

Evaluation and Management of Life-Threatening Headaches in the Emergency Department

Abstract

Headache is the fourth most common reason for emergency department encounters, accounting for 3% of all visits in the United States. Though troublesome, 90% are relatively benign primary headaches --migraine, tension, and cluster headaches. The other 10% are secondary headaches, caused by separate underlying processes, with vascular, infectious, or traumatic etiologies, and they are potentially life-threatening. This issue details the important pathophysiologic features of the most common types of life-threatening headaches, the key historical and physical examination information emergency clinicians must obtain, the red flags that cannot be missed, and the current evidence for best-practice testing, imaging, treatment, and disposition.

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Case Presentations

A previously healthy 30-year-old man presents to the ED complaining of the “worst headache of my life.” He describes it as sharp, nonradiating, with an abrupt onset 5 hours ago. You are concerned for subarachnoid hemorrhage. You provide pain medication and obtain a noncontrast CT scan of the head, which is negative. The patient is feeling better and wants to go home. You wonder whether a negative CT is sufficient to rule out an SAH or whether a lumbar puncture should be done...

A 55-year-old man with history of nonsmall cell lung cancer who is on cisplatin presents with an acute headache and lethargy for 6 hours. His vital signs are remarkable for a blood pressure of 210/120 mm Hg, heart rate of 70 beats/min, and a temperature of 36.7°C (98°F). His physical exam reveals a lethargic patient with no localizing neurologic signs and no meningismus. You order a noncontrast CT of the head and consider lowering this patient's blood pressure, though you wonder how much and how fast it should be reduced...

A 45-year-old woman presents to the ED complaining of a severe occipital headache, neck pain, and dizziness. Earlier in the day, she was involved in a motor vehicle crash and suffered “whiplash.” Her neurologic exam is normal, including no nystagmus and normal cerebellar function, but you are concerned that this patient may have a vertebral artery dissection, and you order a CTA head and neck. You wonder: if it's positive, should the treatment include anticoagulation or antiplatelet therapy...or both?

Introduction

The third edition of the International Classification of Headache Disorders (ICHD-3), published in January 2018, is the most up-to-date and widely accepted standard criteria for the classification of headaches.¹ The ICHD-3 classifies headaches into 3 distinct categories: (1) primary headache disorders, including migraine, tension, and cluster headaches; (2) secondary headaches, including potentially life-threatening forms of headaches such as those secondary to vascular disorders, traumatic injury, and disorders in hemostasis; and (3) cranial neuropathies, such as trigeminal neuralgia.

The National Hospital Ambulatory Medical Care Survey reviewed over 10,000 patients presenting to emergency departments (EDs) for acute headache and found that 2% represented secondary headaches.² Although they are rare, life-threatening headaches require prompt diagnosis and treatment, as delays in some diagnoses can have a mortality rate approaching 50%.³

Evaluating complaints of acute headache is a common practice in the ED, and distinguishing benign from serious pathology can be a diagnostic challenge. A focused workup begins with a careful,

well-organized clinical history and physical examination. Physical examination findings such as abnormal vital signs, papilledema, cranial nerve palsies, and neck pain are suggestive of more concerning headache etiologies.⁴ Resources such as ocular ultrasound, neuroimaging, and lumbar puncture are important strategies, but the sensitivity and specificity of the results must be understood in order to apply them correctly. This issue of *Emergency Medicine Practice* focuses on the most commonly encountered causes of life-threatening secondary headaches and provides best-practice recommendations on their initial evaluation and management.

Critical Appraisal of the Literature

A literature search from 1993 to 2018 was conducted using PubMed and Ovid MEDLINE®, with the search terms *headaches AND emergency, sudden onset, fever, visual symptoms, neurologic deficits, high-risk, trauma, immunocompromised, pregnancy, coagulopathy, and life threatening*. The National Guideline Clearinghouse and the Cochrane Database of Systematic Reviews were searched. Guidelines published by the American College of Emergency Physicians (ACEP) and the American Academy of Neurology were searched. International guidelines, including the Canadian and European neurology guidelines, were also reviewed. Over 500 abstracts published within the last 25 years were examined, and 89 of these full-text articles were reviewed and included for reference. Many of the identified articles were prospective studies, meta-analyses, clinical guidelines, and literature reviews.

Etiology and Pathophysiology

Brain parenchyma has no pain receptors. Headaches result from traction to or irritation of the meninges and blood vessels, which are the only innervated central nervous system (CNS) structures. Nociceptors located in surrounding tissue and vasculature may be stimulated by trauma, neurogenic inflammation, edema, tension, or space-occupying lesions. It is the activation of these specific nerve ganglion complexes by neuropeptides, including substance P and calcitonin gene-related peptide, that contributes to headache pain.⁵

Both primary and secondary headaches have common pain pathways, so response to pain medications does not exclude potential life-threatening secondary causes. Multiple case reports document relief with nonsteroidal anti-inflammatory drugs (NSAIDs), triptans, and neuroleptics in patients with subarachnoid hemorrhage or mass lesions.⁶ Therefore, in accordance with the guidelines from ACEP,

excluding dangerous secondary causes of headaches should not be based on response to analgesics (ACEP Level C recommendation).⁷

Elderly patients presenting with headache are at higher risk for secondary causes of headache, such as intracranial hemorrhage, acute angle closure glaucoma, giant cell arteritis, and malignancy.⁸ A large retrospective study that evaluated risk factors for intracranial pathology in patients presenting with headache found that patients aged > 50 years were 4 times more likely to have a pathologic diagnosis.⁴ **Table 1** lists causes of secondary headaches and their initial signs and symptoms.

Differential Diagnosis

The following sections summarize the etiology and clinical features of the most common life-threatening headaches that should be on the differential for a patient with a headache: subarachnoid hemorrhage (SAH); cervical artery dissection (CAD), which includes vertebral artery dissection and carotid artery dissection; cerebral venous thrombosis (CVT); idiopathic intracranial hypertension (IIH); giant cell arteritis (GCA); and posterior reversible encephalopathy syndrome (PRES). Also on the differential and discussed in following sections are meningitis, acute angle closure glaucoma, pre-eclampsia/eclampsia, and carbon monoxide poisoning.

Subarachnoid Hemorrhage

SAH represents approximately 1% of all headaches presenting to the ED and affects nearly 30,000 North Americans each year.⁹ Nontraumatic or spontaneous SAH is most commonly caused by aneurysm rupture; other etiologies include arteriovenous malformations and idiopathic etiologies.¹⁰ Misdiagnosis can be catastrophic, with case-fatality rates up to 50%.¹¹

Perhaps the most distinctive historical feature of SAH is a headache described as “abrupt in onset,” a feature that is documented in 75% of SAH patients. Approximately 25% of these patients describe a transient alteration or complete loss of consciousness.¹² Additional symptoms include neck stiffness, vomiting, and double vision. Approximately 20% of patients with SAH have warning signs of a sentinel bleed preceding the major hemorrhage. Symptoms often occur within days to weeks of rupture and include: (1) headaches lasting several hours or days, (2) associated cranial nerve palsies, (3) neck pain, and (4) nausea and vomiting.^{13,14} A recent meta-analysis of 22 studies offered important historical and examination findings seen in SAH.¹⁵ (See **Table 2**, page 4.)

