CHAPTER 17

Headache

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PERSPECTIVE

Headache is consistently among the top reasons for visit to the emergency department (ED). The vast majority of patients who have a primary complaint of headache do not have a serious medical cause for the problem. Most common primary headache etiologies are benign, such as tension headache and migraine. A minority of headaches will be secondary to an underlying medical or surgical condition; a patient may present with headache due to a serious life-threatening disease requiring prompt diagnosis and treatment. The low incidence of serious disease can create a "needle in the haystack" phenomenon, and headache is disproportionately represented in emergency medicine malpractice claims despite widespread overuse of imaging for benign headache conditions. Although representing only 0.5% to 6% of presentations of acute headache to the ED, the most important and commonly encountered life-threatening cause of severe sudden head pain is subarachnoid hemorrhage (SAH).[1] Unfortunately, this is a diagnosis that is also missed on first presentation over 25% of the time.[2] The other significant, potentially life-threatening causes of headache occur even less frequently. As is the case for SAH, these other serious disorders (ie, meningitis, carbon monoxide poisoning, temporal arteritis, acute angle-close glaucoma, intracranial hemorrhage [ICH], cerebral venous sinus thrombosis, and increased intracranial pressure) can often be linked with specific historical elements and physical findings that facilitate their diagnosis.

Pathophysiology

The brain parenchyma is insensitive to pain. The pain-sensitive areas of the head include the meninges, the arteries and veins supplying the brain, and the various tissues lining the cavities within the skull. The ability of the patient to specifically localize head pain is often poor. Much of the pain associated with headache, particularly with vascular headache and migraines, is mediated through the fifth cranial nerve. Such pain may proceed back to the nucleus and then be radiated through various branches of the fifth cranial nerve to areas not directly involved. Inflammation in a specific structure (eg, periapical abscess, sinusitis, or trigeminal neuralgia) is much easier to localize than the relatively diffuse pain that may be generated by tension or traction headaches. Pains in the head and neck may easily overlap. They should be thought of as a unit when complaints of headache are considered.

DIAGNOSTIC APPROACH

Differential Diagnosis Considerations

The differential diagnosis of headache is complex due to the large number of potential disease entities and the diffuse nature of many types of pain in the head and neck region (Table 17.1). In evaluating the patient with a primary complaint of headache, the top priority is to exclude the causes with significant morbidity and mortality: SAH, ICH, meningitis, encephalitis, and mass lesions. Carbon monoxide is an exogenous toxin, the effects of which may be reversible by removing the patient from the source and administering oxygen. Carbon monoxide poisoning is a rare example of a headache in which relatively simple interventions may quickly improve a critical situation; however, returning the patient to the poisoned environment without a diagnosis could be lethal (see Chapter 153).

Pivotal Findings

Physical findings may be minimal or nonspecific, even in serious causes of headache, so the history is the pivotal part of the evaluation (Table 17.2).

1. Determine the *pattern and the onset* of the pain. Patients may remember having had frequent and recurrent headaches similar to the one they have on the current ED visit; a marked variation in the headache pattern can signal a new or serious problem. A rapid and severe onset of pain ("thunderclap") has been associated with serious causes of headache, and this warrants strong consideration of a cerebrovascular etiology.[3] Slow onset of headache should not be solely relied on to rule out a potentially life-threatening cause, and the nature of the onset usually is not possible to ascertain if the headache came on during sleep.

   Almost all studies dealing with subarachnoid bleeding report that patients moved from the pain-free state to severe pain within seconds to minutes. The thunderclap headache is common in acute presentations of SAH but is not highly specific. If the patient with moderate or severe headache can indicate the precise activity in which he or she was engaging at the time of the onset of the headache, the suddenness of onset warrants consideration of SAH. Careful questioning about the onset of headache may lead to the correct diagnosis of SAH, even if the pain is improving at the time of evaluation.
2. The patient's *activity at the onset of the pain* may be helpful. Headaches that come on during exertion have a relationship to vascular bleeding.[4] Additionally, although the syndrome of postcoital headache is well known, coitus is also recognized as an activity associated with SAH, so a pattern of previous postcoital headache is key, as is understanding whether the current headache fits that pattern. Postcoital headaches require the same evaluation on initial presentation as any other exertion-related head pain.
3. If there is a history of head trauma, the differential diagnosis shifts markedly toward epidural and subdural hematoma, traumatic SAH or intraparenchymal hemorrhage, skull fracture and closed head injuries, such as concussion and diffuse axonal injury.
4. The *intensity of head pain* is difficult to quantify objectively. Almost all patients who come to the ED consider their headaches to be severe. Use of a pain scale with appropriate explanation may help differentiate patients initially but has more value in monitoring their response to therapy. Rapid resolution

