with the direction gaze
    2/ Positional nystagmus: No latency/ No fatigability / less intensity, impairment VOR suppression (different of BPPV)
    3/ Can be find between attacks
Nystagmus during acute vestibular migraine

(Hartman Polensek & Tusa 2010)

(n = 26)
Positional nystagmus during acute vestibular migraine

(Hartman Polensek & Tusa 2010)

Positional nystagmus was sustained and started without latency in 75%
Videonystagmography findings in the asymptomatic interval: peripheral

Unilateral decrease of caloric response: 8-60%

Directional preponderance: 20-25%

Spontaneous nystagmus: 0-16%

VOG findings in the asymptomatic interval: central

- Decreased smooth pursuit 25-30% (controversial)
- Saccadic dysmetria 20% (controversial)
- Impaired VOR suppression 13% (controversial)

Child’s forms

- 15-20% of vertigo of childhood
- Benign paroxysmal vertigo is considered as one the first manifestation of Migraine.
- But also abdominal pain, cyclic vomiting, benign paroxysmal torticollis.
Diagnostic pitfalls

- Think to VM when:
  - Atypical BPPV with no latency, longer, and small central signs

- Think to Posterior circulation stroke when one or more signs:
  - Skew deviation, HIT normal, torsional nystagmus, acute unilateral deafness, dysarthria, severe gait ataxia and altered Oculo motor testing +++

In cases of first episode MRI is required.

Don’t forget to perform the HINTS
Differential diagnosis: VM and Meniere's disease

Diagnosis is sometimes difficult, based on a spectrum of arguments:

- Clinical history +++
- Associated signs, triggers,
- Clinical exam,
- Deafness evolution

Differential diagnosis: VM and Meniere's disease

Signs and symptoms can often be correlated

In favor of Vestibular Migraine:

➢ Short crisis < 15 minutes or longer > 24 h
➢ Photo-phonophobia, anxiety, frequent palpitations
➢ High visual sensitivity
➢ Central signs from clinical exam +++
➢ Fluctuating deafness in low frequency often bilaterally but rarely permanent nor evolutive. +++
Differential diagnosis: VM and Meniere's disease

Dc criteria of both diseases are recognized in 13% of all patients:

- Crisis where symptoms associated with low or no auditory signs are predominant
- Crisis with auditory signs, auditory fluctuation documented, plenitude, tinnitus, even if migraine headaches occur during crisis.

ICHDI classification could include a new entity with overlap of MV and Meniere!?
TAKE HOME MESSAGE

Differentiate MM and MV

- Crisis duration
- Central signs during clinical examination (especially positional) during crisis
- Can be observed out of crisis
- Moderate deafness doesn’t exclude diagnosis
- associated signs: photo-phonophobia
- to triggering factors

→ Both diseases can co-exist thus, treatment needs to be adapted
Differentiate MV and M Menière

- Place of cervical VEMPS?
- Place of MRI hydrops protocol?
cVEMPS

Vestibular evoked myogenic potentials to sound and vibration: characteristics in vestibular migraine that enable separation from Menière’s disease

Rachael L Taylor¹, Alessandro S Zagami², William PR Gibson¹, Deborah A Black¹, Shaun RD Watson², G Michael Halmagyi¹ and Miriam S Welgampola¹

Figure 5. Mean (+2SE) asymmetry ratios for Controls, VM and MD across stimulus frequencies. Asymmetry ratios are higher for MD compared to Controls and VM.

Figure 3. Effect of cone-burst frequency on oVEMP and corrected cVEMP amplitudes. Mean ± 1 SE cVEMP (A) and oVEMP (B) reflex amplitudes for MD (affected and unaffected ears), VM and normal control ears. cVEMP amplitudes are corrected for baseline electromyographic activity.
Can Vestibular-Evoked Myogenic Potentials Help Differentiate Ménière Disease from Vestibular Migraine?

M. Geraldine Zuniga, MD\textsuperscript{1,2}, Kristen L. Janky, AuD, PhD\textsuperscript{1}, Michael C. Schubert, PT, PhD\textsuperscript{1}, and John P. Carey, MD\textsuperscript{1}

Can be interesting
Remains to define the method
TB 500Hz on AC or on BC seems to be the best stimulation.
Clinic case: Mrs B. Nathalie  52 years old

**ATCD:**
- Treated migraine with B Blockers (Lopressor)
- Depressive and anxious ground treated with Escitalopram

**Clinical history:** symptoms start around 25 years old with crisis evoking a left Meniere disease?

**Evolution:** new crisis typical of Meniere and others more atypical
- **Dizziness** and **left aural fullness**, humming and whistling tinnitus more or less marked
- Deafness on low frequencies
- Variable duration: **a few hours to a few days**
- Frequent **headaches** pre or post crisis
- Sometimes **bilateral** auditory symptoms

**Vestibular clinical examination** normal out of crisis
Paraclinical assessment

- Caloric: 50% left deficit without directional preponderance of the nystagmus.
- VHIT: Normal
- TDM and MRI: Normal
Mrs B. New Medical Care

- Introduction flunarizine
- 1/day for 2 months then progressive decrease over 2 months if symptoms improve.
- Beta blocker stop
- Acetazolamide ½ cp twice a day for its osmotic action.
Evolution after 6 months

Flunarizine and Acetazolamide

- No more crisis or dizziness
- Hearing improvement.
- Tinnitus disappearance

Stop flunarizine at 6 months because reurgence of the depressive syndrom.
IRM Protocole HYDROPS

