Migraine on the rise time to create awarness during pregnancy and its medical treatment

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Abstract

Migraine headaches result from a combination of blood vessel enlargement and the release of chemicals from nerve fibers that coil around these blood vessels. During the headache, an artery enlarges that is located on the outside of the skull just under the skin of the temple (temporal artery). This causes a release of chemicals that cause inflammation, pain, and further enlargement of the artery. A migraine headache causes the sympathetic nervous system to respond with feelings of nausea, diarrhea, and vomiting. Migraine headaches have a female predominance with a peak in prevalence in the third and fourth decades of life. Women of reproductive age are liable to develop their first migraine while pregnant or exhibit changes in the character, frequency or severity of their headaches during pregnancy and the puerperium. The purpose of this Review is to examine the pathophysiology underlying the development of migraine headaches and the association of this pathophysiology with pregnancy-related complications. We present the case of a woman with a history of migraine headaches before pregnancy, whose symptoms progressed during pregnancy in part because of increasing exposure to narcotic medications. The abortive medications for moderate or severe migraine headaches are different than OTC analgesics. Instead of relieving pain, they abort headaches by counteracting the cause of the headache, dilation of the temporal arteries. In fact, they cause narrowing of the arteries. Examples of migraine-specific abortive medications are the triptans and ergot preparations. We also discuss the diagnosis and management of migraine headaches that precede pregnancy or develop *de novo* during pregnancy, placing an emphasis on the distinction between primary migraine headache and headache secondary to pre-eclampsia—a relatively frequent complication of pregnancy and the puerperium.

Introduction

A migraine headache is a form of vascular headache. Migraine headache is caused by a combination of vasodilatation (enlargement of blood vessels) and the release of chemicals from nerve fibers that coil around the blood vessels. During a migraine attack, the temporal artery enlarges. (The temporal artery is an artery that lies on the outside of the skull just under the skin

of the temple.) Enlargement of the temporal artery stretches the nerves, that coil around the artery and cause the nerves to release chemicals. The chemicals cause inflammation, pain, and further enlargement of the artery. The increasing enlargement of the artery magnifies the pain. Migraine attacks commonly activate the sympathetic nervous system in the body. The sympathetic nervous system is often thought of as the part of the nervous system that controls primitive responses to stress and pain, the so-called "fight or flight" response. The increased sympathetic nervous activity in the intestine causes nausea, vomiting, and diarrhea. Sympathetic activity also delays emptying of the stomach into the small intestine and thereby prevents oral medications from entering the intestine and being absorbed. The impaired absorption of oral medications is a common reason for the ineffectiveness of medications taken to treat migraine headaches. The increased sympathetic activity also decreases the circulation of blood, and this leads to pallor of the skin as well as cold hands and feet. The increased sympathetic activity also contributes to the sensitivity to light and sound sensitivity as well as blurred vision. A migraine is a very painful type of headache. People who get migraines often describe the pain as pulsing or throbbing in one area of the head. During migraines, people are very sensitive to light and sound. They may also become nauseated and vomit. Migraine is three times more common in women than in men. Some people can tell when they are about to have a migraine because they see flashing lights or zigzag lines or they temporarily lose their vision. Migraine headaches predominantly affect women and show a peak in prevalence in the third and fourth decades of life.[1] In women of reproductive age, pregnancy and the puerperium are associated with an increased likelihood of developing a first migraine or showing changes in the character, frequency, or severity of such headaches. Pregnancy is characterized by elevated estrogen and progesterone levels, which both suddenly decline after delivery. Menstrual migraines have been hypothesized to occur when estrogen levels decline after a period of sustained estrogen exposure.[2] On the basis of this assumption, migraine symptoms would be expected to improve during pregnancy and recur during the puerperium.

Symptoms of migraine

Following are the major **migraine symptoms**:

1. Photophobia
2. Throbbing pain in half of head
3. Eyes become red and patient perceives of burning eyes
4. Nausea sensation and vomiting
5. Loss of appetite
6. Patient wishes to stays all alone during and feels comfortable in silent and dark room
7. Depression and irritability
8. Numbness or weakness in an arm or leg.

