Evaluation of the adult with headache in the emergency department

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INTRODUCTION — Patients with headache constitute up to 4.5 percent of emergency department (ED) visits [1,2]. The differentiation of the small number of patients with life-threatening headaches from the overwhelming majority with benign primary headaches (ie, migraine, tension, or cluster) is an important problem in the ED. Failure to recognize a serious headache can have potentially fatal consequences.

A careful history and physical examination remain the most important part of the assessment of the headache patient; they enable the clinician to determine whether the patient is at significant risk for a dangerous cause of their symptoms and what additional workup is necessary.

This topic will discuss how to approach adults presenting with headache in the ED with an emphasis on those components of the history and physical examination that characterize high-risk headaches. A flow chart to help guide this evaluation is provided (algorithm 1A-B). Detailed discussions of specific causes of headache are found elsewhere. (See "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage" and "Headache, migraine, and stroke" and "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Acute treatment of migraine in adults" and "Preventive treatment of migraine in adults".)

HIGH-RISK HISTORICAL FEATURES — The essential job of the emergency clinician is to determine which patients are at high-risk for a dangerous underlying cause of their headache. Such headaches are commonly referred to as secondary to distinguish them from benign intrinsic causes (ie, migraine, cluster, and tension), which are referred to as primary. The following historical features are warning signs to the presence of a secondary headache (table 1) [3].

Sudden onset — A severe persistent headache that reaches maximal intensity within a few **seconds** or **minutes** after the onset of pain warrants aggressive investigation [4,5]. Subarachnoid hemorrhage (SAH), for example, often presents with the abrupt onset of excruciating pain. Other serious etiologies of sudden-onset headache include carotid and vertebral artery dissections, venous sinus thrombosis, pituitary apoplexy, acute angle-closure glaucoma, and hypertensive emergencies (table 2). In contrast, migraine headaches generally begin with mild to moderate pain and then gradually increase to a maximal level over one to two hours.

Cluster headache may sometimes be confused with a serious headache, since the pain from a cluster headache can reach full intensity within minutes. However, cluster headache is transient (usually lasting less than one to two hours) and is associated with characteristic ipsilateral autonomic signs, such as tearing or rhinorrhea. (See "Cluster headache: Epidemiology, clinical features, and diagnosis".)

Of note, pain may increase gradually with some dangerous causes of headache, such as herpetic or Lyme meningitis and idiopathic intracranial hypertension.

No similar headaches in the past — The absence of similar headaches in the past is another finding that suggests a serious disorder. The "first" or "worst headache of my life" is a description that sometimes

accompanies an intracranial hemorrhage or central nervous system (CNS) infection. A new or unusual headache in a patient with AIDS or cancer is particularly worrisome, as it suggests an intracranial lesion or infection [6]. On the other hand, patients suffering from migraine usually have had similar headaches in the past. (See 'HIV and immunosuppression' below.)

Concomitant infection — Infection in a nonintracranial location (such as the lungs or the paranasal or mastoid sinuses) may serve as a nidus for the development of meningitis or intracranial abscess. (See "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Pathogenesis, clinical manifestations, and diagnosis of brain abscess".)

Altered mental status or seizure — Any change in mental status, personality, or fluctuation in the level of consciousness suggests a potentially serious abnormality. Syncope or near syncope is suggestive of SAH. Headache associated with seizure is also concerning for intracranial pathology. Preeclampsia can cause a headache and may present following delivery. (See "Stupor and coma in adults" and "Evaluation of the first seizure in adults" and "Preeclampsia: Clinical features and diagnosis".)

Headache with exertion — The rapid onset of headache with exertion (eg, sexual intercourse, exercise), especially when minor trauma has occurred, raises the possibility of carotid artery dissection or intracranial hemorrhage. (See "Headache, migraine, and stroke" and "Exertional headache".)

