Migraine associated dizziness

- Clinical Manifestation
  - Occurs with a peak around the third and fourth decades.
  - Vestibular manifestation can range from light-headiness to severe vertigo.
  - Vertigo last few seconds to several hours.
  - Patients are easily subject to motion sensitivity and movement disequilibrium outside of migraine attacks.
  - May be associated with mild and diffuse headaches to severe unilateral headaches.
  - Headaches do not always occur with vestibular symptoms.
  - Aura does not always occur with vestibular symptoms.
  - Aura such as photophobia and phonophobia may be associated with dizziness.
  - Fluctuating low frequency sensorineural hearing loss is more common with Meniere's disease.
  - Tinnitus is usually present which can be unilateral or bilateral.

Migraine associated dizziness

- Not included in the International Headache Society or American Academy of Otolaryngology classification.
- Neuhauser proposed criteria for migrainous vertigo

Neuhauser Criteria

Definite migrainous vertigo:

A. Recurrent episodic vestibular symptoms of at least moderate severity.
B. Current or previous history of migraine according to the criteria of the International Headache Society.
C. One of the following migrainous symptoms during at least two vertiginous attacks: migraine headache, photophobia, phonophobia, visual or other auras.
D. Other causes ruled out by appropriate investigations.

Neuhauser Criteria

Probable migrainous vertigo:

A. Recurrent episodic vestibular symptoms of at least moderate severity.
B. One of the following:
    1. Current or previous history of migraine according to the criteria of the International Headache Society.
    2. Migrainous symptoms during greater than 2 attacks of vertigo
    3. Migraine-precipitants before vertigo in more than 50% of attacks: food triggers, sleep irregularities, hormonal changes.
    4. Response to migraine medication in more than 50% of attacks.
C. Other causes ruled out by appropriate investigations.

Audiometric testing

- Battista Study
  - Compared audiometrics with Meniere's disease and Migraine associated dizziness patients.
    - Migraine associated dizziness rarely demonstrates progressive sensorineural hearing loss.
    - 80% of basilar migraine patients will demonstrate fluctuating low frequency sensorineural hearing loss similar to Meniere's disease patients.
    - Meniere disease will over time demonstrate fluctuating and progressive sensorieneural hearing loss.

Battista R. Audiometric findings of patients with migraine-associated dizziness. Otology & Neurotology 2004; 25:987-992.

Vestibular testing

- Cass Study
  - Compared vestibular testing with Meniere's disease and Migraine associated dizziness patients.
    - Spontaneous and positional nystagmus are not diagnostic for Meniere's disease or migraine associated dizziness.
    - Ocular motor abnormalities with pursuit tracking or optokinetic testing abnormalities have slightly increased prevalence in migraine associated dizziness. This indicates central vestibular involvement with the cerebellum and brainstem.
    - Caloric testing demonstrates reduced vestibular response in approximately 18-20% of migraine associated dizziness patients. Findings for Meniere's patients will vary depending on the early or late stages of their disease.
    - Rotational chair asymmetry is the most frequent finding for migraine associated dizziness.

Cass et al. Migraine-related vestibulopathy. Ann Otol Rhinol Laryngol. 1997; 106:182-189.

Meniere's disease Pathophysiology

- Endolymphatic hydrops
  - There is distension of the membranous labyrinth due to excessive endolymphatic fluid.
    - Due to impaired resorption of endolymph in the endolymphatic duct and sac.
    - Histopathologic evidence of reduced vascularization and fibrosis in the perisaccular tissue which shows the reduction of resorptive capacity of the endolymphatic sac. Additionally there has been accumulation of proteinaceous debris in the lumen.[1]
    - Possible obstruction of the endolymphatic duct.[2]
    - Possible overproduction of endolymph by stria vascularis in the cochlea or dark cells in the labyrinth.[3]
- Reisner's membrane
  - Schuknecht discovered through histopathologic examination that there was bowing and rupture of Reisner's membrane in the pars inferior portion of the inner ear.[4]

1. Fukuda et al., The development of endolymphatic hydrops following CMV inoculation of the endolymphatic sac. 1988 Laryngoscope 98:439.

