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# Migraine variants:

## Nursing considerations

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**Abstract:** Migraine is a common, chronic disorder that often manifests in childhood. This article discusses the prevalence, diagnosis, and clinical management of uncommon subtypes of migraine with aura, including hemiplegic migraine, retinal migraine, and abdominal migraine.

**Keywords:** abdominal migraine, aura, classic migraine, FAPD, functional abdominal pain disorders, functional gastrointestinal disorders, hemiplegic migraine, migraine, retinal migraine

MIGRAINE IS A COMMON, CHRONIC disorder that often manifests in childhood, peaking between adolescence and early adulthood.<sup>1</sup> It is the third most prevalent disorder worldwide and the second most prevalent disabling disorder.<sup>2-4</sup> Females are affected at a rate three times higher than males. Although migraine onset typically occurs early in life, most of those affected are between the ages of 35 and 45.<sup>2,5</sup>

Often severe and disabling, migraine attacks affect up to 11% of the global adult population (more than 1 billion people) annually.<sup>1,3</sup> They present significant challenges due to their unpredictable onset, episodic pattern, intensity, and duration.<sup>1,6</sup> Although the exact mechanism is unknown, both genetic and environmental factors may be involved in the pathology (see *Common migraine triggers*).<sup>5</sup>

The International Headache Society discusses several distinct migraine syndromes in the *International Classification of Headache Disorders, 3rd edition (ICHD-3)*.<sup>7</sup> Common migraine syndromes include those with and without aura, which is described as a sensory, speech, motor, or most often visual disturbance that develops gradually over the course of an hour. This article discusses the prevalence, diagnosis, and clinical management of three uncommon migraine variants: hemiplegic migraine, retinal migraine, and abdominal migraine.

### Hemiplegic migraine

Hemiplegic migraine (HM) is a migraine with aura that is characterized by significant unilateral reversible muscle and/or motor weakness (rather than paralysis). HM may be

familial or sporadic, and onset typically occurs between ages 12 and 17, with females affected at a rate up to three times higher than males.<sup>8,9</sup> The characteristic muscle weakness can last from hours to days, but it generally lasts between 4 and 72 hours.<sup>7,8</sup> In some individuals, this weakness may also persist for weeks.<sup>8,10</sup>

Typically, migraine auras produce visual symptoms; motor symptoms are rare. HM is a subtype of migraine in which the aura manifests as motor weakness with at least one other typical migraine aura symptom, such as visual field defects, paresthesias, ataxia, or lethargy.<sup>8</sup>

HM may be precipitated by triggers similar to other forms of migraine, such as acute stress, insufficient or excessive sleep, exertion, and head trauma.<sup>8,9,11</sup> Neurologic impairments seen in HM may last from 4 to 72 hours, but they are usually reversible.<sup>5,7</sup> Auras typically last less than an hour but may last up to a day, with reversible hemiplegia lasting up to 4 weeks in rare cases.<sup>9</sup> Most individuals with HM also report an associated headache.<sup>8</sup> Additionally, cortical spreading depression (CSD) is “a self-propagating wave of neuronal and glial depolarization that spreads across the cerebral cortex” that may play an important role in the motor aura symptoms of HM.<sup>12,13</sup>



**HM motor symptoms can develop acutely and mimic stroke symptoms in some cases.**

Motor signs and symptoms associated with HM often start unilaterally, gradually spreading from one hand up into the arm and face. Unilateral weakness may change between or even during attacks, affecting the other side of the body. In some cases, individuals with HM may also experience bilateral motor weakness simultaneously or in succession.<sup>8</sup>

HM motor symptoms typically occur during a 20- to 30-minute period, but they can last anywhere from a few hours to several days and even weeks before resolving. They can also develop acutely and mimic stroke symptoms in some cases. Severe HM attacks may be associated with encephalopathy or coma and can cause brain injury, cerebral infarction, cognitive decline, and death.<sup>8</sup>

**HM diagnosis**

To diagnose HM, providers must rule out other possible etiologies such as stroke, transient ischemic attack, and meningitis.<sup>5</sup> The ICHD-3 diagnostic criteria for HM include aura consisting of motor weakness and visual, sensory, and/or speech and language symptoms. All are fully reversible. According to the ICHD-3, patients with HM also present with at least two of the following four characteristics:<sup>7</sup>