Pathogenesis

The brain's susceptibility to migraine attacks is fundamentally associated with the excitability of the neuronal cell membranes of the occipital cortex. Cortical spreading depression is the most widely accepted mechanism for the development of aura. In cases of headache following aura,

cortical spreading depression is hypothesized to lead to activation of the trigeminal nucleus caudalis—part of the central pathway of migraine pain generation.
The pathogenic mechanism of migraine pain in the absence of aura is still hotly debated and is an active area of investigation. The main area of dispute is whether migraine pain originates peripherally, from activation of the trigeminal nerve via release of vasoactive factors from blood vessels, or centrally, in the trigeminal nucleus in the brainstem. The exact mechanisms for the central origination of pain in the brainstem have yet to be elucidated, so the activity observed in the brainstem during migraine could be a consequence of peripheral activation and not the cause. The other possible mechanism is one in which peripheral activation is combined with a change in central pain modulation.

Regardless of the source of signaling—central or peripheral—the common mechanism underlying migraine headache seems to be dilatation of the large cranial vessels beneath the dura mater. These vessels are innervated by the trigeminal nerve and form part of the trigeminovascular system. Several vasoactive peptides have been identified in the cell bodies of trigeminal neurons, including calcitonin gene-related peptide (CGRP), substance P, and neurokinin A. Of these peptides, CGRP seems to be the most important in the pathogenesis of migraine headache.[8]

Complications of migraine with aura include prolonged aura without infarction and migrainous stroke. The pathophysiological mechanisms that have been linked to ischemia and the development of true migrainous infarction include neurologically mediated cortical spreading depression, reversible vasoconstriction, intracranial arterial dissection, and arterial embolism.

Migraine during pregnancy

Epidemiology

Pregnancy has a variable effect on the frequency and intensity of migraines. The available literature indicates that most women seem to experience either an improvement or no change in migraine frequency during pregnancy. The percentage of women who show improvement during pregnancy ranges from 18% to 86%, although studies performed to date have not established an objective set of criteria that can predict which patients will improve.
Studies have consistently found that migraine with aura is less likely to improve during pregnancy than migraine without aura. The reason for this finding is not obvious, but it could be related to increased endothelial reactivity in patients with migraine and aura, compared with those without aura. The additional, and potentially adverse, effect of pregnancy on endothelial reactivity might lead to an increase in the frequency of attacks in patients with migraine and aura. The case report in Box 1features a patient with migraine and aura, whose attacks were frequent enough to warrant preventive therapy throughout pregnancy.

In a study of 98 women undergoing induced ovulation, those with a history of migraines (25 individuals) had a higher frequency of headaches during the phase of hypothalamus–pituitary–ovary-axis downregulation than those without migraines. The downregulation phase of induced ovulation is characterized by very low 17β-estradiol serum levels. Similarly, levels of estrogens

have been observed to fluctuate in the perimenopausal state. The observation that migraine attacks increase during perimenopause supports the concept that fluctuations and abrupt changes in estrogen levels can act as triggers for migraine.

Migraine headaches do not seem to directly affect the outcome of pregnancy, although they could exert an indirect effect. Patients with migraine have a higher risk of developing pre-eclampsia than individuals without migraine. Of the limited number of studies that evaluated other pregnancy outcomes in patients with migraine, most did not report an increase in the incidence of other poor outcomes, such as preterm labor, low birth weights, or congenital anomalies.

Additional indirect associations between migraine and outcomes in women of reproductive age were noted in an analysis of a national database of hospital discharges. This analysis showed that the prevalence of migraine in hospitalized women was only 185 per 100,000 deliveries, whereas the expected prevalence for women of childbearing age is 12–18%. The prevalence of migraine in pregnant women might have been underestimated by this study, as only women actively affected by severe migraines were assigned a migraine discharge diagnosis. Studying only the discharge codes during pregnancy also led to an underestimation of peripartum migraine prevalence because many women do not experience migraines during pregnancy or have only very mild attacks.

Neuroimaging

Neuroimaging might be considered in the evaluation of women with headaches that are of new onset or different from their usual headaches. In most patients with established migraine, however, neuroimaging is not usually necessary. Evidence-based guidelines issued by the American Academy of Neurology suggest that neuroimaging should be considered in the following patients with nonacute headache: patients with an unexplained abnormal finding on neurological examination; patients with atypical headache features or headaches that do not fulfill the strict definition of migraine or other primary headache disorders (or who have an additional risk factor, such as immune deficiency); and patients with sudden severe headache.

A number of features signify an increased risk of finding an abnormality on neuroimaging in patients with a nonacute headache. These indicators include a rapidly increasing headache frequency, ataxia, localized neurological signs, and the occurrence of a severe headache that either awakens a patient from sleep or can be described as the worst headache the patient has ever experienced. A head CT scan (with and without contrast) is sufficient in many patients when neuroimaging is deemed necessary. MRI is indicated when a posterior fossa lesion or cerebrospinal fluid leak is suspected. MR angiography and MR venography are indicated when an arterial or venous lesion (for example, cerebral venous thrombosis) is considered in the differential diagnosis.