Cervical Artery Dissection

CAD is a diagnosis that includes carotid artery dissections and vertebral artery dissections, and it is estimated to be the cause of 2% of all strokes and 20% of strokes in adults aged ≤ 50 years.¹⁶ CAD

Table 1. Life-Threatening Secondary Causes of Headache

Secondary Headache Cause	Signs and Symptoms
Subarachnoid hemorrhage	- Sudden onset, onset during exertion - Loss of consciousness - Neck pain or stiffness, limited neck flexion on examination - Age ≥ 40 years - CN III palsy with associated ipsilateral pupillary dilation
Carotid artery dissection	- Neck pain - Anterior circulation stroke symptoms such as hemiplegia and slurred speech, partial Horner syndrome (ptosis and miosis)
Vertebral artery dissection	- Neck pain - Recent neck flexion/extension trauma - History of Ehlers-Danlos or Marfan syndromes - Posterior circulation stroke symptoms such as vertigo, dizziness, dyscoordination
Central venous thrombosis	- Pregnant, post partum, other hypercoagulable state - Signs of intracranial pressure, such as papilledema and CN VI palsy - Neurologic findings may be present, including hemiparesis, ataxia, and seizure
Idiopathic intracranial hypertension	- Changes in body position exacerbate symptoms - CN VI palsy and horizontal binocular diplopia (double vision induced with lateral gaze) - Pulsatile tinnitus
Giant cell arteritis	- Temporal artery beading and tenderness - Jaw claudication - Diplopia
Posterior reversible encephalopathy syndrome	- Acute-onset headache, elevated blood pressure, altered level of consciousness - Visual disturbances - Tonic-clonic seizures may also be present
Meningitis	- Fever, neck pain, nuchal rigidity - Immunocompromised state
Acute glaucoma	- Advanced age - Eye pain, redness - Unilateral blurry vision or vision loss
Pre-eclampsia	- Patient pregnant or post partum - Hypertension, proteinuria, peripheral edema, thrombocytopenia, liver and/or renal impairment
Carbon monoxide poisoning	- Peripheral cyanosis - Confusion - Blurry vision - Nausea and vomiting

can occur spontaneously, but is more commonly associated with a traumatic event such as seat-belt injury, hanging, vigorous physical activity, and chiropractic manipulation.¹⁷ Disruption of the blood vessel wall leads to intimal bleeding, hematoma formation, and eventually thromboembolic stroke. Risk factors for CAD include Ehlers-Danlos syndrome, osteogenesis imperfecta, and Marfan syndrome.¹⁸ For more information on CAD, see the July 2016 issue of *Emergency Medicine Practice*, "Cervical Artery Dissection: Early Recognition and Stroke Prevention," available at: www.ebmedicine.net/CAD

Cerebral Venous Thrombosis

CVT commonly presents as a gradual-onset headache, although some cases have been documented as CVT presenting with a thunderclap or sudden-onset headache.¹⁹ Typically the result of thrombotic disease, risk factors for CVT include oral contraceptive use, pregnancy and postpartum states, Factor V Leiden deficiency, and lupus. Infections spreading from the sinuses, ears, face, and mouth can also cause a sinus thrombus to develop.²⁰

Idiopathic Intracranial Hypertension

Typically associated with obese females of child-bearing age, the incidence of IIH has been increasing due to the rise in obesity in the population.²¹ Hypervitaminosis A from excessive dietary intake has been a long-known cause of IIH.²² Other retinoids used in the treatment of dermatologic conditions and cancer therapy, all-trans-retinoic acid, retinol, isotretinoin, etretinate, and tretinoin have also been reported to be associated with IIH. Tetracyclines have been linked to IIH in a number of cases, often appearing shortly after onset of treatment.²²

Giant Cell Arteritis

GCA is a rare diagnosis with a prevalence of < 1%.²³ It is 3 times more likely in women and almost exclusively found in patients older than 50 years.²⁴ Common clinical features of GCA include

Table 2. Historical and Physical Examination Findings Associated with Subarachnoid Hemorrhage¹⁶

Findings	Positive Likelihood Ratio
Neck stiffness (objective)	6.6
Neck stiffness (subjective)	4.1
Focal neurologic deficit	3.2
Photophobia	2.3
Loss of consciousness	1.9
Onset during exertion	1.7
Sudden onset	1.3
Onset during intercourse	1.2

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systemic symptoms such as fever, fatigue, and myalgia, and localized symptoms such as headache, jaw claudication, and visual symptoms such as diplopia and amaurosis fugax.²⁵ Polymyalgia rheumatica is present in more than half of all GCA patients; therefore, history should include questions related to chronic muscle pain, particularly in the shoulders and hips.²⁶

Posterior Reversible Encephalopathy Syndrome

PRES is a form of hypertensive emergency that is reversible.²⁷ The presumed pathogenesis of PRES suggests that severe hypertension leads to cerebral autoregulatory failure, vasodilatation, interstitial extravasation of fluid, and subsequent vasogenic brain edema.²⁸ The most common conditions associated with PRES are hypertensive encephalopathy, eclampsia, and the use of immunosuppressive agents such as cyclosporine, tacrolimus, and cisplatin.²⁹ The clinical syndrome of PRES is typically characterized by elevated blood pressure, acute-onset headache, and altered level of consciousness. Generalized tonic-clonic seizures occur in up to 75% of these patients.³⁰

Prehospital Care

The prehospital approach to the patient with headache includes: (1) conducting a primary survey and eliciting a basic history, particularly with regard to the time of onset, provocation, quality, and severity; (2) conducting a focused neurologic examination using the Cincinnati Prehospital Stroke Scale (CPSS); and (3) assessing for red flags; ie, signs and symptoms of dangerous causes of headache. (See Table 3.)

The CPSS provides a framework for the prehospital assessment of acute headache, as several life-threatening headaches present with neurologic deficits.³¹ Patients with neurologic deficits and patients with severe, sudden-onset headache should be transported immediately to the nearest available stroke center. Acetaminophen may be offered for

Table 3. Red Flags for Life-Threatening Headaches

- Focal neurologic deficits
- Sudden-onset or "thunderclap" headache
- New-onset headache in patients aged ≥ 50 years
- Neck pain or neck stiffness
- Changes in visual acuity
- Fever or immunocompromised state
- History of malignancy
- Pregnancy or postpartum status
- Syncope
- Seizure

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initial pain management during transport; opioids and NSAIDs should be avoided.

Emergency Department Evaluation

A thorough history and physical examination will guide the emergency clinician in determining the need for laboratory testing and imaging studies.

History

The history should include the time of onset of the headache and its location, its severity, and associated symptoms. **Table 4** lists historical factors that are concerning for a life-threatening etiology. Associated signs and symptoms, particularly those considered to be red flags for life-threatening headaches, should be considered when eliciting the history of a patient presenting with headache. (See **Tables 2 and 3, page 4.**)

Physical Examination

The physical examination in the patient with headache includes an assessment of the following 5 components: (1) vital signs, (2) neurologic function, (3) cranial nerves, (4) head and neck, and (5) fundoscopic evaluation.

Table 4. Historical Factors and Concerning Descriptors

Historical Factors	Concerning Descriptors
Onset	Sudden-onset headaches or those elicited during exercise, straining, or orgasm are concerning for SAH, ICH, or CVT.
Provocation	Headaches exacerbated by changes in position, particularly the supine position or coughing, are concerning for decompensated elevated ICP.
Quality	Change in the quality, pattern, or intensity of a known pre-existing headache syndrome requires the same evaluation as a new-onset headache.
Radiation	Headaches with associated pain that radiates to the neck should prompt consideration of SAH and CAD.
Severity	"Thunderclap" or "worst headache" descriptors should prompt the clinician to have a high index of suspicion for SAH, CVT, and ICH.
Temporal	Chronic headaches that have changed over time should raise concern for structural abnormalities such as an intracranial mass or tumor.

Abbreviations: CAD, cervical artery dissection; CVT, cerebral venous thrombosis; ICH, intracranial hemorrhage; ICP, intracranial pressure; SAH, subarachnoid hemorrhage.

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Vital Signs

A complete set of vital signs must be obtained in all patients presenting to the ED with an acute headache. Abnormal vital signs, particularly with regard to elevated blood pressure and temperature, may be associated with life-threatening etiologies.

Acute headache in the setting of severe hypertension should prompt a search for signs of end-organ damage such as hypertensive encephalopathy, intracranial hemorrhage, PRES, and pre-eclampsia in pregnant women.

Patients presenting with increased intracranial pressure (ICP) may have an associated vasopressor response. This is also known as the *Cushing reflex*, which is a triad of increased blood pressure, irregular respirations, and bradycardia. These findings should prompt the emergency clinician to look for causes of ICP that include SAH, acute stroke, and IIH.