- one aura symptom, such as unilateral motor weakness, which spreads gradually over a period of at least 5 minutes; and/or at least two symptoms such as unilateral motor weakness and visual alterations occurring in succession<sup>7,8</sup>
- nonmotor symptoms lasting between 5 minutes and an hour, and motor symptoms lasting less than 3 days
- at least one unilateral aura symptom
- aura accompanied or followed by a headache within 30 minutes.<sup>7,8</sup>

HM attacks last between 4 and 72 hours and include at least two of the following diagnostic criteria: unilateral location, pulsating pain, moderate-to-severe intensity, and aggravation with routine physical activity.<sup>7</sup> Patients also experience at least one of the following:<sup>3</sup>

- nausea and/or vomiting
- photophobia
- phonophobia.

**Common migraine triggers<sup>4,5,8,14,19</sup>**

Migraine attacks have been linked to many environmental, medication, dietary, and behavioral triggers.

<b>Environmental</b>	noise, odor, bright or flickering lights
<b>Medications</b>	oral contraceptives, menopausal hormone therapy, hydrogen ion blockers
<b>Dietary</b>	cheese, wine, chocolate, citrus food, caffeine, carbonated drinks, food colorings and flavorings, prolonged fasting
<b>Behavioral</b>	sleep deficits, excessive sleep, school or family stressors

A genetic variation is known as familial hemiplegic migraine (FHM). Those diagnosed with FHM have at least one first- or second-degree relative who has experienced migraine attacks that fulfill the HM diagnostic criteria.<sup>8</sup> Certain types of FHM follow an autosomal dominant pattern of transmission, leaving patients with a 50% chance of inheritance from a parent.<sup>8,9</sup> HM that develops in individuals with no familial connection or, in some cases, an asymptomatic parent, is known as sporadic hemiplegic migraine.<sup>5,8,9</sup>

### Managing HM

Establishing the diagnosis and initiating proper treatment can help patients manage and prevent future attacks. Severe HM attacks, which can include fever, depressed level of consciousness, or seizures, may require additional measures such as hospitalization.<sup>9</sup>

Most but not all patients with HM experience headaches as part of the attack.<sup>8</sup> Prophylactic treatments for migraine should be considered in patients who are experiencing more than 3 migraine headaches or at least 8 headache days in a month, as well as in those with migraine variants such as HM.<sup>14</sup> Verapamil, a calcium channel blocker, has demonstrated success as both a prophylactic and an abortive agent for HM. Other prophylactic medications that can be considered include ketamine, lamotrigine, botulinum toxin, acetazolamide, I.V. naloxone, and flunarizine outside of the US.<sup>8,9</sup> Intranasal ketamine administered at the onset of an attack has shown benefits for patients with FHM.<sup>8,9</sup>

Patients with HM may be treated with the same abortive and preventive medications used for typical migraines with aura. However,

pharmacologic agents that may exacerbate cerebral ischemia, such as vasoconstrictors, should be avoided. Treatment of acute HM consists of nonsteroidal anti-inflammatory drugs (NSAIDs) and antiemetic medications. The triptan drug class is usually contraindicated in HM, as are ergotamine derivatives, due to the potential for cerebral vasoconstriction.<sup>8</sup>

For most patients with HM, aura symptoms such as motor weakness may be prolonged but will resolve completely. In rare cases, however, HM may lead to permanent neurologic deficits, cerebral infarctions, cognitive decline, or death. Poor outcomes are often associated with an earlier onset of HM with severe attacks, seizures, or coma. The frequency of HM attacks typically decreases after age 50 and usually evolves into more typical migraine attacks without motor symptoms.<sup>8</sup>

### Retinal migraine

Retinal migraine (RM) is characterized by repeated monocular visual disturbances that can result in scintillations, or bright flashing lines, and/or scotomas, or blind spots.<sup>2,7</sup> RM is a rare disorder with an unknown prevalence, but 29% of patients with RM have a clinical history of migraine headaches and 50% have a family history.<sup>2</sup> Visual symptoms are associated with the painful throbbing headache of classic migraine attacks.<sup>10</sup>

Precipitating factors are also similar to classic migraine attacks with and without aura. These include emotional stress, hypertension, hormonal contraception, exercise, high altitudes, dehydration, smoking, hypoglycemia, and hyperthermia. Patients with comorbidities such as lupus, atherosclerosis, and sickle cell disease may be at an increased risk.<sup>2</sup>

