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Evidence-Based Approach To Diagnosis And Management Of Aneurysmal Subarachnoid Hemorrhage In The Emergency Department

You walk into a crowded evening shift in the emergency department (ED). Your first patient is a middle-aged woman lying with her hands clutching her head, complaining of the "worst headache of her life." You are worried about a subarachnoid hemorrhage (SAH). You treat her pain and order a noncontrast head computed tomography (CT), which is negative. She now says that her headache is better and that she needs to go home to pick up her kids. Does she really need a lumbar puncture (LP)? She eventually agrees to stay for an LP, which is also negative. Can she go home now? Does she need any additional workup?

While you are thinking about this, another patient with a history of migraine arrives complaining of sudden-onset, severe headache that has lasted 12 hours. Is this headache her usual migraine or could this be an SAH? After further history is obtained, you are concerned about an SAH and you obtain a CT, which is normal. You perform an LP, which shows some clearing of red blood cells (RBCs) from tube 1 to tube 4, and you think it may have been a traumatic tap, but how can you be sure? Just as you are pondering this, the lab calls to say there is xanthochromia. You make the diagnosis of SAH. After calling for neurosurgical consultation, what else should you do in the ED to treat this patient?

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CME Objectives

Upon completion of this article, you should be able to:

1. Describe the classic presentation of an SAH as well as discuss the wide spectrum of presentation.
2. Describe the diagnostic approach to a patient suspected of having an SAH.
3. Identify the major limitations in interpreting the diagnostic modalities.
4. Discuss general principles of acute SAH management in the ED.
5. Identify common pitfalls in the diagnosis of SAH.

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Headache is the presenting complaint in roughly 2% of ED visits.[1] Of all those with headache, about 1% will have SAH,[2-4] meaning 99% of ED headache patients do not have SAH. This makes selecting those patients a major diagnostic challenge. Among those with sudden-onset, severe headache with normal neurologic examination, 10% to 16%[5-10] have SAH. Diagnosing and accurately managing SAH remain pressing challenges for emergency clinicians. Estimates of misdiagnosis, previously as high as 32%, are now thought to be lower but still range from 5% to 15%.[11-15] Patients with delayed diagnosis have worse outcomes, highlighting the importance of early recognition.[12-14,16] Unfortunately, SAH claims the lives of 40% of its victims and leaves 30% with substantial neurological disability.[17]

When a patient presents with a classic "worst-of-life headache" and maximal symptoms at onset, the next steps of management, including CT followed by LP, are straightforward. However, patients often present with vague headaches or with underlying primary headache disorders, which make diagnosis more challenging. Although an extensive workup of every headache patient is impractical and not called for, the diagnosis is unlikely to be made if not considered. Deciding who to evaluate for SAH is difficult. Once the decision has been made, translating the CT and LP results may be challenging, as the sensitivity for subarachnoid bleeding depends on several factors, including the emergency clinician's accurate testing interpretation and knowledge of test limitations.[18]

The emergency clinician must be able to quickly and accurately identify SAH in order to optimize outcome. This issue of *Emergency Medicine Practice* focuses on the challenge of diagnosing and managing SAH, using the best available evidence from the literature.

Abbreviations Used In This Article

ABC: Airway, Breathing, Circulation
AHA: American Heart Association
BP: Blood Pressure
CI: Confidence Interval
CSF: Cerebrospinal Fluid
CT: Computed Tomography
CTA: Computed Tomographic Angiography
DSA: Digital Subtraction Angiography
ECG: Electrocardiogram
ED: Emergency Department
GCS: Glasgow Coma Scale
ICP: Intracranial Pressure
ICU: Intensive Care Unit
INR: International Normalized Ratio
IV: Intravenous
LP: Lumbar Puncture
MR: Magnetic Resonance
MRI: Magnetic Resonance Imaging
MRA: Magnetic Resonance Angiography
PCC: Prothrombin Complex Concentrates
PT: Prothrombin Time
PTT: Partial Thromboplastin Time
RBC: Red Blood Cell
RCT: Randomized Controlled Trial
SAH: Subarachnoid Hemorrhage
SBP: Systolic Blood Pressure