Fever in the setting of acute headache should raise concern for CNS infections that include meningitis, brain abscess, and encephalitis. In particular, patients presenting with headache, fever, altered mental status, and neck stiffness should prompt consideration of meningitis. Ninety-five percent of patients with bacterial meningitis will present with a combination of 2 of these 4 symptoms.³²

Neurologic Function

A focal neurologic deficit in the setting of an acute headache is the single highest predictor for the presence of intracranial pathology.³³ Careful attention should be paid to evaluating the patient's mental status, including level of alertness, orientation, and attention (eg, reciting the months of the year in reverse). The neurologic examination should also include assessment of motor strength, coordination, reflexes, sensory function, and gait. Neurologic abnormalities suggestive of a lesion involving the anterior circulation (such as dysarthria and cognitive impairment) should prompt consideration of carotid artery dissection, particularly if Horner syndrome is also present. Conversely, lesions of the posterior circulation can cause dizziness, vision changes, and limb weakness and are symptoms that may be seen in strokes from a vertebral artery dissection.³⁴

Cranial Nerves

The focused neurologic examination in patients with a chief complaint of headache includes a careful assessment of select cranial nerves (CN). The optic nerve (CN II) controls the afferent pupillary reflex and can be tested using the swinging flashlight test, which involves shining a bright light into each eye and watching for brisk pupillary constriction. Paradoxical dilation indicates an afferent pupillary defect, also known as a *Marcus Gunn pupil*. Conditions with an afferent pupillary defect include optic neuritis, GCA, and central retinal artery occlusion.³⁵

CN III, IV, and VI are tested by having the patient follow, with his eyes, a finger that is drawing an “H” pattern without moving the head. In the setting of headache, oculomotor nerve palsy (CN III), ipsilateral mydriasis, ptosis, and abnormal extraocular movements (“down-and-out eye”) are concerning for a posterior communicating aneurysm and can also be seen in the setting of SAH.³⁶

Abducens nerve (CN VI) deficits may be elicited when a patient develops diplopia with lateral gaze. The patient may also have convergent strabismus, where one eye points toward the nose. CN VI deficits may be present in patients with ICP, such as with IIH and CVT. These patients may also have impaired visual acuity, visual field defects, and tunnel vision due to papilledema.³⁷

Head and Neck Examination

A thorough head and neck examination should accompany the physical examination for all patients presenting with headache. Findings such as neck rigidity and stiffness should raise concern for possible meningitis or SAH.

Patients presenting with headache and neck pain should raise concern for CAD. Partial Horner syndrome may also be present in patients with CAD, leading to miosis and ptosis. Anhidrosis is absent, as sympathetic fibers innervating the facial sweat glands are not affected. Patients may present initially with head or neck pain but without any neurologic deficits, as neurologic symptoms may take several days to develop.³⁸

Unilateral tenderness and beading to the temporal artery in a patient presenting with acute headache may suggest GCA. Smetana et al published an excellent review on clinical examination findings for GCA. Symptoms with the highest positive likelihood ratio (LR) include jaw claudication (LR, 4.2), diplopia (LR, 3.4) Physical examination findings include temporal artery beading (LR, 4.6), and temporal artery tenderness (LR, 2.6).²⁴

Fundoscopy Examination

Examination of a patient presenting with acute headache should include a fundoscopic examination. (See Table 5.) If possible, pharmacologic dilation will enhance visualization of the disk, macula, and proximal vessels. Dilation can be achieved by using 1 drop of 1% tropicamide. Fundoscopy can reveal papilledema, a common finding in IIH, malignant hypertension, and CVT.

To view a video demonstration of a fundoscopic examination, go to: <https://stanfordmedicine25.stanford.edu/the25/fundoscopy.html> or scan the QR code with an enabled device.



Diagnostic Studies

Laboratory Testing

Routine laboratory testing, such as a complete blood cell count and a metabolic panel, are typically of low utility in aiding in the diagnosis of headaches. However, if a life-threatening headache is suspected, there are several circumstances that warrant specific laboratory testing. For example, there is utility in obtaining a serum blood sugar level for a patient with headache and altered mental status, or a pregnancy screen for a female patient with acute headache and elevated blood pressure.

Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are often obtained on patients being evaluated for GCA. However, several retrospective studies have demonstrated their poor sensitivity and specificity to diagnose GCA.^{40,41} While an elevated ESR and CRP makes the diagnosis of GCA more likely, patients for whom there is a high suspicion of GCA should be treated, and consultation obtained for a biopsy even if both the ESR and CRP results are negative.

If there is concern for carbon monoxide (CO) exposure, carboxyhemoglobin (COHb) level can be obtained with a co-oximeter, a device that spectrophotometrically reads the percentage of total hemoglobin saturated with CO (blood gas).⁴² Either a venous or arterial blood sample can be used to detect COHb.

Several small studies have looked at the utility of D-dimer to risk stratify patients presenting to the ED with headache suspicious for CVT.⁴³ One prospective study that looked at > 300 patients determined that a D-dimer level < 500 mcg/L had

Table 5. Stepwise Approach to the Fundoscopic Examination³⁹

1. In a dimly lit examining room, have the patient remove glasses, sit upright, look forward, and focus on an object at least 10 feet away.
2. Set the illuminated lens disk on the ophthalmoscope to zero, the aperture to small, and the scope brightness to maximum. Brightness may be lowered for patient comfort.
3. Standing to the right of the patient, use your right hand to place the ophthalmoscope to your right eye. Place your index finger on the lens disk. Place your left hand on the patient's forehead and use your left thumb to elevate the patient's eyelid.
4. Position the scope 6 inches away from the patient at a 20° angle to the temporal side. Direct the light beam into the pupil, locate the red reflex, and keep it in focus as you move closer to the patient. The optic disk and macula should come into view when you are about 2 inches away.
5. Rotate the lens to best visualize the optic nerve and macula. For hyperopic (farsighted) patients, rotate the disk in the positive (green) direction. For myopic (nearsighted) patients, rotate the disk in the negative (red) direction.

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a 97% sensitivity and a negative predictive value of 99%.⁴⁴ A 2015 retrospective study and literature review of more than 600 patients found similar sensitivities and negative predictive value.⁴⁵ Thus, D-dimer may be used to exclude CVT in low-risk patients (defined as headache patients with a normal neurological examination, normal standard head computed tomography (CT) scan, and absence of risk factors such as pregnancy or puerperium).⁴⁶ High-risk patients, such as those who are pregnant or those with active malignancy, should proceed to magnetic resonance venography.

Radiographic Imaging

Despite the frequent utilization of brain CT, its diagnostic yield is low, ranging from 2.5% to 10% in patients presenting with nontraumatic headaches.^{47,48} Many disease processes will likely not be identified on CT scan, such as carbon monoxide poisoning, IIH, or meningitis/encephalitis. Noncontrast head CT should be obtained in patients suspected of having an acute intracranial hemorrhage; contrast-enhanced CT should be considered when there is concern for a vascular lesion or a space-occupying lesion (eg, tumor or abscess) or infection (eg, toxoplasmosis or neurocysticercosis).

CT angiography or magnetic resonance imaging (MRI) of the head and neck are the 2 recommended initial imaging modalities for CAD.⁴⁹ Digital subtraction angiography has classically been used as the gold-standard imaging for vascular dissections; however, it is invasive, resource-intensive, and has decreased sensitivity, particularly with regard to luminal abnormalities.⁴⁹

In cases of suspected CVT, a noncontrast CT may be the initial imaging obtained and may help rule in or rule out an alternative diagnosis. However, magnetic resonance venography is recommended when clinical suspicion for CVT is high.⁵⁰

For suspected PRES, neuroimaging is the most important diagnostic tool. Typical findings are symmetrical white matter edema in the posterior cerebral hemispheres.⁵¹ Although vasogenic edema can be visualized in some patients using noncontrast CT, brain MRI is much more sensitive.^{51,52}

Current ACEP Clinical Policy recommendations regarding emergent neuroimaging on patients presenting to the ED with acute headache are:⁷

Level B Recommendations (Moderate Strength of Evidence)

- Patients presenting with headache and new abnormal findings in a neurologic examination (eg, focal deficit, altered mental status, altered cognitive function)
- Patients presenting with new, sudden-onset severe headache
- HIV-positive patients with a new type of headache

Level C Recommendations (Weak Strength of Evidence)

- Patients who are aged > 50 years and presenting with new type of headache but with a normal neurologic examination should be considered for urgent neuroimaging study

Patients with a sudden-onset, severe headache who have negative findings on a head CT, normal opening pressure, and negative findings in cerebrospinal fluid analysis do not need emergent angiography and can be discharged from the ED, with follow-up recommended. (ACEP Level B recommendation.) A recent prospective study found that the combination of a negative head CT and negative lumbar puncture has been found to have a negative predictive value of 100%.⁵³

