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Migraine in adults: A head start

BY VINCENT M. VACCA, JR., MSN, RN

Abstract: Migraine is a chronic episodic disorder typically characterized by a debilitating headache. This article offers tools to identify potential triggers and prevent or reduce the occurrence of attacks.

Keywords: aura, headache, migraine, postdrome, prodrome

SEBASTIAN KAULITZKI, FREEDA / SHUTTERSTOCK

MIGRAINE IS A SEVERE, disabling, episodic disorder reported to affect more than one billion people around the world annually.[1] Frequently described as moderate-to-severely intense, throbbing, unilateral headaches that can last from 4 to 72 hours, migraine can also be associated with nausea, vomiting, photophobia, and phonophobia. Each of these can worsen with physical activity.[1-3]

Migraine is a chronic disorder that presents significant challenges due to its unpredictable onset, episodic pattern, intensity, and duration.[1,2] This article offers insight to help patients identify and understand migraine, as well as nonpharmacologic and pharmacologic therapies to manage them.

Epidemiology

According to the World Health Organization, migraine is the sixth most disabling health disorder worldwide.[1,2] The highest prevalence of migraine occurs in adults ages 30 to 39. A 2016 report from the American Association of Neuroscience Nurses categorized migraine as the third highest cause of disability globally for both males and females under age 50.[3,4] Migraine can lead to significant disability and socioeconomic impact, with a combination of lost productivity and direct medical costs leading to an estimated annual cost of $19.6 billion in the US and €27 billion in the European Union.[1,5]

Although migraine can begin at any age, onset typically occurs during early to mid-adolescence. In the US, approximately 12% of individuals age 12 and older are affected.[1] Before puberty, the annual incidence of migraine attacks between males and females is similar at approximately 4%; after puberty, the rates increase to 18% in females and just

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6% in males, resulting in a 3:1 ratio.[6] Many who experience migraine attacks at a younger age continue to experience them into adulthood.[7]

Pathophysiology

The associated signs and symptoms of migraine result from disordered sensory processing in the central nervous system. Although brain tissue is devoid of pain sensing or nociceptive nerve fibers, they innervate surrounding brain structures such as the meninges, especially the dura mater and cerebral blood vessels. When central nervous system nociceptive fibers are exposed to mechanical, chemical, and/or electrical stimulation, the signals cause migraine pain and associated symptoms.[1]

Cortical spreading depression (CSD) is an alteration of brain activity that consists of brief excitation followed by prolonged depression. It also initiates the headache phase by activating the nociceptive pathways and providing sensory innervation to the meninges, specifically the dura mater. CSD is thought to be associated with, or cause, the aura preceding migraine.[8,9]

Genetic components

Migraine is often hereditary, and researchers have identified the KCNK18 and CSNK1D genes as potential contributors to the disorder, among other factors. Common migraine types may be caused by more complex genetic disorders, however, in which multiple genetic or environmental characteristics play a role.

A strong genetic component in migraine has been observed in clinical practice reports and supported by population-based family studies. These studies demonstrate that immediate family members of individuals who experience migraine have a substantially increased risk of suffering from migraine.[10] As such, identifying the corresponding genetic components is vital to the management of migraine.[4]

Although migraine can begin at any age, onset typically occurs during early to mid-adolescence.

Clinical manifestations

A typical migraine attack progresses through four phases: the prodrome, the aura, the headache, and the postdrome.[1] The ***migraine prodrome*** occurs in most patients who experience migraine, precedes a migraine attack, and signals that migraine is on the way.[10,11] Although the prodrome is not always recognized as signaling an impending migraine attack, it consists of affective or vegetative symptoms that appear 24 to 48 hours before headache onset. Frequently reported prodromal symptoms include increased yawning, euphoria, depression, irritability, food cravings, constipation, and neck stiffness.[4]

About 25% of people with migraine experience one or more focal neurologic symptoms in the second phase, called the ***migraine aura***.[4] The migraine aura may occur simultaneously, independently, or in the absence of a headache.[10,12] Auras are most often visual, but can also be sensory, speech, or motor disturbances. They are typically characterized by gradual development, a duration of no longer than 1 hour, a mix of positive and negative features, and complete reversibility.