Age over 50 — Patients over 50 years of age with new onset or progressively worsening headache are at significantly greater risk of a dangerous cause of their symptoms, including an intracranial mass lesion and temporal arteritis [1,4,7]. (See "Clinical manifestations of giant cell (temporal) arteritis".)

HIV and immunosuppression — HIV and other immunosuppressed patients with headache are at significant risk for intracranial disease, including toxoplasmosis, stroke, brain abscess, meningitis, and malignancy of the central nervous system. Clinicians should have a low threshold to perform aggressive workups on such patients, particularly if high-risk features such as new-onset seizure or altered mental status is present [6]. (See "Approach to HIV-infected patients with central nervous system lesions".)

Visual disturbances — Occasionally patients with significant ophthalmologic disease, most notably acute narrow angle glaucoma (ANAG), will present with a complaint of headache. A careful history and physical examination, including measurement of intraocular pressures when warranted, is usually sufficient to determine whether this is the source of pain. Headache associated with visual disturbances may also be associated with giant cell arteritis. (See "Angle-closure glaucoma", section on 'Clinical presentation' and "Clinical manifestations of giant cell (temporal) arteritis".)

Location of pain — Head pain that spreads into the lower neck (ie, occipitonuchal headache) and between the shoulders may indicate meningeal irritation due to either infection or subarachnoid blood; it is not typical of a benign process [1]. Some secondary headaches are well localized. As an example, headache from acute narrow angle glaucoma is commonly centered around the involved eye, while headache from temporal arteritis, as implied by its name, can be focused in a temple, although not always. Generally, however, headache location is nonspecific and should not be relied upon for diagnosis.

Family history — The headache patient with a family history of subarachnoid hemorrhage (SAH) among first or second-degree relatives is at significantly greater risk of SAH [8]. (See "Aneurysmal subarachnoid hemorrhage: Epidemiology, risk factors, and pathogenesis", section on 'Genetic risk'.)

Medications — Clinicians should inquire about medication use, particularly anticoagulants, glucocorticoids, and analgesics. Use of anticoagulants or nonsteroidal anti-inflammatory drugs, including aspirin, increases the risk of intracranial bleeding. Sympathomimetics are also associated with intracranial bleeding. Analgesics can mask severe symptoms or sometimes exacerbate migraine headache through a rebound effect (medication overuse headache). (See "Acute treatment of migraine in adults".)

Illicit drugs — A number of illicit drugs, including cocaine, methamphetamine, and other sympathomimetic agents, increase the risk of stroke and intracranial bleeding. (See "Evaluation and management of the cardiovascular complications of cocaine abuse", section on 'Stroke'.)

Toxic exposure — Headaches that involve multiple family members or coworkers and improve rapidly in the emergency department without intervention, particularly during winter months, raise the possibility of carbon monoxide poisoning. (See "Carbon monoxide poisoning".)

Additional history — Other historical factors to consider when investigating the cause of headache include head trauma, possible toxic exposures (eg, carbon monoxide), and comorbidities known to put patients at higher-risk for critical secondary causes of headache. Such comorbidities include malignancy with a risk of intracranial metastasis and polycystic kidney disease or connective tissue disease, both of which increase the risk of aneurysms with resultant SAH (see "Management of acute severe traumatic brain injury"). Jaw claudication suggests temporal arteritis as the cause of headache. (See "Clinical manifestations of giant cell (temporal) arteritis".)

Comorbidities that adversely affect coagulation increase the risk of headache from dangerous causes. As examples, liver disease or clotting disorders may predispose patients to intracranial bleeding, while hypercoagulable states may increase the risk of stroke or cerebral venous thrombosis. Some regions may have high rates of particular infections that may present with headache as a prominent symptom (eg, Lyme disease). (See "Clinical manifestations of Lyme disease in adults" and "Etiology, clinical features, and diagnosis of cerebral venous thrombosis".)