RM attacks can start as early as age 7, but most patients first experience

symptoms between ages 10 and 19 and the incidence peaks between ages 30 and 39.<sup>2</sup>

The pathophysiology remains controversial. One theory suggests RM may be due to vasospasm in the retinal and/or ciliary vasculature. This theory is contentious due to the complexity of the retinal vascular supply. The retina has dual circulation, including the central artery, which supplies the inner retinal layers, and choroidal circulation, which supplies the posterior retina such as photoreceptors.<sup>2</sup> Another theory is that RM results from a spreading depression of the retinal neurons, similar to CSD.<sup>2</sup>

### Signs and symptoms of RM

Patients with RM report a wide range of symptoms, including flashing lights and scintillating scotomas, which may be black, shaded, or white areas of different sizes. In one study, 50% of patients with RM reported complete vision loss in one eye, about 20% reported blurring, 13% reported a scotoma, 12% reported incomplete vision loss, and 7% reported dimming. More than 75% of patients also reported a headache on the same side as the visual disturbance within an hour of onset.<sup>2</sup>

RM attacks may occur multiple times in a day and usually last between 5 and 20 minutes. These are similar to classic migraines with aura, but the RM visual symptoms do not typically last as long. RM should be suspected if patients experience visual disturbances along with a headache or neurologic symptoms such as a stroke, but a cranial nerve exam would be otherwise normal.<sup>2</sup>

### Diagnosing RM

For an RM diagnosis, the patient's clinical presentation must fulfill the diagnostic criteria for migraines with

aura in which the aura is fully reversible, monocular, and confirmed via visual field assessment; for example, patients may be asked to draw a clock, which would include a missing detail consistent with the visual disturbances of RM.<sup>2</sup> The diagnostic criteria include two or three of the following:<sup>2</sup>

- a gradual onset over 5 minutes or longer
- a duration between 5 and 60 minutes
- a headache within 60 minutes of the visual symptoms.

### Managing RM

In general, pharmacologic treatment is not needed if RM attacks occur infrequently; for example, one per month. Abortive pharmacologic therapies are not used to treat RM because of the episodic nature and brief duration of these attacks.<sup>2</sup> Patients with infrequent RMs are advised to manage known and suspected triggering factors such as emotional stress, hypertension, hypoglycemia, alcohol, caffeine, dehydration, smoking, and hormonal contraception.

If the attacks occur more frequently than once per month, however, both nonpharmacologic and pharmacologic treatments may be indicated. Preventive therapies for patients with frequent RM attacks may be taken daily to reduce the frequency and severity of the attacks. For example, a calcium channel blocker may be prescribed to prevent potentially irreversible vision loss.<sup>2</sup>

Although calcium channel blockers such as verapamil are considered a first-line pharmacologic therapy, they may be contraindicated in patients with heart failure, hypotension, sick sinus syndrome, cardiac conduction defects, and renal or hepatic failure. Aspirin and antiepileptic drugs (AEDs) such as topiramate



**RM should be suspected in patients experiencing visual disturbances with a headache or neurologic symptoms.**

or valproate may be indicated to reduce RM severity.<sup>15</sup>

Pharmacologic treatment may reduce the recurrence of attacks, but medications such as triptans and ergotamine derivatives should be avoided in patients with migraines associated with transient vision loss due to a risk of vasoconstriction and irreversible vision loss.<sup>2</sup> Similarly, beta-blockers should be avoided in patients over age 60 and those who smoke due to an increased risk of stroke and other cardiovascular complications.<sup>16</sup>

Complications associated with RM attacks may include central retinal artery and venous occlusion, retinal infarction, branch retinal artery occlusion, choroid or optic nerve ischemia, vitreous hemorrhage, and retinal hemorrhage leading to edema.