Positive features indicate active discharge from central nervous system neurons. Typical positive symptoms can be visual (such as bright lines, shapes, objects), auditory (such as tinnitus, noises, music), somatosensory (such as burning, pain, paresthesia), or motor (such as jerking or repetitive rhythmic movements). Negative features indicate an absence or loss of function, such as loss of vision, hearing, feeling, or ability to move a part of the body.[4]

The ***headache of migraine*** is often but not always unilateral and tends to have a throbbing or pulsatile quality, especially as the intensity increases.[4] As the attack severity increases over the course of one to several hours, patients may experience nausea and sometimes vomiting. Many report photophobia or phonophobia during attacks, leading them to seek relief by lying down in a darkened, quiet room. Other migrainous features such as osmophobia and cutaneous allodynia may occur during attacks.

Additional symptoms may follow the headache and persist up to 48 hours. Common symptoms associated with migraine include:[13]

- cognitive dysfunction
- sensory hypersensitivity
- visual disturbances
- facial or sinus pressure
- temperature dysregulation
- muscle stiffness or spasms
- vertigo
- imbalance

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• psychological and/or emotional disturbances.

Once the spontaneous throbbing of the headache resolves, the patient may experience a ***migraine postdrome***, during which sudden head movement transiently causes pain in the location of the antecedent headache. During the postdrome, most patients feel drained or exhausted, although some report a feeling of mild elation or euphoria.[4]

Select migraine syndromes

In the International Classification of Headache Disorders, 3rd edition (ICHD-III), the International Headache Society identifies the following migraine syndromes.[3,14]

Migraine without aura is most commonly characterized by a unilateral frontotemporal headache, but may occur in any region of the brain. This migraine is typically described as throbbing or pulsating. In younger patients, it is often bilateral and associated with nausea, photophobia, and phonophobia, each of which is aggravated by physical activity.[3,12]

Up to 80% of those who experience migraine without an aura report various symptoms, such as tiredness, irritability, reduced concentration, and yawning. These can occur from hours to days before the onset of the headache.[6]

Migraine with aura occurs in approximately one-third of individuals who experience migraine attacks.[1,2] The ICHD-III defines migraine aura as one or more transient, fully reversible neurologic deficits of unilateral localization, which develop over 5 minutes or longer and last up to an hour.[1] As previously discussed, auras may feature positive and/or negative features and typically appear 30 to 60 minutes before the headache.[1,3,4] Reversible visual disturbances include blind (scotoma) or bright and flashing (scintillating) spots in the center of the visual field.[1,3]

Common migraine triggers[1,10,24]

Behavioral	sleep deficit, excessive sleep, stress
Dietary	cheese, wine, chocolate, citrus, caffeine, carbonated drinks, food colorings and flavorings, prolonged fasting
Environmental	noise, odors, bright or flickering lights
Medications	oral contraceptives, menopausal hormone therapy

Hemiplegic migraine is characterized by significant motor weakness but not paralysis as a manifestation of aura in at least some attacks. Motor weakness associated with hemiplegic migraine typically lasts less than 72 hours but may persist for weeks in some patients.