Computed Tomography and Subarachnoid Hemorrhage

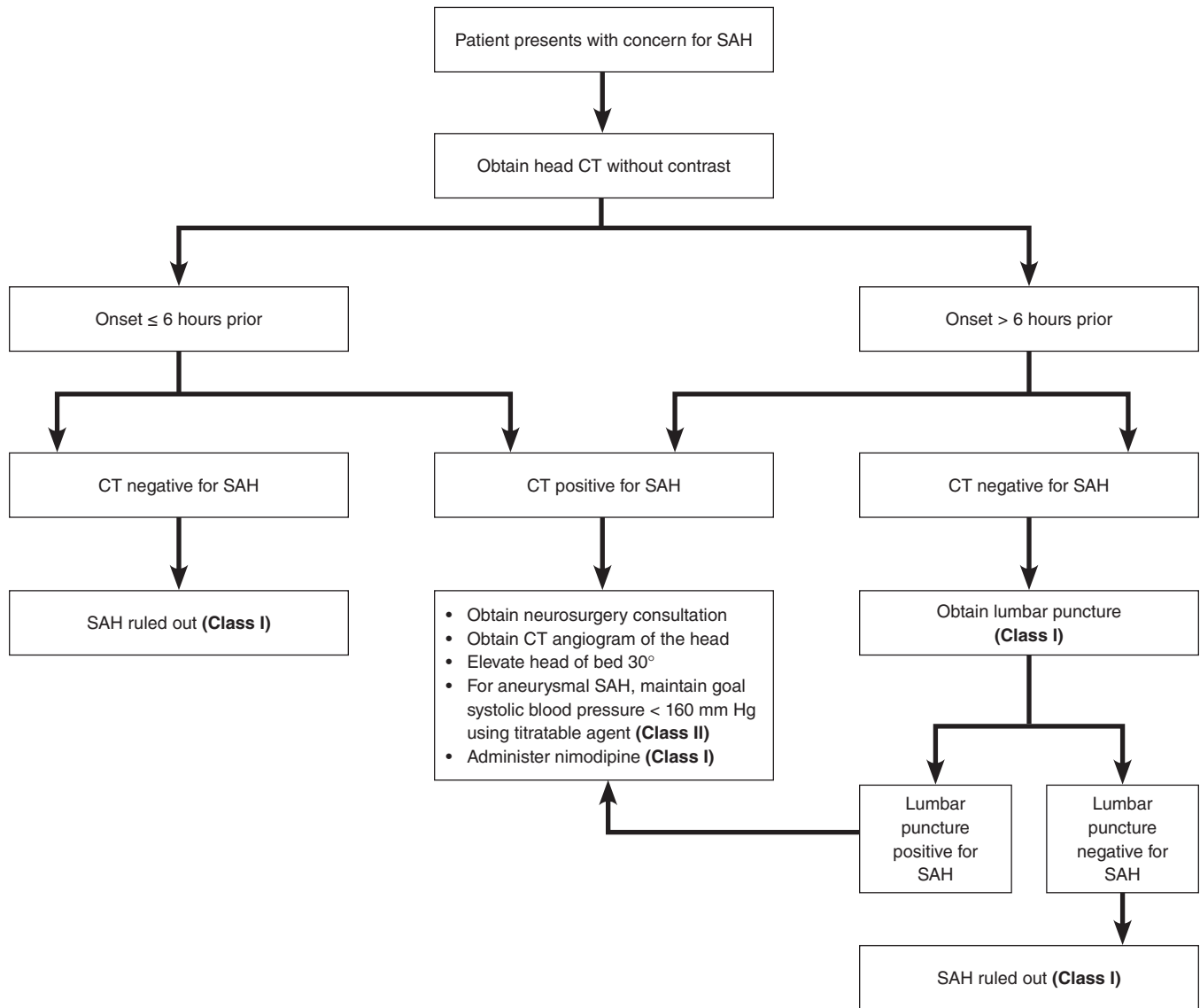
Perhaps the greatest limitation of CT for the diagnosis of SAH is that its sensitivity is time-dependent. Recent literature, including a prospective study that enrolled more than 3000 patients, found the sensitivity of CT conducted within 6 hours of symptom onset to have a sensitivity of 100% (97%-100%), a specificity of 100% (99.5%-100%), and a negative predictive value of 100% (99.5%-100%).⁵⁴ Two large 2016 meta-analyses demonstrated that CTs that were conducted within 6 hours of headache onset and whose results were read by an attending radiologist as negative had a LR of 0.01, essentially ruling out the diagnosis of SAH.^{16,55}

The 2008 ACEP Clinical Policy Guidelines (currently under revision) as well as the 2012 AHA Guidelines recommend a lumbar puncture in all patients being evaluated for SAH who have a negative noncontrast CT, regardless of time of onset (Level B recommendation).^{7,56} However, the most up-to-date literature supports a CT-only approach in patients presenting within 6 hours of headache onset.^{54,55}

Lumbar Puncture

Lumbar puncture may be utilized as a diagnostic tool in patients with headache and historical or examination findings suggestive of SAH, IIH, or meningitis. Lumbar puncture carries the risk of possible herniation, particularly in patients with headache and signs of increased ICP (eg, papilledema, absent venous pulsations on fundoscopic examination, altered mental status, focal neurologic deficits, or signs of meningeal irritation). Patients exhibiting these symptoms should undergo a neuroimaging study before having a lumbar puncture. In the absence of clinical findings suggestive of increased ICP, a lumbar puncture may be performed without obtaining a neuroimaging study. (ACEP Level C recommendation)

Clinical Pathway for Emergency Department Management of Subarachnoid Hemorrhage



Abbreviations: CT, computed tomography; SAH, subarachnoid hemorrhage.

Class of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II

- Safe, acceptable
- Probably useful

Level of Evidence:

- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III

- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate

- Continuing area of research
- No recommendations until further research

Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Patients with suspected IIH should have an opening pressure obtained with the patient lying in the lateral decubitus position, as performing the test on a patient in the sitting position can give falsely elevated readings. Patients with IIH have normal cerebrospinal fluid composition and, often, an elevated opening pressure (> 25 cm H_2O). Removal of a volume of cerebrospinal fluid can provide temporary relief of symptoms. Removal of 1 mL of cerebrospinal fluid will lower the cerebrospinal fluid pressure by approximately 1 cm H_2O .⁵⁷

Lumbar puncture findings for SAH include an elevated opening pressure > 20 cm H_2O and a persistent number of red blood cells (RBCs), usually in the thousands, from tubes 1 through 4. There is no guideline for a cutoff number of RBCs to diagnose SAH. A 2013 retrospective study looked at over 4000 patients who received a lumbar puncture for suspicion of SAH. The study found no patients with SAH who had RBCs < 100 in the final tube; RBCs $> 10,000$ increased the odds of SAH by a factor of 6.⁵⁸

Xanthochromia is produced by the breakdown of hemoglobin, and it produces a yellow-tinged color in the cerebrospinal fluid. Xanthochromia is time-dependent, and takes 2 to 12 hours to develop, so its absence in patients presenting within this timeframe may not be helpful. A 2015 prospective study found that patients with no xanthochromia and < 2000 RBCs were effectively ruled out of having SAH, with a combined sensitivity of 100%.⁵⁹

Patients with a sudden-onset, severe headache who have negative findings on a head CT, normal opening pressure, and negative findings in cerebrospinal fluid analysis do not need emergent angiography and can be discharged from the ED, with follow-up. (ACEP Level B recommendation.)

With respect to IIH, brain imaging with CT or MRI can be helpful in excluding other causes of secondary headache that may mimic IIH, such as CVT and intracranial mass. However, the brain parenchyma and ventricles generally appear normal on CT in patients with IIH.⁶⁰

Ocular Ultrasonography

Ocular ultrasonography can expedite the diagnosis and management of a variety of ocular emergencies in the setting of headache and can also be potentially used to assess for increased ICP and thus increase the safety of performing a lumbar puncture without CT. Several studies have demonstrated good correlation between ICP and sonographic optic nerve sheath diameter measurement.^{61,62}

A normal optic nerve sheath diameter measures up to 5.0 mm in adults. The measurement is obtained 3 mm posterior to the globe for both eyes. Three measurements should be obtained and averaged. Values > 5.0 mm are predictive of ICP > 20 mm Hg; increased ICP should be suspected with values above this threshold.⁶³ (See Figure 1.)

There is not enough evidence at this time to completely exclude increased ICP with normal optic sheath diameters on bedside ocular ultrasonography; thus, if clinical suspicion is high for increased ICP, brain CT should be performed first before proceeding with a lumbar puncture.