Many of these complications can lead to irreversible vision loss.<sup>2</sup> Although RM is generally considered benign, some complications may lead to permanent vision loss after the acute attacks.<sup>2</sup>

Nurses must educate patients about the triggers, signs and symptoms, and clinical management of RM attacks. Patients, families, and caregivers should understand the warning signs of possible permanent compromise or loss of vision; for example, monocular vision loss is described as sudden darkness and requires immediate medical attention. Besides RM, this could be a sign of another irreversible eye condition or even a stroke and requires emergency intervention. Patients should be educated that the visual symptoms associated with RM may come without a headache or pain. Patients may also keep a diary to help evaluate the success of therapies.<sup>2,10</sup>

### Abdominal migraine

Most common in children, abdominal migraine (AM) affects between 1% and 9% of this population and is a common cause of functional abdominal pain in children. It is characterized by episodes of moderate-to-severe, poorly localized, or diffuse abdominal pain lasting more than an hour. This abdominal pain is usually severe enough to interfere with activities of daily living and is often associated with other classic migraine signs and symptoms such as headache, photophobia, anorexia, and nausea and vomiting.<sup>7,17</sup>

The average age of onset for AM ranges between ages 3 and 10, peaking at age 7. Many children with AM will continue to experience migraine headaches into adulthood, even after they no longer experience gastrointestinal (GI) symptoms.<sup>7,17,18</sup> The prevalence is higher among females

than males.<sup>8,19</sup> A history of migraine headache in an immediate family member has also been reported in most AM patients. Episodes of AM can be separated by weeks to months, but there appears to be an individualized pattern and associated symptom profile.<sup>19</sup>

### Signs and symptoms of AM

Abdominal symptoms of AM typically present with a headache, and the relationship between recurrent abdominal pain and migraine headaches has been well established. In 2004, the ICHD-3 recognized AM as a pediatric migraine equivalent.<sup>19</sup> In 2013, the ICHD-3 (beta version) categorized AM as an episodic syndrome that may be associated with migraine.<sup>7</sup>

Globally, an estimated 13.5% of pediatric patients suffer from recognized functional abdominal pain disorders (FAPDs) such as AM, functional dyspepsia, irritable bowel syndrome (IBS), and functional abdominal pain. The pathophysiology of all known FAPDs involves GI dysmotility, visceral hypersensitivity, dysregulation of the mucosal immune system, altered gut microbiota, and complex bidirectional interactions in the brain-gut axis, which links emotional and cognitive brain function with peripheral intestinal functions.<sup>19-22</sup>

AM has a complex pathophysiology and further research is necessary to develop a clearer understanding.<sup>19</sup> However, visceral hypersensitivity refers to heightened sensation in response to stimulus.<sup>19,23</sup> Based on an association between the enteric nervous system and central nervous system (CNS) due to a common embryonic origin, visceral hypersensitivity is the most accepted theory regarding all functional GI disorders, which are characterized by any combination of

abdominal pain, dyspepsia, regurgitation, bloating, constipation, diarrhea, and incontinence.<sup>19,23,24</sup>

Patients with AM may have abnormal gut motility, which can lead to abdominal pain from both distension of the GI tract and intestinal contractions.<sup>19</sup> Gut permeability may also be altered in patients with AM. Recent research has demonstrated that in patients with AM, gut mucosal permeability was significantly increased compared with that of healthy individuals. It also decreased as symptoms improved and increased as symptoms worsened. Based on this understanding, NSAIDs are not beneficial in patients with AM because they can increase gut mucosal permeability.<sup>19</sup>

Dietary factors may also contribute to the symptoms of AM. For example, certain ingredients may trigger migraines, including chocolate, citrus, nuts, dairy products, tomatoes, onions, alcohol, caffeine, monosodium glutamate, aspartame, nitrites, and gluten. These may not be related to patient allergic reactions, however, and serologic testing is required to determine allergens.<sup>25</sup> Excitatory and inhibitory amino acid imbalances in the brain-gut axis, such as inflammatory responses to increased intestinal permeability, are associated with classic migraines, and similar neurotransmitter imbalances may also be involved in AM.<sup>19,25</sup> Limiting food consumption, such as by skipping meals or fasting, can also lead to migraines.<sup>25</sup>

Additionally, genetic mutations can lead to dysregulation of ion channels, neurotransmitter metabolism, and mitochondrial metabolism in the CNS, which contribute to the pathophysiology of migraine headaches. There is also a likely genetic predisposition to the development of functional abdominal pain. Genetic factors that

may contribute to AM are supported by a family history of migraine or chronic abdominal pain in most patients. Ongoing research may further define and identify these factors.<sup>19</sup>

### Diagnosing AM

In the ICHD-3, AM is defined as an idiopathic cause of moderate-to-severe chronic, recurrent abdominal pain that is midline, periumbilical (or poorly localized), and described as dull or sore.<sup>7</sup> The duration of an attack can range between 2 and 72 hours without treatment. Between attacks, patients are typically symptom-free.<sup>7</sup>