Retinal migraine is characterized by repeated monocular visual disturbances, such as scintillating or bright flashing lines and blind spots. These symptoms are associated with the classic unilateral frontotemporal headache.[3]

Menstrual migraine is named for the correlation between the menstrual cycle and migraine occurrence. Menstrual migraine syndrome is typically long-lasting, without an aura, and associated with severe nausea and increased pain compared with nonmenstrual migraine attacks.[3,15]

Precipitating and exacerbating factors

Migraine has been linked to many environmental, pharmaceutical, behavioral, and hormonal triggers (see *Common migraine triggers*).[10] For example, exposure to certain common and benign sensory stimuli, such as light, sound, touch, and smell, is interpreted as abnormally intense and noxious in patients with migraine. Exposure to excessive sensory stimulation, such as flashing lights, may trigger a migraine attack.[5]

Nursing assessment

Nurses must obtain a patient's health history and perform a physical assessment. The history includes gathering information on the onset, location, duration, and characteristics of the migraine, as well as any associated, aggravating, and relieving factors. Additionally, all treatments and subsequent patient responses should be documented in the electronic health record. The physical assessment should include a focused neurologic assessment to identify abnormalities. Other relevant findings or contributing factors include:[4]

• cranial or cervical muscle tenderness
• Horner syndrome (relative miosis with 1 mm to 2 mm of ptosis on the same side as the headache)
• conjunctival injection
• tachycardia or bradycardia
• hyper- or hypotension
• hemisensory or hemiparetic neurologic deficits
• Adie-type pupils (poor light reactivity with light-near dissociation).

Nurses should also perform medication reconciliation, including any over-the-counter (OTC) medications, to ensure safety and prevent possible drug interactions with prescribed medications. Additionally, patients should be advised to limit their use of OTC medications as directed by both the manufacturer and the provider.[3]

Diagnosis

Migraine is a clinical diagnosis based on a patient's health history, physical assessment findings, and the criteria specified by ICHD-III (see *Diagnostic criteria for migraine*).[4,12] Typically, neuroimaging is unnecessary unless other risk factors

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or comorbidities are suspected, including nonacute headaches that do not meet migraine criteria, immune deficiency, abnormal neurologic findings, and potential subarachnoid hemorrhage.[4,16]

Certain "red-flag" signs and symptoms are consistent with migraine but may be associated with other serious neurologic disorders. For example, a sudden severe "worst headache of my life" could indicate a subarachnoid hemorrhage and should be evaluated to rule out cerebral aneurysm. A headache associated with a stiff neck and fever could signify meningitis. Headache associated with severe or projectile vomiting could indicate increased intracranial pressure. If the headache occurs later in life (beyond age 50), a brain tumor should be considered.[3]

Diagnostic neuroimaging should be considered if there are any cognitive changes or alterations in a standard neurologic examination, as these are not associated with migraine but with other neurologic disorders. Diagnostic neurologic monitoring, such as an electroencephalogram, could be indicated for headaches associated with seizures to rule out epilepsy. Neuroimaging may also be indicated for new daily headaches or those that are progressive in intensity. For patients with existing or suspected cancer, headaches may be associated with metastatic brain lesions and should be evaluated.[3]

Management

Daily lifestyle recommendations for migraine management include the avoidance of known triggers, including lack of sleep, emotional stress, strong odors, and skipped meals. Regular meals and exercise, adequate hydration, and proper sleep can help prevent or reduce migraine attacks. Hydration recommendations may vary slightly depending on age and activity level, but an average of 8 to 10 cups of noncaffeinated, nonalcoholic fluid per day and increased fluid intake with physical activity is ideal. Exercising 30 minutes or more three to five times a week is recommended, as is sleeping and waking around the same time on a consistent basis.[3,7,17]

Many patients report that they will go to bed, turn off the lights, and avoid any type of sensory stimulation from light, sound, touch, or smells to limit both the intensity and duration of migraine.[10] The use of ice packs over the forehead, side of the head, or eyes may be beneficial during migraine.[3,7,17]

The application of heat, cold, and massage may also be beneficial, especially with pain or muscle stiffness in the neck and/or upper back. Relaxation therapies, such as stretching, are designed to maintain tone and strength through low-impact activities. Patients may also consider meditation. A referral to a physical therapist or specialist, such as a chiropractor, may be appropriate for some patients.[3]

Psychological management of migraine has been evaluated. The most common psychotherapy for migraine is cognitive-behavioral therapy, which includes the following skills and techniques:[7,17]

- behavioral relaxation strategies, such as deep diaphragmatic breathing
- muscle relaxation
- meditation
- guided imagery.

