MIGRAINE HEADACHE: DIAGNOSIS AND TREATMENT

By

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Abstract

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Migraine headache occurs in 6% of males and 18% of females. The process of migraine is believed to be one of vascular, neurochemical, neuronal and hormonal changes. Severe migraine pain can be disabling, and mild to moderate pain can interfere with one's normal daily activities. Research has demonstrated a connection with depression and altered quality of life in migraineurs. Diagnosis of migraine is based on history, patient presentation, and exclusion of other pathology. Treatment options include preventive and abortive therapies. The goal of management is to reduce frequency and severity of episodes, and limit the impact of this chronic pain condition.
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Algorithm of headache diagnostics
Introduction

Migraine headache occurs in 6% of males and approximately 18% of females in the United States (Lipton & Stewart, 1993). Affecting approximately 23 million Americans per year (Johnson, 1999), practitioners see patients with migraine as a frequent reason for consultation. Migraine headaches are the cause of 35% of headache complaints in the primary care setting (Derman, 1991).

Migraine headache may interfere with one's normal activities. Lipton and Stewart (1997) found 85% of women and 82% of men with migraine reported some disability resulting from the episodes. Women report more frequent, and more severe attack than males (Lipton & Stewart, 1993). Monthly, it is estimated that 14% of females and 8% of males will miss school or work as a result of disabling migraine (Linet, Stewart, Celentano, Ziegler, & Sprecher, 1989).

Research has shown some connection between migraine and depression (Kolotylo & Broome, 2000). Other research has supported findings of altered quality of life as a result of this chronic pain condition (Allen, Haririfar, Cohen, & Henderson, 2000).

Proper diagnosis and treatment offers hope to patients who live with this prevalent, potentially debilitating illness. The purpose of this paper is to examine the pathway to diagnosis and treatment options for migraine.

Pathophysiology

The pathophysiology of migraine has historically been accepted to be a process of vasoconstriction, resulting in ischemia of brain tissue, followed by vasodilation of the cerebral arteries (Johnson, 1999). This theory was believed to be supported by the focal neurologic changes often seen in migraine (Johnson, 1999). Further research has shown
some evidence of vascular changes which occur with migraine (Diener & May, 1996), with evidence of changes in neurochemical levels (Cady & Farmer-Cady, 2000).

Some theories include involvement of sensory neurons in the trigeminal nerve, which connects cerebral blood vessels with the brain stem. Moskowitz and Cutrer (1993) conducted experiments which suggest migraine pain may originate from inflammation of trigeminal sensory fibers. Once stimulated, these fibers release substance P and calcium gene-related peptide. These neurochemicals produce inflammation, extravasation of plasma and vasodilatory changes which lead to a pulsatile headache (Silberstein, Lipton, & Goadsby, 1998). Initial vasoconstriction of intracranial and extracranial arteries causes a transient ischemia that may result in prodrome symptoms such as confusion, vertigo, and hemiparesthesias (Johnson, 1999).

Serotonin (5-hydroxytryptamine or 5-HT) is believed to play an important role in mediating cerebral dilatory changes and inflammation. Release of serotonin triggers the stimulation of pain fibers (Cunningham, 1999). Manipulation of 5-HT levels impacts perceived pain, but does not affect vasoconstriction or dilation (Ferrari, 1998).

Menstrual and postpartum migraines are believed to be initiated by rapidly falling levels of estrogen which trigger a vasospasm. This theory may help explain the higher prevalence of migraine in the female population. Some theorists have proposed that a decrease in estradiol and estrone may be the trigger for perimenopausal headaches (Moloney, Matthews, Scharbo-Dehann, & Strickland, 2000).

While proposed theories attempt to isolate a specific pathophysiology, the interaction of vascular, neurochemical, hormonal and neuronal influences remains the best explanation. Triggers for migraine may offer some clue to the process. Some
potential triggers that may initiate migraine include stress, exercise, altered sleep cycle, hormonal changes, and foods that contain tyramine, nitrites, phenylethylamine or other additives. (Taylor, 2000). Chronic use of abortive medication, leading to rebound headache, and caffeine have been implicated as well (Cunningham, 1999). Table 1 offers a list of potential triggers.