TABLE 17.1

Headache Etiologies and Associated Spectrum of Severity of Disease by System

ORGAN SYSTEM	CRITICAL	EMERGENT	NONEMERGENT
CNS, neurologic, vessels	SAH Carotid dissection Venous sinus thrombosis	Shunt failure Traction headaches Tumor or mass Subdural hematoma Reversible cerebral vasoconstriction syndrome	Migraine, various types Vascular headache, various types Trigeminal neuralgia Post-traumatic (concussion) Post LP headache
Toxic/metabolic, environmental	Carbon monoxide poisoning	Mountain sickness	
Collagen vascular disease	Temporal arteritis		
Ocular/ENT		Glaucoma	Sinusitis Dental problems TMJ disease
Musculoskeletal			Tension headache Cervical strain
Allergy			Cluster or histamine headaches
Infectious disease	Bacterial meningitis Encephalitis	Brain abscess	Febrile headaches, non-neurologic source
Pulmonary or oxygen		Anoxic headache Anemia	
Cardiovascular		Hypertensive crisis	Hypertension (rare)
Unspecified		Preeclampsia IIH	Effort-dependent or coital headaches

CNS, Central nervous system; *ENT,* ear, nose, and throat; *IIH,* idiopathic intracranial hypertension; *LP,* lumbar puncture; *SAH,* subarachnoid hemorrhage; *TMJ,* temporomandibular joint.

TABLE 17.2

Signs and Symptoms of Various Headache Etiologies

SYMPTOM	FINDING	POSSIBLE DIAGNOSIS
Sudden onset of pain	"Thunder clap" with any decreased mentation, any positive focal finding, meningismus or intractable pain	SAH, cervical artery dissection, cerebral venous thrombosis
Sudden onset of pain	Recurrent thunder clap episodes, may be associated with stroke-like symptoms	Reversible cerebral vasoconstriction syndrome
"Worst headache of my life"	Associated with sudden onset	SAH, cervical artery dissection, cerebral venous thrombosis
Near syncope or syncope	Associated with sudden onset	SAH, cervical artery dissection, cerebral venous thrombosis
Increased with jaw movement	Clicking or snapping; pain with jaw movement	TMJ disease
Facial pain	Fulminant pain of the forehead and area of maxillary sinus; nasal congestion	Sinus pressure or dental infection
Forehead and/or temporal area pain	Tender temporal arteries	Temporal arteritis
Periorbital or retro-orbital pain	Sudden onset with tearing	Temporal arteritis or acute angle closure glaucoma

SAH, Subarachnoid hemorrhage; *TMJ,* temporomandibular joint.

of pain in the ED, either from time or therapy, should not be relied on to rule out serious causes of headache.[5]

5. The *character of the pain* (eg, throbbing, pressure), although sometimes helpful, may not be adequate to differentiate one type of headache from another.
6. The *location of head pain* at onset and as the pain progresses is helpful when the patient can identify a specific area. It is certainly useful to direct the examination to evaluate for externally visible contributing factors, such as an infectious process. Unilateral pain is more suggestive of migraine or localized inflammatory process in the skull (eg, sinus) or soft tissue. Muscle tension headache often starts at the base of the skull and can extend over the entire head, following the occipital-frontal aponeurosis. Temporal arteritis, temporomandibular joint (TMJ) disease, dental infections, and sinus infections frequently have a highly localized area of discomfort. Meningitis, encephalitis, SAH, and even severe migraine, although intense in nature, are usually more diffuse in their localization.

