Global Year Against HEADACHE
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Migraine

Clinical Picture

What Symptoms Occur During a Migraine Attack?

- Migraineurs have recurrent, severe, and disabling attacks of headache, often unilateral and pulsating, along with symptoms of sensory disturbance, such as light, sound, and odor sensitivity. Nausea and neck stiffness are other common symptoms, and symptoms can be aggravated by movement.
- Some patients experience dizziness during attacks.
- About 20–30% of patients experience aura and neurological symptoms (e.g., visual disturbances), which usually precede the headache phase of an attack.
- Premonitory symptoms such as yawning, irritability, tiredness, cravings, and difficulty concentrating sometimes precede headache onset.

What Is Migraine Aura, and What Symptoms Can Occur?

- An aura is any neurological symptom that occurs shortly before the headache attack. Visual symptoms (e.g., flickering lights or zigzag phenomena), somatosensory symptoms (e.g., paresthesias), speech problems, and rarely, motor symptoms can occur during aura.
- Symptoms usually last >5 and <60 minutes.
- Before migraine can be diagnosed, other possible neurological deficits must first be excluded.
- Cortical spreading depression (see below) is thought to be the pathophysiological cause.

What Can Trigger a Migraine?

- Menstruation
- Shortness of sleep, irregular sleep, or too much sleep
- Stress (or in some patients, relaxation from stress)
- Alcohol (e.g., red wine)
- Caffeine (e.g., coffee, chocolate)
- Foods containing glutamate or aspartame
- Dehydration
- Vasodilating drugs (e.g., nitrates)

Epidemiology

How Many People Are Affected by Migraine?

- Women: about 13–18% of the population
- Men: about 5–10% of the population
- Numbers may be lower in Asian populations

Chronic Migraine

About 4% of the adult population experiences chronic headache, i.e., headache on 15 or more days a month. About half of this group has chronic migraine, and the other half has chronic tension-type headache [7].

Disorders/Abnormalities That Can Be Comorbid with Migraine

- Anxiety
- Depression
- Fibromyalgia

- Back pain
- Hypertension
- Stroke and cardiac disease
- Childhood vomiting

Pathophysiology

Migraine Pathophysiology

Migraine was considered to be vascular in origin for much of the 20th century [6], but today it is considered to be a disorder of the brain, with abnormalities in the vasculature occurring secondary to the primary neuronal events. Migraine has a strong inherited component, and a large genetic study suggests the involvement of glutamate pathways in migraine pathogenesis [1]. Genetic variations on chromosome 19 have been reported for rare forms of familial hemiplegic migraine (FHM1 and 2).

Migraine Aura Pathophysiology

Cortical spreading depression (CSD), a process transiently compromising cortical function at a speed of about 3 mm per minute [5] is thought to be the underlying mechanism of aura [2]. It is possible that CSD is also implicated in the generation of migraine headache.

Definition/Diagnosis/Differential Diagnosis

The International Classification of Headache Disorders

- Distinguishes primary headache syndromes from secondary, symptomatic headache diseases. Migraine and tension-type headache are the most common types of primary headache. The classification of primary headache disorders is based on a phenomenological (operational) categorization. In contrast, secondary headaches, which have to be ruled out, are classified according to their etiology (e.g., headache attributed to a tumor or a vascular abnormality). Some types of migraine can be classified based on genetic markers (familial hemiplegic migraine).
- Defines episodic migraine as migraine attacks occurring on fewer than 15 days a month and chronic migraine as attacks occurring on 15 or more days a month.
- Other potential causes of headache such as intracranial/spinal pathology, musculoskeletal pathology, inflammatory/autoimmune diseases, systemic illnesses, or drug-related headaches have to be ruled out by clinical history, neurological examination, and additional diagnostic tests, when appropriate, in order to diagnose migraine.

Migraine: Diagnostic Criteria (ICHD-2) [4]

A. At least 5 attacks fulfilling criteria B-D
B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated)
C. Headache has at least 2 of the following characteristics:
    - unilateral location
    - pulsating quality
    - moderate or severe pain intensity
    - aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D. During headache at least 1 of the following:
    - nausea and/or vomiting
    - photophobia and phonophobia
E. Not attributed to another disorder

Therapy

How Can Migraine Attacks Be Treated?

