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Evaluation of the adult with nontraumatic headache in the emergency department

AUTHORS: F Michael Cutrer, MD, Franz J Wippold II, MD, FACR, Jonathan A Edlow, MD, FACEP
SECTION EDITORS: Korilyn S Zachrison, MD, MSc, Jerry W Swanson, MD, MHPE
DEPUTY EDITORS: Jonathan Grayzel, MD, FAAEM, Richard P Goddeau, Jr, DO, FAHA

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INTRODUCTION

Patients with headache unrelated to trauma constitute approximately 2 percent of emergency department (ED) visits, though some studies suggest a rate as high as 4 percent [1-3]. 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 serious consequences, including permanent neurologic deficits, loss of vision, and death.

A careful history and physical examination remain the most important parts of the assessment of the headache patient; they help 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 to the ED with headache, with an emphasis on those components of the history and physical examination that characterize high-risk headaches. Diagnostic tables to help guide this evaluation are provided (table 1 and table 2).

Discussions of headache following trauma and other specific causes of headache are found separately:

- Trauma: (see "Acute mild traumatic brain injury (concussion) in adults" and "Initial evaluation and management of facial trauma in adults")
- Concussion: (see "Sequelae of mild traumatic brain injury" and "Postconcussion syndrome")
- Intracranial hemorrhage: (see "Spontaneous intracerebral hemorrhage: Pathogenesis, clinical features, and diagnosis" and "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Subdural hematoma in adults: Etiology, clinical features, and diagnosis" and "Intracranial epidural hematoma in adults")
- Infection: (see "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Aseptic meningitis in adults" and "Viral encephalitis in adults")
- Migraine: (see "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults" and "Acute treatment of migraine in adults")

IDENTIFYING HIGH-RISK PATIENTS

Patients who have one or more high-risk historical features or examination findings are considered to have a life-threatening condition requiring urgent diagnosis (table 2). The essential job of the emergency clinician is to identify these patients (algorithm 1). Headaches in such patients are commonly referred to as secondary to distinguish them from benign intrinsic causes (ie, migraine, cluster, and tension), which are referred to as primary.

High-risk historical features — The following historical features are warning signs to the presence of a secondary headache (table 1) [4,5]:

- **Sudden-onset** headache – While some dangerous causes of headache may present with a gradual increase in pain, any severe, persistent headache that reaches maximal intensity within a few **seconds** or **minutes** after the onset of pain warrants aggressive investigation [6-8]. Subarachnoid hemorrhage (SAH), for example, often presents with the abrupt onset of excruciating pain. Other serious etiologies of sudden-onset headache include reversible cerebral vasoconstriction syndromes, carotid and vertebral artery dissections, venous sinus thrombosis, pituitary apoplexy, acute angle-closure glaucoma, unruptured cerebral aneurysms, colloid cyst of the third ventricle, and hypertensive emergencies (table 3). In contrast, migraine headaches generally begin with mild to moderate pain and then gradually increase to a maximal level over one to two hours. (See "Overview of thunderclap headache".)

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, miosis, or rhinorrhea. (See "Cluster headache: Epidemiology, clinical features, and diagnosis".)

Of note, many dangerous causes of headache can present with gradual-onset headache, including herpetic or Lyme meningitis, brain tumor, brain abscess, hypertensive encephalopathy, posterior reversible encephalopathy syndrome (PRES), and idiopathic intracranial hypertension. Furthermore, dangerous causes may present with atypical timing, and symptoms may overlap. As examples, most patients with cervico-cranial artery dissections and venous sinus thrombosis present with gradual-onset headaches, and some patients with acute-angle closure glaucoma develop headaches that simulate migraine and can be intermittent [9]. (See "Aseptic meningitis in adults" and "Cerebral venous thrombosis: Etiology, clinical features, and diagnosis" and "Pathogenesis, clinical manifestations, and diagnosis of brain abscess" and "Moderate to severe hypertensive retinopathy and hypertensive encephalopathy in adults" and "Reversible posterior leukoencephalopathy syndrome" and "Idiopathic intracranial hypertension (pseudotumor cerebri): Clinical features and diagnosis".)

- **No similar headaches in the past** – The absence of similar headaches in the past is another finding that suggests a serious disorder. The "first" and "worst headache of my life" are descriptions that sometimes accompanies an intracranial hemorrhage or central nervous system (CNS) infection. A new or unusual headache in a patient with acquired immunodeficiency syndrome (AIDS) or cancer is particularly worrisome, as it suggests an intracranial lesion or infection [10]. On the other hand, patients suffering from migraine usually have had similar headaches in the past, and by definition, one cannot definitively make a diagnosis of migraine without at least four prior episodes. (See "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults".)

- **Concomitant infection** – Infection in an extracranial location (such as the paranasal or mastoid sinuses, pharynx, or inner ear) 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 at headache onset is suggestive of SAH. Headache associated

with seizure is also concerning for intracranial pathology. Preeclampsia and PRES can cause headache and seizure and may present up to six weeks following delivery. In the patient with a headache and altered mental status, it is important to check for hypoglycemia using a point-of-care (eg, fingerstick) glucose concentration. (See "Stupor and coma in adults" and "Evaluation and management 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), raises the possibility of carotid artery dissection, reversible cerebral vasoconstriction, or intracranial hemorrhage. (See "Migraine-associated stroke: risk factors, diagnosis, and prevention" and "Exercise (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 giant cell arteritis [1,3,6,11]. (See "Clinical manifestations of giant cell 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 CNS. 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 are present [10,12]. (See "Approach to the patient with HIV and central nervous system lesions".)

- **Visual disturbances** – Occasionally, patients with significant ophthalmologic disease, most notably acute angle-closure glaucoma, present with a complaint of headache. A careful history and physical examination, including measurement of intraocular pressures, is usually sufficient to determine whether this is the source of pain. Headache may precede visual changes or eye pain in acute angle-closure glaucoma. Headache associated with visual disturbances may also be associated with giant cell arteritis, idiopathic intracranial hypertension, and PRES. (See "Angle-closure glaucoma", section on 'Clinical presentation' and "Clinical manifestations of giant cell arteritis" and "Idiopathic intracranial hypertension (pseudotumor cerebri): Clinical features and diagnosis" and "Reversible posterior leukoencephalopathy syndrome".)

- **Pregnancy and postpartum state** – Although primary headache syndromes remain the most common cause of headaches in this group, other pregnancy-related diagnoses should be considered. Preeclampsia is the most common of these; the presentation of preeclampsia can overlap with PRES [13]. Less common etiologies include venous sinus

thrombosis, pituitary apoplexy, and reversible cerebral vasoconstriction [13]. Venous sinus thrombosis is most common postpartum. Worse headache upon standing postpartum suggests either a postdural puncture headache (if the patient had a spinal anesthetic) or spontaneous intracranial hypotension (in patients without a spinal anesthetic) due to dural tears sustained during labor-related pushing [13]. (See "Headache during pregnancy and postpartum" and "Preeclampsia: Clinical features and diagnosis" and "Cerebral venous thrombosis: Etiology, clinical features, and diagnosis".)