Most neuroimaging diagnostic techniques pose a negligible risk to the fetus. The average radiation to which the mother is exposed from a head CT scan is <0.01 Gy, whereas the threshold for fetal damage with ionizing radiation directed towards the maternal pelvis is >0.1–0.2 Gy. To maintain a margin of safety, the National Council for Radiation Protection and

Measurements has set the upper limit of acceptable radiation in all CT scans at 0.05 Gy. Other forms of electromagnetic radiation, such as that produced in MRI, do not pose the same level of risk associated with ionizing radiation.

Gadolinium-based contrast agents have been associated with the development of nephrogenic systemic sclerosis. This condition is rare and has been reported to occur in patients with compromised renal function. Gadolinium can cross the placenta into the fetal circulation and, subsequently, is excreted into the amniotic fluid, where the agent can remain for an extended period of time. No prospective studies with large numbers of patients have evaluated the risk of teratogenic or mutagenic effects. Animal studies have demonstrated that high doses of gadolinium administered over long periods of time can have adverse effects. The American College of Radiology Guidance Document for Safe MR Practices recommends that a pregnant patient should only receive gadolinium-based contrast agents after careful consideration of the risk–benefit ratio.

Iodinated CT contrast agent has been associated with contrast-induced nephropathy in as many as 21% of patients who had a baseline glomerular filtration rate of <50 ml/min/1.73 . Nephropathy induced by iodinated CT contrast agent is usually reversible, but the condition can be associated with nonrenal complications that can prolong hospital stays and increase in-hospital mortality. Free iodide in the contrast medium given to the mother has the potential to depress fetal and neonatal thyroid function. Neonatal thyroid function should, therefore, be checked after delivery in such patients. The risk associated with absorption of contrast medium during lactation is small and can be considered insufficient to warrant stopping of breastfeeding.

Causes of migraine

Following are the major **migraine causes**:

1. Stress and over burdening of mind
2. Tensions
3. Acidity, Indigestion or Constipation
4. Excessive smoking and taking of alcohol
5. Low blood sugar, low blood pressure
6. General body weakness
7. Menstruation in women
8. Nutritional deficiency
9. Consistent overwork
10. Improper sleep and rest.

Migraine headache diagnosis

Migraine headaches are usually diagnosed when the symptoms described above are present. Migraine generally begins in childhood to early adulthood. While migraines can first occur in an individual beyond the age of fifty, advancing age makes other types of headaches more likely. A family history is usually present, suggesting a genetic predisposition in migraine sufferers. In addition to diagnosing migraine from the clinical presentation there is usually an accompanying

normal examination. Patients with the first headache ever, worst headache ever, or where there is a significant change in headache or the presence of nervous system symptoms, like visual or hearing or sensory loss, may require additional tests. The tests may include blood testing, brain scanning (either CT or MRI), and a spinal tap.

Approaches of migraine headaches prevention

Prevention of migraine

There are two ways to prevent migraine headaches: 1) by avoiding factors ("triggers") that cause the headaches, and 2) by preventing headaches with medications (prophylactic medications). Neither of these preventive strategies is 100% effective. The best one can hope for is to reduce the frequency of headaches.

Migraine triggers

A migraine trigger is any factor that causes a headache in individuals who are prone to develop headaches. Only a small proportion of migraine sufferers, however, clearly can identify triggers. Examples of triggers include stress, sleep disturbances, fasting, hormones, bright or flickering lights, odors, cigarette smoke, alcohol, aged cheeses, chocolate, monosodium glutamate, nitrites, aspartame, and caffeine. For some women, the decline in the blood level of estrogen during the onset of menstruation is a trigger for migraine headaches. The interval between exposure to a trigger and the onset of headache varies from hours to two days. Exposure to a trigger does not always lead to a headache. Conversely, avoidance of triggers cannot completely prevent headaches. Different migraine sufferers respond to different triggers, and any one trigger will not induce a headache in every person who has migraine headaches.

Sleep and migraine

Disturbances such as sleep deprivation, too much sleep, poor quality of sleep, and frequent awakening at night are associated with both migraine and tension headaches, whereas improved sleep habits have been shown to reduce the frequency of migraine headaches. Sleep also has been reported to shorten the duration of migraine headaches.