**Clinical Features / Presentation**

Onset of migraine is generally between the ages of 15 to 25 years. Many patients present with eyes closed, or wearing dark glasses, holding their head, and speaking softly. They are often nauseated and have deliberate, slow movements. Often patients (20%-50%) will relate a family history of migraine, and state recurrence of the same type of headache. This is an important history assessment (Johnson, 1999). Prevalence of migraine increases to age 40, with a decrease in attack frequency seen after that age, except with perimenopausal migraine (Moloney, Matthews, Scharbo-Dehann, & Strickland, 2000).

Perhaps the most important diagnostic tool for migraine is the criteria generated by the Headache Classification Committee of the International Headache Society (1988). This document classifies and describes diagnostic criteria for migraine with aura (meaning accompanied or preceded by focal neurological symptoms) and migraine without aura. Table 2 addresses the classification of headaches.

A variety of focal neurological symptoms including scotomas (black spots in front of the eyes), unilateral weakness of the extremities or speech difficulty may be present with the diagnosis of migraine with aura. The criteria for diagnosis includes one or more reversible aura symptoms, at least one aura that develops over 4 minutes and does not last
longer than 60 minutes, and the headache follows the aura by not less than 60 minutes (Headache Classification Committee, 1988).

Migraine without aura, as well as with aura, is defined by an attack lasting 4-72 hours, with a unilateral location and a pulsating moderate to severe intensity, and increasing symptoms with activity. During the headache, at least one symptom of nausea and/or vomiting or photophobia and phonophobia must be present (Headache Classification Committee, 1988).

**Differential Diagnosis**

Primary headaches involve inflammation, pressure, muscle spasm and vascular changes, with no identifiable pathologic abnormality. Secondary headaches, which occur in 2-10% of headaches, are attributed to organic origin, such as a tumor (Uphold & Graham, 1998). Choice of initial diagnostic tests will be determined by patient presentation, history and physical examination. Other important factors in considering evaluation include gender, age, family history, onset and development of the headache (Edmeads, 1998).

Differential diagnosis must include consideration of potentially life threatening conditions. Of paramount concern are embolic or hemorrhagic cerebrovascular accident (CVA), intracranial tumor, abscess, meningitis, and arteriovenous malformation (AVM) (Jackson, 1998). The practitioner must determine if this is the first headache, the worst headache or a result of trauma. A positive response to these questions indicates the immediate need for a Computed Tomography (CT) scan. One must also consider changes in level of consciousness (LOC), nuchal rigidity, pupillary changes or other focal neurological signs to be ominous, indicating the need for CT (Edmeads, 1998). Table 3
lists differential diagnosis for migraine. Figure 1 details an algorithm for migraine diagnosis.

The presence of focal neurological changes which accompany CVA and AVM may be similar to migraine. Motor, sensory or cognitive deficits may be present, in varying degree dependent on the area and extent of infarct. Hemiparesis, inability to swallow, or aphasia are more indicative of cerebral infarct, and an immediate CT scan is the diagnostic test of choice (Gruenthal, 2001).

Viral and bacterial meningitis, and cerebral abscess headache will most often be accompanied by a fever. Nuchal rigidity, photophobia, phonophobia, lethargy, and vomiting may also be present. Diagnostic evaluation includes gram stain, white blood cell count and culture of cerebral spinal fluid, and complete blood count with white blood cell differential. A CT scan should be done if focal neurological changes develop (Mikolich, 2001).

A headache of recent onset, that is more severe or frequent, or that awakens the patient with pain may indicate an intracranial tumor. Nighttime or morning nausea and vomiting, with more frequent pain suggest the need for a CT scan. Patient report of head pain related to tumor is more common with a history of previous headache (Silberstein, Lipton, & Goadsby, 1998).