2. Franklin et al. Meniere's symptoms resullting from bilateral otosclerosis occlusion of the endolymphatic duct: an analysis of the causual relationship between otosclerosis and Meniere's disease. 1990 J Otol. 11(2):135.

3. Henriksson et al. Pathophysiology of Meniere's disease. 1986.Controversial aspects Meniere's disease. Georg. Thieme. Verlag.

4. Schuknecht, Pathology of the ear, Boston, 1974 Harvard University

Migraine Pathophysiology

- Vascular theory (Wolff Hypothesis 1963)
  - Initially there is vasoconstriction during the prodromal phase of migraine followed by vasodilatation in the headache phase.
  - Neurotransmitter release
    - Serotonin, histamine, catecholamine, and prostaglandins have been implicated as part of the cause of migraines.
    - Medication such as calcium channel blockers may reduce migraine symptoms.
  - Autonomic nervous system
    - Cerebral vessels are richly innervated with norepinephrine-containing neurons whose cell bodies are located in the brainstem, superior cervical ganglia, the locus coeruleus; these have connection to the hypothalamus.
    - Activation of alpha-adrenergic receptors results in vasoconstriction and decreases cerebral blood flow, while beta-adrenergic receptor stimulation leads to vasodilatation and increases cerebral blood flow.
    - Appenzeller postulated that migraine patients demonstrate abnormal vascular responsiveness manifested as vasoconstriction, as a result of an abnormal adrenergic drive , abnormal hypersensitivity of adreno-receptors, or some other sympathetic system abnormalities.[1]
    - Medications such as beta-blockers may reduce migraine symptoms.

1. Appenzeller, et al., Clinical and experimental studies in migraines. Res. Clin Stud. Headaache, 1978; 6:160-166.

Migraine Pathophysiology

- Vascular theory
  - Prostaglandins produced by the cyclo-oxygenase pathway
    - Can cause vasoconstriction or vasodilatation associated with aura and headache.
    - Increases the effects of bradykinin and other chemically induced pain transmitters by sensitizing pain receptors.
    - Mediates inflammatory response
    - Modulates neurotransmitters release from nerve terminals.
    - Medication such as NSAIDS may reduce symptoms of migraines.

Migraine Pathophysiology

- Neurological theory
  - Primary dysfunction of the brain that leads to secondary vascular abnormalities.
  - Described by Leao: First a wave of short increased cortical or brainstem neuronal activity followed by a wave of depression of the cortical neuronal activity.
  - Potassium ions and the neurotransmitter glutamate enhance neuronal excitation, coupled with firing in a localized region of the cortex which results in the accumulation of potassium ions in the extracellular space. The increased extracellular potassium results in depolarization of the adjacent inactive neurons concentration which plays a crucial role in creating and maintaining central nervous system depression.
  - The potassium ion concentration cannot be rebalanced by reuptake, diffusion or transportation away from the active site by the glial potassium buffering system.
  - Glutamate is released by the depolarization which also increases the intracellular calcium during the spreading wave of depression.
  - Neural theory suggests that the instability created by the ionic and neurotransmitter disturbance causes the release of vasoactive amines, serotonin, bradykinin, histamine and substance P that have a role in the vasodilation.

Migraine Pathophysiology

- Intracranial vascular vessels, venous sinuses and dura mater derive innervations from the ophthalmic division of the trigeminal ganglion.
- Contributions also occur from upper cervical dorsal roots.
- Involves of the caudal brainstem and high cervical spinal cord extending into the thalamus.