- **Location of pain** – Location of the pain is not particularly useful in the diagnosis. However, 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 angle-closure glaucoma is commonly centered around the involved eye, while headache from giant cell arteritis is often, but not always, focused in a temple. (See "Angle-closure glaucoma" and "Diagnosis of giant cell arteritis".)

- **Family history** – The headache patient with a family history of SAH among first- or second-degree relatives is at significantly greater risk of SAH [14]. Simultaneous headaches in multiple family members suggest carbon monoxide poisoning. (See "Aneurysmal subarachnoid hemorrhage: Epidemiology, risk factors, and pathogenesis", section on 'Genetic risk'.)

- **Medications** – Clinicians should inquire about medication use, particularly anticoagulants, glucocorticoids, oral contraceptives, and analgesics. Use of anticoagulants or nonsteroidal antiinflammatory drugs (NSAIDs), including aspirin, increases the risk of intracranial bleeding. Another consideration in patients taking anticoagulants is a cerebral venous sinus thrombosis, if the indication for the medication was a venous thrombotic event. 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" and "Cerebral venous thrombosis: Etiology, clinical features, and diagnosis".)

    - **Anticoagulants** – All anticoagulants confer an increased bleeding risk, including bleeding into the brain. Patients on warfarin have a higher risk of intracranial hemorrhage compared with those on direct oral anticoagulants (DOACs), and patients on any oral anticoagulant plus antiplatelet agents have an increased risk compared with those not on these agents. Details pertaining to the risk of bleeding associated with particular medications are provided separately. (See "Risks and prevention of bleeding with oral anticoagulants".)

All anticoagulated patients with head trauma, even minor trauma, should undergo computed tomography (CT). Many patients with a new nontraumatic headache will fall into the older age group for whom imaging is already recommended. Other anticoagulated patients with an intracranial hemorrhage will fall into another high-risk category (eg, thunderclap onset or new neurological deficits). Apart from age and trauma, there are no high-quality data to help determine whether to perform a routine cranial CT on anticoagulated patients. Factors that influence this judgment include other known risk factors such as age, hypertension, degree of anticoagulation, and prior stroke or known vascular lesions such as cerebral amyloid angiopathy.

- **Illicit drugs** – A number of illicit drugs, including cocaine, methamphetamine, and other sympathomimetic agents, increase the risk of stroke and intracranial bleeding. If the drugs are used intravenously (IV), brain abscess is another possibility, even in the absence of fever. (See "Clinical manifestations, diagnosis, and management of the cardiovascular complications of cocaine abuse", section on 'Stroke' and "Methamphetamine: Acute intoxication".)

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

Additional relevant history — Other historical factors to consider when investigating the cause of headache include chiropractic neck manipulation, 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 possibly increased intracranial pressure [ICP]) and polycystic kidney disease or connective tissue disease, both of which increase the risk of aneurysms with resultant SAH. Jaw claudication suggests giant cell arteritis as the cause of headache. (See "Spinal manipulation in the treatment of musculoskeletal pain", section on 'Risks of spinal manipulation' and "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Clinical manifestations of giant cell arteritis".)

Recent rash compatible with erythema migrans (picture 1) suggests Lyme meningitis, and concurrent petechial (picture 2) or purpuric (picture 3) rash suggests bacterial meningitis. Postural or positional headache raises concern for spontaneous intracranial hypotension or colloid cyst of the third ventricle. (See "Clinical manifestations of Lyme disease in adults", section on 'Erythema migrans' and "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Spontaneous intracranial hypotension: Pathophysiology, clinical features, and

diagnosis" and "Overview of thunderclap headache", section on 'Colloid cyst of the third ventricle'.)

A social history, inclusive of hobbies and occupational exposures, may reveal potential causes of headache. A variety of compounds such as carbon monoxide, hydrocarbons, mineral spirits, and formaldehyde, along with pesticides, chlorine bleach, and many other chemicals, may cause headache following exposure.

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 geographic 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 "Cerebral venous thrombosis: Etiology, clinical features, and diagnosis".)

Headaches that worsen upon standing are usually due to postdural puncture (eg, following a lumbar puncture [LP]) or spontaneous intracranial hypotension (if a procedure violating the dura has not been performed). These headaches are due to low ICP from cerebrospinal fluid (CSF) leaks. Headaches that worsen with cough or Valsalva suggest the possibility of brain tumor or other causes of elevated ICP. (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".)

- **Abnormal vital signs** – Fever makes a diagnosis of migraine or tension-type headache highly unlikely. Fever may be due to CNS infection or inflammation and may be due to a several-day-old SAH (table 4). Although rare, severe hypertension (diastolic blood pressure ≥120 mmHg) can manifest as headache [7]. Although new hypertension in a previously normotensive patient can be due to pain or anxiety, it may also be a compensatory finding from elevated ICP of any cause. (See "Moderate to severe hypertensive retinopathy and hypertensive encephalopathy in adults".)

- **Toxic appearance** – An acutely ill patient complaining of headache who manifests other concerning signs of illness, such as lethargy, altered mental status, poor perfusion, pallor, fever, or sweats, may have a systemic illness or infection that is secondarily affecting the CNS.

- **Decreased level of consciousness** – Obtundation and confusion are not seen in benign headaches and increase the likelihood of meningitis, encephalitis, SAH, or results of a space-occupying lesion. (See "Stupor and coma in adults".)

- **Neurologic abnormalities** – The patient with any new focal or nonfocal neurologic abnormality must be evaluated for serious illness. Abnormal findings on neurologic examination remain the single best clinical predictor of intracranial pathology [1,6,15]. The findings may be quite subtle and go unnoticed by the patient (eg, slight pupillary asymmetry, unilateral pronator drift, visual field cut, or extensor plantar response) or pronounced and obvious (eg, 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 ischemic stroke, intracranial hemorrhage, brain tumor, brain abscess, acute angle-closure 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 or encephalitis, toxins such as carbon monoxide, and metabolic derangements such as hypoxia. In the patient with a headache and altered mental status, it is important to check for hypoglycemia using a point-of-care (eg, fingerstick) glucose concentration (hypoglycemia may cause focal neurologic deficits).

  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 prior migraines. Any new or atypical focal neurologic deficit is considered a high-risk finding and should be investigated urgently until the cause is identified. (See 'Evaluation of patients with high-risk features' below.)

  One concept that helps to distinguish symptoms or signs due to migraine rather than ischemia, infarct, or other destructive processes is that of "positive" versus "negative" phenomena, as described in the following table (table 5). As a general rule, positive phenomena are due to migraine and seizure whereas negative findings are found in destructive lesions.

- **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 [16]. (See "Clinical features

and diagnosis of acute bacterial meningitis in adults", section on 'Presenting manifestations'.)

- **Ophthalmologic findings** – Papilledema, detected by blurring of the optic disks (picture 4), is indicative of increased ICP, 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 giant cell arteritis or carotid artery dissection, or as a result of increased intraocular pressure in acute angle-closure glaucoma. Examination of the eye in acute angle-closure glaucoma often shows an edematous ("steamy") cornea and may reveal ciliary flush (picture 5) and sluggish pupillary light response. Field cuts can be seen with any process that involves the optic nerve, chiasm (especially with pituitary apoplexy), optic radiations (any process), or occipital cortex (PRES or posterior cerebral artery ischemic stroke). (See "Overview and differential diagnosis of papilledema" and "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Angle-closure glaucoma".)