Treatment

Initial treatment for patients presenting with suspicion of a severe life-threatening headache includes a primary assessment of their airway, breathing, and circulation. Patients should be in a monitored setting and vital signs obtained quickly. Often, blood pressure normalizes once pain is treated. Patients with persistently elevated blood pressure and signs of neurologic end-organ damage (eg, confusion, lethargy, seizure) should be evaluated for potential hypertensive emergency. These patients need emergent blood pressure management.

The treatment goal for hypertensive emergencies is to lower the mean arterial pressure (MAP) = one-third systolic blood pressure + two-thirds diastolic blood pressure by approximately 25% in the first hour. If acute ischemic stroke is suspected, the

Figure 1. Ocular Ultrasound Evaluating Elevated Intracranial Pressure



The measurement is obtained 3 mm posterior to the globe for both eyes (line 1). A normal optic nerve sheath measures up to 5.0 mm in diameter in adults (line 2). Diameter values > 5.0 mm are predictive of intracranial pressure > 20 mm Hg.

Image courtesy of David Zodda, MD.

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blood pressure should be lowered below 185/110 mm Hg prior to the administration of thrombolytic therapy (for those patients who are candidates for thrombolytic therapy).⁶⁴

Results from the INTERACT-2 and ATACH-2 trials demonstrated that intensive blood pressure management does not appear to impact rates of death or disability. Rather, the INTERACT-2 trial demonstrated that a target systolic blood pressure (SBP) < 140 mm Hg was associated with improved functional outcomes. Based on this, guidelines recommend that for ICH patients presenting with SBP between 150 and 220 mm Hg and without contraindication to acute blood pressure treatment, acute lowering of SBP to 140 mm Hg is safe. (Class I, Level A recommendation.)^{65,66} The choice of antihypertensive agent and the goals of therapy should be tailored to the individual patient, with careful consideration of contraindications and adverse effects. (See Table 6.)

Subarachnoid Hemorrhage

Several clinical decision tools have been developed to aid in risk stratifying patients for SAH. The most well-known tool is the Ottawa subarachnoid hemorrhage rule. (See Table 7.) The original Ottawa SAH rule study demonstrated 100% sensitivity for SAH, and 15.3% specificity.⁶⁷ This study has since been prospectively validated, and although it has been challenged for its interobserver variability, it does provide helpful red flags for clinicians to consider when evaluating patients with headache.^{68,69}

A neurosurgical consultation and CT angiogram of the head should be obtained immediately when SAH is diagnosed. ED management focuses on monitoring the patient's airway, breathing, circulation, and mental status. Appropriate analgesia and antiemetics should be provided as needed. Elevating the head of the bed to 30° may improve venous drainage.

For aneurysmal SAH, the American Stroke Association guidelines currently recommend obtaining a target goal SBP of 160 mm Hg with a titratable agent that includes nicardipine or clevidipine.⁵⁶

Nimodipine, a calcium-channel blocker, should be provided to patients with aneurysmal SAH to improve neurologic outcomes. Nimodipine is typically administered 60 mg orally every 4 hours. The use of prophylactic antiepileptic drugs is controversial, and little literature has been published recently. One study demonstrated worse outcomes based on the Glasgow Outcome Scale when anti-epileptic drugs were administered. Phenytoin accounted for about half of the antiepileptic drugs used in that study.⁷⁰ Levetiracetam is used frequently, though data are limited. Anticonvulsant prophylaxis may be considered in the immediate posthemorrhagic period and should be limited to a 3- to 7-day course. Longer courses may be considered for patients with prior seizure, intracerebral hematoma, intractable hypertension, infarction, or aneurysm at the middle cerebral artery.^{56,71}

An online tool for using the Ottawa Subarachnoid Hemorrhage Rule is available from MDCalc at: <https://www.mdcalc.com/ottawa-subarachnoid-hemorrhage-sah-rule-headache-evaluation>



Table 7. Ottawa Subarachnoid Hemorrhage Rule

Inclusion Criteria:

- Age ≥ 15 years
- Glasgow coma scale score = 15
- New-onset severe headache, maximum intensity within 1 hour

Exclusion Criteria:

- Age ≥ 40
- Neurologic deficits, trauma, history of cerebral aneurysm, subarachnoid hemorrhage, brain tumors, chronic headaches, or papilledema
- Neck pain or stiffness
- Witnessed loss of consciousness
- Onset during exertion
- Thunderclap headache
- Limited neck flexion

If all are negative, subarachnoid hemorrhage is unlikely (100% sensitive)

Table 6. Intravenous Antihypertensive Medications for Treating Hypertensive Emergencies

Antihypertensive Medication (Dosage)	Mechanism of Action	Contraindications	Adverse Effects
Nicardipine (5-15 mg/hr)	Dihydropyridine calcium-channel blocker	Aortic stenosis	Reflex tachycardia
Labetalol (20-40 mg every 10 min, up to 300 mg)	Alpha1 and nonselective beta blocker	Asthma, chronic obstructive pulmonary disease	Bradycardia, bronchoconstriction
Esmolol (start at 50 mcg/kg/min; increase infusion by 50 mcg/kg/min increments to a maximum of 200 mcg/kg/min)	Cardioselective beta blocker	Decompensated congestive heart failure, heart block > first degree, sick sinus syndrome	Bradycardia, heart block
Nitroglycerin (10-200 mcg/min)	Venodilator	Right ventricular infarction; recent use of phosphodiesterase type 5 inhibitor	Reflex tachycardia

Cerebral Venous Thrombosis

Treating CVT focuses on managing the initiating cause of the thrombosis. Broad-spectrum antibiotics should be employed if an infectious source is suspected. Treating CVT with anticoagulation is controversial, as it is common for CVT to present with intracerebral hemorrhage or hemorrhagic transformation.^{19,72} Ferro et al documented that as many as one-third of these patients had hemorrhage on CT or MRI.²⁰ However, the data may be slightly biased, as providers may have avoided anticoagulation in patients with poorer prognosis;⁷² more robust studies are needed. Despite the controversy and the significant risk of bleeding, per guideline recommendations, anticoagulation is the standard therapy for this disease, with full-dose anticoagulation of low-molecular-weight heparin (eg, enoxaparin) or heparin bridge to warfarin therapy.^{72,74}

Idiopathic Intracranial Hypertension

The modified Dandy criteria can aid in the clinical diagnosis of IIH.⁷⁵ (See Table 8.)

A lumbar puncture can be performed for not only diagnostic purposes, but also for therapeutic purposes, although the relief is typically temporary.⁷⁶ Weight loss may be recommended for obese IIH patients. A prospective study of obese IIH patients found that weight loss led to reduced symptoms, signs, and ICP.⁷⁷

Acetazolamide, 250 mg to 500 mg orally twice daily, is considered the first-line pharmacotherapy for IIH. Acetazolamide is a carbonic anhydrase enzyme inhibitor that decreases cerebrospinal fluid production in the choroid plexus, thereby lowering ICP. A multicenter double-blinded randomized controlled trial found that the use of acetazolamide with a low-sodium weight-reduction diet resulted in modest improvement in visual field function.⁷⁸ Potential adverse effects of the drug include flushing, hypersensitivity reactions (eg, Stevens-Johnson syndrome/toxic epidermal necrolysis or agranulocytosis). If acetazolamide therapy fails or there is a contraindication to its use, topiramate or

Table 8. Modified Dandy Criteria for Diagnosing Idiopathic Intracranial Hypertension

- Symptoms and signs of increased intracranial pressure (eg, headache, transient visual obscurations, pulse synchronous tinnitus, papilledema, visual loss)
- No other neurologic abnormalities or impaired level of consciousness
- Elevated intracranial pressure with normal cerebrospinal fluid composition
- A neuroimaging study that shows no etiology for intracranial hypertension
- No other cause of intracranial hypertension apparent

furosemide are alternative therapies. Starting doses are not well established, but typical regimens may be topiramate 25 mg orally daily and furosemide 20 mg orally daily.⁷⁹

Surgical options are available for refractory cases, such as cerebrospinal fluid shunting, venous sinus stenting, and optic sheath fenestration.