The differential should also include sinusitis and temporal arteritis (Jackson, 1998). Patients with sinusitis often report facial pressure, nasal drainage or congestion, nocturnal cough and low-grade fever, as well as headache. Water’s view x-ray may be used to confirm diagnosis, though CT scan is more reliable (Eason, 2001).
Temporal arteritis, most common in males over the age of 50, is a granulomatous inflammation of the arteries. The patient will present with headache, often reporting scalp tenderness and visual changes. Pulsation in the upper extremities may be decreased due to skip lesions in the arteries. Elevated erythrocyte sedimentation rate and alkaline phosphatase, and granulomas changes on biopsy of the temporal artery may also be noted (Ferri, 2001).

Hypertension and tension headaches should be considered in the differential as well (Edmeads, 1998). Tension headache is generally described as a tight band around the head, and the pain is generally not accompanied or preceded by nausea, vomiting, photophobia and phonophobia. Diagnosis is made by patient presentation and exclusion of other etiology (Cunningham, 1999).

Cluster headaches are so named because headaches occur in close episodes then disappear for months or years. Cluster headaches often cause unilateral pain, lacrimation and nasal congestion. The attack will generally occur nocturnally, awakening the patient with severe pain. In contrast to migraine, the patient will be unable to remain still and often moans or yells loudly. Diagnosis is partially made by relief of pain with 100% oxygen, and by exclusion of more ominous conditions (Gruenthal, 2001).

Diagnostics, Cost, Sensitivity and Specificity

The approximate cost of a brain CT is $400 - $500, though this may vary by region and with the use of contrast. The scan takes a few minutes, and little patient preparation is necessary. This diagnostic exam, though expensive, is highly sensitive and specific to rule-out life threatening brain disease or injury such as intra-cranial tumor or hemorrhage, abscess, AVM or CVA. For this reason, it should be considered as the first
radiological exam for fitting patient presentation (Jackson, 1998). Patient teaching for diagnostic testing is discussed in Table 4.

Magnetic resonance imaging (MRI) scan of the brain should be considered if the patient presentation and physical examination point to a potential brain stem lesion or infarct (Jackson, 1998). The cost is approximately $900 - $1,000.

Waters view x-ray of the sinuses may be considered if the patient presents with sinus pressure, fullness or drainage (Jackson, 1998). While the cost is minimal, approximately $80, the study is low in specificity and sensitivity, and not generally considered other than to verify an infectious process. Diagnosis by CT is more reliable and should be the imaging tool of choice for patients of fitting presentation.

Lumbar puncture (LP) for spinal fluid culture costs approximately $400 and should be considered if the patient presents with nuchal rigidity or fever, and CT scan has already ruled out the possibility of a cranial bleed. Sudden onset of LOC changes, as well as vital sign changes, should first be studied with CT, then LP (Uphold & Graham, 1998). Sensitivity and specificity are high for bacterial or viral infection, though results may take up to 48 hours for culture. The presence of red blood cells or white blood cells can be known within an hour.

Abortive Therapy

Trial and error is the theme of abortive therapy. Migraine that is mild to moderate can often be treated with non-prescriptive analgesics (such as naproxen or aspirin/acetaminophen/caffeine combination drugs). Even a patient who suffers from severe migraine may be able to decrease or eliminate symptoms, if the headache is treated in the prodrome with over-the-counter analgesics (Johnson, 1999).
Failure of non-prescriptive analgesics to abort or ameliorate a migraine suggest the use of a 5-HT agonist as the next choice. Unless contraindicated (as in pregnancy or coronary artery disease), medication options include dihydroergotamine mesylate injection (D.H.E. 45) and nasal spray (Migranal); sumatriptan succinate (Imitrex) tablets, nasal spray, and subcutaneous injection; rizatriptan (Maxalt) tablets; zolmitriptan (Zomig) tablets; and naratriptan (Amerge) tablets; and ergotamine tartrate/caffeine (Caftergot) tablets (Jones, 1999).