Fasting and migraine

Fasting possibly may precipitate migraine headaches by causing the release of stress-related hormones and lowering blood sugar. Therefore, migraine sufferers should avoid prolonged fasting.

Bright lights and migraine

Bright lights and other high intensity visual stimuli can cause headaches in healthy subjects as well as patients with migraine headaches, but migraine patients seem to have a lower than normal threshold for light-induced pain. Sunlight, television, and flashing lights all have been reported to precipitate migraine headaches.

Caffeine and migraine

Caffeine is contained in many food products (cola, tea, chocolates, coffee) and OTC analgesics. Caffeine in low doses can increase alertness and energy, but caffeine in high doses can cause insomnia, irritability, anxiety, and headaches. The over-use of caffeine-containing analgesics causes rebound headaches. Furthermore, individuals who consume high levels of caffeine regularly are more prone to develop withdrawal headaches when caffeine is stopped abruptly.

Chocolate, wine, tyramine, MSG, nitrites, aspartame and migraine

Chocolate has been reported to cause migraine headaches, but scientific studies have not consistently demonstrated an association between chocolate consumption and headaches. Red wine has been shown to cause migraine headaches in some migraine sufferers, but it is not clear whether white wine also will cause migraine headaches. Tyramine (a chemical found in cheese, wine, beer, dry sausage, and sauerkraut) can precipitate migraine headaches, but there is no evidence that consuming a low-tyramine diet can reduce migraine frequency. Monosodium glutamate (MSG) has been reported to cause headaches, facial flushing, sweating, and palpitations when consumed in high doses on an empty stomach. This phenomenon has been called Chinese restaurant syndrome. Nitrates and nitrites (chemicals found in hotdogs, ham, frankfurters, bacon and sausages) have been reported to cause migraine headaches. Aspartame, a sugar-substitute sweetener found in diet drinks and snacks, has been reported to trigger headaches when used in high doses for prolonged periods.

Female hormones and migraine

Some women who suffer from migraine headaches experience more headaches around the time of their menstrual periods. Other women experience migraine headaches only during the menstrual period. The term "menstrual migraine" is used mainly to describe migraines that occur in women who have almost all of their headaches from two days before to one day after their menstrual periods. Declining levels of estrogen at the onset of menses is likely to be the cause of menstrual migraines. Decreasing levels of estrogen also may be the cause of migraine headaches that develop among users of birth control pills during the week that estrogens are not taken.

Medication of migraine sufferers

Individuals with mild and infrequent migraine headaches that do not cause disability may require only OTC analgesics. Individuals who experience several moderate or severe migraine headaches per month or whose headaches do not respond readily to medications should avoid triggers and consider modifications of their life-style. Life-style modifications for migraine sufferers include:

Betablockers

Beta-blockers are a class of drugs that block the effects of beta-adrenergic substances such as adrenaline (epinephrine). By blocking the effects of adrenaline, beta-blockers relieve stress on the heart by slowing the rate at which the heart beats. Beta-blockers have been used to treat high blood pressure, angina, certain types or tremors, stage fright, and abnormally fast heart beats

(palpitations). They also have become important drugs for improving survival after heart attacks. Beta-blockers have been used for many years to prevent migraine headaches. It is not known how beta-blockers prevent migraine headaches. It may be by decreasing prostaglandin production, though it also may be through their effect on serotonin or a direct effect on arteries. The beta-blockers used in preventing migraine headaches include propranolol (Inderal), atenolol (Tenormin), metoprolol (Lopressor, Lopressor LA, Toprol XL), nadolol (Corgard), and timolol (Blocadren).Beta-blockers generally are well-tolerated. They can aggravate breathing difficulties in patients with asthma, chronic bronchitis, or emphysema. In patients who already have slow heart rates (bradycardias) and heart block (defects in electrical conduction within the heart), beta-blockers can cause dangerously slow heartbeats. Beta-blockers can aggravate symptoms of heart failure. Other side effects include drowsiness, diarrhea, constipation, fatigue, decrease in endurance, insomnia, nausea, depression, dreaming, memory loss, impotence.