Migraine and Meniere's disease A Common Pathophysiology

- Vestibulocortical system includes the vestibular nuclei, the thalamus, and the cerebral cortex.
- Vestibulothalamic tracts originate from neurons in the superior and lateral vestibular nuclei and ascends to the thalamus through a dominant anterior tract and smaller posterior tract.
- Vestibulocortical tracts proceed to the parieto-insular vestibular cortex.
- Thalamic and cortical regions that involve the vestibulothalamocortical tracts integrate proprioceptive, vestibular, and visual stimulations to provide a conscious awareness or "body oriented map" of orientation.
- Neurotransmitters (calcitonin-gene related peptide, serotonin, noradrenaline, dopamine) are know to modulate the activity of vestibular neural pathways.
- Migraine associated dizziness may be considered both a peripheral vestibular and central nervous system disorder.

Migraine associated dizziness

- Treatment:
  - Determine if patient has Meniere's disease or migraine associated dizziness.
  - Differentiation between Meniere's disease and migraine associated dizziness is important because the treatments for each condition are different.
  - Follow-up with additional history, audiograms and effectiveness of treatment is essential.

Meniere's disease

- Medical treatment:
  - Diet: 2000mg low sodium diet, caffeine reduction to 50-75mg per day, alcohol and nicotine restriction.
  - Weather changes
  - Meniere's log
  - Diuretics: Maxzide, HCTZ, Lasix, Diamox
  - Vasodilators: Histamine (Serc), Papaverine (Pavabid)
  - Calcium Channel Blocker: Verapamil
  - Steroids: Prednisone
- Emergency vertigo kit
  - Valium and Phenergan

Meniere's disease

- Surgical treatment:
  - Intratympanic gentamicin injections
  - Intratympanic dexamethasone infusions
  - Endolymphatic sac shunt
  - Vestibular nerve section
  - Labyrinthectomy

Migraine

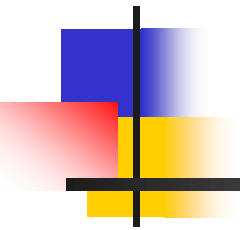
- Diet:
  - Avoid aged, canned, cured or processed meats, including bologna, game ham, herring, hot dogs, pepperoni, and sausage, aged cheese, meat tenderizer, monosodium glutamate, aspartame, chocolate, coffee, nuts, peanut butter, red wine, avocados, beans, brewer's yeast, raisins, pickles, figs, lentils, canned soups, sauerkraut, seasoned salts, snow peas, red plums, papaya, pickled, preserved or marinated foods i.e. olives and cultured dairy products i.e. buttermilk and sour cream.
- Weather changes, menstrual cycle, pregnancy, eating disorders fasting or binges, sleep disorders
- Migraine log
- Symptomatic treatment:
  - Over the counter medication (OTC) analgesics such as: acetaminophen, aspirin, ibuprofen, and naproxen.
  - Antimetic: Phenergan
  - Sympathomimetic vasoconstrictor
    - Isometheptene, dichloraphenazone, and acetaminophen (Midrin)
    - Aspirin, caffeine, and butalbital (Fiorinal)

Migraine

- Abortive treatment:
  - Serotoninergic vasoconstrictors
    - Cafergot
    - Dihydroergotamine
    - Triptans
  - Preventive treatment:
    - Tricyclics: Amitriptyline, Doxepin, Nortriptyline
    - Beta blockers: Atenolol, Propanolol
    - Calcium channel blockers: Verapamil
    - Anticonvulsants: Tegretol, Neurontin, Topamax

Migraine associated dizziness versus Meniere's disease

Symptoms	Meniere's	Migraine (MAD)
Gender	equal	mostly females
Hearing loss	fluctuate, progressive, low frequency	fluctuate, low frequency
Family Hx	10-20%	60-70%
Age onset	variable	teenage to 20 years
Headaches	variable	5 or more attacks
Episodic	always	always
Diet triggers	salt, caffeine, MSG	preservatives, MSG, red wine, cheese, etc.
Vertigo	almost always	dizzy to vertigo



THE END

QUESTIONS ?

Hearing & Balance Center

- Making the World Sound Good.
- Maintaining Balance and Equilibrium.