Cognitive-behavioral therapy teaches patients how to manage pain, reduce the associated symptoms, and prevent migraine attacks.[7,17,18]

Diagnostic criteria for migraine

Migraine without aura

A. at least five attacks fulfilling criteria B through D
B. headache attacks lasting 4 to 72 hours (untreated or unsuccessfully treated)
C. headache has at least two of the following characteristics:
- unilateral location
- pulsating quality
- moderate or severe pain intensity
- aggravation by or causing avoidance of routine physical activity

D. during headache, at least one of the following:
- nausea, vomiting, or both
- photophobia and phonophobia

E. not more appropriate to another ICHD-III diagnosis

Migraine with aura

A. at least two attacks fulfilling criterion B and C
B. one or more of the following fully reversible aura symptoms:
- visual
- sensory
- speech and/or language
- motor
- brainstem
- retinal

C. at least three of the following six characteristics:
- at least one aura symptom spreads gradually over a period greater than or equal to 5 minutes
- two or more symptoms occur in succession
- each individual aura symptom lasts 5 to 60 minutes
- at least one aura symptom is unilateral
- at least one aura symptom is positive
- the aura is accompanied or followed within 60 minutes by headache

D. Not better accounted for by another ICHD-III diagnosis

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Noninvasive neuro-modulation is also a developing field in the management of migraine. This technique involves stimulating the nervous system with an electric current or fluctuating magnetic field to modulate pain transmission via pathways involved in migraine-associated headaches.[18]

Additionally, acupuncture is a treatment option for the reduction or prevention of migraine. Using the Cochrane database, a 2018 meta-analysis concluded that acupuncture is safe, effective, and without adverse effects in prophylaxis and treatment.[12]

Pharmacologic management

Both abortive and preventive medications are frequently used and recommended for migraine management across all ages.[19,20] The goals of these treatments include:

- reducing the frequency, severity, and duration of migraine
- improving the responsiveness to treatment of acute attacks
- reducing the level of disability during attacks
- maintaining and controlling cost of care for migraine management
- reducing the overuse of abortive and/or preventive medications.

Typically more effective when administered early in the course of the migraine, abortive medications, also known as symptomatic medications, are taken to reduce and stop symptoms.[19] A single large dose of abortive medication is more effective than repeated smaller doses.[19,20] Common abortive medications include acetaminophen, triptans, and nonsteroidal anti-inflammatory drugs such as ibuprofen and naproxen (see *Medication summary*).

Triptans inhibit the release of vasoactive peptides, promote vasoconstriction, and block pain pathways in the brainstem.[19] Despite excellent efficacy in the acute management of migraine, triptans do not prevent attacks. They are not effective for everyone, contraindicated in some, and can be complicated by headache recurrence in 15% to 40% of patients.[20] Currently, many triptans are available, but not all are approved for use in children.[5,21] They are also contraindicated in pregnant and breastfeeding patients, those with forms of hemiplegic migraine, and those age 65 or older.[5,20,21]

Certain "red-flag" signs and symptoms that are consistent with migraine may also be associated with serious neurologic conditions.

Due to their vasoconstrictive effects, triptans are also contraindicated in those with preexisting cardiovascular, cerebrovascular, or peripheral vascular disease.[14,22] Before providers prescribe a triptan, the FDA recommends that they evaluate for silent myocardial ischemia in patients with risk factors such as hypertension, dyslipidemia, smoking, obesity, and diabetes.[8,21]

Preventive medications are taken regularly between attacks to reduce the severity and/or frequency of migraine.[19] Multiple classes of medications are used for prevention and approved for all age groups. These include beta-blockers, antidepressants, antiepileptics, and calcium-channel blockers.[5,7,23] Cannabinoids are also a promising class of compounds, with antiepileptic, analgesic, antiemetic, and anti-inflammatory effects for the acute and prophylactic treatment of migraine symptoms.[10]

It may take several months before a decrease in the frequency and/or severity of migraine is achieved with preventive treatments. Preventive medications should be gradually tapered after a period of sustained

Medication summary[19,20]

Pharmacologic agents used for the treatment of migraine can be classified as abortive, which alleviate the acute phase, or prophylactic, which are preventive.