Several less common headache syndromes associated with specific symptoms or signs may be associated with dangerous underlying etiologies. As examples, headache induced by cough or valsalva may be caused by malignancy or cerebrovascular disease, and headache associated with positional hypotension may be caused by leaking cerebrospinal fluid (CSF). (See "Primary cough headache" and "Spontaneous intracranial hypotension: Pathophysiology, clinical features, and diagnosis".)

HIGH-RISK EXAMINATION FINDINGS — The following findings on physical examination may suggest a life-threatening cause of headache (table 1). (See "The detailed neurologic examination in adults".)

Neurologic abnormalities — The patient with any new focal or nonfocal neurologic abnormality must be evaluated for serious illness. Abnormal findings on neurologic exam remain the single-best clinical predictor of intracranial pathology [1,4,9]. The findings may be quite subtle, such as slight pupillary asymmetry, unilateral pronator drift, or extensor plantar response, or pronounced, such as unilateral vision loss, ataxia, or seizure (see "The detailed neurologic examination in adults"). Focal neurologic findings can accompany a number of secondary causes of headache, including intracranial hemorrhage, acute narrow angle glaucoma, and carotid or vertebral artery dissection. Nonfocal alterations in mental status more commonly characterize other secondary causes of headache, including SAH, infectious processes such as meningitis, toxins such as carbon monoxide, and metabolic derangements such as hypoxia.

Neurologic abnormalities can also occur with migraine headaches. As an example, a visual field cut in both eyes within the same hemifield bounded by scintillations is characteristic of migraine with visual aura (see "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults", section on 'Migraine aura'). However, a focal neurologic deficit should not be assumed to be related to migraine unless similar deficits have occurred with migraine in the past. Any new or atypical focal neurologic deficit is considered a high-risk finding and should be investigated urgently in the emergency department. (See 'Evaluation' below.)

Decreased level of consciousness — Obtundation and confusion are not typical of a benign headache and increase the likelihood of meningitis, encephalitis, subarachnoid hemorrhage (SAH), or other space occupying lesion. (See "Stupor and coma in adults".)

Meningismus — Meningismus may indicate meningitis or SAH. It can be subtle. This sign is also less sensitive and less specific in adults older than 60 years [10]. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Presenting manifestations'.)

Abnormal vital signs — Fever can reflect CNS infection. It is not characteristic of migraine headache; it may, however, follow an SAH by a few days (table 3). Although rare, severe hypertension (diastolic bp ≥120 mmHg) can manifest as headache [5]. (See "Moderate to severe hypertensive retinopathy and hypertensive encephalopathy in adults".)

Toxic appearance — A toxic appearing patient may be suffering from a CNS infection or a systemic illness affecting the CNS.

Ophthalmologic findings — Papilledema, detected by blurring of the optic disks, is indicative of increased intracranial pressure, possibly due to a tumor or other structural abnormality. Retinal or subhyaloid hemorrhage can result from SAH. Decline or loss of vision can occur as a result of vascular compromise in temporal arteritis or carotid artery dissection, or as a result of increased intraocular pressure in acute narrow angle glaucoma (ANAG). Ciliary flush and sluggish pupillary light response can also occur with ANAG. (See "Overview and differential diagnosis of papilledema" and "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage" and "Angle-closure glaucoma".)

Traumatic findings — External findings consistent with trauma raise concern for intracranial hemorrhage as the cause of headache. Such findings may include scalp or facial lacerations or contusions, hemotympanum, and periorbital or preauricular ecchymosis. Victims of abuse or other violence may not volunteer a history of trauma. Headache caused by a chronic subdural hemorrhage, although likely secondary to distant and perhaps mild trauma, will lack such immediate findings. (See "Skull fractures in adults" and "Subdural hematoma in adults: Etiology, clinical features, and diagnosis".)