Tricyclicantidepressants

Tricyclic antidepressants (TCAs) prevent migraine headaches by altering the neurotransmitters, norepinephrine and serotonin, that the nerves of the brain use to communicate with one another. The tricyclic antidepressants that have been used in preventing migraine headaches include amitriptyline (Elavil), nortriptyline (Pamelor, Aventyl), doxepin (Sinequan), imipramine (Tofranil), and protriptyline.The most commonly encountered side effects associated with TCAs are fast heart rate, blurred vision, difficulty urinating, dry mouth, constipation, weight gain or loss, and low blood pressure when standing.TCAs should not be used with drugs that inhibit monoamine oxidase such as isocarboxazid (Marplan), phenelzine (Nardil), tranylcypromine (Parnate), and procarbazine (Matulane), since high fever, convulsions and even death may occur. TCAs are used with caution in patients with seizures, since they can increase the risk of seizures. TCAs also are used with caution in patients with enlargement of the prostate because they can make urination difficult. TCAs can cause elevated pressure in the eyes of some patients with glaucoma. TCAs can cause excessive sedation when used with other medications that slow the brain's processes, such as alcohol, barbiturates, narcotics, and benzodiazepines, e.g. lorazepam (Ativan), diazepam (Valium), temazepam (Restoril), oxazepam (Serax), clonazepam (Klonopin), zolpidem (Ambien). Epinephrine should not be used with amitriptyline, since the combination can cause severe high blood pressure

Antiserotonin medications

Methysergide (Sansert) prevents migraine headaches by constricting blood vessels and reducing inflammation of the blood vessels. Methylergonovine is related chemically to methysergide and has a similar mechanism of action. They are not widely used because of their side effects. The most serious side effect of methysergide is retroperitoneal fibrosis (scarring of tissue around the ureters that carry urine from the kidneys to the bladder). Retroperitoneal fibrosis, though rare, can block the ureters and cause backup of urine into the kidneys. Backup of urine into the kidneys can cause back and flank (the side of the body between the ribs and hips) pain and ultimately can lead to kidney failure. Methysergide also has been reported to cause scarring around the lungs that can lead to chest pain, and shortness of breath.

Calcium channel blockers

Calcium channel blockers (CCBs) are a class of drugs that block the entry of calcium into the muscle cells of the heart and the arteries. By blocking the entry of calcium, CCBs reduce contraction of the heart muscle, decrease heart rate, and lower blood pressure. CCBs are used for treating high blood pressure, angina, and abnormal heart rhythms (e.g., atrial fibrillation). CCBs also appear to block a chemical within nerves, called serotonin, and have been used occasionally to prevent migraine headaches. The CCBs used in preventing migraine headaches are diltiazem (Cardizem, Dilacor, Tiazac), verapamil (Calan, Verelan, Isoptin), and nimodipine.

The most common side effects of CCBs are constipation, nausea, headache, rash, edema (swelling of the legs with fluid), low blood pressure, drowsiness, and dizziness. When diltiazem or verapamil are given to individuals with heart failure, symptoms of heart failure may worsen because these drugs reduce the ability of the heart to pump blood. Verapamil and diltiazem may reduce the elimination and increase the blood levels of carbamazepine (Tegretol), simvastatin (Zocor), atorvastatin (Lipitor), and lovastatin (Mevacor). This can lead to toxicity from these drugs.

Anticonvulsants

Anticonvulsants (antiseizure medications) also have been used to prevent migraine headaches. Examples of anticonvulsants that have been used are valproic acid, phenobarbital, gabapentin, and topiramate. It is not known how anticonvulsants work to prevent migraine headaches.

Prophylactic medications to prevent migraine headaches

Not all migraine sufferers need prophylactic medications; individuals with mild or infrequent headaches that respond readily to abortive medications do not need prophylactic medications. Individuals who should consider prophylactic medications are those who:

- Require abortive medications for migraine headaches more frequently than twice weekly.
- Have two or more migraine headaches a month that do not respond readily to abortive medications.
- Have migraine headaches that are interfering substantially with their quality of life and work.
- Cannot take abortive medications because of heart disease, stroke, or pregnancy, or cannot tolerate abortive medications because of side effects.

Effective for prophylactic medications

Prophylactic medications can reduce the frequency and duration of migraine headaches but cannot be expected to eliminate migraine headaches completely. The success rate of most prophylactic medications is approximately 50%. Success in preventing migraine headaches is defined as more than a 50% reduction in the frequency of headaches. Prophylactic medications usually are begun at a low dose that is increased slowly in order to minimize side effects. Individuals may not notice a reduction in the frequency, severity, or duration of their headaches for 2-3 months after starting treatment.