Posterior Reversible Encephalopathy Syndrome

If PRES is caused by a specific medication (such as chemotherapy drugs), this medication should be discontinued temporarily. The management of hypertensive episodes and maintenance of normal blood pressure is an essential component of treatment.⁸⁰ The choice of antihypertensive drugs is up to the discretion of the treating clinician and includes vasodilators such as nitroglycerin, nitroprusside, or particularly nicardipine, due to its predictable response in primary neurologic events, or adrenergic inhibitors such as labetalol. Starting doses are variable and should be based on patient-specific factors including age, current blood pressure, and heart rate.²⁹ Nitroglycerin is contraindicated when there is a concern for increased ICP. A reduction of MAP by 25% within the first hour is a reasonable goal.

Cervical Artery Dissection

Whether traumatic or spontaneous, medical management for stroke prevention is the mainstay of treatment for carotid artery dissection and vertebral artery dissection. The superiority of antiplatelets versus antithrombotics is not established. The landmark CADISP study found no difference in mortality or neurologic improvement when anticoagulation versus antiplatelet therapy were compared.⁸¹ Based on the best available evidence, we recommend using intravenous (IV) heparin followed by warfarin/direct oral anticoagulant in patients with extracranial dissections, and to use antiplatelet therapy (aspirin or clopidogrel) in patients with intracranial dissections and in patients in whom systemic anticoagulation is contraindicated.⁸²

Acute Angle Closure Glaucoma

According to the American Optometric Association Consensus Panel, acute angle closure glaucoma is typically unilateral and most commonly found in either elderly or hyperopic patients. Signs and symptoms may include headache, pain, redness, tearing, photophobia, nausea/vomiting, blurred vision, and seeing halos around lights. Pain has been noted to be associated with the rapid rise in intraocular pressure (IOP). IOP that increases above the normal usual range (10-21 mm Hg) can lead to rapid progression of symptoms, which may even include vision loss. Immediate treatment is essential,

and includes consultation with an ophthalmologist. Treatment of acute angle closure glaucoma includes prompt lowering of IOP in order to preserve the patient's vision. Options for IOP reduction include medical (pharmaceutical) therapy, laser therapy, and surgery. Medication therapies in an acute attack include topical miotics, beta blockers, and alpha agonists. Treatment options are outlined in **Table 9**.⁸³⁻⁸⁵

Giant Cell Arteritis

Patients for whom there is a high level of suspicion for GCA should be treated with high-dose methylprednisolone 15 mg/kg/day IV for 1 to 3 days, followed by oral prednisone 40 mg/day. Arrangements should be made to obtain a biopsy, even if both ESR and CRP are negative.⁸⁶

Pre-Eclampsia

The American College of Obstetricians and Gynecologists (ACOG) recommends that the diagnosis of pre-eclampsia be based on hypertension and associated symptoms and/or laboratory findings.⁸⁷ (See **Table 10.**) Postpartum pre-eclampsia may occur up to 4 weeks post partum.⁸⁸

Pre-eclampsia is defined as elevated blood pressure with proteinuria or other severe symptoms in a pregnant patient at ≥ 20 weeks' gestation. Proteinuria is no longer necessary to diagnose pre-eclampsia if other severe symptoms are present. *Severe pre-eclampsia* is defined as pre-eclampsia plus 1 of the following symptoms: thrombocytopenia, liver or renal impairment, pulmonary edema, or new-onset headache.⁸⁷

Treatment of severe pre-eclampsia includes IV magnesium sulfate and antihypertensive agents. Typical dosing of magnesium sulfate includes a 4- to 6-gram load given over 15 to 20 minutes, immediately followed by an infusion of 1 to 2 grams/hour.⁸⁹ Patients without findings to suggest severe pre-eclampsia do not require IV magnesium, but should receive antihypertensive

Table 9. Medications for Treatment of Acute Angle Closure Glaucoma

Medication (Class)	Dosage and Directions
Timolol (beta blocker) 0.5% ophthalmic solution	1 drop into affected eye(s), repeated in 1 hour if needed, then 1 drop every 12 hours
Pilocarpine (miotic) 1% or 2% ophthalmic solution	1 drop into the affected eye(s) up to 3 times over a 30-minute period
Apraclonidine (alpha-2 agonist) 0.5% ophthalmic solution	1-2 drops at time of diagnosis; may be repeated 1 time in 1 hour

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treatment. Agents and starting doses may include labetalol 10 to 20 mg IV, hydralazine 5 mg IV, or nifedipine 10 to 20 mg orally.⁸⁷

Controversies and Cutting Edge

An important aspect of medical care is to determine the acceptable threshold for testing and the potential miss of a life-threatening condition. In other words, at what threshold does further testing do more harm than good? A miss rate of 1% in many diseases is deemed acceptable. Once this threshold of 1% is crossed, further testing may cause harm to the patient rather than providing a benefit.

This leads to the need for risk stratification and the use of a shared decision-making model. Thus, a negative head CT in a patient presenting with headache within 6 hours of onset does not rule out SAH, but it does risk stratify the patient to a group with a $< 1\%$ chance of having SAH. The emergency clinician and the patient should negotiate a plan for further diagnosis and management in line with the patient's values and preferences, with the clinician presenting the information and discussing the risks and benefits of further testing if the initial CT is negative, as well as all the risks associated with a lumbar puncture. This strategy applies not just to SAH, but to other neurologic emergencies, including meningitis/encephalitis, IHH, and hyperbaric oxygen therapy for CO poisoning.

Table 10. Criteria for the Diagnosis of Pre-Eclampsia (≥ 20 Weeks' Gestation)⁸⁷

Hypertension

- SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg (2 readings, 4 hours apart)

or

- SBP ≥ 160 mm Hg and/or DBP ≥ 110 mm Hg (1 reading)

Plus One of the Following:

Proteinuria

- Excretion of ≥ 300 mg of protein in 24-hour urine collection
- Protein/creatinine ratio ≥ 0.3 mg/dL
- 1+ urine dipstick (if other options are not available)

Thrombocytopenia*

- Platelet count $< 100,000/\text{mL}$

Liver Impairment*

- Elevated liver transaminases to at least 2 times normal

Renal Insufficiency*

- Elevated serum creatinine > 1.1 mg/dL
- 2 times baseline in absence of renal disease

Pulmonary Edema*

New-Onset Headache*

*Represent severe pre-eclampsia.

Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.

Risk Management Pitfalls for Emergency Department Management of Severe, Sudden-Onset Headache

1. **"I got a CT scan, and the lumbar puncture revealed no xanthochromia, so I discharged him, thinking he didn't have a SAH."**
Xanthochromia is time-dependent and takes 2 to 12 hours to develop, so its absence in patients presenting within this timeframe may not be helpful. If the diagnosis for SAH is still unclear after noncontrast CT and lumbar puncture, additional diagnostic imaging may include CT angiogram and magnetic resonance angiography.
2. **"When evaluating the pregnant patient for CVT, I didn't want to subject her to any radiation, so I obtained a D-dimer in lieu of a CT scan."**
The diagnosis of CVT should be made using the clinical examination and imaging studies. Several small studies have looked at the utility of D-dimer to screen patients presenting to the ED with headache suspicious for CVT. Pregnancy is a risk factor for CVT, so this patient is not low-risk.
3. **"My patient complaining of headache and neck pain had no focal neurologic deficits, so I had a very low clinical suspicion for carotid or vertebral artery dissection."**
Patients with CAD may initially present with head or neck pain, but without any neurologic deficits; the goal is to diagnose and treat before the dissection causes thrombus, which can embolize and cause stroke.
4. **"The 55-year-old patient I evaluated for a new-type headache had no neurologic deficits, so I suspected that the etiology was benign, and I did not obtain imaging."**
ACEP Clinical Policy recommends that patients aged > 50 years who present with a new type of headache and a normal neurologic examination should be considered for an urgent neuroimaging study (Level C recommendation).
5. **"She was 7 days post partum and came in complaining of new-onset headache and with a blood pressure of 186/92 mm Hg. Her urinalysis was negative for protein, so I ruled out pre-eclampsia."**
ACOG recommends that diagnosis of severe pre-eclampsia includes new headache and hypertension. Proteinuria is no longer necessary to diagnose pre-eclampsia if other symptoms are present. Postpartum pre-eclampsia and eclampsia may occur up to 4 weeks post partum. Treatment with IV magnesium and antihypertensives is indicated for this patient.
6. **"My 60-year-old patient presented with signs of an anterior stroke; however, her last known well time was 12 hours ago, so I did not consult neurosurgery, since she was out of the window for thrombolytics."**
The 2018 AHA Guidelines for endovascular therapy in acute ischemic stroke recommends that, in select patients with signs of acute stroke whose onset is within 6 to 24 hours, mechanical thrombectomy is reasonable (Level IIa recommendation).
7. **"I was evaluating a patient I highly suspected of having GCA, but both the ESR and CRP were negative, so I was able to rule it out."**
ESR and CRP are poor screening tests for GCA. While a greater ESR and CRP makes the diagnosis of GCA more likely, when there is high suspicion of GCA, it should be treated, and consultation for possible biopsy obtained, even if both ESR and CRP are negative.
8. **"I have a patient I've diagnosed with PRES that I think is caused by her tacrolimus. I was able to manage her blood pressure, so I told her she can continue her tacrolimus."**
If PRES is caused by a specific medication, it should be discontinued temporarily. Immediate follow-up should be scheduled with the provider who prescribed the medication.
9. **"I am suspecting IIH in a 27-year-old woman with headache and bilateral blurry vision. However, her vision is 20/20 bilaterally, so I was less concerned for IIH."**
Subjective blurry vision in IIH is due to papilledema, but visual acuity is typically preserved in these patients. Visual fields are affected first. If the clinician has been trained, bedside ultrasound should be attempted to assess for optic sheath enlargement.
10. **"He presented to the ED with a new-onset headache, but it was mild and the neuro exam was normal. His only past medical history included HIV, so I treated him for pain and discharged him without obtaining imaging."**
The ACEP Clinical Policy on the evaluation and management of adult patients presenting to the ED with acute headache includes emergent neuroimaging on HIV-positive patients with a new type of headache (Level B recommendation).