Ergotamines are effective in approximately 50% of migraine without aura patients. Medication may be administered by oral, nasal, rectal, intramuscular, intravenous, subcutaneous or sublingual routes. These varied options allow the patient to choose their most convenient and effective dosing. This class is also one of the least expensive of the prescription therapies (Johnson, 1999). Important patient teaching includes taking the medicine at the onset of symptoms, keeping medication available at all times, and not exceeding 6 milligrams per week. Patients should also be instructed not to combine this drug another vasoconstrictor, and in the short shelf life of this medication. Ergotamines, believed to work through vasoconstrictive properties, may also decrease serotonin levels (Johnson, 1999).

Sumatriptan succinate (Imitrex), available in tablet and subcutaneous injection form, is a vasoconstrictor that offers approximately 55% of migraineurs relief. The most frequent side effects include dizziness, paresthesias, and localized injection site reaction. Fetal harm or death may occur with use of this drug, therefore pregnancy is an absolute contraindication to use of sumatriptan (Johnson, 1999).
If acute treatment fails, antiemetics such as metoclopramide (Reglan) can be added to increase absorption and efficacy, as well as help control nausea. The clinician should also evaluate other causes for treatment failure such as sub-therapeutic dosing, rebound headache from frequent analgesic use, and incorrect diagnosis (Diener, Kaube & Limmroth, 1999).

Patient Education

There is much a patient can do to help prevent the occurrence of migraine. Identifying triggers, avoidance of dietary caffeine and alcohol, limiting stress, massage therapy, if there is a stress/tension component, and regular sleep habits may be helpful. Avoiding foods such as red wine, aged cheese and certain food additives, such as monosodium glutamate may help reduce the occurrence of migraine (Taylor, 2000).

As a first step in identifying triggers, the clinician should recommend maintaining a headache log. Documentation should include foods eaten prior to migraine, how often migraine occurs, lasts and what helps resolve the headache (Cady, 2001). The clinician should include evaluation of chronic use of analgesic medication in the event of frequent headache, as rebound symptoms can occur (Gruenthal, 2001).

Preventive Therapy

Preventive therapy for migraine should be considered for the patient with more than three episodes of migraine in a month, pain that is refractory to acute therapy, or those with contraindications to acute treatment regimens (Jones, 1999). Prophylaxis of migraine requires commitment to a daily regimen of dosing for a minimum of two to three months before effectiveness is known (Moloney, Matthews, Scharbo-Dehann, & Strickland, 2000). This may be a useful option for the patient who has more than three
headaches per month (Aminoff, 2001), as at least 50% of patients report a decrease in occurrence and/or severity (Johnson, 1999).

Alternative oral therapy for prophylaxis is a promising area of research. Murphy, Heptinstall & Mitchell (1988) conducted a study of the effectiveness of the herb feverfew in migraine prophylaxis. The researchers noted a 24% reduction in the number of migraine attacks. Two leaves (Murphy, Heptinstall, & Mitchell, 1988) of dried fever enclosed in capsules has been the traditional preparation, and remains the recommended form for dosing (Awang, 1998).

Magnesium deficiency has also been related to the occurrence of migraine. One study has shown a decrease in total serum magnesium during attack in patients with migraine and tension type headache (Sarchielli et al., 1992). A supplement of 300 to 600 milligrams per day has been suggested as the level for prophylaxis, especially if there is known hypomagnesemia (Jones, 1999).

Preventive pharmacological therapy options include beta blockers (such as propranolol/Inderal), calcium channel blockers (verapamil/Calan), and tricyclic antidepressants (amitriptyline/Elavil). Other treatment options include the use of NSAID’s (naproxyn/Naprosyn), selective serotonin reuptake inhibitors (such as paroxetine hydrochloride/Paxil), and anticonvulsants (such as divalproex/Depakote) (Taylor, 2000). The clinician should endeavor to find the most effective drug, at the lowest dose, with the fewest side effects (Diener, Kaube & Limmroth, 1999).

Unless contraindicated (as in patients with bradycardia, asthma, or diabetes mellitus), beta blockers are generally the first drug selected for prophylaxis (Jones, 1999). Beta blockers are about 60% effective (Moloney, Matthews, Scharbo-Dehaan, &
Strickland, 2000). Potential adverse effects of beta blockers include bronchospasm, bradycardia and altered sleep cycle. An effective and generally well tolerated drug, propranolol (Inderal) showed a 65% reduction of occurrence of migraine (Diener, Kaube, & Limmroth, 1999).