Proper way to use preventive medications

Doctors familiar with the treatment of migraine headaches should prescribe preventive medications. Decisions about which preventive medication to use are based on the side effects of the medication and the medical conditions that the patient may have. Propranolol (Inderal) often is used first, provided that the patient does not have asthma, COPD or heart disease. Amitriptyline (Elavil) also is used commonly. Preventive medications are begun at low doses and gradually increased to higher doses if needed. This minimizes side effects from the medications. Preventive medications are to be taken daily for months to years. When they are stopped, the dose needs to be gradually reduced rather than abruptly stopped. Abruptly stopping preventive medications can lead to headaches. In some instances, more than one drug may be needed. Non-medication and behavioral therapies also may be needed.

Treatment of migraine

- Avoid factors that cause a migraine attack (for example, lack of sleep, fatigue, stress, certain foods, vasodilators).
- Treat accompanying conditions (for example, anxiety, depression).
- Oral birth control agents (contraceptives) may increase the frequency of headaches in females. Women may be advised to discontinue oral contraceptives (or to use a different form) for a trial period to see if they are a factor.

Migraine Headaches, Abortive treatment

Abortive treatments stop migraines quickly. Many drugs are now available for immediate treatment of migraine attacks. The goal is rapid and effective relief of headache. The most effective drugs for stopping a migraine are the triptans, which specifically target serotonin receptors. They are all very similar in chemical structure and action. The following is a list of triptans:

1. Sumatriptan (Imitrex, Imigran)
2. Zolmitriptan (Zomig, Zomig-ZMT)
3. Naratriptan (Amerge, Naramig)
4. Rizatriptan (Maxalt, Maxalt-MLT)
5. Almotriptan (Axert)
6. Frovatriptan (Frova)
7. Eletriptan (Relpax)

The following nontriptans also act on the serotonin receptors. They also act on some other receptors, most likely on those for dopamine and noradrenalin. Sometimes, they are effective when the triptans fail.

1. Ergotamine tartrate (Cafergot)
2. Dihydroergotamine (D.H.E. 45 Injection, Migranal Nasal Spray)
3. Acetaminophen-isometheptene-dichloralphenazone (Midrin)

The following are primarily used when nausea is a complicating factor in migraine headache. In some cases, they also help relieve the headache.

1. Prochlorperazine (Compazine)
2. Promethazine (Phenergan)

Combination drugs like butalbital-acetaminophen-caffeine (Fioricet), butalbital-aspirin-caffeine (Fiorinal), or acetaminophen with codeine (Tylenol With Codeine) are general painkillers in the narcotic class. They can help relieve any kind of pain to some degree, whereas the triptans, ergotamines, and Midrin are used specifically for headaches and do not help relieve arthritis, back pain, or menstrual cramps.

Treatment strategies are more successful if they are tailored to the individual patient and are initiated early in the headache.Patients with severe nausea and vomiting at the onset of an attack may at first respond best to intravenous prochlorperazine. These patients may be dehydrated; adequate fluid intake is necessary.Vasoconstrictors (agents that narrow the blood vessels), such as ergotamines or triptans, should not be given to patients with known complicated migraine without the advice of a headache specialist. Instead, acute attacks should be treated with one of the other available agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs) or prochlorperazine.Mild and infrequent attacks may not always require the use of ergotamines or triptans and may be adequately treated with acetaminophen (Tylenol), NSAIDs, propoxyphene (Darvon, Darvocet), or a combination of these.About 40% of all attacks do not respond to triptans or any other substance. If all else fails, migraineurs with an attack lasting more than 72 hours (status migrainous) can be treated with intravenous medications. Brief hospitalization may be needed.

Migraine headaches, preventive treatment

Patients who have frequent acute migraine attacks and report that the attacks affect their quality of life should consider preventive therapy as a supplement to the specific headache-stopping drugs (abortive treatments) they use.The goals of preventive therapy include decreasing the frequency and severity of acute attacks and improving quality of life.Patients with complicated migraine headaches who have a history of neurological symptoms associated with their attacks are definite candidates for preventive therapy. For these patients, even a single previous complicated migraine episode qualifies them for long-term preventive therapy. The choice of preventive medication should be tailored to the individual's profile, taking into account comorbidities (concurrent medical conditions) such as depression, weight gain issues, exercise tolerance, asthma, and pregnancy plans. All medications have side effects; therefore, selection must be individualized.Preventive drugs include beta-blockers, tricyclic antidepressants, some anticonvulsants, calcium channel blockers, cyproheptadine (Periactin), and NSAIDs such as naproxen (Naprosyn). Unlike the specific headache-stopping drugs (abortive drugs), most of these were developed for other conditions and have been coincidentally found to have headache preventive effects. The following drugs also have preventive effects; unfortunately, they also have more side effects:

- Methysergide maleate (Sansert): This drug has many side effects.
- Lithium (Eskalith, Lithobid): This drug has many side effects.
- Indomethacin (Indocin): This drug can cause psychosis in some people with cluster headaches.
- Steroids: Prednisone (Deltasone, Meticorten) works extremely well for some people and should be tried if other therapies fail.