Time- And Cost-Effective Strategies

- Noncontrast head CT should not be routinely ordered on all patients presenting to the ED with nontraumatic headache. The diagnostic yield is low, ranging from 2.5% to 10%.^{47,48}
- Routine laboratory testing, such as a complete blood cell count and a metabolic panel, are also of low utility in aiding in the diagnosis of headaches. Targeted testing should be performed.
- The 2008 ACEP Clinical Policy Guidelines as well as the 2012 AHA Guidelines recommend a lumbar puncture in patients being evaluated for SAH who have a negative noncontrast CT scan, regardless of the time of onset (Level B recommendation).^{7,56} However, the most current literature appears to support a CT-only approach in patients presenting within 6 hours.^{54,55}
- Ocular ultrasonography can expedite the diagnosis and management of several ocular emergencies, including elevated ICP secondary to IIH. Several studies have demonstrated good correlation between ICP and sonographic optic nerve sheath diameter.⁶¹⁻⁶³
- ESR and CRP are poor screening tests for GCA.^{40,41} While a greater ESR and CRP makes the diagnosis of GCA more likely, when there is a high suspicion of GCA, the patient should be treated and a biopsy should be obtained even if both ESR and CRP are negative.
- Acetazolamide is considered the first-line pharmacotherapy for IIH. As a carbonic anhydrase enzyme inhibitor, cerebrospinal fluid production in the choroid plexus is decreased and lowers pressure. A multicenter double-blinded randomized controlled trial found that the use of acetazolamide with a low-sodium weight-reduction diet resulted in modest improvement in visual field function.⁷⁵

Disposition

Emergency clinicians are encouraged to obtain emergent consultation on life-threatening headaches that are diagnosed in the ED. Consultations are dependent upon the etiology of the specific medical condition and may include consultations from neurosurgery, ophthalmology, obstetrics, and infectious disease. Nearly all patients diagnosed with a severe, life-threatening headache will require admission to or transfer to a facility with access to a 24-hour neurology critical care team. These patients often require intensive care unit admission for frequent monitoring of airway, breathing, and circulation, and frequent neurologic assessment. Efforts should be made to transfer these

patients to the intensive care unit as efficiently and expeditiously as possible, as delays in transfer and ED boarding have been shown to lead to increased rates of morbidity and mortality.

Summary

Headache is one of the most common chief complaints in patients presenting to the EDs in the United States. Secondary headaches represent approximately 2% of all headache presentations to the ED, and though they are rare, they can represent life-threatening emergencies. These headaches may be the result of vascular, infectious, or traumatic etiologies.

The most important factor in evaluating a patient presenting to the ED with headache is to obtain a thorough history and physical examination. Historical clues and examination findings are the foundation of the assessment and treatment plan, and should help guide decisions with regard to obtaining laboratory and imaging studies and patient disposition. Advances in diagnostic testing and imaging, such as the 100% sensitivity of CT to rule out SAH within 6 hours, can aid emergency clinicians in their workup and assessment of life-threatening headaches.

Emergency clinicians are on the front lines of cutting-edge critical care management. We now have more access to diagnostic imaging and treatment modalities than ever thought possible. And yet, perhaps the most important tools we have are our ability to listen to our patients, conduct a quality examination, and incorporate evidence-based decision-making into our clinical practice.

Case Conclusions

Given the history your first patient provided, your suspicion and immediate evaluation for subarachnoid hemorrhage was appropriate. The most recent literature supports a CT-only approach in patients presenting within 6 hours. Because this patient's symptoms had resolved, additional imaging and neurology consultation were not indicated. You utilized a shared decision-making strategy with the patient and discussed the risks and benefits of obtaining additional testing. You discharged him and cautioned him that he should return to the ED if his headache returned.

Regarding your second patient, you recognize that this cancer patient's change in mental status and severely elevated blood pressure was likely the result of PRES. You obtained a CT of the head, which revealed white-matter changes in the posterior cerebral hemispheres. Utilizing IV nicardipine, you lowered the patient's MAP by 25% over the first hour. In addition, you temporarily discontinued his chemotherapy medication. He subsequently became more alert and responsive.

Based upon your third patient's history of a motor

vehicle crash, particularly her complaints of headache, neck pain, and dizziness following the neck trauma, you obtained a CT angiogram of the neck. The imaging revealed an extracranial dissection of the left vertebral artery. You promptly initiated anticoagulation therapy utilizing IV heparin to be followed by oral warfarin. In addition, you obtained a vascular surgery consultation and admitted the patient to the surgical intensive care unit.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study is included in bold type following the references, where available. The most informative references cited in this paper, as determined by the author, are noted by an asterisk (*) next to the number of the reference.

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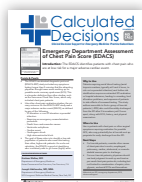


1. Which of the following historical or examination findings is most suggestive of subarachnoid hemorrhage?
 - a. Sudden onset
 - b. Neck stiffness on examination
 - c. Focal neurologic deficit
 - d. Loss of consciousness
2. A pregnant patient presents with a severe, sudden-onset headache. You suspect cerebral venous thrombosis. What would you expect to find on clinical examination?
 - a. Horner syndrome
 - b. Hyporeflexia
 - c. Cranial nerve VI palsy
 - d. Clonus
3. Regarding the clinical features of posterior reversible encephalopathy syndrome, which of the following is TRUE?
 - a. Onset of symptoms typically develops within hours to days.
 - b. Seizures are rarely associated with PRES.
 - c. Radiological findings typically do not change.
 - d. Nitroprusside is the antihypertensive drug of choice.
4. Which of the following is a common characteristic in patients presenting with suspected carotid artery dissection?
 - a. Carotid bruit
 - b. Hemianopsia
 - c. Horner syndrome
 - d. Delayed neurologic findings
5. Which of the following findings has the highest positive likelihood ratio for the diagnosis of giant cell arteritis?
 - a. Temporal artery beading
 - b. Elevated erythrocyte sedimentation ratio
 - c. Jaw claudication
 - d. Diplopia
6. Regarding laboratory testing for a potential life-threatening headache, which of the following is TRUE?
 - a. Co-oximetry for carboxyhemoglobin should be performed with an arterial blood gas.
 - b. Erythrocyte sedimentation rate and C-reactive protein are good screening tests for giant cell arteritis.
 - c. D-dimer testing is a good screening test for low-risk patients suspected of having cerebral venous thrombosis.
 - d. Routine laboratory testing, such as complete blood cell count, have a high yield in diagnosing life-threatening headaches.
7. Which patient does the ACEP Clinical Policy on ED headache evaluation and management recommend acute imaging for?
 - a. A patient with a new abnormal finding on a neurologic examination
 - b. A patient presenting with headache and neck pain
 - c. An HIV-positive patient presenting with a headache
 - d. A pregnant patient presenting with headache
8. What is the accuracy for the Ottawa subarachnoid hemorrhage rule?
 - a. Highly sensitive and highly specific
 - b. Highly sensitive and not specific
 - c. Not sensitive and highly specific
 - d. Not sensitive and not specific
9. Which of the following is a diagnostic criterion for idiopathic intracranial hypertension?
 - a. Signs of hydrocephalus on brain imaging
 - b. Raised cerebrospinal fluid pressure > 20 cm H₂O
 - c. Obesity
 - d. Localized neurologic findings
10. A 25 weeks' pregnant patient presents with a blood pressure of 180/120 mm Hg. Which of the following associated symptoms requires IV magnesium therapy for the treatment of severe pre-eclampsia?
 - a. Proteinuria
 - b. New-onset headache
 - c. Protein/creatinine ratio ≥ 0.3 mg/dL
 - d. Serum creatinine 0.8 mg/dL