Additional findings — Abnormalities of the temporal artery (eg, diminished pulse, swelling, or tenderness) are highly suggestive of temporal arteritis. Nausea and vomiting can accompany increased intracranial pressure, intracranial hemorrhage, or acute narrow angle glaucoma. A carotid bruit may accompany carotid artery dissection. Nasal discharge associated with sinus tenderness or signs of dental infection may reflect the cause of headache. (See "Diagnosis of giant cell (temporal) arteritis" and "Acute sinusitis and rhinosinusitis in adults: Clinical manifestations and diagnosis" and "Complications, diagnosis, and treatment of odontogenic infections".)

LOW-RISK PATIENTS — Patients with a history of prior headaches who present to the emergency department due to failure of their standard therapy regimen and who meet the following criteria can be considered at low-risk for dangerous headache:

- No substantial change in their typical headache pattern
- No new concerning historical features (eg, seizure, trauma, fever)
- No focal neurologic symptoms or abnormal neurologic examination findings
- No high-risk comorbidity

Routine neuroimaging is not needed in these patients [11].

EVALUATION — Once historical and examination criteria have determined those patients with high-risk headache features, further evaluation is performed.

Neuroimaging and CSF evaluation — The presence of one or more high-risk features in a patient with an acute headache increases the possibility of a serious underlying illness and warrants more urgent evaluation with lumbar puncture, neuroimaging, or both (algorithm 1A-B). On the other hand, the yield of

neuroimaging is very low if no high-risk historical feature is present and the neurologic examination is normal.

As an example, a meta-analysis of published articles on the utilization of CT and MR scanning in patients presenting with headache conducted by the Quality Standards Subcommittee of the American Academy of Neurology revealed that abnormalities were present in only 2.4 percent of patients with a normal neurologic examination [12]. The incidence of pathology was even lower (0.4 percent) in patients whose headaches were typical of migraine and whose physical examination was normal.

In patients who have a history or physical examination suggestive of serious pathology, the diagnostic approach depends upon the most likely suspected cause. A noncontrast head CT is most helpful for identifying intracranial lesions or bleeding; a lumbar puncture is most helpful for diagnosing infection.

Lumbar puncture (LP) should **always** be performed in patients with suspected SAH in whom the CT scan is normal. One of the tragedies of contemporary emergency care is that those patients with SAH most amenable to treatment are the ones most frequently misdiagnosed at initial presentation. Such patients generally appear less ill and do not have neurologic deficits; misdiagnosis stems from a lack of appreciation of the range of possible presentations of patients with SAH [8]. (See "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage", section on 'Diagnosis of subarachnoid hemorrhage'.)

For patients in whom suspicion for CNS infection is higher than for intracranial lesion, LP may be the first test of choice. Documented cases of herniation following LP have made some clinicians reluctant to perform the procedure without prior imaging in patients suspected of having intracranial pathology. In most such patients, however, LP alone is safe. In one prospective observational study that evaluated the need for cranial CT scan prior to LP, only 3 of 113 consecutive emergency department patients (2.7 percent) in whom an urgent lumbar puncture was thought to be necessary (not only for headache) had a CT abnormality that was a contraindication to LP [13]. Three clinical findings were associated with such an abnormality: papilledema, focal neurologic findings, and altered mental status. Another prospective study evaluating the need for preliminary cranial CT scan in patients with suspected meningitis found similar results [14]. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Lumbar puncture'.)

The clinical policy statement on acute headache of the American College of Emergency Physicians gives a class C recommendation (ie, suggestion based upon weak evidence) to the performance of LP without prior neuroimaging in patients without evidence of increased intracranial pressure (ie, papilledema, absent venous pulsations on funduscopy, altered neurologic status, or focal neurologic deficits) [11]. An opening pressure should be measured when the LP is performed.

Treatment with antibiotics **must NOT be delayed** in patients with suspected meningitis, regardless of the decision of whether to perform an imaging study. Detailed discussions of the interpretation of head CT and LP results are found elsewhere. (See "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage" and "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Cerebrospinal fluid: Physiology and utility of an examination in disease states".)