Critical Appraisal Of The Literature

A literature search was performed using Ovid MEDLINE and PubMed from 1950 to December 2008. Search terms included subarachnoid hemorrhage, aneurysm, thunderclap headache, sentinel headache, lumbar puncture, xanthochromia, emergency department, head CT, CTA, angiography, MRI, nimodipine, risk factors, prehospital care, diagnosis, management, analgesia, treatment, rebleeding, vasospasm, hypertension, antiepileptic, and combinations of these keywords. The search was limited to the English language and human studies. More than 250 articles were reviewed, which provided background for further literature review. During the final editing phase, the American Heart Association (AHA) published updated 2009 guidelines pertaining to SAH, which were incorporated into this article.

During the literature review process, the highest value was placed on scientific articles, especially clinical trials, larger prospective cohort studies, and meta-analyses of clinical trials. Secondary evidence was collected from retrospective studies, case-control studies, and other meta-analyses. Finally, expert consensus and case reports were reviewed. The Cochrane Database of Systematic Reviews and the National Guideline Clearinghouse (www.guidelines.gov) were also consulted.

Guidelines helpful for the practicing emergency clinician are presented in **Table 1**. Some guidelines are supported by many organizations, as indicated by multiple citations. The most relevant guidelines for emergency clinicians are the 2008 American College of Emergency Physician's (ACEP) clinical policy on acute headache (**Table 1A**)[19] and the most recent 2009 AHA guidelines (**Table 1B**).[20]

Etiology And Pathophysiology

Incidence

The international incidence of SAH varies according to region but affects approximately 7 to 9 of 100,000 people per year.[28,29] Although classic teaching is that women are more likely than men to suffer from SAH,[29,30] newer epidemiologic research reveals that this difference occurs only after the sixth decade.[28,31,32]

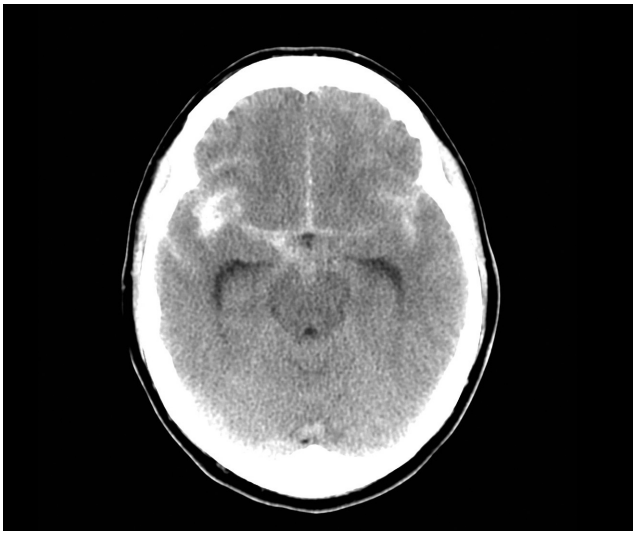
Etiology

Trauma is the most common cause of all SAH.[33] It is usually a diffuse process that results from rupture of corticomeningeal vessels and from hemorrhagic contusions of the brain.[34] Most spontaneous cases are related to aneurysm rupture,[35,36] which is the focus of this article. See **Figure 1** for the typical appearance of a spontaneous SAH on noncontrast head CT. Distinguishing traumatic from spontaneous SAH may be difficult in some cases because the trauma may have been unwitnessed; however, this distinction is important.

One retrospective study comparing 99 patients with traumatic SAH and 114 patients with aneurysmal SAH found traumatic cases to have more diffuse hemorrhage, earlier resolution of bleed, and decreased delayed complication rates,[34] illustrating mechanistic differences leading to the onset and evolution of SAH and the need for distinct management. When in doubt, cerebrovascular imaging should be obtained to determine if an underlying aneurysm is present, since this finding is important for definitive repair.