Calcium channel blockers (CCB’s) are the next classification of drug recommended in prophylaxis (Diener, Kaube, & Limmroth, 1999). This class has been shown to approach 60% effectiveness (Moloney, Matthews, Scharbo-Dehaan, & Strickland, 2000), with minimal side effects (Jones, 1999). Use of CCB’s is contraindicated in any patient with arrhythmia’s (Diener, Kaube, & Limmroth, 1999).

The next class for consideration in prevention is tricyclic antidepressants. Providing an approximate 60% effectiveness (Moloney, Matthews, Scharbo-Dehann, & Strickland, 2000), this drug frequently causes drowsiness, but this side effect will usually abate within a few weeks.

If a patient appears to have a purely menstrual migraine, and episodes of occurrence are predictable, therapy with estrogen may be effective. Estrogen supplementation during menses, or estrogen suppression during the late luteal phase (when estrogen is at its peak, thereby preventing a rapid decline) are possible therapies (Jones, 1999). Patients should be instructed in the increased risk of stroke and thromboembolism with the use of estrogen. Patients who smoke should be counseled in cessation (Diamond, 2000). Triphasic oral birth control should be avoided as estrogen levels vary throughout the cycle, and tolerance of therapy may be lower (Diamond, 2000).
Divalproex sodium (Depakote) has shown mid-range effectiveness (Moloney, Matthews, Scharbo-Dehaan, & Strickland, 2000). In one study, Matthew et al., (1995) found a 48% reduction in headache with daily administration of 1,000 mg of divalproex. Migraine induced vomiting, photophobia and phonophobia were also ameliorated. Reported adverse effects in the study participants were somnolence, and nausea and vomiting not connected to migraine.

Alternative Therapies

Non-pharmacological therapy for migraine prophylaxis and treatment has included chiropractic, massage, essential oil preparation, oxygen therapy, therapeutic touch, and intraoral chilling of the trigeminal nerve (by use of an experimental device). Results are varied, and patients must use trial and error to determine the most effective route of control for their headache.

Conclusion

Accurate diagnosis of this condition is the first step to helping the patient develop an effective treatment plan to control this illness. Quality of life can be greatly affected by this potentially debilitating condition, and the primary care provider must be willing to explore many treatment options. As with any illness, treatment should be individualized with consideration to cost, effectiveness, adverse effects and patient compliance.

The need for further research and development of new medications is evident. A 50% effectiveness rate for the prophylactic therapies is low, with many patients unable to find relief.
References


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<th>Category</th>
<th>Triggers</th>
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<tr>
<td>Physical</td>
<td>Menstruation, Exercise, Altered Sleep pattern, Irregular meals</td>
</tr>
<tr>
<td>Food</td>
<td>Red wine, Aged cheese, Bacon, Chocolate, Liver, Alcohol, Aspartame, Monosodium Glutamate, Caffeine</td>
</tr>
<tr>
<td>Emotional</td>
<td>Anxiety, Stress, Excitement, Anger</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Chronic pain medication usage, Cocaine use and withdrawal, Tobacco use</td>
</tr>
</tbody>
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(Cady, 2001; Johnson, 1999; Taylor, 2000)
### Table 2

**International Headache Society Criteria for Migraine**

**Migraine without aura:**
- At least 5 attacks, lasting 4-72 hours, at least two from Group A, and one from Group B
- **Group A**
  - Pain is unilateral
  - Pain is pulsatile or throbbing
  - Pain is aggravated by normal activity (such as climbing stairs, bending over)
  - Moderate to severe pain that limits normal activity
- **Group B**
  - Nausea and/or vomiting
  - Photophobia and phonophobia