Abortive medications

- selective serotonin receptor (5-HT1) agonists (triptans)
- ergot alkaloids
- analgesics
- nonsteroidal anti-inflammatory drugs (NSAIDs)
- combination products
- antiemetics

Prophylactic medications

- antiepileptic drugs
- beta-blockers
- tricyclic antidepressants
- calcium channel blockers
- selective serotonin reuptake inhibitors
- NSAIDs
- serotonin antagonists
- botulinum toxin
- calcitonin gene-related peptide inhibitors

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benefit.[19,20] Due to the risk of rebound headaches and other adverse reactions, patients should not stop taking these medications suddenly.

Pharmacologic modulation of calcitonin-gene related peptide (CGRP) activity is a new and recently approved pharmacologic treatment for the prevention of acute migraine. CGRP is a vasoactive neuropeptide released in the brain that leads to inflammation and pain associated with migraine. CGRP antagonists inhibit vasodilation and inflammation by blocking the release of CGRP at all locations within the migraine pathway. CGRP monoclonal antibodies have been demonstrated as effective in migraine prevention. Currently, three CGRP monoclonal antibody medications have been approved for treatment of migraine in adults, with several more in development.[22]

Patient education

Nurses can teach patients to identify and recognize potential migraine triggers, including emotional factors, poor sleep hygiene, environmental factors, hormonal fluctuations, medication use and overuse, dietary factors, and alcohol, especially red wine. Nurses should encourage patients to keep a migraine diary and offer lifestyle strategies to reduce, limit, or avoid triggers (see *Migraine diary criteria*).

Both abortive and preventive medications are frequently recommended for migraine management.

Additionally, nurses can educate patients about the available pharmacologic interventions, including the safe and proper use of abortive, preventive, and OTC medications. Nurses should emphasize the importance of treating migraine early on, avoiding overuse of medications, and taking all medications as prescribed to achieve maximum benefit. Nurses should also warn patients not to discontinue medication unless properly guided by a healthcare provider.

Nurses should provide patients with educational handouts, brochures, and other resources as available.[13] Nurses should also support healthy sleep practices, appropriate nutrition and hydration, routine exercise, and stress management and relaxation techniques, such as cognitive-behavioral therapy.

Ongoing support

The clinical presentation of migraine is similar regardless of age, and individuals may benefit from a combination of pharmacologic and nonpharmacologic interventions.[7] Education and support from nurses can maximize patient control of migraine leading to prevention or decreased frequency, effective and safe pain management, desired lifestyle maintenance, and improved functionality in daily living. ■

Migraine diary criteria

As recommended by the healthcare provider, patients may consider tracking their migraine characteristics to identify migraine triggers, as well as the timing of and response to treatment in each attack. Key factors to document include:

- date
- exact times of the onset and ending
- potential triggers
- prodromal symptoms
- severity of headache on an intensity scale of 0 (no pain) to 10 (worst pain imaginable)
- location of the headache
- description of headache pain
- other relevant symptoms
- medication taken, including frequency, dose, and route
- pain relief on a scale of 0 to 10.

List adapted from: Hickey JV. *The Clinical Practice of Neurological and Neurosurgical Nursing*. 6th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott, Williams, and Wilkins; 2009.

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Vincent M. Vacca, Jr., is a member of the adjunct faculty at the University of Massachusetts in Boston, Mass.

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