Traumatic lumbar puncture — A dilemma sometimes confronted in the ED is determining what additional work-up and disposition are appropriate for patients at risk for SAH whose head CT is unrevealing and whose initial LP is presumed to be traumatic [15]. The clearing of blood in CSF collection tubes is **NOT** a reliable means of excluding SAH, unless a late or final collecting tube specimen is normal (ie, red blood cell count approaches zero). Xanthochromia, while useful, is not a foolproof means of diagnosing SAH. Interpretation of the LP is discussed in detail separately. (See "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage", section on 'Lumbar puncture'.)

One approach in such cases is to repeat the LP one intervertebral level cephalad to where the initial attempt was made (but no higher than L3/4) or under fluoroscopic guidance. The presence of blood in CSF obtained from two LPs suggests SAH, possibly from a nonaneurysmal cause, while a normal specimen from the repeat LP makes SAH less likely. In cases where suspicion for SAH remains substantial, the head CT is unrevealing, and LP results are difficult to interpret, we recommend admission for further evaluation by neurology or neurosurgery. (See "Clinical manifestations and diagnosis of aneurysmal subarachnoid hemorrhage", section on 'Diagnosis of subarachnoid hemorrhage' and "Nonaneurysmal subarachnoid hemorrhage".)

Laboratory tests — Specific laboratory tests can be helpful in evaluating possible causes of severe headache and are generally indicated by the patient's history and examination. Such tests can include complete blood count, electrolytes, glucose, BUN and creatinine, erythrocyte sedimentation rate and protein C (frequently elevated in temporal arteritis), coagulation studies, arterial blood gas, and carboxyhemoglobin.

DISPOSITION — Patients in whom a secondary cause of headache is discovered are admitted or referred to the appropriate setting. Patients in whom a secondary cause was suspected, but a thorough, appropriate workup was normal, should be treated symptomatically and may be discharged with primary care or neurologic follow-up. Of note, patient response to analgesics should not be used as a diagnostic tool and should **NOT** dissuade performance of LP when indicated by history or examination.

TREATMENT OF UNDIFFERENTIATED HEADACHE IN THE EMERGENCY DEPARTMENT — Relief of symptoms is an important part of management for patients presenting to the emergency department (ED) with severe headache, even when a thorough work-up reveals no clear underlying process. The large majority of these patients will ultimately be diagnosed with either a migraine or cluster headache. Details of migraine and cluster headache treatment are discussed separately (table 4). (See "Acute treatment of migraine in adults" and "Cluster headache: Treatment and prognosis".)

For those patients with a primary headache disorder that does not clearly meet criteria for migraine or cluster headache, symptomatic treatment should be provided. Few studies are available to guide empiric management of undifferentiated headache in the ED; treatment remains symptom-based and largely nonspecific. Nevertheless, many of the treatments used for acute migraine headaches provide some relief in patients with a severe undifferentiated headache [16,17].

The authors of a systematic review of headache management in the ED propose the use of a parenterally administered nonsteroidal anti-inflammatory drug (NSAID) and a dopamine antagonist (DA) [2]. Their goal is to relieve pain, and allow the patient to return to baseline mental function without drowsiness. In the United States, this treatment would probably be ketorolac 30 mg IV and prochlorperazine 10 mg. Chlorpromazine 0.1 mg/kg IV might be used in place of prochlorperazine. Pretreatment with 12.5 mg of diphenhydramine or 1 mg of benztropine is suggested to avoid akathisia. In a small randomized trial, prochlorperazine was shown to be as effective as subcutaneous sumatriptan [18].

For headaches unresponsive to treatment with a combination of NSAID and DA that have some migrainous features (eg, photophobia), dihydroergotamine 1 mg IV may be effective. Other medications used to treat undifferentiated headache in the ED include sumatriptan, olanzapine, metoclopramide, and droperidol [19-21]. The use of injectable opioids, while not encouraged, may be necessary for patients with contraindications to NSAIDs or medications with vasoconstrictive effects (eg, dihydroergotamine).