BOX 17.1

Emergent Causes of Headache and Associated Risk Factors

1. Carbon monoxide poisoning
   a. Breathing in enclosed or confined spaces with engine exhaust or ventilation of heating equipment
   b. Multiple household members with the same symptoms
   c. Wintertime and working around machinery or equipment producing carbon monoxide (eg, furnaces)
2. Meningitis, encephalitis, abscess
   a. History of sinus or ear infection or recent surgical procedure
   b. Immunocompromised state
   c. General debilitation with decreased immunologic system function
   d. Acute febrile illness—any type
   e. Extremes of age
   f. Impacted living conditions (eg, military barracks, college dormitories)
   g. Lack of primary immunization
3. Temporal arteritis
   a. Age >50
   b. Females more often than males (4 : 1)
   c. History of other collagen vascular diseases (eg, systemic lupus)
   d. Previous chronic meningitis
   e. Previous chronic illness, such as tuberculosis, parasitic or fungal infection
4. Glaucoma—acute angle closure
   a. Not associated with any usual or customary headache patterns
   b. History of previous glaucoma
   c. Age >30
   d. History of pain increasing in a dark environment
5. Increased intracranial pressure
   a. History of previous benign intracranial hypertension
   b. Presence of cerebrospinal fluid (CSF) shunt
   c. History of congenital brain or skull abnormalities
   d. Female gender
   e. Obesity
6. Cerebral venous sinus thrombosis
   a. Female gender
   b. Pregnancy, peripartum, hormone replacement therapy or oral contraceptive use
   c. Prothrombotic conditions
7. Reversible cerebral vasoconstriction syndrome
   a. Episodic sudden severe pain, with or without focal neurological findings or seizure
   b. Recurrent episodes over a period up to several weeks
   c. Exposure to adrenergic or serotonergic drugs
   d. Postpartum state
8. Intracranial hemorrhage (ICH)
   a. Subarachnoid hemorrhage (SAH)
      i. Sudden and severe pain; "worst headache of life"
      ii. Acute severe pain after sexual intercourse or exertion
      iii. History of SAH or cerebral aneurysm
      iv. History of polycystic kidney disease
      v. Family history of SAH
      vi. Hypertension—severe
      vii. Previous vascular lesions in other areas of the body
      viii. Young and middle-aged
   b. Subdural hematoma
      i. History of alcohol dependency with or without trauma
      ii. Current use of anticoagulation
   c. Epidural hematoma
      i. Traumatic injury
      ii. Lucid mentation followed by acute altered mentation or somnolence
      iii. Anisocoria on physical examination

7. *Exacerbating or alleviating factors* may be important. Patients whose headaches rapidly improve when they are removed from their environment or recur each time they are exposed to a particular environment (eg, basement workshop) may have carbon monoxide poisoning. Most other severe causes of head pain are not rapidly relieved or improved when patients get to the ED. Intracranial infections, dental infections, and other regional causes of head pain tend not to be improved or alleviated before therapy is given.
8. *Associated symptoms and risk factors* may relate to the severity of headache but rarely point to the specific causes (Box 17.1). Nausea and vomiting are nonspecific symptoms seen in both primary and secondary headaches, but they are rare in simple muscle tension headache. Migraine headaches, increased intracranial pressure, temporal arteritis, and glaucoma can all manifest with severe nausea and vomiting, as can some systemic viral infections with headache. Such factors may point toward the intensity of the discomfort but are not specific in establishing the diagnosis. Immunocompromised patients are at risk for unusual infectious causes of headache, which may present with deceptively low grade symptomatology. Toxoplasmosis, cryptococcal meningitis, and abscess are very rare but may be seen in patients with a history of human immunodeficiency virus (HIV) or other immunocompromised state. This subset of patients may have a serious central nervous system infection without typical signs or symptoms of systemic illness (eg, fever and meningismus).

