

HEADACHE

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Up to 40% of people have a significant headache at some time in their lives. One in five Americans has a headache at least once weekly. As many as 10% of people see a healthcare provider for episodic headache, and non-traumatic headache accounts for 12% of all emergency department visits. There are approximately 300 pathological etiologies for headache, making the definitive diagnosis of headache etiology quite challenging.

HEADACHE ETIOLOGY

Headaches present in four general patterns. First is a simple minor headache for which the patient requires no treatment, does not seek medical attention, and recognizes on his or her own that this is a benign, self-limiting process. This patient does not present for emergency care because his or her perception of the problem is that whatever the cause, he or she can manage well enough on their own. This points to the fundamental difference from this patient and those presenting for care in the emergency department; the perception is that this headache is either too severe or caused by a problem too serious for them to manage at home.

The remaining headaches then are considered to be of a somewhat greater severity and can be divided into three groups based on pattern of the headache. Some patients will present with a first occurrence of an acute headache and have no history of severe headache (therefore no prior work-up). Other patients will present with complaints of a chronic recurrent headache (such as migraine, tension) and will have had a thorough work-up conducted previously. There is no change in the headache pattern and this patient will have presented for pain control. The remaining patients will have headache as part of a systemic syndrome.

1. **Serious Etiologies**

- Subarachnoid Hemorrhage (SAH)
- Infection
- Arteritis
- Increased Intracranial Pressure
- Glaucoma

2. **Uncomfortable, Benign**

- Vascular/Migraine
- Tension
- Cluster
- Other

3. **Other Secondary Headaches**

- Dental
- Sinus
- Toxin (caffeine, cocaine)

EMERGENCY DEPARTMENT EVALUATION

An extensive work-up is both costly and time consuming. These general guidelines are helpful to decide who is likely to require an extensive work-up.

Clinical presentations that suggest secondary headaches and should prompt further work-up:

- Sudden onset headache
- Headache occurring for the first time in a patient > 50 years old

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- A change in the character of a chronic or recurrent headache syndrome
- Any headache described as the “worst headache of my life”, especially if associated with nuchal rigidity and/or focal neurologic signs
- Headaches associated with fever, photophobia, and/or neck stiffness
- Headaches that worsen under observation
- Headaches associated with an abnormal neurologic exam (including focal neurologic findings, changes in mental status, or changes in personality)
- Headaches of increasing severity over days to weeks

GOLDEN RULES

When evaluating patients with headache in the emergency department it may be helpful to consider two basic rules:

1. Every patient has a serious life threatening etiology for his headache until proven otherwise
2. If the patient ever passes out, do not send him home.

Critical History

Physical examination is often non-diagnostic in the search for the cause of a headache. This makes a thorough history essential. Certain historical points are critical to making an accurate diagnosis. Timing of onset of symptoms can be quite helpful. Sudden, severe headache sometimes described as a “thunderclap” headache perhaps following vigorous exercise or intercourse suggests the possibility of subarachnoid hemorrhage. Symptoms may demonstrate a regular pattern or duration, perhaps in conjunction with prodromal symptoms. Symptoms may be correlated with ingestion of certain foods, medications, or with menstrual cycling. Presence of a prodrome itself begins to suggest migrainous or vascular etiologies for the headache.

Location of the headache will not clinch the diagnosis but may be helpful. Two-thirds of migraine headaches are unilateral. A headache that recurs in the same place every time may suggest a mass lesion. In contrast, tension headaches may be bilateral or circumferential.

The quality of the pain may vary from the throbbing pain associated with vascular headache (thought to be vasodilatation of extracranial arteries) to the transient pain of trigeminal neuralgia. Cluster headaches are usually associated with a deep piercing unilateral pain. Associated symptoms such as photophobia or fever are also helpful. If the patient notes fever only at home this must not be discounted. Family history and other acute or chronic illness should be elicited as well.

Other Key History Points

Certain questions must always be asked to prevent overlooking serious etiologies of headache. Every patient must be asked if there was a loss of consciousness. Any history of trauma, regardless of how trivial it may seem to the patient, must be noted. Not all patients recall what medications they take so it is worth while to ask specifically if they take warfarin or other “blood thinning” medications.

Attempting to narrow the differential diagnosis, it is beneficial to try and summarize the history and place it within a broad category. For example, a patient with a continuous headache that is progressive over weeks to months should be considered for evaluation for the presence of mass or tumor. A patient with a headache different from usual recurrent headaches, or new onset in persons over age 40-50 with a new onset headache, are somewhat more likely to have a serious organic cause for the etiology.