A SIMPLIFIED CLASSIFICATION BASED ON SACCULAR MORPHOLOGY

Attié et al., Eur Radiol 2016: « 1st advance in knowledge: Half of MD population presented with Saccular Hydrops.»
Transient endolymphatic hydrops after an attack of vestibular migraine: a longitudinal single case study
Valerie Kirsch et al.
2018
Endolymphatic hydrops: a common finding in vestibular migraine

Kirsch V (en cours de publication)

61% MV et 100% des M Méniere définies
Pattern différent en prévalence, design et symétrie et degré moyen
Physiopathology of Migraine

Journal international de médecine
Migraine pathophysiology

- Involves the trigeminothalamic vascular (TVS) insuring connection between:
  - Sensitive and nociceptive innervation of meningeal vessels on the surface of the cortex
  - Pain central network.
- Includes:
  - Afferents passing through the 1st neurone of the V and 2 first dorsal horns of the spinal cord
  - A cerebral entry point: trigemino-cervical complex that brings together the V and 2 first dorsal horns

Thèse de Cedric GOLLON 2017

Thèse: Locus coeruleus activity and cerebral auto-regulation in the migraine with aura: multimodal study in MRI and DTC.
Pathophysiology MV

- Remains unknown
- Implies STV and based on migraine’s
- DCE is considered as one of the potential pathophysiological explanation for short duration crisis.
- Neuropeptides CGRP, serotonin, noradrenaline implied in the central vestibular system and the vestibular nerve? Could explain the inner ear dysfunction.
- Serotonin: extravasation of plasma and liquids of the inner ear in the rat?
- Genetic deficit non-demonstrated but suspected to be correlated with family periodic ataxias (gene CACNA1A)
- Painful *Stim* of the V, provoks a nystagmus in MV patients

→ *Currently considered as a pathophysiological connection between the migraine system and the vestibular system via the STV?*
MV treatment: great principals

1/ **Explain** the pathophysiology in simple words and **reassure the patient**

2/ **Adapt medicine treatments** at the frequency, intensity and crisis symptoms;
   - intense and rare: Symptomatic treatment (anti dizziness, antiemetic, AINS)
   - frequent: Prophylactic treatment, taking into account comorbidities (asthma, HTA or hTA,, weight, anxiety, depression) and invalidating characteristics of the symptoms.
   - Entanglement MV and MM

3/ **Stress, sleep disorders and lifestyle**, avoid triggers, regular exercise advised ,relaxation....
Traitement prophylactique de 1 ère intention

1/ Calcium antagonist:
   - Flunarizine 5 à 10mg ad bedtime  AE/ weight gain and drowsiness.
     Attention to extra pyramidal signs and depression (if previous state) Priority to short periods
   - Verapamil 120mg  CI/ hypotension,IC (bilan cardio) AE/ oedèmes MI

2/ Betablockers :
   - Propanolol 40 mg /80 mgLP ou Métoprolol
     CI/ hypotension,bronchospasme,bradycardie
Anti-épileptiques

- Molécule : Topiramate 150 à 200mg
  - Indication : si céphalées plus invalidantes que les vertiges
  - Propriétés : bloque canaux sodiques, bloque l’effet excitateur du glutamate au niveau Récepteur AMPA, potentialise activité GABAergique, inhibiteur anhydrase carbonique

- **Cl**: dépression, insuffisance rénale, FdR, lithiase rénale,
- **El**: somnolence, confusion, tb concentration, cytolyse hépatique, amaigrissement
Anti-dépresseurs

- Indication: associée à tb sommeil, anxiété, stress
- Molécules= tricycliques, Amitriptyline (4-10 gtttes le soir)
- CI: glaucome angle fermé, < 25 ans, tb psy, risque de rétention urinaire aigue

**Diamox** aurait action positive en cas de

- Formes familiales
- Ataxie périodique type 1
TTT non pharmacologiques

- Rééducation vestibulaire douce ++++
  - Peur par anticipation, perte de confiance, évitement de la marche
  - Troubles visuels associés
  - Cinétose

- Règles hygiéno-diététiques
  - Diète, Hygiène de sommeil
  - Relaxation, TCC, sophrologie
  - Éviter déclencheurs migraine
Conclusion

➢ Very frequent cause of dizziness
➢ Clinical history is the key of diagnosis
➢ Central vestibular signs are frequent between crisis
➢ Positional nystagmus is the most classical presentation on examination findings
➢ Tests findings are poor specificity
➢ Place of l’IRM protocole hydrops would be interesting for differential diagnosis
➢ Meniere’s disease and Vestibular Migraine can exist and it’s not rare.
➢ Treatments are fonction of frequency of crisis, of prevalence of vertigo or headache and of the impact of quality of life keeping in mind possible adverse events and contre indications.
Thanks for your attention
Epidémiology

- Cause la + fréquence chez l’adulte après le VPPB
- Prévalence variable
  - ORL: 4,3% à 29%
  - Clinique des vertiges: 6 à 25%
  - Clinique céphalées: 9 à 12%
- Prévalence sur 1 an des femmes 40 – 54 ans: 5%
- 50% des formes MV probable évoluent vers forme de MV
20 families with vestibular migraine of autosomal dominant inheritance

**Linkage analysis:**

Genetically heterogenous,
Subgroup showed association with 22q12 (LOD Score 4.02)

Lee et al. 2006

1 family, with 10/23 affected by VM,
Locus maps to 5q35 (LOD score 4.2)

Bahmad et al. 2009