   Another special population to considers is the pregnant and peripartum woman, in whom preeclampsia, idiopathic intracranial hypertension (IIH), and reversible cerebral vascular syndrome should be considered, as well as the even more serious causes of headache including venous sinus thrombosis, pituitary apoplexy, cervical artery dissection, and stroke.[5-7]

   Patients on medications containing estrogen are also at higher risk for thrombotic events, such as cavernous venous thrombosis, and this should be considered in the differential diagnosis.
9. A *prior history of headache,* although helpful, does not rule out current serious problems. One important consideration is the association of migraine headaches and stroke, with particular consideration of carotid dissection.[8] Previous evaluation for serious disease can be useful to guide the current evaluation. Prior visits to an ED or outpatient setting, computed tomography (CT), magnetic resonance imaging (MRI), and other forms of testing can provide support for, or help rule out, a specific diagnosis. Patients with migraine, cluster, and tension headaches tend to have stereotypical recurrent patterns. Adherence to these patterns is also helpful in deciding the degree to which a patient's symptoms are pursued.

Signs

There are signs that may be elicited on physical examination that can be particularly high yield. For example, deficits of extraocular movements localizing to cranial nerves (CNs) III, IV, and VI may indicate the presence of increased intracranial pressure due to mass lesion or IIH. When headache is associated with an acutely red eye, this finding should prompt consideration of acute angle closure glaucoma and further investigation with testing of

intraocular pressure. Any focal neurological deficit found on examination, regardless of subtlety, warrants further investigation. Not all signs associated with headache contribute greatly to final determination of diagnosis, but they may serve as cues for further consideration of a serious intracranial process. Nausea and vomiting are often associated with migraine, but they are also associated with intracranial mass, acute angle closure glaucoma, intracranial bleeding, and carbon monoxide poisoning. Additional physical findings associated with various forms of headache are listed in Table 17.3.

Ancillary Testing

The vast majority of headache patients do not require additional testing (Table 17.4). Advanced imaging should be directed toward the specific disease of concern in the differential diagnosis and not

TABLE 17.3

Signs and Symptoms Associated With Different Headache Etiologies

SIGN	FINDING	POSSIBLE DIAGNOSES
General appearance	Nonfocal mental status changes	Meningitis, encephalitis, SAH, subdural hematoma, anoxia, increased intracranial pressure, carbon monoxide poisoning
	Mental status changes with focal findings	Intraparenchymal bleed, tentorial herniation, stroke
	Severe nausea, vomiting	Increased intracranial pressure, acute-angle closure glaucoma, SAH, carbon monoxide poisoning
Vital signs	Hypertension with normal heart rate or bradycardia	Increased intracranial pressure, SAH, tentorial herniation, intraparenchymal bleed, preeclampsia, reversible cerebral vasoconstriction syndrome
	Tachycardia	Anoxia, anemia, febrile headache, exertional or coital headache
	Fever	Febrile headache, meningitis, encephalitis
HEENT	Tender temporal arteries	Temporal arteritis
	Increased intraocular pressure	Acute angle closure glaucoma
	Loss of venous pulsations on funduscopy or papilledema	Increased intracranial pressure, mass lesions, subhyaloid hemorrhage, SAH, cerebral venous thrombosis
	Acute red eye (severe ciliary flushing) and poorly reactive pupils	Acute angle closure glaucoma
Neurologic	Enlarged pupil with third nerve palsy	Tentorial pressure cone, mass effect (aneurysm, bleed, abscess, or tumor)
	Lateralized motor or sensory deficit	Stroke, subdural hematoma, epidural hematoma, hemiplegic or anesthetic migraine (rare), reversible cerebral vasoconstriction syndrome, central venous thrombosis
	Balance and coordination deficits	Cervical artery dissection, acute cerebellar hemorrhage, acute cerebellitis (mostly children), chemical intoxication of various types
	Extraocular movement deficits (CN III, IV, and VI)	Mass lesion, neurapraxia (post-traumatic headache), IIH

CN, Cranial nerve; *HEENT,* head, eyes, ears, nose, and throat; *IIH,* idiopathic intracranial hypertension; *SAH,* subarachnoid hemorrhage.