Presence of certain accompanying symptoms can also be helpful. Nausea and vomiting are seen most commonly with migraine headache and after head trauma. Fever is usually only seen in the presence of infection but can be seen with subarachnoid hemorrhage. Facial weakness or neurological symptoms (such as numbness, paresthesia, and blurred or double vision) support the diagnosis of mass or tumor but can be seen in the migraine patient.

One other area to consider is precipitating factors. Emotional stress and depression may lead to tension headaches. Migraine headaches may be precipitated by menses, exposure to carbon monoxide, or even by caffeine withdrawal.

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Physical Examination

The majority of headache patients will have normal vital signs. This makes it very necessary to explain abnormal vital signs in a patient presenting with headache. Hypertension may be associated with subarachnoid hemorrhage. Tachycardia is nonspecific and may be associated with severe pain. Tachypnea can also be associated with severe pain, but consideration should be given to carbon monoxide exposure or hypoxia. The presence of even a low-grade fever should prompt consideration of infectious processes to include meningitis, encephalitis, or abscess.

The bulk of the examination of patients presenting with headache is comprised by a thorough neurologic examination, and examination of the head and neck in general. There are several fundoscopic findings that must be sought out. Papilledema suggests intracranial pressure as does lack of retinal venous pulsation in an erect patient. This is especially important if the patient had venous pulsation on earlier examination. Acute hemorrhagic exudates are consistent with hypertensive encephalopathy.

Palpation of the head enables the examiner to recognize occult trauma, detect muscle spasm, or elicit tenderness of the temporal arteries. Tenderness when palpating and percussing the sinuses, dental tenderness to percussion, or tenderness in the trigeminal nerve distribution may be enlightening regarding etiology. Regardless of findings from this examination the patient has a heightened sense of thoroughness and caring on the part of the practitioner, creating genuine therapeutic benefit.

During examination of the neck, attention is given to signs of trauma or nuchal rigidity. To elicit nuchal rigidity it is helpful to have the patient lay supine and be as relaxed as possible. The examiner then raises the head gently from the bed. Nuchal rigidity is defined as an involuntary resistance to flexion when the examiner flexes the neck. This is often a subtle finding, and not simply pain with flexion. With marked meningismus (particularly in the child or infant) the neck musculature will spasm firmly to protect against the pain associated with flexion. It may be possible to literally lift the patient off the bed with an attempt at flexion.

Even subtle involuntary resistance to flexion should be noted and not ignored. Additionally, involuntary hip flexion as the neck is flexed (Brudzinski sign) or pain in the neck and resistance as the knee is extended (Kernig sign) may be useful in eliciting nuchal rigidity.

Patients will often present with a complaint of a "stiff" neck. This complaint may be triaged verbatim by paramedics and/or support staff. The lay public is cautioned through the media that headache, fever, and stiff neck are warning signs of possible meningitis. Reports of community outbreaks may prompt individuals to present to the ED with specific concerns about meningitis. Many of these patients may have fairly mild symptoms due to some other process, but have a high level of anxiety. Most patients would define stiff neck as one that would not move because of pain. In medical terminology we would more exactly refer to stiffness as a joint which should be mobile and cannot be moved. However, it would seem preferable to have these patients evaluated in the emergency department and be screened and relieve their concerns, rather than not disseminating this information to the public.

Certain signs should be considered when evaluating the peripheral nervous system. Pronator drift is a good indicator of subtle abnormalities. To elicit drift have the patient stand with arms extended to the front, palms up, eyes closed for a period of 10 seconds. Note any drift or pronation of the extremities. When considering a posterior fossa mass the patient should be specifically examined regarding gait (heel walking, toe walking, tandem walking, and walking on outer edges of the feet) and cerebellar function (finger to nose, heel to shin).

HEADACHE SYNDROMES

Subarachnoid Hemorrhage

Although this is not the most common headache syndrome, subarachnoid hemorrhage has such severe consequences it is a concern that must be addressed in every headache presentation to the emergency department. This is a frequently misdiagnosed headache etiology. The mortality rate from subarachnoid hemorrhage approaches 50-60%, yet when warning signs are identified and the patient receives prompt treatment good outcomes are possible. Untreated, 50% of those who survive the first 24 hours of a subarachnoid hemorrhage will die within two weeks. Interestingly, approximately 5% of the population have a pre-existing berry aneurysm, increasing risk of subarachnoid hemorrhage.