To meet the diagnostic criteria for AM, patients must experience a minimum of two associated symptoms during an AM attack, including anorexia, nausea, vomiting, or pallor, as well as at least five instances of abdominal pain as described above.<sup>7,8,19</sup>

### Managing AM

The initial management of AM includes nonpharmacologic interventions, such as education and support, avoidance of triggers, and dietary modifications. Medication therapies may be considered when these initial interventions fail.<sup>19,26</sup> Patient and family education should discuss the episodic nature of AM attacks, possible triggers and relieving factors, and the association with cephalic migraines both with and without aura and functional GI disorders.<sup>19</sup> Nurses should confirm the absence of other abdominal pathologies and establish a positive outlook that AM typically improves on its own in most patients to offer hope and help patients cope.<sup>19</sup>

One study demonstrated that AM that starts early in the morning can be prevented by consuming a breakfast cereal before bed. This “breakfast at bedtime” strategy uses a high-fiber

cereal to prolong its glycemic effect.<sup>19,27</sup> Additionally, cognitive behavioral therapy may be effective in some patients. Similarly, hypnotherapy, family therapy, and yoga can be beneficial for pediatric patients with functional abdominal pain, including AM and IBS. Additional research focused specifically on AM is needed to confirm the efficacy of the “breakfast at bedtime” intervention and other options.<sup>19</sup>

Abortive and prophylactic pharmacologic treatments are beneficial for some patients. Abortive treatments may include triptans (serotonin 5-HT receptor agonists), acetaminophen, ibuprofen, and antiemetics.<sup>19,21,28,29</sup> Prophylactic treatments include beta-blockers such as propranolol (in patients under age 60 who are not smokers); GABA agonists such as valproate; calcium channel blockers such as verapamil; and serotonin antagonists such as cyproheptadine.<sup>9,19,30</sup>

AM is also a precursor to the development of classic migraine headaches with and without aura, and many treatment strategies have been effective for both.<sup>19</sup> It shares many clinical, epidemiologic, and pathophysiologic similarities with classic migraines. For example, a history of migraine headaches in an immediate family member has been described in 34% to 90% of patients with AM. Similarly, a personal history of migraine headaches occurs in 24% to 47% of these patients. AM and classic migraines also share common triggers and similar relieving factors.

Although infrequent, AM can persist into adulthood in some cases. However, the abdominal pain associated with AM typically resolves completely in most patients as they age.<sup>19</sup> One study followed 54 patients with AM for 10 years and found that abdominal pain-



### Nurses can help patients develop an individualized treatment plan and identify candidates for prophylactic treatment.

related symptoms had resolved completely in 61% of these individuals. In addition, 70% of the patients developed migraine with or without aura compared with 20% in the matched control group.<sup>19,31</sup>

Although precise diagnostic criteria are present, AM remains an underdiagnosed disorder. Increased awareness and research on the epidemiology, pathophysiology, effective treatment, and long-term prognosis of AM could help improve patient quality of life and limit healthcare utilization.<sup>19</sup>

### Enhancing outcomes

Classic or variant forms of migraines are common causes of visits to the ED and clinic and represent significant financial and healthcare challenges. Approxi-

mately 28 million patients over age 12 experience migraine headaches in the US, but less than 13% of all patients with migraines are receiving prophylactic treatment, although many could potentially benefit from this therapy.

Patient education is essential in helping to identify migraine type and frequency. Nurses are in a position to teach and assist patients, help develop a successful individualized treatment plan, and identify candidates for prophylactic treatment. They can also provide information related to both nonpharmacologic and pharmacologic interventions to treat, limit, terminate, and reduce the frequency and severity of migraine attacks.<sup>14</sup>

Additional considerations such as the efficacy, adverse reactions, contraindications, and cost of a medication, as well as patient adherence are essential. Healthcare professionals must also consider comorbidities and potential interactions between prescription medications, nonprescription drugs, and even nutritional or herbal supplements. Once treatment begins, it should be reinforced to patients that efficacy is typically assessed after 2 to 3 months and may take up to 6 months. Successful treatments result in a minimum 50% reduction in migraine frequency, so nurses can also help manage patient expectations for realistic outcomes. ■

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