Neuro-otologic Findings In Acute Migrainous Vertigo

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Introduction

• Patients with both episodic vertigo and migraine have been reported from the early days of clinical neurology (quoted by Kayan and Hood, 1984).

• However, systematic studies on the interrelations of migraine and vertigo have been undertaken in the last two decades only (Neuhauser and Lempert, 2004).
The association of migraine with vertigo is assigned to one of three categories: (Reploeg and Goebel, 2002)

(i) vertigo that is causally related to migraine (migrainous vertigo, MV).

(ii) vertigo syndromes that are not caused by migraine but show a statistical association with migraine as Meniere’s disease.

(iii) vertigo that coexists with migraine in the same patient just by coincidence.

Migrainous vertigo (MV) presents with attacks of spontaneous or positional vertigo lasting seconds to days in association with migrainous symptoms during the attack (Neuhauser and Lempert, 2004).

MV is presently not included in the International Headache Society classification of migraine.
Neuhauser et al. (2001) defined two separate diagnostic categories for MV:
- **Definite MV**
- **Probable MV**

**Definite MV**

A. Recurrent episodic vestibular symptoms at least moderate of severity

B. Current or previous history of migraine according to the criteria of the IHS

C. One of the following migrainous symptoms during at least two vertiginous attacks: headache, photophobia, phonophobia, visual or other auras

D. Other causes ruled out

**Probable MV**

One of the following:
1) Current or previous history of migraine according to the criteria of the IHS
2) Migrainous symptoms during ≥2 attacks of vertigo
3) Migraine-precipitants before vertigo in > 50% of attacks
4) Response to migraine ttt in > 50% of attacks

C. Other causes ruled out
• MV is a common disorder with a lifetime prevalence of about 1% (Neuhauser, 2007).

• Prevalence of MV in specialized dizziness and migraine clinics (7–9%) (Neuhauser et al., 2001; 2006).

Unfortunately, the pathophysiology of MV is still a matter of speculation.

• In any paroxysmal disorder, the physical examination during the acute episode is indispensable for an understanding of the underlying pathophysiology.

• Reports on clinical findings in patients with MV during the acute episode are scarce (Moretti et al., 1980; Lempert et al., 1993; Dieterich and Brandt, 1999; von Brevern et al., 2005).
The aim of this prospective study was to record and describe the spectrum of clinical findings during acute MV and to clarify which structures of the vestibular system are involved.
Methodology

Subjects:

1. Study group
   30 adult
   Subjects with Definite MV
   in acute symptomatic phase

Symptoms of at least 2 hours
Duration

Acute phase
Symptom free interval
(After 7-10 days)
Subjects:

1. Study group
   30 subjects with Acute Definite MV
   Mean age = 32 ± 9 years

2. Control group
   15 subjects with Acute migraine + no Life time vertigo
   Mean age = 34 ± 8 years

Methodology:

- Patients were recruited from the neurology clinic, vestibular clinic, emergency Dept.
- Patients were collected over one year period
- Patients were subjected to neuro-otologic examination by a neurologist and an audiologist
- All patients were still symptomatic when arrived for both examinations
Methodology

1. **Full neurological evaluation**

2. **Brain imaging studies**

3. **Audio-vestibular evaluation**

   - **Acute phase** '1,2,3'
   - **Symptom free interval** (After 7-10 days) '1,3'

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**Methodology**

1. **Neurological Work up**
   - Semi-structured interview for diagnosis of migraine according IHS classification
   - Thorough neurological examination
   - **Cerebellum:** The finger-nose test, heel-shin test & diadochokinesis
   - **Gait evaluation**
2. Brain imaging studies
   • Magnetic Resonance Imaging MRI
   • Magnetic Resonance Angiography MRA

FOR:
1. To exclude central neurological problem
2. Assessment of the arterial system

3. Audio-vestibular work up
   • Detailed vertigo history
   • Otological examination
   • Hearing evaluation (PTA, speech audiometry)
   • Vestibular test battery:
     - Clinical office vestibular tests
     - Standard VNG test battery including Bithermal caloric test
Clinical office vestibular tests

1. Head Impulse Test
   ?? findings

2. Head Shake Test
   ?? findings

3. Fukuda stepping test
   ?? findings

Standard VNG test battery

- Spontaneous nystagmus
- Oculomotor tests:
  - Gaze evoked nystagmus
  - Saccade
  - Tracking (Pursuit)
  - Optokinetic
- Dix-Hallpike (positioning) test
- Positional tests
- Water caloric test
In the current study

- MV was common in middle aged females with history of migraine without aura.
- The presence of associated migrainous symptoms during attacks are of diagnostic importance, however they need to be specifically inquired.
- Neuro-otologic manifestations of acute MV were heterogeneous.
- 80% of MV patients presented with pathologic positional nystagmus in the acute symptomatic phase.
In the current study

• Migraine should not longer be considered a central disorder with occasional peripheral signs, rather the underlying processes seem to affect both the brain and the inner ear.

MV

WHY & HOW????
Pathophysiology

1. Central disturbance of electrical activity: in the brain stem including the dorsolateral pons (affecting central vestibular neuronal activity).

Pathophysiology

2. Trigeminovascular hypothesis:

- In the migraine course, the trigeminal efferent nerve endings release inflammatory peptides such as substance P, Calcitonin gene related peptide into the dura producing neurogenic inflammation & pain.
- Vass et al. (2001) demonstrated that stimulation of the V nerve ganglion can cause vertebrobasilar and cochlear vascular permeability changes (peripheral, central, both).
Pathophysiology

3. Channelopathy: most promising

- Gene defect in encoding ion channels: CACNA1A, Na ATPase, ATP1A2, SCN1A genes.
- The brain and inner ear may share critical ion channels that are affected in migraine attacks leading to both central and peripheral symptoms and signs (Balogh, 1997; Esteve, 2006).

Pathophysiology

- 4. During migraine attack, activation of the rostral brainstem – locus coerules (the main central noradrenergic nucleus) & midbrain dorsal raphe (the main serotonin-containing nucleus in the brain stem was demonstrated (Weiller 1995, Bahra 2001).
- Vestibular nuclei receive projections from these loci. Activation of these structures in migraine might affect the central vestibular processing (Furman et al., 2003).
Thank You

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