Additional relevant findings — Abnormalities of the temporal artery (eg, diminished pulse, swelling, or tenderness) are highly suggestive of giant cell arteritis. Nausea and vomiting can accompany increased ICP, intracranial hemorrhage, or acute angle-closure glaucoma but are also common with migraine. Vomiting in a migraineur who has never vomited with prior headaches raises concern for a secondary cause of the new headache. A carotid bruit may accompany carotid artery dissection but is more commonly due to atherosclerotic plaque. Nasal discharge associated with sinus tenderness or signs of dental infection may reflect the cause of headache. (See "Diagnosis of giant cell arteritis" and "Acute sinusitis and rhinosinusitis in adults: Clinical manifestations and diagnosis" and "Complications, diagnosis, and treatment of odontogenic infections".)

CHOICE OF IMAGING EXAM

In a patient with a headache with high-risk features, imaging may be needed.

Computed tomography (CT) or magnetic resonance imaging (MRI) of the head is the preferred examination if the headache requires imaging evaluation [17]. Choice of modality and need for intravenous (IV) contrast depends upon the indication, availability of MRI, and patient contraindications. For imaging of the vessels, cerebral and cervical angiography using computed tomography with angiography (CTA) or magnetic resonance angiography (MRA) is performed as an added examination to head CT or MRI and usually requires IV contrast

administration. CTA or MRA examinations image the arteries, veins, or both, depending on the indication.

Examinations tailored for imaging the orbits and ear (encompassing the skull base and pituitary), face and maxilla (encompassing the paranasal sinuses), or the temporomandibular joint are sometimes added to the head imaging if an underlying diagnosis that localizes anatomically is suspected. Approximate effective radiation dose is 2 mSv for a head CT and 4 to 5 mSv for head CTA [18,19].

Factors to consider when choosing the appropriate imaging examination include diagnostic performance for the most likely diagnosis, availability of the technology, radiologist expertise, and safety considerations. The choice of when to image and with what modality for many suspected etiologies of headache is discussed here and in other related UpToDate topics. In addition, the ACR Appropriateness Criteria provides general guidance for many common clinical scenarios of headache [20]. When the decision is not obvious, consultation with the radiologist is helpful to facilitate patient referral.

EVALUATION OF PATIENTS WITHOUT HIGH-RISK FEATURES

Patients with a history of prior headaches who present to the emergency department (ED) 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, fever)
- No focal neurologic symptoms or abnormal neurologic or ophthalmologic examination findings
- No high-risk comorbidity

An extensive diagnostic workup and routine imaging in the ED are not needed in these patients [21]. However, some of these patients should be referred for evaluation for non-life-threatening but treatable causes of headache. (See "Evaluation of headache in adults".)

The yield of imaging is 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 computed tomography (CT) and magnetic resonance imaging (MRI) 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 [22]. The incidence of pathology was even lower (0.4 percent) in

patients whose headaches were typical of migraine and whose physical examinations were normal.

Another analysis of 10 years of data from the National Hospital Ambulatory Medical Care Survey (NHAMCS) reported that, of the 14 percent of ED patients with headache who had brain imaging at the discretion of the ED provider, 5.5 percent had a "pathological diagnosis" [3].

EVALUATION OF PATIENTS WITH HIGH-RISK FEATURES

Once historical and examination criteria have determined those patients with high-risk headache features, further evaluation is performed to diagnose the specific cause (algorithm 1). Laboratory tests, imaging, intraocular pressure measurements, and lumbar puncture (LP) for cerebrospinal fluid (CSF) are the options for further workup. In patients with hard neurologic examination findings whose testing is non-diagnostic, a neurology consultation may facilitate both the acute evaluation and follow-up. Critical diagnoses for the emergency department (ED) patient presenting with headache are described in the following table (table 2).

Sudden-onset "thunderclap" headache — Severe headache of sudden onset (ie, that reaches maximal intensity within a few **seconds** or **less than one minute** after the onset of pain) is known as "thunderclap headache" because its explosive and unexpected nature is likened to a clap of thunder. Approximately 8 percent of emergency department (ED) patients with a thunderclap headache have a subarachnoid hemorrhage [8,23]. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Nonaneurysmal subarachnoid hemorrhage" and "Overview of thunderclap headache".)

A head computed tomography (CT) without contrast should be performed and, if negative, an LP should follow in most cases. If either are positive for hemorrhage, subsequent evaluation is directed toward identifying the underlying cause (most often a cerebral aneurysm or an arteriovenous malformation) with computed tomography with angiography (CTA), magnetic resonance angiography (MRA), or catheter angiography. Because the consequences of missing subarachnoid hemorrhage (SAH) are potentially dire, most guidelines state that an LP should be performed in all patients with suspected SAH in whom the CT is normal. One exception is that if a high-quality CT is obtained within six hours of the onset of symptoms and interpreted by an expert radiologist to be normal, LP is not necessary. These issues are discussed in detail separately. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis", section on 'Evaluation and diagnosis'.)

Equally important to obtaining appropriate diagnostic testing are initiating actions to prevent acute complications and immediate consultation with a neurosurgeon or other cerebrovascular specialist.

Failure to evaluate patients with thunderclap headache thoroughly and expeditiously can result in misdiagnosis with resultant poor outcomes due to rebleeding, early hydrocephalus, or vasospasm. Misdiagnosed 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 and failure to do a CT or LP [14]. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis", section on 'Evaluation and diagnosis'.)

Traumatic lumbar puncture — A dilemma sometimes confronted in the ED is determining what additional workup and disposition are appropriate for patients at risk for SAH whose head CT is unrevealing and whose initial LP is presumed to be traumatic [24]. Several approaches are available to help distinguish between a traumatic tap and true SAH.

First, finding an elevated opening pressure (greater than 20 cm of water) suggests a pathologic process.

Of note, the clearing of blood from sequential tubes of CSF 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). A useful technique to increase the likelihood of obtaining a red cell count in the last tube that approaches zero is to waste several milliliters of CSF between the first and last tubes (this is not harmful, as 10 mL of CSF are produced within 20 to 30 minutes). Another technique is to test for xanthochromia, the presence of which strongly suggests a true SAH (though this too can be falsely positive). Interpretation of CSF analysis is discussed in detail separately. (See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis", section on 'Lumbar puncture'.)

Another approach 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, while a normal specimen from the repeat LP makes SAH less likely.

In cases where suspicion for SAH remains (ie, normal head CT and ambiguous CSF results), we recommend cerebrovascular imaging to resolve the ambiguity. Ideally, this includes head magnetic resonance imaging (MRI) without and with contrast and craniocervical MRA, or alternatively a head CT with contrast and craniocervical CTA. Neurologic consultation in the ED or admission for further evaluation by neurology or neurosurgery are also appropriate options.

(See "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis", section on 'Evaluation and diagnosis' and "Nonaneurysmal subarachnoid hemorrhage".)