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more

detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topic (see "Patient information: Headache (The Basics)")
- Beyond the Basics topics (see "Patient information: Headache treatment in adults (Beyond the Basics)" and "Patient information: Headache causes and diagnosis in adults (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- A careful history and physical examination remain the most important part of the assessment of the headache patient.
- Historical and clinical features associated with dangerous causes of headache are summarized in the table (table 1). (See 'High-risk historical features' above and 'High-risk examination findings' above.)
- Abnormal findings on neurologic examination remain the single best clinical predictor of intracranial pathology. This includes alterations in mental status, visual changes, and changes in a migraine patient’s typical pattern.
- The presence of one or more high-risk features in a patient with an acute headache increases the possibility of a serious underlying illness and warrants urgent evaluation with lumbar puncture, neuroimaging, or both. A flow chart to help guide the emergency department evaluation of the patient with headache is provided (algorithm 1A-B). (See 'Evaluation' above.)
- Relief of symptoms is important, regardless of the headache’s underlying cause. Treatment options are described in the text. (See 'Disposition' above and 'Treatment of undifferentiated headache in the emergency department' above.)

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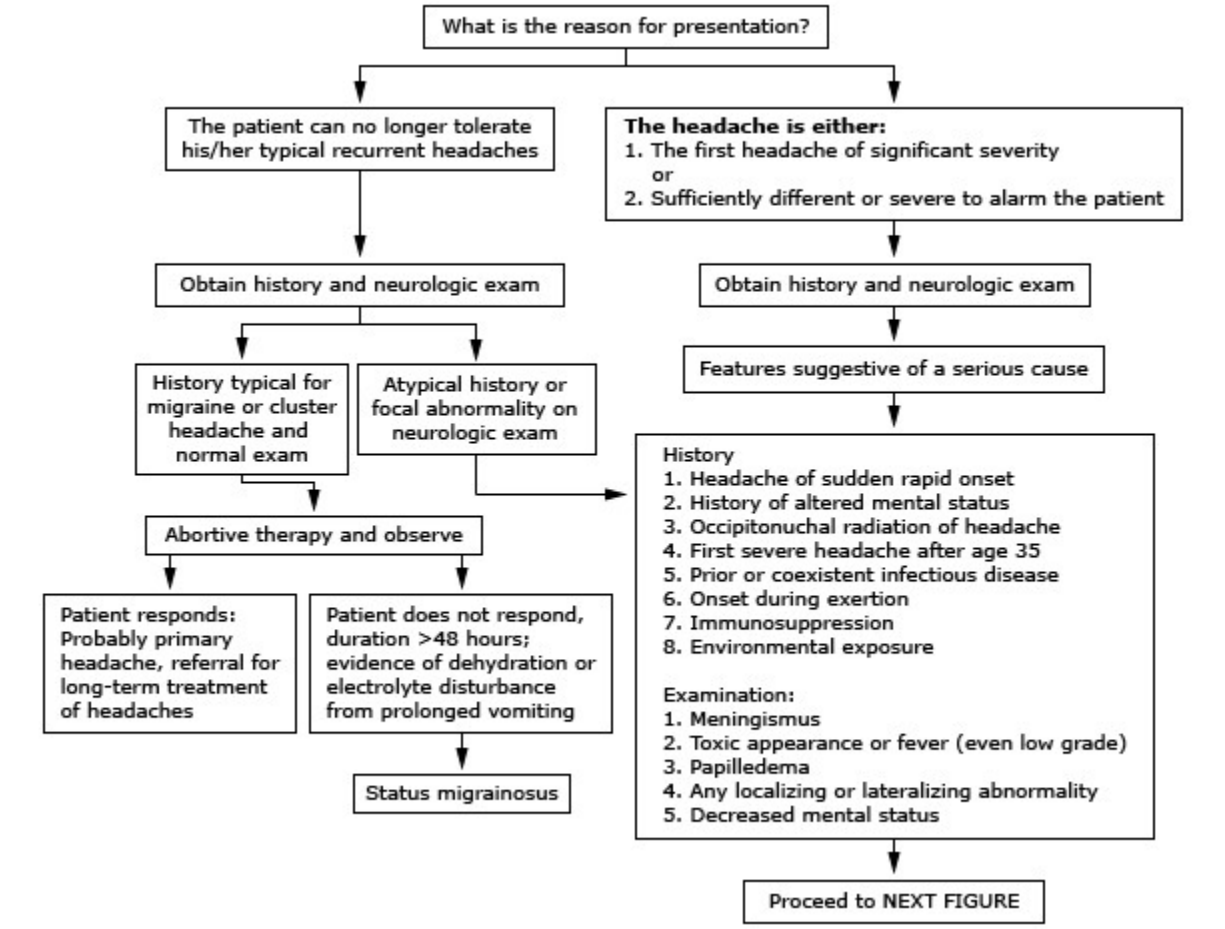
Topic 287 Version 14.0