Warning symptoms of subarachnoid hemorrhage result from a variety of causes. Aneurysmal expansion may result in cranial nerve palsy (especially oculomotor, CN III), localized head pain, or visual

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field defects. A sentinel bleed may result in generalized headache with or without nausea and vomiting, but generally without photophobia or meningismus.

Frequently presenting symptoms include sudden severe headache (“thunderclap”) which is focal at onset and then spreads, seizures, diminished level of consciousness, focal neurologic deficits, dizziness, vertigo, and vomiting. Common signs include clouding of consciousness, fundoscopic changes, nuchal rigidity, delirium, and other neurologic and cardiac effects. Because of the low specificity of presenting symptoms, subarachnoid hemorrhage is often misdiagnosed as a systemic infection, migraine, “flu”, sinusitis, tension headache, cervical disc herniation, or hypertensive encephalopathy.

When evaluating these patients up to 15% will have normal non-contrast CT scans. Contrast actually reduces the sensitivity of identifying subarachnoid hemorrhage. For this reason when subarachnoid hemorrhage is being considered in earnest, a lumbar puncture must be obtained after the scan to rule out the presence of xanthochromia. Some patients with an expanding aneurysm may have both negative scans and lumbar punctures requiring cerebral angiography to make the definitive diagnosis. Patients requiring such an evaluation would be those with a remote history of a possible aneurysm or a strong family history of intracranial aneurysm.

Treatment of subarachnoid hemorrhage consists of analgesia, nimodipine to reduce spasm, and measures directed to reduce intracranial pressure. The use of intubation and subsequent hyperventilation to induce hypocapnia can be helpful to lower intracranial pressure. In some populations agents such as mannitol or furosemide are used as diuretics to reduce intracranial pressure but are used much less commonly than in the past. Hypertension should be controlled but mean arterial pressure generally does not need to be reduced below approximately 120-140 mm Hg. Reducing mean arterial pressure to normal or below normal levels in the face of increased intracranial pressure may result in cerebral hypoperfusion and have serious consequences. Definitive management in the OR may be undertaken based on the results of the CT scan or may require cerebral angiography to better define the source of bleeding prior to surgery.

Meningitis

Headache associated with meningitis usually involves the entire head. It is usually associated with fever and a stiff neck (nuchal rigidity). Diagnosis and treatment of meningitis are discussed elsewhere.

Temporal Arteritis

Headache associated with temporal arteritis is usually severe, throbbing in nature and located over the frontotemporal region. There may be jaw claudication or strong association with polymyalgia rheumatica. The involved temporal artery may be nonpulsatile or tender, or have a diminished pulse. The most serious complication of temporal arteritis is non-reversible loss of vision.

Treatment should begin immediately when there is strong suspicion of temporal arteritis in order to prevent blindness. Begin oral steroid therapy with 40-60 mg of prednisone daily and urgently refer for temporal artery biopsy.

Space Occupying Lesions

Mass lesions causing increased intracranial pressure include intracranial hemorrhages (epidural and subdural hematoma), tumors, and abscesses. The brain parenchyma is not pain sensitive, so in order to have symptoms (headache) pressure must involve pain sensitive structures such as meninges or vasculature.

Suspicion of tumor should increase if there is pain upon awakening, pain worsens with a valsalva maneuver, or the headache is new and associated with nausea and vomiting. Focal neurologic deficits may or may not be present.

CT scan with contrast makes the diagnosis.

Brain abscess will present similarly to other space occupying lesions but may have accompanying fever and infection such as frontal sinusitis.

Pseudotumor Cerebri (benign intracranial hypertension) should also be considered in the differential diagnosis. This is a non-specific headache accompanied by visual complaints and papilledema on examination. CT scan reveals slit-like ventricles without mass effect. Diagnosis (elevated opening pressure) and treatment (relief of pressure) are made by lumbar puncture.

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Glaucoma

Acute glaucoma may present as a headache accompanied by nausea, vomiting, and orbital pain described as a headache. Patients with iritis or optic neuritis may describe eye or supraciliary pain as a headache.

Careful history should aid in confirming these types of diagnoses. Thorough eye examination to include measuring intraocular pressures makes the diagnosis.