TABLE 17.4

Diagnostic Findings in Emergent Causes of Headache

TEST	FINDING	DIAGNOSIS
Erythrocyte sedimentation rate (ESR)	Significant elevation	Temporal arteritis
Electrocardiogram (ECG)	Nonspecific ST/T wave changes	SAH Increased intracranial pressure
Complete blood count (CBC)	Severe anemia	Anoxia
Computed tomography (CT) scan: Head	Increased ventricular size	Increased intracranial pressure
	Blood in subarachnoid space	SAH
	Blood in epidural or subdural space	Epidural or subdural hematoma
	Bleeding into parenchyma of brain	Intraparenchymal hemorrhage
	Areas of poor vascular flow	Pale infarct
	Structural, mass lesion	Traction headache secondary to mass effect
Lumbar puncture (LP) and cerebrospinal fluid (CSF) analysis	Increased opening pressure	IIH Mass lesion Shunt failure Cryptococcal meningitis
	Increased protein	Tumor or other structural lesions, infection
	Increased RBCs	SAH
	Increased WBCs	Infection
	Positive Gram's stain	Infection
	Decreased glucose	Infection

IIH, idiopathic intracranial hypertension; *RBC,* red blood cell; *SAH,* subarachnoid hemorrhage; *WBC,* white blood cell.

as a default process in the investigation of headache in general. For example, a head CT scan is not indicated for muscle tension headache or recurrent migraine, and it may not be sufficient to assess for cerebral venous thrombosis or for a posterior circulation stroke. A CT scan performed within 6 hours of onset of headache has been shown to be sufficiently sensitive to exclude the diagnosis of SAH when using a third-generation CT scanner. Outside this window, sensitivity declines, and additional testing must be undertaken for appropriate evaluation for SAH.[9]

Lumbar puncture (LP) with measurement of the opening pressure and cerebrospinal fluid (CSF) analysis is indicated when assessing for an infectious process, such as meningitis or encephalitis, IIH, or SAH. Although evidence for this is scant, it is widely believed that LP may increase the likelihood of herniation in certain cases with elevated intracranial pressure caused by a mass lesion. This is the genesis of the common dictum of "CT before LP" when a mass lesion or abscess is a consideration. In reality, this concern is likely misguided, and the compelling reason to obtain a CT scan first in such patients is that it may provide the diagnosis and make the LP unnecessary.

DIAGNOSTIC ALGORITHM

Key elements of the history of present illness, past medical history, and examination are used to narrow the differential diagnosis and choose the appropriate diagnostic pathway. Figure 17.1 outlines a diagnostic algorithm for assessment of headache patients.

If it is clear from the evaluation that the diagnosis is a primary headache disorder (eg, migraine) or of minor severity and gradual onset (eg, typical tension headache) with normal neurological examination findings, then symptomatic treatment is provided without need for further diagnostic evaluation.

If the history or examination findings are clearly indicative of a particular etiology (eg, angle closure glaucoma), then directed testing is indicated—in this case, intraocular pressure determination.

It is cases in which there are highly concerning elements of history but no definitive diagnosis that are the most challenging in terms of choosing the appropriate evaluation. Indications of patients at higher risk for serious cause of headache who are candidates for more comprehensive evaluation include (1) sudden onset of headache, (2) patient description of the headache as "the worst ever," (3) altered mental status, (4) meningismus, (5) unexplained fever, (6) focal neurological deficit on examination, (7) symptoms refractory to appropriate treatment or worsening despite treatment, (8) onset of headache during exertion, (9) history of immunosuppression, or (10) pregnancy or peripartum state. In these potentially critically ill patients, head CT scan is indicated, and a LP often is needed for those in whom imaging does not reveal the etiology of their symptoms.

Sequential evaluation of the patient's condition and assessment of ancillary data will confirm a working diagnosis or trigger a reconsideration of alternatives, including more serious conditions (Table 17.5).