Suspected meningitis or encephalitis — Fever and altered mental status, with or without nuchal rigidity, can indicate central nervous system (CNS) infection. If bacterial meningitis is considered, blood cultures should be obtained, then empiric antimicrobial therapy (with or without intravenous [IV] dexamethasone) should be instituted (table 6). Management is discussed in detail separately, but in addition to the standard agents for bacterial meningitis, empiric treatment with IV acyclovir for herpes encephalitis and IV doxycycline for tick-borne diseases (eg, Rocky Mountain spotted fever, anaplasmosis) should be given if these entities are serious considerations. (See "Treatment of bacterial meningitis caused by specific pathogens in adults", section on 'Approach to therapy' and "Dexamethasone to prevent neurologic complications of bacterial meningitis in adults" and "Aseptic meningitis in adults".)

Other potential infections to consider in the immunocompromised host or some resource-limited settings include fungal (eg, cryptococcal) and tuberculous meningitis. (See "Aseptic meningitis in adults" and "Central nervous system tuberculosis: An overview".)

After antibiotics have been given, perform a head CT without contrast to look for contraindications to LP prior to the procedure. A CT prior to a LP is mandatory in the following groups of patients (see "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Indications for CT scan before LP'). Note that LP should **not be delayed** to await a head CT in the **absence** of these criteria:

- Immunocompromised state (eg, HIV infection, immunosuppressive therapy, solid organ or hematopoietic stem cell transplantation)
- Active CNS disease (eg, acute stroke, mass lesion, abscess) causing intracranial mass effect
- New-onset seizure (within one week of presentation)
- Papilledema
- Abnormal level of consciousness
- Focal neurologic deficit

In the absence of CT findings that would contraindicate a safe LP (eg, a mass lesion or signs of generalized cerebral edema), one can proceed with LP, measuring the opening pressure. If LP is contraindicated on the basis of concerning CT findings, the patient should be admitted to an intensive care unit (ICU), antimicrobials continued, and a neurologist consulted. If CT is unavailable and the patient has one or more of the above criteria, we recommend that LP **not** be performed without obtaining further data, such as CT with contrast or MRI. If CT is unavailable in patients with seizure, papilledema, or other signs of increased intracranial

pressure (ICP), we recommend treating with antimicrobials as above and transferring to a facility where imaging is available. The airway should be secured as necessary for patients prior to transfer.

Rare documented cases of herniation following LP have made some clinicians reluctant to perform the procedure without prior imaging. In most patients, however, imaging is unnecessary prior to LP. In one prospective observational study that evaluated the need for head CT prior to LP, only 3 of 113 consecutive ED patients (2.7 percent) in whom an urgent LP was thought to be necessary (not only for headache) had a CT abnormality that was a contraindication to LP [25]. 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 in patients with suspected meningitis found similar results [26]. (See "Clinical features and diagnosis of acute bacterial meningitis in adults", section on 'Cerebrospinal fluid examination'.)

The clinical policy statement on acute headache of the American College of Emergency Physicians makes a recommendation based upon weak evidence that an LP without prior imaging may be performed in patients without signs of increased ICP (ie, papilledema, absent venous pulsations on funduscopy, altered neurologic status, or focal neurologic deficits) [21]. The presence of venous pulsations on funduscopic examination is strong evidence of normal ICP. (See "Approach to diagnosis and initial treatment of eye injuries in the emergency department" and "Slit lamp examination".)

Focal neurologic deficit or papilledema and new headache — Headache is the primary symptom of increased ICP, which should be suspected when accompanied by papilledema, focal neurologic deficit, or repeated episodes of nausea and vomiting without another explanation. Head MRI without and with contrast should be obtained to evaluate for an intracranial mass lesion (eg, primary or metastatic neoplasm, abscess, hematoma), communicating or obstructive hydrocephalus, or cerebral edema from ischemia or infarction (ie, stroke).

If MRI is not immediately available, head CT without contrast is an option to evaluate for findings associated with elevated ICP (eg, hydrocephalus, hemorrhage, and mass effect) prior to LP, and MRI without and with contrast is performed when the modality becomes available. CT without and with contrast is the second-line option and only performed if MRI is contraindicated or not available at all. If the patient has contraindications to both gadolinium-based contrast of MRI and iodinated contrast of CT (eg, eGFR<30 mL/min/1.73 m^2 and not on dialysis), MRI without contrast is preferred over CT without contrast.

If imaging does not reveal a structural cause or anatomical changes associated with elevated ICP, LP is performed to measure the opening pressure and to evaluate for underlying infection. Note that in patients with suspected idiopathic intracranial hypertension, most of whom will have papilledema, an LP with opening pressure is a necessary part of the evaluation, but it should **follow** brain imaging. (See "Overview and differential diagnosis of papilledema" and "Overview of the evaluation of stroke".)

Suspected carbon monoxide poisoning — If carbon monoxide (CO) poisoning is suggested by the history (eg, multiple family members or coworkers affected simultaneously; patient brought from enclosed space with an identified source such as gasoline or kerosene generator; rapid improvement when removed from source without other intervention), high-flow oxygen should be administered immediately and the diagnosis confirmed by measuring a carboxyhemoglobin concentration with cooximetry using an arterial blood gas sample. Standard pulse oximetry (SpO_2) does **not** differentiate carboxyhemoglobin from oxyhemoglobin. In addition, the partial pressure of oxygen (PaO_2) in an arterial blood gas is normal, as it measures dissolved oxygen (which is not affected by CO), not oxygen bound to hemoglobin (which is). Chronic low-dose CO exposure may also cause headache, more often during winter months. (See "Carbon monoxide poisoning".)

Suspected acute angle-closure glaucoma — An acute headache accompanied by eye pain or diminished vision, typically unilateral, suggests acute angle-closure glaucoma (sometimes referred to as acute narrow-angle glaucoma). Examination typically reveals a red eye with ciliary flush and no discharge, a pupil fixed in mid-dilation, and a cornea that is edematous or "steamy" appearing (not normally translucent) (picture 5). Elevated intraocular pressure confirms the diagnosis. Unnecessary neurologic workup and imaging delay treatment of this sight-threatening emergency. (See "Angle-closure glaucoma".)

Neck pain and/or Horner syndrome associated with new headache — Headache that radiates into the neck may be the result of carotid or vertebral artery dissection. Horner syndrome is seen in approximately 39 to 47 percent of those with carotid and up to 19 percent of those with vertebral artery dissection [27,28].

Most patients with an arterial dissection do not have a thunderclap headache (3.2 percent for carotid dissection and 9 percent for vertebral dissection) [29]. Nearly 60 percent of patients have brain ischemia or infarction [29]. However, nearly 10 percent of patients present with isolated headache or neck pain [30]. In the absence of a thunderclap onset, Horner syndrome, or symptoms or signs of brain ischemia or infarction, other clues include recent (even minor) trauma and the history (given by almost all patients with migraine who describe the headache from a dissection) as different from prior headaches [30].