How long a person should follow a preventive therapy plan is a function of his or her response to the drug being taken. If headaches completely stop, it is reasonable to gradually reduce the dosage so long as headaches do not recur.

Cluster Headaches

Cluster headaches have been called histamine cephalalgia, Horton neuralgia, and erythromelalgia. The causes of cluster headaches are not known with certainty. The mechanisms by which the body produces cluster headache pain and other symptoms are also not known for sure.

Cluster Headaches, Prevalence

Cluster headaches are rare. (Less than 1% of the population experience them.) People who do have such headaches usually start to have them when aged 20-40 years. Males get them more often than females (by a ratio of 5-8:1). Usually, no family history of cluster headaches is noted.

Cluster Headaches, Clinical features

Typically, cluster headaches come on with no warning. The signs and symptoms may include intense burning or penetrating pain, often described as a stabbing or hot poker sensation, in or around one eye or temple, occasionally spreading to the forehead, nose, cheek, or upper gum and jaw.

Cluster headaches usually occur on one side of the head. Pain is often penetrating and lasts from 15 minutes to 4 hours. Cluster headaches often cause people to awaken in the middle of the night. During a cluster headache, people are restless and may find relief in pacing or crying. Cluster headaches start rapidly over a few minutes. Periodicity (occurring at regular intervals) is characteristic of cluster headaches. Clusters of headaches are experienced, each cluster lasting as long as several months, once or twice a year. Using alcohol, histamines, or nitroglycerine during a cluster headache may worsen the attack.

Certain personality and physical characteristics have been associated with cluster headaches. A leonine (lionlike) appearance is one of them. Strong associations with smoking, alcohol use, and previous head and face trauma have been noted.

Cluster Headaches, Abortive treatment

Most of the headache-stopping drugs (abortive drugs) effective in treating migraine headaches are also effective in stopping cluster headaches, suggesting that the two types are related.

Oxygen therapy: This is the treatment of choice and is very safe and effective. Early in an attack, oxygen delivered through a face mask has been known to either stop an attack or diminish its intensity. Why this works is unknown.
Occipital nerve steroid injection (methylprednisolone acetate [Depo-Medrol]): An injection of this drug may stop a cluster headache attack.

Cluster Headaches, Preventive therapy
As with the abortive drugs, most of the preventive drugs effective in treating migraine headaches are also effective in preventing cluster headaches, again suggesting that the two types are related.

Daily Chronic Headache
Daily chronic headache is defined as a headache that is present for more than 15 days a month and for at least 6 months a year. Three main types are noted: chronic tension-type headache, migraine chronic tension-type headache complex, and rebound (analgesic abuse) headache. How the body produces chronic daily headaches is not well understood. They have been associated with depression, anxiety, bipolar disorders, panic attacks, mouth/jaw problems, stress, and drug overuse.

Chronic tension-type headache
Chronic tension-type headaches are not associated with a history of migraine or cluster headaches. Patients report almost constant daily headaches of mild-to-moderate intensity. The headache is described as a feeling of tightness or pressure that is not worsened, and may actually be improved, by activity. Patients with chronic tension-type headaches can carry on their daily activities. Nausea and photophobia (sensitivity to light) may occur, but vomiting usually does not. A small group of patients may have head and neck tenderness.

Chronic tension-type headache, Treatment

Patients who are less responsive to previous treatment and those with conditions like depression and stress may be good candidates for psychological treatments. Biofeedback has been successful in patients with tension headache. They are taught how to relax their tense muscles. Thermal biofeedback, in which patients are taught to increase their body temperatures to improve their headaches, has also worked. Other less conventional treatments, such as relaxation training and stress-coping training, may be helpful in the long term.

Transformed migraine
Migraine transformation has been a term used by some experts to describe when intermittent migraines become daily migraines. This type of headache is believed to be associated with analgesic or ergotamine overuse. Patients report intermittent typical migraine attacks along with the daily chronic headaches.