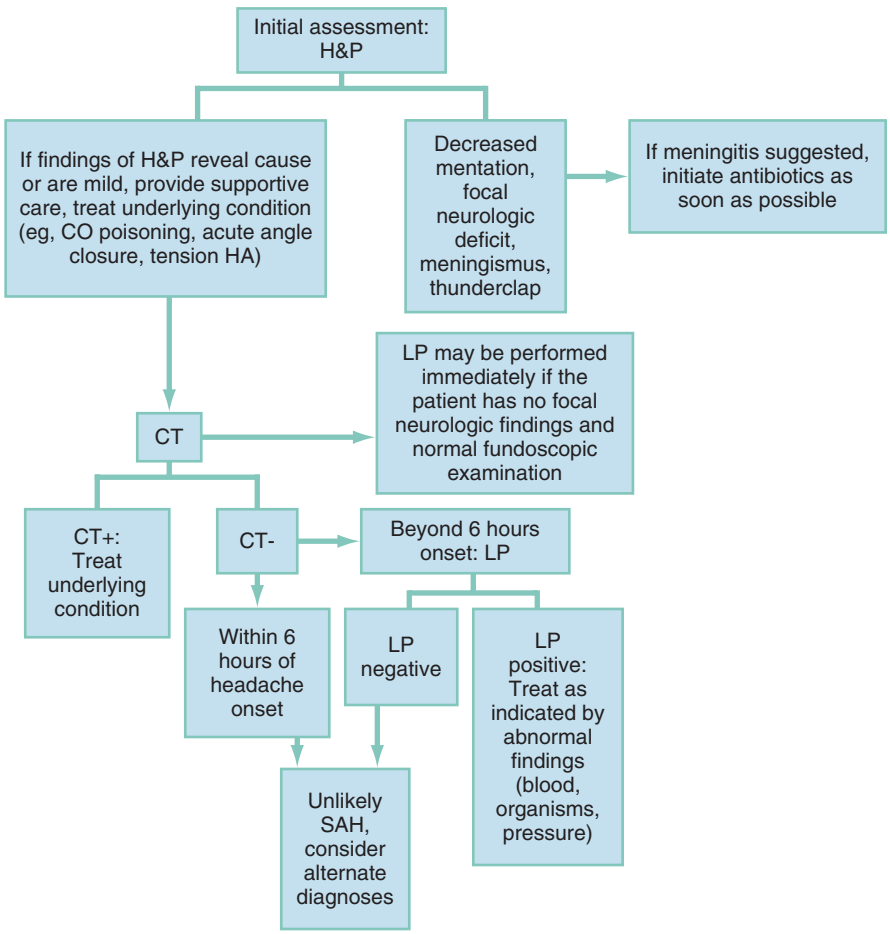

Fig. 17.1. Evaluation algorithm for presentation of headache. *CO,* Carbon monoxide; *CT,* computed tomography; *H&P,* history and physical examination; *HA,* headache; *LP,* lumbar puncture; *SAH,* subarachnoid hemorrhage.

TABLE 17-5

Causes and Differentiation of Potentially Catastrophic Illness Manifesting With Nontraumatic Headache

DISEASE ENTITIES	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORT HISTORY	PREVALENCE
Carbon monoxide poisoning	Usually gradual, subtle, dull, nonfocal throbbing pain	May wax and wane as individual leaves and enters the involved area of carbon monoxide; throbbing may vary considerably	Exposure to engine exhaust, old or defective heating systems, most common in winter months	Rare
Subarachnoid hemorrhage (SAH)	Sudden onset, "thunderclap," severe throbbing	Symptoms variable; may present from relatively asymptomatic to altered mental status or focal neurological deficit	History of polycystic kidney disease; history of HTN	Uncommon
Meningitis, encephalitis, abscess	Gradual; as general symptoms increase, headache increases. Nonfocal pain	Decreased mentation prominent, irritability prominent. With abscess, focal neurologic findings may be present	Recent infection, recent facial or dental surgery or other ENT surgery, unimmunized state	Uncommon
Temporal arteritis	Pain often develops over a few hours from mild to severe, almost always localized to temporal area(s)	Decreased vision, nausea, vomiting may be intense and confound diagnosis	Age over 50; other collagen vascular diseases or inflammatory diseases	Uncommon
Acute angle closure glaucoma	Sudden in onset	Nausea, vomiting, decreased vision	History of glaucoma; history of pain increasing in dark areas	Rare
Increased intracranial pressure syndromes	Gradual, dull, nonfocal	Vomiting, decreased mentation	History of CSF shunt or congenital brain or skull abnormality	Uncommon

CSF, Cerebrospinal fluid; *ENT,* ear, nose, and throat; *HTN,* hypertension.