Because headache (and neck pain) are so common, establishing the diagnosis of arterial dissection is very difficult in patients who present with pain **only** (ie, without neurologic findings) and who do not have any of the clues described above. As it is neither feasible nor desirable to evaluate every such patient with cerebrovascular imaging, occasionally patients may be misdiagnosed on a first visit. This highlights the importance of giving clear, specific discharge instructions about symptoms that should prompt immediate return to the ED.

Noncontrast head CT should be followed by CT and CTA of the head and neck with IV contrast. Alternatively, MRI and MRA of the head and neck without and with IV contrast is an equivalent option. CTA or MRA of the neck extends from the carotid and vertebral artery origins at the thoracic inlet to the circle of Willis at the skull base. Reported accuracies vary somewhat but are similar for CT and MRI. Reported sensitivity ranges from 50 to 80 percent and specificity from 67 to 99 percent for both modalities [31]. (See "Cerebral and cervical artery dissection: Clinical features and diagnosis".)

Older adult with new headache — A new or progressively worsening headache in a patient older than 50 years may suggest underlying tumor or hemorrhage. Head MRI without and with contrast is recommended. If MRI is not immediately available, head CT without contrast is an option to evaluate for findings associated with elevated ICP (eg, hydrocephalus, hemorrhage, and mass effect), and MRI without and with contrast is usually performed when the modality becomes available. CT without and with contrast is the second-line option and only performed if MRI is contraindicated or not available at all. If the patient has contraindications to both gadolinium-based contrast of MRI and iodinated contrast of CT (eg, eGFR<30 mL/min/1.73 m^2 and not on dialysis), MRI without contrast is preferred over CT without contrast. (See "Subdural hematoma in adults: Etiology, clinical features, and diagnosis", section on 'Clinical manifestations' and "Brain tumor headache".)

Immunosuppressed patient with new headache — Infection, lymphoma, and leukemia are complications of chronic immunosuppression. Head MRI without and with contrast should be obtained to evaluate for abscess or encephalitis. If MRI is not immediately available, head CT without contrast is an option to evaluate for findings associated with elevated ICP (eg, hydrocephalus, hemorrhage, and mass effect), and MRI without and with contrast is performed when the modality becomes available. CT without and with contrast is the second-line option and only performed if MRI is contraindicated or not available at all.

If the patient has contraindications to both gadolinium-based contrast of MRI and iodinated contrast of CT (eg, eGFR<30 mL/min/1.73 m^2 and not on dialysis), MRI without contrast is preferred over CT without contrast. If imaging does not reveal a structural cause or anatomical

changes associated with elevated ICP, LP is performed to measure the opening pressure and to evaluate for underlying infection or leptomeningeal metastases.

Cancer patient with new headache — Metastases and, if the patient is immunocompromised, infection are the primary concerns in this population. Head MRI without and with contrast is recommended. If MRI is not immediately available, head CT without contrast is an option to evaluate for findings associated with elevated ICP (eg, hydrocephalus, hemorrhage, and mass effect), and MRI without and with contrast is performed when the modality becomes available. CT without and with contrast is the second-line option and only performed if MRI is contraindicated or not available at all. If the patient has contraindications to both gadolinium-based contrast of MRI and iodinated contrast of CT (eg, eGFR<30 mL/min/1.73 m^2 and not on dialysis), MRI without contrast is preferred over CT without contrast. Leptomeningeal metastases are another consideration.

New headache in pregnancy — Preeclampsia and eclampsia must be considered as the cause of headache in every pregnant woman over 20 weeks of gestation, in addition to the many other causes of acute headache. Preeclampsia and eclampsia can also occur up to a few weeks postpartum. Cerebral venous sinus thrombosis is another particular concern in the postpartum period. Headache in the pregnant or postpartum patient is discussed separately. (See "Headache during pregnancy and postpartum".)

Orbital, sinonasal, or oromaxillofacial headache — Diagnostic workup for headache with localizing features to the face in the emergency setting should be pursued in patients with high-risk features. (See 'Identifying high-risk patients' above and "Overview of craniofacial pain".)

Headache with visual impairment, periorbital pain, or ophthalmoplegia could indicate acute angle-closure glaucoma, infection, inflammation, or tumor involving the orbits or cavernous sinus. MRI of the head and orbits without and with contrast would be the preferred imaging examination. However, if acute angle-closure glaucoma is suspected, an urgent ophthalmic examination is also indicated. (See "Angle-closure glaucoma" and "Orbital cellulitis" and "Tolosa-Hunt syndrome".)

Headache of sinonasal origin usually does not require imaging for diagnosis as it is best evaluated with nasal endoscopy performed by an ear, nose, and throat (ENT) specialist as an outpatient. If intracranial complications of sinus disease are suspected, head MRI without and with contrast is indicated. (See "Acute sinusitis and rhinosinusitis in adults: Clinical manifestations and diagnosis".)

Headache suspected to originate from maxillofacial conditions such as temporomandibular joint (TMJ) disorders or trigeminal neuralgia is best evaluated with head MRI without and with IV

contrast, generally as an outpatient. Headache suspected of odontogenic origin is better evaluated with CT, especially in the acute setting. Dental amalgam degrades image quality with both CT and MRI, and in such cases, imaging is likely to be of limited quality. (See "Temporomandibular disorders in adults" and "Trigeminal neuralgia" and "Complications, diagnosis, and treatment of odontogenic infections".)

Older adult with temporal artery abnormalities — Giant cell arteritis should be suspected in patients with temporal artery abnormalities (eg, tenderness, decreased pulsations), particularly if associated with jaw claudication, sudden monocular vision loss, or unexplained fever or anemia. The clinical manifestations, evaluation, and treatment of giant cell arteritis are discussed separately. (See "Clinical manifestations of giant cell arteritis" and "Diagnosis of giant cell arteritis" and "Treatment of giant cell arteritis".)

DISPOSITION

Patients with high-risk features in whom a secondary cause of headache is discovered are admitted or referred to the appropriate setting. For many patients in whom a secondary cause was suspected but a thorough, appropriate workup was normal, symptomatic treatment and discharge with timely primary care or neurologic follow-up is reasonable. However, some patients with worrisome high-risk features (eg, altered mental status, a new hard neurologic deficit or associated seizure), should be admitted for observation and further consultation.

Of note, patient response to analgesics should not be used as a diagnostic tool and should **not** dissuade performance of lumbar puncture (LP) when indicated by history or examination [32].

TREATMENT OF PAIN FROM 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 workup reveals no clear underlying process. The large majority of these patients will ultimately be diagnosed with either a migraine or cluster headache (table 7). Details of migraine and cluster headache treatment are discussed separately. (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 [33,34].

The authors of a systematic review of headache management in the ED propose the use of a parenterally administered nonsteroidal antiinflammatory drug (NSAID) and a dopamine antagonist [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 15 to 30 mg intravenously (IV) and prochlorperazine 10 mg. Haloperidol 2.5 to 5 mg IV or 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 or other extrapyramidal symptoms.

Two small randomized trials report that low-dose haloperidol is effective for alleviating severe pain due to a benign cause of headache [35,36]. In a small randomized trial, prochlorperazine was shown to be as effective as subcutaneous sumatriptan [36]. Treating nausea and vomiting with a parenteral agent such as ondansetron may make the patient more comfortable while other diagnostic and therapeutic steps are occurring. NSAIDs should be withheld in patients for whom there remains concern for a hemorrhagic cause of headache or who may require a lumbar puncture (LP).