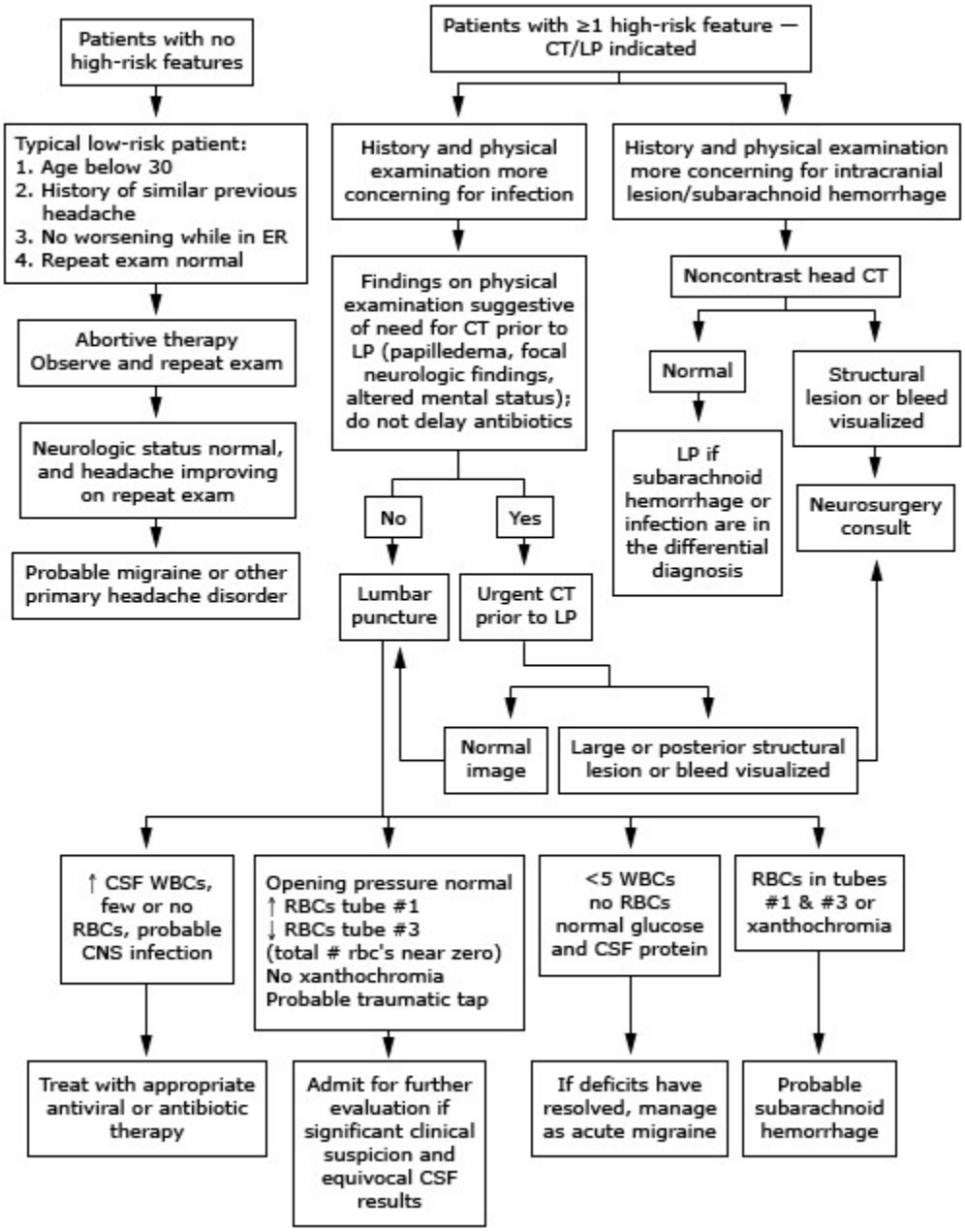
GRAPHICS

Management of headache in the emergency department-I



Graphic 71604 Version 3.0

Management of headache in the emergency department-II



CNS: central nervous system; CSF: cerebrospinal fluid; CT: computed tomography scan; LP: lumbar puncture; RBC: red blood cell; WBC: white blood cell.

Graphic 51917 Version 4.0

Characteristics of headache with serious underlying pathology

History
Explosive onset and severe at onset
No similar headaches in the past
Concomitant infection
Altered mental status
Headache with exertion
Age over 50
Immunosuppression
Physical examination
Neurologic abnormalities
Decreased level of consciousness
Meningismus
Toxic appearance
Papilledema

Graphic 78567 Version 1.0

Differential diagnosis of sudden onset severe headache

Subarachnoid hemorrhage
Expansion or thrombosis of unruptured intracranial aneurysm
Intracerebral hemorrhage
Acute ischemic stroke
Subdural and epidural hematomas
Internal carotid and vertebral artery dissection (with face or neck pain)
Cerebral venous thrombosis
Pituitary apoplexy
Colloid cyst of the third ventricle
Acute expansion of mass lesion in the posterior fossa
Hypertensive encephalopathy
Spontaneous intracranial hypotension
Coital headache
Acute narrow angle glaucoma
Exertional headache
Acute sinusitis (with barotrauma)
Benign thunderclap headaches

Graphic 61027 Version 1.0

Differential diagnosis of headache with fever

Intracranial infection
Meningitis
Bacterial