Other Major Causes of Headache

Other presentations to consider in the differential diagnosis of headache include temporal mandibular joint pain, hyperopic eye strain, sinusitis, trigeminal neuralgia, post concussive headache, muscular lesions, and dental lesions.

Although painful, these etiologies carry far less potential for bad outcome. History and thorough physical examination are key to diagnosis.

Migraine Headache and Variants

Vascular headaches have been traditionally divided into classic migraine (with aura) and common migraine (without aura). Migraine headaches may have a variable aura but tend to have a predictable unilateral pattern. The aura represents transient neurologic changes and it is postulated that these may be due to ischemia during vasoconstriction.

The mechanism for migraine remains unclear and controversial. One theory suggests that stress results in vasospasm of cerebral arteries. Another more recent theory is that serotonin has been implicated as the cause for vasoconstriction of the innervated vascular system. Either of these mechanisms can lead to the aura that is experienced. There is subsequent serotonin absorption followed by reaction with histamine, tyramine, bradykinin, free fatty acids and prostaglandins that result in a sterile perivascular inflammation. Serotonin metabolism results in a rapid decrease in blood serotonin which in turn leads to rebound vasodilatation of arteries and capillaries. This distention and inflammation results in pain.

Classic migraine usually presents with prodromal symptoms that are fairly constant to the patient. Among the most common complaints are scintillating scotoma, homonymous hemianopsia, or photophobia. Patients may also complain of hand or face tingling, extremity weakness, or mild aphasia, all of which suggest a more concerning headache etiology. Many patients experience nausea and vomiting with peak headache intensity. Duration of the headache is typically 6 -12 hours.

Common migraine may not have any of the well-defined neurologic symptoms of classic migraine. There may be only a vague prodrome such as irritability. A positive family history and two additional findings from nausea and vomiting, photophobia, unilateral pain, throbbing, or increased incidence associated with menses strongly suggest the diagnosis of common migraine.

Other causes of vascular headache not thought to be migraine include hypoxia, carbon monoxide exposure, acute anemia, diastolic blood pressure greater than 130 mm Hg, tyramine ingestion, nitroglycerine ingestion, and certain metabolic causes. Some toxic exposures will cause this type of headache as well, such as the headache associated with alcohol hangover (caused by acetaldehyde, an ethanol metabolite and potent vasoconstrictor).

Treatment consists of abortive or preventive therapy. Abortive therapy can be accomplished with many different medications to include dihydroergotamines or other serotonin receptor agonists, antiemetics, anti-inflammatory medications, and fluid hydration. Opioid analgesics should be avoided and may even cause exacerbation of headache. Preventive therapy may be accomplished with many types of medications to include beta-blockers, calcium-channel blockers, tricyclic antidepressants, and non-steroidal anti-inflammatory medications. Preventive therapy should not be started in the emergency department without first obtaining neurological consultation.

Tension Headache

It was previously thought that tension headache resulted from extracranial muscle tension. This relationship has been questioned and current thought is that there is some connection between tension-type headaches and migraine headache via pathophysiology and they simply represent different ends of a spectrum.

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To differentiate tension from migraine tension is defined as bilateral, non-pulsating headache not worsened by exertion or accompanied by nausea and vomiting. However, when severe, tension headache may be accompanied by nausea and vomiting.

Treatment is effected with mild analgesics or non-steroidal anti-inflammatory medications. When severe, treatment may consist of modalities similar to abortive treatment of migraine headache.

Cluster Headache

Cluster headache is generally rare, frequently occurring in men. The onset is usually after 20 years of age and there is usually no significant family history of headache. Trigeminal nerve dysfunction is thought to be the cause of cluster headaches and the fact they respond to serotonin agonists suggests a commonality with migraine headache.

The pain is characterized by sudden, severe unilateral orbital, supraorbital, or temporal pain lasting from a few minutes to three hours. The pain is unrelenting and the patient appears uncomfortable, unable to remain at rest. The pain may be accompanied by one of the following signs on the ipsilateral side: injected conjunctiva, tearing, rhinorrhea, miosis, or ptosis.

Because of the transient nature of the pain any attempt at relief must come from a fast acting modality. 100% oxygen has been shown to be effective relief in up to 70% of these patients. Serotonin agonists (which act rapidly) have also had success in relieving this pain. Oral agents are poorer alternatives since they take longer to sustain the desired effect. Non-steroidal anti-inflammatory medications may be of some use in reducing the frequency or severity of attacks.

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