For headaches unresponsive to treatment with a combination of NSAID and dopamine antagonist 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 [37-39]. Sumatriptan and oxygen are used to treat cluster headache.

The use of injectable opioids is strongly discouraged, but they may be necessary for patients with contraindications to NSAIDs or medications with vasoconstrictive effects (eg, dihydroergotamine), or for patients in whom prochlorperazine and diphenhydramine have not been effective.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Adult with headache in the emergency department".)

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

SUMMARY AND RECOMMENDATIONS

Danger signs and dangerous causes – Historical and clinical features associated with dangerous causes of headache are summarized in the following table and algorithm (table 1 and algorithm 1). Critical diagnoses for the emergency department (ED) patient presenting with headache are described in the following table (table 2). (See 'High-risk historical features' above and 'High-risk examination findings' above.)

- **Importance of neurologic abnormalities** – 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 patient's typical pattern of headache.

Evaluation and diagnostic imaging – 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 (LP), imaging, or both. (See 'Evaluation of patients with high-risk features' above.)

Computed tomography (CT) or magnetic resonance imaging (MRI) of the head is the preferred imaging examination for headache. Choice of modality and need for intravenous (IV) contrast depend upon the suspected diagnosis. For imaging of the vessels, cerebral and cervical computed tomography with angiography (CTA) or magnetic resonance imaging with angiography (MRA) is performed as an added examination to head CT or MRI and usually requires IV contrast administration.

Examinations tailored for imaging the orbits and ear (encompassing the skull base and pituitary), face and maxilla (encompassing the paranasal sinuses), or the temporomandibular joint are sometimes added to the head imaging if the suspected diagnosis localizes anatomically. (See 'Evaluation of patients with high-risk features' above.)

- **Symptom management** – Relief of symptoms is important, regardless of the headache's underlying cause, but pain relief should not be used to mean that the etiology of the headache is benign. Treatment options are described in the text. (See 'Disposition' above and 'Treatment of pain from undifferentiated headache in the emergency department' above.)

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Topic 287 Version 38.0

GRAPHICS

Clinical features of headache associated with serious pathology

History
Explosive onset and severe at onset
No similar headaches in the past
Concomitant infection
Altered mental status or seizure
Associated with exertion (eg, exercise, sexual intercourse)
Age over 50 years
Immunosuppression (eg, HIV, chronic glucocorticoid therapy)
Visual disturbances
Pregnancy or postpartum state
Medications or illicit drugs (eg, anticoagulants, sympathomimetic agents)
Physical examination
Abnormal vital signs
Neurologic abnormalities
Decreased level of consciousness
Meningismus
Toxic appearance
Papilledema or other ophthalmologic findings

HIV: human immunodeficiency virus.

Graphic 78567 Version 3.0

Cannot miss diagnoses in the ED patient with acute or severe headache

Diagnosis	History	Suggestive clinical findings
Subarachnoid hemorrhage	Thunderclap onset; severe pain at onset; transient loss of consciousness; severe neck pain; diplopia	Meningismus; nausea and vomiting
Reversible cerebral vasoconstriction syndromes (RCVS)	Thunderclap headaches of short duration that recur over days to weeks; may have recurrent trigger (eg, exertion, emotional stress, cough)	Neurologic deficits from underlying stroke or edema may develop
Cervical artery dissection	Recent head or neck trauma, even minor; prominent neck pain; new acute dizziness; tinnitus	Nystagmus; Horner syndrome (ptosis, miosis); cervical bruit; cranial neuropathy; stroke or TIA
Cerebral vein and dural sinus thrombosis	Risk factors for venous thromboembolism: highly variable presentation; headache; seizure	Neurologic deficits not consisten with arterial lesion; seizure; papilledema; encephalopathy
Hypertensive encephalopathy/reversible posterior leukoencephalopathy syndrome	Seizures and visual symptoms with headaches of insidious onset; nausea and vomiting	Severe hypertension (relative to patient's baseline); papilledema and retinal hemorrhage; encephalopathy; hematuria and proteinuria
Meningitis/encephalitis NOTE: Patients with Lyme meningitis often do not have severe findings	Ill appearing; fever (often >38°C); neck pain or stiffness; confusion or lethargy (may be sole finding in older adults)	Fever; meningismus; altered mental status; seizure; CN palsies; petechiae or purpura
Brain abscess	Headache (onset may be acute or gradual); recent history of bacteremia or head/neck infection; neck stiffness; fever (possibly)	Fever (often absent); papilledema; focal or cranial neurologic deficits (develop days to weeks after headache onset); depressed mental status
Brain tumor	History of cancer; headache exacerbated by cough, Valsalva, and maneuvers that increase ICP; headache can be sudden onset if bleeding into a tumor; nausea and vomiting	Papilledema; seizure; depressed mental status; neurologic deficits (weakness, sensation, aphasia, visual abnormalities)