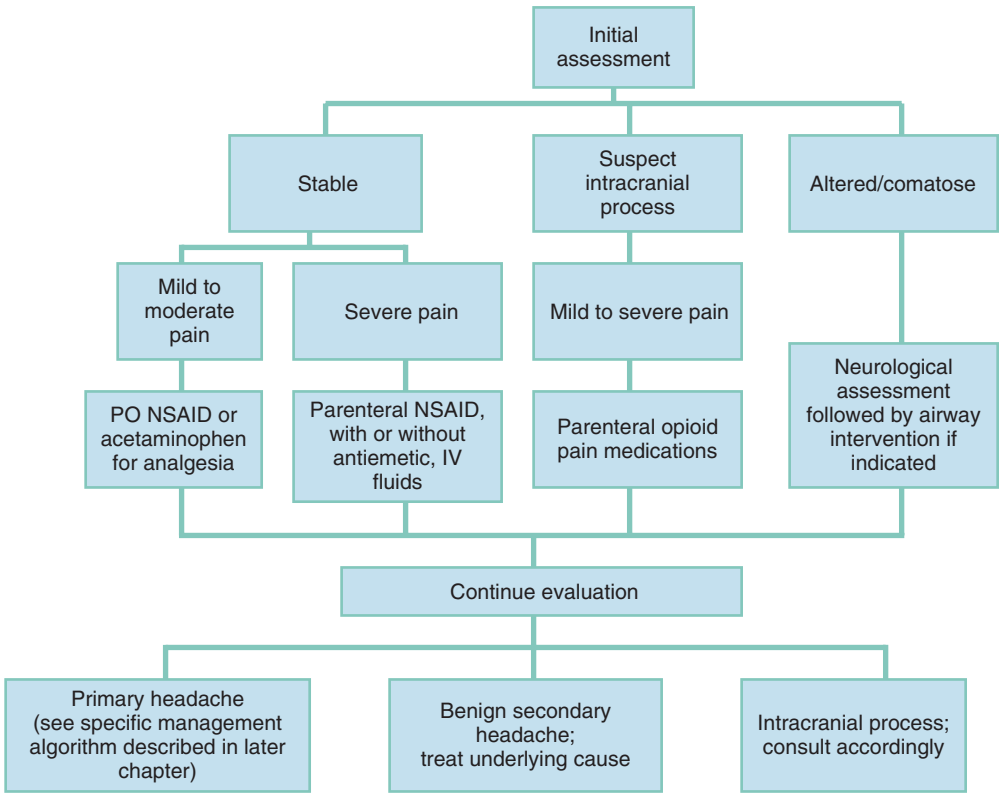

Fig. 17.2. Management algorithm. *IV,* intravenous; *NSAID,* nonsteroidal antiinflammatory drug; *PO, per os* (by mouth).

EMPIRICAL MANAGEMENT

Headache, although a frequent chief complaint, is a nonspecific symptom. The speed and intensity of the initial evaluation and treatment are guided by the presentation and the patient's mental status. Figure 17.2 represents a management algorithm with immediate management pending completion of a full diagnostic evaluation.

For purposes of the initial assessment, headache can be divided into two categories: (1) accompanied by altered mental status and (2) without altered mental status. Whenever a patient's mental status is impaired, brain tissue is initially assumed to be

compromised. The principles of cerebral resuscitation address the seven major causes of evolving brain injury: (1) lack of substrate (glucose, oxygen), (2) cerebral edema, (3) intracranial mass lesion, (4) endogenous or exogenous toxins, (5) metabolic alterations (fever, seizure), (6) ischemia, or (7) elevated intracranial pressure.

Pain is mitigated as soon as possible. The pain medication of choice depends on the working diagnosis of the patient's headache. For nonspecific mild to moderate headache, oral nonsteroidal antiinflammatory medication is appropriate in analgesic doses (eg, 500 mg of naproxen). Opioids are not first-line management for any type of headache pain, except when ICH (including SAH) is thought to be present.