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Abstract
Emergency clinicians must be aware of the current diagnostic and therapeutic recommendations for influenza and the available evidence to guide management. This comprehensive review outlines the classification of influenza viruses, and the pathophysiology, clinical presentation, and laboratory diagnosis of influenza. Several evidence-based management strategies are presented, as well as the challenges in achieving optimal care of high-risk patients. Influenza management in the emergency department is discussed, as well as the challenges in achieving optimal care of high-risk patients. Influenza management in the emergency department is discussed, as well as the challenges in achieving optimal care of high-risk patients.

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Abstract
Timely management of patients presenting to the ED with first trimester of pregnancy complications is important for both the patient and the fetus. Common obstetric problems, emergency and pregnancy of undetermined location, ectopic pregnancy and timing of pregnancy including hyperemesis gravidarum, common, including which obstetric emergencies are life-threatening. Optimal diagnostic approaches and management algorithms are presented, including which obstetric emergencies are life-threatening. Optimal diagnostic approaches and management algorithms are presented, including which obstetric emergencies are life-threatening.

Abstract
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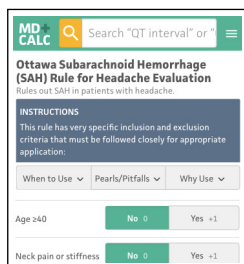
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Ottawa Subarachnoid Hemorrhage Rule

Introduction: The Ottawa subarachnoid hemorrhage rule is used to rule out subarachnoid hemorrhage in patients with headache.

Points & Pearls

- The Ottawa subarachnoid hemorrhage (SAH) rule was developed to be used in patients presenting to the emergency department (ED) with acute nontraumatic headache.
- The rule is 100% sensitive for SAH (ie, it is a rule-out tool).
- The rule should only be used in patients who are alert and oriented, are presenting within 14 days of the headache, and have no history of head trauma or a fall in the past 7 days.
- The Ottawa SAH rule cannot be used in patients who have new neurologic deficits, a previous history of headache syndrome, or intracranial lesions. **(See the Evidence Appraisal section for the complete list of exclusion criteria.)**
- The specificity of the Ottawa SAH rule is low (15%), so it should not be used to diagnose SAH, even in patients for whom all criteria are positive. Clinician judgment should be used to determine further workup, if any, for patients who fail the rule.

Advice

Consider workup for SAH in patients who have **any** positive criteria; however, given the low specificity of the rule, not every patient who fails the rule will require workup for SAH. In patients for whom all criteria are negative, consider avoiding further SAH-specific workup.

CALCULATOR REVIEW AUTHOR

Rachel Kwon, MD
MD Aware

Critical Actions

Patients in whom SAH has been ruled out may still have other causes of headache that require workup or intervention. The differential diagnosis should be broad.

Evidence Appraisal

The first iteration of what is now known as the Ottawa SAH rule was derived by Perry et al in 2010. The study prospectively enrolled 1999 patients with headache who were from 5 Canadian tertiary care centers; 130 of these patients had confirmed SAH. Sixteen variables were identified as predictive for SAH (13 on history and 3 on physical examination). Recursive partitioning was used to identify combinations of these variables and create the 3 separate decision rules with the highest sensitivity for SAH.

Perry et al (2013) prospectively validated these findings in a study of 2131 patients at 10 sites, using the following inclusion and exclusion criteria for enrollment:

Inclusion criteria

- Glasgow coma scale score = 15 of 15 (ie, patient was alert and oriented)
- No history of fall or head trauma in the past 7 days
- Presentation within 14 days of headache onset

Exclusion criteria

- A previously established history of headache syndrome
- Referral from another institution with a confirmed diagnosis of SAH
- Returning for reassessment of a headache that was previously evaluated with computed tomography (CT) and lumbar puncture

- Presence of papilledema
- New focal neurologic deficits
- Previous diagnosis of a cerebral aneurysm, SAH, brain tumor, or hydrocephalus

The variables were again run through recursive partitioning and the final Ottawa SAH rule was found to be 100% sensitive for SAH (95% confidence interval [CI], 25.6%-29.5%). Specificity was 15.3% (95% CI, 13.8%-16.9%).

Not all patients in the validation study underwent a full workup with CT scan and lumbar puncture (80% had a CT scan and 45% had lumbar puncture). The patients who were discharged without undergoing a CT scan and lumbar puncture were assessed using a follow-up tool that included structured telephone interviews and medical records review. The

authors acknowledged that some patients with small nonaneurysmal SAH may have been missed.

Bellolio et al (2015) also externally validated the Ottawa SAH rule by retrospectively applying it to 454 patients who presented to the ED with headache. Sensitivity was 100% (95% CI, 62.9%-100%) but specificity was lower than in the validation by Perry et al (7.6%, 95% CI 5.4%-10.6%), so the authors concluded that the rule's clinical use may be limited.

According to the hierarchy of evidence for clinical decision rules that was developed by McGinn et al (2000), the Ottawa SAH rule is a level 2 clinical decision rule, with established accuracy in at least 1 large prospective study, but no impact analysis completed as of yet.

Instructions

The Ottawa SAH rule has very specific inclusion and exclusion criteria that must be followed closely for appropriate application:

- **Only apply the rule** in alert patients aged ≥ 15 years who present with a new, severe nontraumatic headache that reaches maximum intensity within 1 hour.
- **Do not use the rule** in patients who have new neurologic deficits, prior aneurysm, prior SAH, known brain tumors, or chronic recurrent headaches (≥ 3 headaches of the same character and intensity occurring over a period of ≥ 6 months).

Why to Use

It is challenging to rule out SAH in patients who present with headache and no neurologic deficits. SAH is rare, accounting for approximately 1% of patients presenting to the ED with headache (Vermeulen 1990), but missed diagnoses are potentially devastating results. A tool that reliably rules out SAH is useful to avoid unnecessary workups.

Lumbar puncture is often performed as the confirmatory test if a noncontrast head CT scan is negative but the clinical suspicion for SAH remains high. Lumbar puncture is painful and carries the risk of bleeding and of headache that may be worse than the original presenting headache.

When to Use

Use the Ottawa SAH rule in patients aged ≥ 15 years who present with headache and are neurologically intact.

Next Steps

In patients who have any positive criteria for the Ottawa SAH rule (ie, SAH cannot be ruled out), workup for SAH typically begins with a noncontrast head CT. Consider lumbar puncture and/or cerebral angiography if clinical suspicion remains. In their 2013 validation study, Perry et al provided insight into the appropriate workup for patients with possible SAH.

- A noncontrast head CT obtained within 6 hours of headache onset is sufficient to rule out SAH in most patients.
- Lumbar puncture should be performed for patients who are at particularly high risk for SAH.
 - » If there is no visual xanthochromia and the fourth tube of the lumbar puncture has a red blood cell count $< 2000 \times 10^6/L$, SAH is ruled out except in patients at "ultra-high risk" (described in the study as patients with $> 50\%$ clinical pretest probability for SAH).
 - » If the patient is "ultra-high risk," CT angiography can be performed to evaluate for cerebral aneurysm. Neurosurgical consultation may be helpful in these patients.
- CT angiography can be useful if there was a significant time delay between the patient's presentation to the ED and the initial headache (eg, a headache that occurred last week).

Neurology and neurosurgical consultation should be obtained for patients with suspected or confirmed SAH.

Abbreviations: CT, computed tomography; ED, emergency department; SAH, subarachnoid hemorrhage.

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Calculator Creator

Jeffrey J. Perry, MD, MSc

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