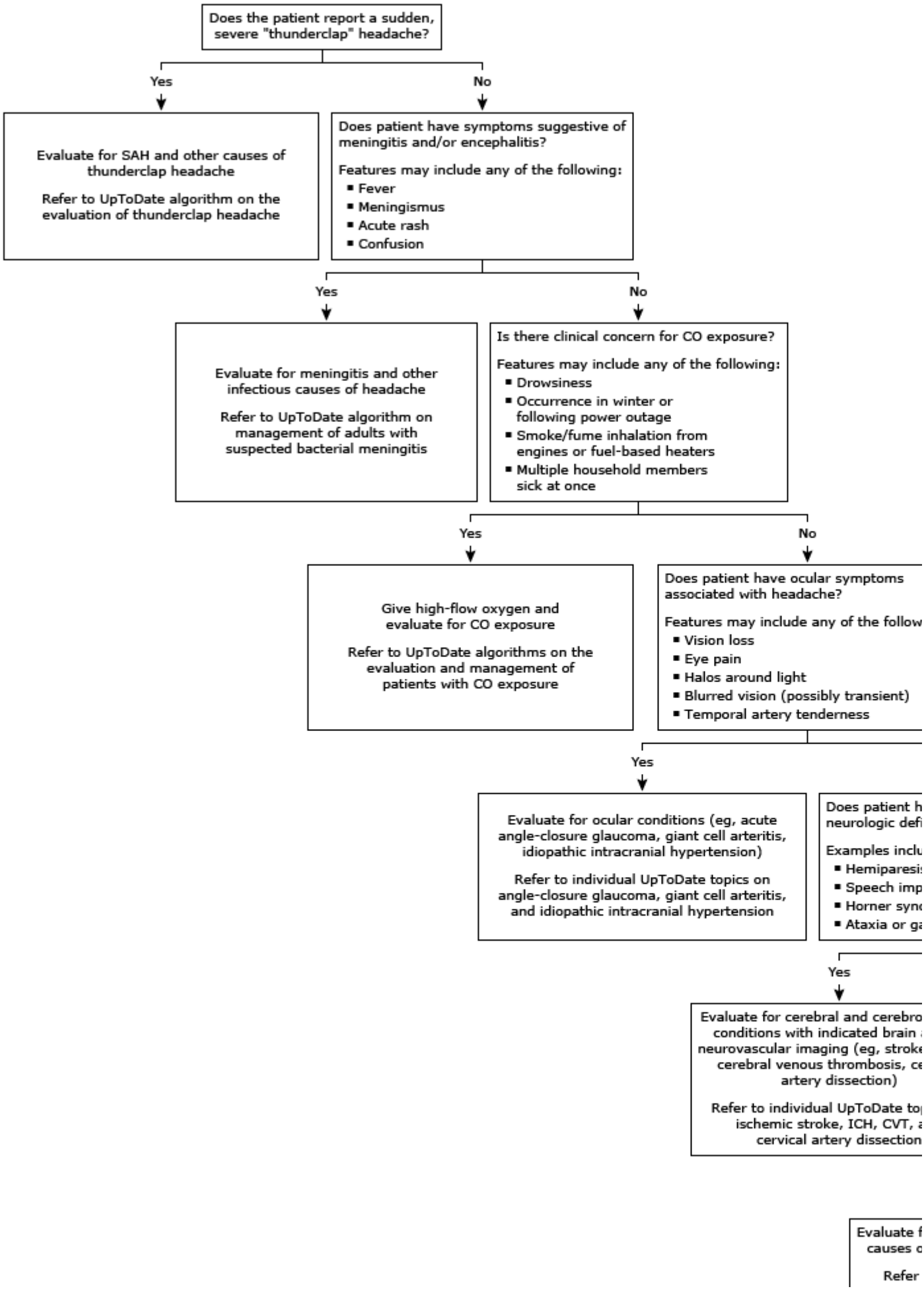
Intracranial, extra-axial hematoma (subdural, epidural)	History of head trauma (even minor); SDH can present weeks after trauma; use of anticoagulant medications	Coma; progressive decline in mental status; hematoma in posterior fossa can present with: vomiting, anisocoria, dysphagia, CN palsies, nuchal rigidity, ataxia
Intraparenchymal hemorrhage (IPH)	Headache usually sudden in onset; use of anticoagulant medications	Findings on neurologic examination depend on site of hemorrhage (examination may be unremarkable)
Colloid cyst of third ventricle	Positional headache that may resolve upon lying flat; confusion; symptoms may be intermittent	Altered mental status with headache
Idiopathic intracranial hypertension	Overweight or obese female of childbearing age; intermittent visual symptoms (eg, decline in vision; flashes, double vision)	Papilledema; sixth cranial nerve palsy; visual deficit (eg, field cut; may be transient); tinnitus; other CN palsies
Spontaneous intracranial hypotension	Severe headache upon standing (often within 15 minutes) that resolves with lying flat; Valsalva exacerbates headache; tinnitus; neck pain; nausea and vomiting	Neurologic examination often normal; wide range of possible findings
Acute narrow-angle glaucoma	Eye pain; red or tearing eye; decreased vision; nausea and vomiting	Red, injected conjunctiva; "steamy" edematous cornea; fixed, mid-dilated pupil (4 to 6 mm); firm globe; elevated IOP (often >30 mmHg)
Giant cell arteritis	Patient 50 years or older with headache, abrupt onset visual disturbances (transient monocular vision loss), and possibly jaw claudication, fever	Palpable or nodular temporal arteries; wide range of possible symptoms and signs; fever; elevated ESR and/or CRP
Carbon monoxide toxicity	History of CO exposure; others at same site affected (eg, worksite or abode); headache resolves or declines when away from CO source	Findings vary with severity of exposure; severe exposure can produce coma, seizures, myocardial ischemia; moderate or mild exposure causes malaise, nausea, dizziness; may see cherry red macula on funduscopy

ED: emergency department; SAH: subarachnoid hemorrhage; RCVS: reversible cerebral vasoconstriction syndromes; TIA: transient ischemic attack; CN: cranial nerve; ICP: intracranial pressure; SDH: subdural

hematoma; EDH: epidural hematoma; IPH: intraparenchymal hemorrhage; IOP: intraorifice pressure; ESR: erythrocyte sedimentation rate; CRP: c-reactive protein; CO: carbon monoxide.

Graphic 118117 Version 2.0

Emergency evaluation of the adult with new nontraumatic headache


Does the patient report a sudden, severe "thunderclap" headache?
Yes
No
Evaluate for SAH and other causes of thunderclap headache
Refer to UpToDate algorithm on the evaluation of thunderclap headache
Does patient have symptoms suggestive of meningitis and/or encephalitis?
Features may include any of the following:
Fever
Meningismus
Acute rash
Confusion
Yes
No
Evaluate for meningitis and other infectious causes of headache
Refer to UpToDate algorithm on management of adults with suspected bacterial meningitis
Is there clinical concern for CO exposure?
Features may include any of the following:
Drowsiness
Occurrence in winter or following power outage
Smoke/fume inhalation from engines or fuel-based heaters
Multiple household members sick at once
Yes
No
Give high-flow oxygen and evaluate for CO exposure
Refer to UpToDate algorithms on the evaluation and management of patients with CO exposure
Does patient have ocular symptoms associated with headache?
Features may include any of the follow
Vision loss
Eye pain
Halos around light
Blurred vision (possibly transient)
Temporal artery tenderness
Yes
Evaluate for ocular conditions (eg, acute angle-closure glaucoma, giant cell arteritis, idiopathic intracranial hypertension)
Refer to individual UpToDate topics on angle-closure glaucoma, giant cell arteritis, and idiopathic intracranial hypertension
Does patient h
neurologic defi
Examples inclu
Hemiparesis
Speech imp
Horner synd
Ataxia or ga
Yes
Evaluate for cerebral and cerebro
conditions with indicated brain
neurovascular imaging (eg, stroke
cerebral venous thrombosis, ce
artery dissection)
Refer to individual UpToDate to
ischemic stroke, ICH, CVT, a
cervical artery dissection
Evaluate f
causes o
Refer

The evaluation of new-onset nontraumatic headache involves assessing for secondary (eg, structural, inflammatory) causes as well as identifying primary headache syndromes.

SAH: subarachnoid hemorrhage; CO: carbon monoxide; ICH: intracranial hemorrhage; CVT: cerebral venous thrombosis; HIV: human immunodeficiency virus.

* Primary headache syndromes include migraine and related conditions, tension-type headaches, trigeminal autonomic cephalalgias (cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks, and hemicrania continua), and less common primary headache disorders (eg, new persistent daily headache, primary cough headache, primary exercise headache). Refer to UpToDate topics for additional details.