Transformed migraine, Treatment

Stopping all analgesics and headache-related medications is best done in an inpatient setting. Doctors may prescribe a clonidine (Catapres) patch to lessen withdrawal symptoms if narcotic analgesics are involved. Preventive: Preventive treatments for transformed migraine headaches are identical to those used for the other types of migraine headache.

Other uncommon chronic headaches

Hemicrania continua and chronic paroxysmal hemicrania are uncommon forms of chronic headache. Chronic paroxysmal hemicrania is a severe chronic headache similar to cluster headache. It has a male predominance. The headaches are paroxysmal (pulsing), with pain in the temple/eye region lasting 20-30 minutes. The paroxysms occur several times a day. This type of headache can last several years. Treatment with indomethacin (Indocin) results in a dramatic response.

Secondary Headaches

Secondary headaches are related to physical problems and include the following:

- Space-occupying intracranial (inside the head) lesions: The headaches associated with intracranial tumors are initially paroxysmal. Classic headaches of this type wake a person from sleep at night and are associated with projectile vomiting. With time, the headaches may become continuous and intensify with activities that increase intracranial pressure (for example, coughing, sneezing).
- Meningeal irritation: Meningitis, especially the chronic forms (tuberculous, fungal), can irritate the meninges (membrane covering the brain and spinal cord) and result in chronic headaches. The headaches are often diffuse (spread out).
- Posttraumatic headache: Headache can be part of a postconcussion syndrome. Patients may report vague headaches, fatigue, memory problems, and irritability for months or years after the traumatic event.
- Temporal arteritis: This is an inflammation of some of the arteries of the extracranial (outside the skull) arteries. The headache is generally localized to the affected side and may be worsened by chewing.
- Post-lumbar puncture (spinal tap) headache: Lumbar puncture can cause a headache that is worsened by sitting up from a lying position. It usually goes away by itself after the person drinks fluids and has caffeine in some form.
- Referred pain: Headache may be a form of referred pain from neighboring structures. Dental disease can cause chronic headaches. Upper neck diseases or arthritis can also cause headaches. People with acute sinus or jaw problems can experience headaches; however, uncomplicated chronic sinusitis does not cause headaches.
- Idiopathic intracranial hypertension (benign intracranial hypertension, pseudotumor cerebri): This disorder, most common in young women, is due to increased intracranial (within the head) pressure in the absence of any structural central nervous system abnormality or obstruction to the flow of cerebrospinal fluid.

Conclusion

Migraine is often under-diagnosed and under-treated. There is no cure for migraine. Nevertheless, there are numerous interventions that may help restore an improved life for migraine sufferers. These measures should consider the various aspects of the particular patient's condition. Triggering factors, nerve inflammation, blood vessel changes and pain are each addressed aggressively. Up to 18% of pregnant women are affected by migraine. In most of these patients, a diagnosis of migraine is made before pregnancy. Differentiation of migraines from

other complications that develop during pregnancy and the puerperium can be difficult, but is of the utmost importance. The clinician is often confronted by coexisting migraine in the setting of pre-eclampsia. Distinguishing between these two conditions is crucial because while migraine does not necessitate delivery, which can place the fetus at risk of prematurity-related complications, persistent headache in the context of pre-eclampsia can be an indication for delivery to prevent further maternal complications.Individualizing treatment is essential for optimal outcome. The most effective treatment is to identify and avoid the trigger-factors. Aspirin and other analgesics can be taken. Soluble aspirin is better as it is absorbed faster than others and hence if taken at the first sign of headache the severity of the attack is reduced considerably. Many newer drugs are available for the treatment of migraine but are costly and should be kept in reserve. These must be on prescription by the doctor. Alternative therapies like acupuncture, improving posture, stress management, massage of the tensed muscles, etc. are also effective is some cases. This consists of the treatment of any identified underlying cause or disease. The application of cold or heat compresses is done to relieve symptoms. Elimination of food or environmental allergens will help to prevent headaches caused by these factors. Counselling and psychological treatment may also be required. Stress management and biofeedback will also be helpful. Migraine headache is thought to be due to vascular disturbances. Cluster headaches are also vascular in nature. The exact cause of migraine has not been clearly defined. Vascular disturbances can also be caused by exposure to toxic chemicals like alcohol, lead, arsenic, and carbon monoxide, and are also causes of headache. The most common type of headache is tension headache, but their precise cause is not well-defined. However, most are related to muscle tension, minor trauma, increased stress or anxiety, food and environmental allergens, infection or lesions of the oral or nasal cavity, ear infections, or eyestrain.

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