Fungal
Viral
Lymphocytic
Encephalitis
Brain abscess
Subdural empyema
Systemic infection
Bacterial infection
Viral infection
HIV/AIDS
Other systemic infection
Other causes
Familial hemiplegic migraine
Pituitary apoplexy
Rhinosinusitis
Subarachnoid hemorrhage
Malignancy of central nervous system

Graphic 80966 Version 3.0

Characteristics of migraine, tension-type, and cluster headache syndromes

Symptom	Migraine	Tension-type	Cluster
Location	Unilateral in 60 to 70 percent; bifrontal or global in 30 percent	Bilateral	Always unilateral, usually begins around the eye or temple
Characteristics	Gradual in onset, crescendo pattern; pulsating; moderate or severe intensity; aggravated by routine physical activity	Pressure or tightness which waxes and wanes	Pain begins quickly, reaches a crescendo within minutes; pain is deep, continuous, excruciating, and explosive in quality
Patient appearance	Patient prefers to rest in a dark, quiet room	Patient may remain active or may need to rest	Patient remains active
Duration	4 to 72 hours	Variable	30 minutes to 3 hours
Associated symptoms	Nausea, vomiting, photophobia, phonophobia; may have aura (usually visual, but can involve other senses or cause speech or motor deficits)	None	Ipsilateral lacrimation and redness of the eye; stuffy nose; rhinorrhea; pallor; sweating; Horner's syndrome; focal neurologic symptoms rare; sensitivity to alcohol

Graphic 68064 Version 4.0

Disclosures

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