- Simple analgesics (e.g., aspirin, acetaminophen/paracetamol)
- NSAIDs (e.g., naproxen, ibuprofen, diclofenac)
- Triptans (e.g., sumatriptan)
- Second choice treatment: ergot derivatives (e.g., dihydroergotamine)
- In the future, CGRP-receptor antagonists such as telcagepant may be a new option for the treatment of acute attacks [3].
- Compound analgesics, such as those containing aspirin, acetaminophen/paracetamol, and caffeine have been shown to be more effective than single analgesics. However, they are thought to increase the risk of medication overuse headache (see below).

What to Do When Nausea Is an Issue

- Antiemetics/prokinetics, such as domperidone or metoclopramide, can improve nausea.
- These medications are sometimes given before the analgesics to improve absorption of the medication.

Preventive Pharmacological Therapy in Episodic Migraine

- Preventive therapy is suitable for patients with frequent and/or severe, disabling attacks.
- A large number of different medications with differing mechanisms of action have been tested in randomized placebo-controlled trials and are available for treatment. Propranolol, topiramate, valproate, flunarizine, and amitriptyline are examples of medications that are frequently used and have proven efficacy [3].
- On average, a 50% decrease in headache frequency can be expected for half the patients using one preventive medication.

Preventive Pharmacological Therapy in Chronic Migraine

- Best scientific evidence exists for use of topiramate or Botulinum toxin injections for pharmacological prevention of chronic migraine.
- Specialist treatment is useful in patients with chronic migraine or refractory migraine.

Medication Overuse

- Is defined as consumption of triptans, ergotamine, opioids, or combination analgesics on 10 days or more per month, or simple analgesics on 15 days or more a month.
- Is an important issue in migraine therapy and needs to be identified and managed.
- Can produce rebound headache.
- Can reduce the efficacy of preventive therapy.
- Can mask the headache phenotype.

Nonpharmacological Management of Migraine

- Patient education
- Identify and avoid triggers
- Maintain regular daily activities
- Maintain a regular sleep/wake cycle
- Aerobic exercise
- Consider weight reduction in overweight patients
- Biofeedback
- Acupuncture
- Relaxation techniques
- Cognitive-behavioral therapy
- There is currently no evidence supporting homeopathy for migraine treatment
- There is no convincing evidence regarding efficacy of closure of patent foramen ovale (PFO) for migraine treatment. PFO closure should not be performed outside of clinical trials.
- “Migraine surgery,” such as surgical cauterization of the superficial blood vessels of the scalp or the removal of muscles or nerves thought to be “trigger sites,“ is advocated by some plastic surgeons, but there is poor scientific evidence and no convincing rationale to support this measure. “Migraine surgery” is currently not recommended outside of clinical trials.

Neuromodulation Approaches

- The efficacy of migraine therapy using electrostimulation of peripheral nerves is currently being investigated in studies (see the fact sheet on Neuromodulation in Primary Headaches). Results are pending.

References

[1] Anttila V, Stefansson H, Kallela M, et al. Genome-wide association study of migraine implicates a common susceptibility variant on 8q22.1. Nat Genet 2010;42:869–73.

[2] Dalkara T, Nozari A, Moskowitz MA. Migraine aura pathophysiology: the role of blood vessels and microembolisation. Lancet Neurol 2010;9:309–17.

[3] Goadsby PJ, Sprenger T. Current practice and future directions in the prevention and acute management of migraine. Lancet Neurol 2010;9:285–98.

[4] Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders, 2nd edition. Cephalalgia 2004;24(Suppl 1):9–160.

[5] Lashley K. Patterns of cerebral integration indicated by the scotomas of migraine. Arch Neurol Psychiatry 1941;46:331–9.

[6] Moskowitz MA, Buzzi MG, Sakas DE, Linnik MD. Pain mechanisms underlying vascular headaches: progress report 1989. Rev Neurol (Paris) 1989;145:181–93.

[7] Scher AI, Stewart WF, Lipton RB. Epidemiology of chronic daily headache. In: Goadsby PJ, Silbersten SD, Dodick DW, editors. Chronic daily headache for clinicians. Ontario: BC Decker; 2005. p. 3–11.