**Migraine with aura:**
- At least 2 attacks, gradual onset of reversible focal neurological symptoms, headache lasting 4-72 hours, at least 3 from Group A and one from Group B
- **Group A**
  - One or more fully reversible neurological symptoms (such as scotomas, paraesthesias, weakness)
  - No aura symptom lasts longer than 60 minutes
  - Headache follows aura free period of not more than 60 minutes
- **Group B**
  - History and exam do not suggest other disorder
  - Other disorders are ruled out by thorough investigation
  - Other disorders are present, but not temporally related to the migraine
<table>
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<tr>
<th>Diagnosis</th>
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<tr>
<td>Cerebrovascular Accident</td>
<td>Vascular occlusion or hemorrhage; sudden onset of focal neurological changes; evaluate patient with physical exam and CT.</td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage</td>
<td>Vascular hemorrhage; sudden onset with vomiting, nausea, altered level of consciousness; may be trauma related; evaluate with physical exam, CT.</td>
</tr>
<tr>
<td>Meningitis</td>
<td>Result of intracranial infection; patient will be febrile and often has nuchal rigidity; evaluate patient with physical exam, CT, LP, and complete blood count with differential.</td>
</tr>
<tr>
<td>Intracranial Tumor</td>
<td>Gradual onset of progressively worsening symptoms; may see focal neurological changes, projectile vomiting, and a headache that began as mild and intermittent is now constant and worse; evaluate with physical exam, CT and MRI.</td>
</tr>
<tr>
<td>Intracranial Abscess</td>
<td>Focal neurological changes accompanied by fever and occasionally a toxic appearance; evaluate with physical exam, CT with and without contrast.</td>
</tr>
<tr>
<td>Temporal Arteritis</td>
<td>Unilateral temporal tenderness, poorly pulsatile artery; evaluate by physical exam, erythrocyte sedimentation rate, and consider temporal artery biopsy.</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>Facial pressure, often worse in the morning and with bending over, daily moderately severe headache, nasal drainage or congestion; evaluate by physical exam, Water’s view x-ray, and CT.</td>
</tr>
<tr>
<td>Cluster Headache</td>
<td>Vascular in nature, often relieved with 100% O2; evaluate patient with physical exam, CT if no prior history.</td>
</tr>
<tr>
<td>Tension Headache</td>
<td>Tension and stress related; muscle relaxation or mild analgesics provide relief; evaluate patient with physical exam.</td>
</tr>
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(Cady, 2001; Derman, 1991; Mikolich, 2001)
Table 4
Patient Teaching

Computed Tomography
The test takes approximately 15 minutes for exam without contrast. Assess the patient for claustrophobia and offer anxiolytics as necessary.

Lumbar Puncture
Obtain informed consent. Explain that local anesthetic is used, but the test may be uncomfortable. Offer pain medication. A baseline and post procedure assessment of neurological function will be performed. Instruct the patient to lay prone for 6-12 hours after the procedure, and to notify the provider if a persistent headache develops. Culture of cerebrospinal fluid may take up to 72 hours. The presence of blood and other abnormal components may be known within the hour.

Magnetic Resonance Imaging
Explain to the patient there is no radiation exposure. Assess the patient for claustrophobia, and offer anxiolytics as necessary. Instruct the patient to remove all metal objects. The patient must remain motionless throughout the exam, and they may hear the thumping sound of the machine. Offer ear plugs.

Laboratory Blood Studies
Inform the patient that blood will be drawn and evaluated for white blood cell elevation and erythrocyte sedimentation rate as indicators of infection and inflammation. Results are generally available within the hour.

(Cady, 2001; Ferri, 2001; Gruenthal, 2001)
Figure 1
Algorithm of Headache Diagnostics

Headache
  Vital signs, headache history and physical exam
    Headache history with no change
    Vital signs and PE normal
      Analgesics, monitor
    No headache history
      Acute onset, vital sign or LOC changes
        CT Scan
          Febrile, Nuchal Rigidity
            CT Normal
              Neurologist Referral
              LP, CBC
                Normal
                  Monitor
                Abnormal
                  Antibiotics, supportive measures
            CT Abnormal
              Neurologist Referral
          Changed LOC
            CT Normal
              Neurologist Referral
              Analgesics, Monitor
            CT Abnormal
              Neurologist Referral
          Acute onset
            CT Normal
              Neurologist Referral
            CT Abnormal