Graphic 107496 Version 3.0

Etiologies of thunderclap headache

Most common causes of thunderclap headache:
Subarachnoid hemorrhage
Reversible cerebral vasoconstriction syndromes (RCVS)
Conditions that less commonly cause thunderclap headache:
Cerebral infection (eg, meningitis, acute complicated sinusitis)
Cerebral venous thrombosis
Cervical artery dissection
Spontaneous intracranial hypotension
Acute hypertensive crisis
Posterior reversible leukoencephalopathy syndrome (PRES)
Intracerebral hemorrhage
Ischemic stroke
Conditions that uncommonly or rarely cause thunderclap headache:
Pituitary apoplexy
Colloid cyst of the third ventricle
Aortic arch dissection
Aqueductal stenosis
Brain tumor
Giant cell arteritis
Pheochromocytoma
Pneumocephalus
Retroclival hematoma
Spinal epidural hematoma
Varicella zoster virus vasculopathy
Vogt-Koyanagi-Harada syndrome
Disputed causes of thunderclap headache:
Sentinel headache (unruptured intracranial aneurysm)*
Primary thunderclap headache¶

* Sentinel headache due to an unruptured intracranial aneurysm is a possible cause of thunderclap headache, but supporting data are weak.

¶ There is controversy as to whether thunderclap headache can occur as a benign and potentially recurrent headache disorder in the absence of underlying organic intracranial pathology.

Graphic 81710 Version 8.0

Erythema migrans

Erythema migrans with central clearing and a necrotic center.

Courtesy of Dori F Zaleznik, MD.

Graphic 81270 Version 1.0

Petechiae

Courtesy of Leslie Raffini, MD.

Graphic 73905 Version 2.0

Acute meningococcemia

Skin lesions in acute meningococcemia can begin as papules but quickly progress to petechiae and purpura. As seen here, the purpuric lesions can coalesce.

Courtesy of Charles V Sanders. (The Skin and Infection: A Color Atlas and Text, Sanders CV, Nesbitt, LT Jr [Eds], Williams & Wilkins, Baltimore, 1995).

Graphic 52107 Version 6.0

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

HIV/AIDS: human immunodeficiency virus/acquired immunodeficiency syndrome.

Graphic 80966 Version 5.0

"Positive" versus "negative" neurologic phenomena with headache*

Finding	Positive finding	Negative finding
Visual	Scintillating scotomata Fortification pattern	Blindness (absence of vision)
Motor	Involuntary movement of muscles	Weakness (absence of strength)
Sensory	"Pins and needles" or tingling ("like when your leg falls asleep")	Numbness ("like local anesthetic given a the dentist's office")
Speech	Saying nonsensical words	Decreased speech output

* As a simple rule of thumb, the positive findings above suggest a migrainous or seizure etiology, whereas negative findings are associated with destructive lesions, especially ischemia or infarction. To emphasize, this is a general guide, and exceptions occur. In migraine patients, for example, initial symptoms are typically positive but then may switch to negative. Thus, a careful history remains essential.

Graphic 132622 Version 1.0

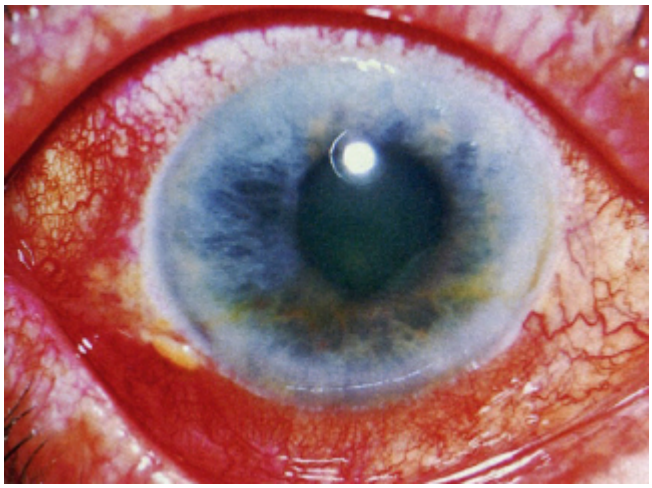
Papilledema

Papilledema, characterized by blurring of the optic disc margins, loss of physiologic cupping, hyperemia, and fullness of the veins, in a 5-year-old girl with intracranial hypertension due to vitamin A intoxication.

Courtesy of Gerald Striph, MD.

Graphic 50378 Version 1.0

Acute angle-closure glaucoma



The conjunctival vessels are dilated, especially near the cornea (ciliary flush) and the cornea is slightly hazy (edematous).

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Graphic 80395 Version 14.0

Recommendations for empiric antimicrobial therapy for purulent meningitis based on patient age and specific predisposing condition*

Predisposing factor	Common bacterial pathogens	Antimicrobial therapy
Age		
<1 month	*Streptococcus agalactiae*, *Escherichia coli*, *Listeria monocytogenes*	Ampicillin plus cefotaxime; OR ampicillin plus an aminoglycoside
1 to 23 months	*Streptococcus pneumoniae*, *Neisseria meningitidis*, *S. agalactiae*, *Haemophilus influenzae*, *E. coli*	Vancomycin plus a third-generation cephalosporin¶ Δ ◇
2 to 50 years	*N. meningitidis*, *S. pneumoniae*	Vancomycin plus a third-generation cephalosporin¶ Δ ◇
>50 years	*S. pneumoniae*, *N. meningitidis*, *L. monocytogenes*, aerobic gram-negative bacilli	Vancomycin plus ampicillin plus a third-generation cephalosporin¶ Δ
Head trauma		
Basilar skull fracture	*S. pneumoniae*, *H. influenzae*, group A beta-hemolytic streptococci	Vancomycin plus a third-generation cephalosporin¶ Δ
Penetrating trauma	*Staphylococcus aureus*, coagulase-negative staphylococci (especially *Staphylococcus epidermidis*), aerobic gram-negative bacilli (including *Pseudomonas aeruginosa*)	Vancomycin plus cefepime; OR vancomycin plus ceftazidime; OR vancomycin plus meropenem
Postneurosurgery	Aerobic gram-negative bacilli (including *P. aeruginosa*), *S. aureus*, coagulase-negative staphylococci (especially *S. epidermidis*)	Vancomycin plus cefepime; OR vancomycin plus ceftazidime; OR vancomycin plus meropenem
Immunocompromised state	*S. pneumoniae*, *N. meningitidis*, *L. monocytogenes*, aerobic gram-negative bacilli (including *P. aeruginosa*)	Vancomycin plus ampicillin plus cefepime; OR vancomycin plus meropenem§

* For recommended doses, refer to the UpToDate content on treatment of bacterial meningitis in children and adults.

¶ Ceftriaxone or cefotaxime.

Δ Some experts would add rifampin if dexamethasone is also given.

◇ Add ampicillin if meningitis caused by *Listeria monocytogenes* is suspected.

§ Meropenem provides sufficient coverage for *Listeria* when used as part of an initial regimen. However, if *Listeria* is identified, the patient should generally be switched to a regimen that includes ampicillin. Refer

to the UpToDate topic that discusses treatment of *Listeria* for a discussion of regimen selection.

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Graphic 71968 Version 12.0

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

Symptom	Migraine	Tension-type	Cluster
Location	Adults: Unilateral in 60 to 70%, bifrontal or global in 30% Children and adolescents: Bilateral in majority	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	30 minutes to 7 days	15 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 syndrome; restlessness or agitation; focal neurologic symptoms rare; sensitivity to alcohol

Graphic 68064 Version 7.0