Other than symptomatic relief of pain, empirical treatment does not precede diagnostic studies in most cases, because the treatment must be targeted to the specific cause of the headache. A significant exception to this is when bacterial meningitis is a consideration. Treatment of bacterial meningitis is time-sensitive, and empirical antibiotics should be administered as soon as possible and before results are available to confirm the diagnosis.

Disposition

Patients who are not thought to have a serious cause for their head pain requiring hospitalization but who are without a specific diagnosis are provided with appropriate return precautions and recommendations for follow-up care. Some patients many benefit from beginning a headache journal to facilitate further outpatient evaluation.

KEY CONCEPTS

- When a patient with a known headache disorder presents with a change in the pattern of the headache, evaluate for potential serious causes.
- The physical examination in the headache patient focuses on cranial nerves (CNs) II, III, IV, and VI.
- Opioid medication is almost never the analgesic of choice for headache. Simple headache is treated with nonsteroidal analgesic medication, and specific antimigraine therapies are used for migraine.
- Most patients with headache do not require neuroimaging. When obtained, neuroimaging should be tailored to the specific elements of the differential diagnosis of concern.
- The differential diagnosis of sudden severe headache includes subarachnoid or other intracranial hemorrhage (ICH), cerebral venous thrombosis, and cervical artery dissection.
- In those patients for whom there is concern for subarachnoid hemorrhage (SAH), a normal head CT scan obtained using a high resolution scanner within 6 hours of onset is sufficient to rule out SAH. Patients outside this window require lumbar puncture (LP) to achieve appropriate sensitivity in the evaluation.
- Antibiotics should be given prior to LP being performed when bacterial meningitis is suspected.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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CHAPTER 17: QUESTIONS & ANSWERS

17.1. The most appropriate initial evaluation of a patient with nontraumatic headache is:
- **A.** CT scan of brain
- **B.** EEG
- **C.** MRI scan of brain
- **D.** Thorough neurological evaluation
- **E.** Trial of NSAIDs for pain relief

Answer: D. A thorough neurological examination may reveal deficits not seen on gross evaluation, prompting expansion of the differential diagnosis to include more concerning etiologies. Depending on the history and remainder of the physical, a normal neurological examination may be reassuring and obviate need for advanced imaging studies.

17.2. In the setting of headache, the presence of nausea and vomiting are diagnostic of which of the following as an underlying cause?
- **A.** Glaucoma
- **B.** Increased intracranial pressure
- **C.** Migraine
- **D.** Temporal arteritis
- **E.** None of the above

Answer: E. Nausea and vomiting are completely nonspecific. Migraine headaches, increased intracranial pressure, temporal arteritis, and glaucoma can all be manifested by severe nausea and vomiting, as can some systemic viral infections with headache. Such factors may point toward the intensity of the discomfort but are not specific in establishing the diagnosis.

17.3. Which of the following causes of headache has a constellation of risk factors that include age older than 50 years, female gender, history of lupus, and previous chronic meningitis?
- **A.** Abscess
- **B.** Encephalitis
- **C.** Increased intracranial pressure
- **D.** SAH
- **E.** Temporal arteritis

Answer: E. Risk factors associated with temporal arteritis include age older than 50 years; female gender (ratio 4 : 1); history of other collagen vascular diseases, such as lupus; previous chronic meningitis; and previous chronic illness, such as tuberculosis, parasitic infection, and fungal infection.

17.4. A history of polycystic kidney disease is an associated risk factor for which of the following potentially catastrophic causes of headache?
- **A.** Cerebral venous sinus thrombosis
- **B.** Increased intracranial pressure
- **C.** SAH
- **D.** Subdural hematoma
- **E.** Temporal arteritis

Answer: C. A history of polycystic kidney disease is a risk factor for SAH. Other historical details and risk factors for SAH are sudden severe pain, acute severe pain after sexual intercourse or straining, history of SAH or cerebral aneurysm, family history of SAH, severe hypertension, previous vascular lesions in other areas of the body, and being young or middle aged.