Rupture of saccular aneurysms located on intracranial vessels at the base of the brain account for

Figure 1. Subarachnoid Hemorrhage On Noncontrast Head Computed Tomography



There is diffuse hyperdense subarachnoid blood surrounding the basal cisterns and extending into the Sylvian fissures bilaterally. (Reprinted with permission from Lisa Thomas, MD.)

Table 1. Clinical Practice Guidelines On Subarachnoid Hemorrhage

A. 2008 American College Of Emergency Physicians Clinical Policy On Acute Headache (Evidence-Based Recommendations)[19]

1. Emergent head computed tomography **IS** the initial diagnostic test recommended in the diagnosis of any new, sudden-onset, severe headache or suspected case of subarachnoid hemorrhage* (*Class I, Level B recommendation*).[20-26]
2. Lumbar puncture **IS** recommended for patients with suspected subarachnoid hemorrhage after negative noncontrast head computed tomography* (*Class I, Level B recommendation*).[20-22]
3. Angiography is **NOT** recommended in patients with sudden-onset, severe headache who have negative findings on head computed tomography, normal opening pressure, and negative cerebrospinal fluid findings (*Class II, Level B recommendation*).
4. Patients with a negative workup including negative computed tomography and lumbar puncture **CAN** be safely discharged from the emergency department, with outpatient follow-up recommended (*Class II, Level B recommendation*).
5. Response to analgesia should **NOT** be used as the sole indicator to the etiology of an acute headache (*Class III, Level C recommendation*).

**Also supported by 2009 American Heart Association guidelines.*

B. 2009 American Heart Association Guidelines For Management Of Subarachnoid Hemorrhage (Evidence-Based Recommendations)[20]

Recommendations for diagnostic studies

1. Once subarachnoid hemorrhage is diagnosed, urgent cerebral angiography **IS** needed to detect the underlying cerebral aneurysm (*Class I, Level B recommendation*).[20,21]
2. When conventional angiography cannot be performed in a timely fashion, magnetic resonance angiography and computed tomographic angiography **MAY** be considered (*Class III, Level B recommendation*).[20]

Recommendations for management

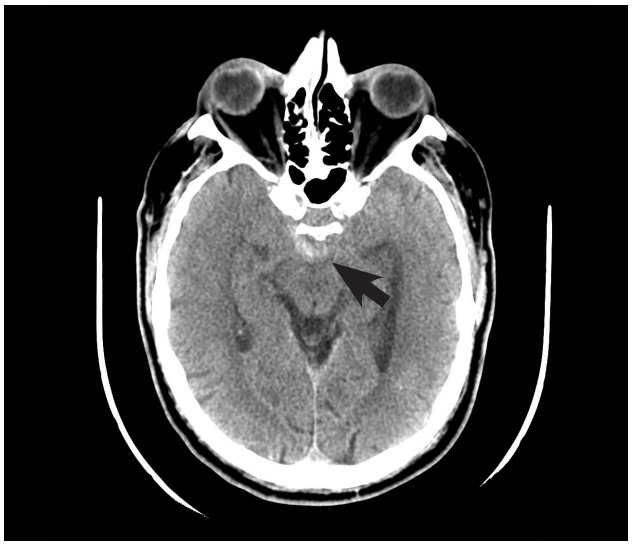
1. Patients with subarachnoid hemorrhage should be treated in an intensive care unit setting with cardiac and blood pressure monitoring (*Class I, Level B recommendation*).[20,21,25]
2. Control of elevated blood pressure **IS** recommended to balance the risk of stroke, rebleeding, and maintenance of cerebral perfusion pressure (*Class II, Level B recommendation*).[20,25]
3. Oral nimodipine **IS** strongly recommended to reduce poor outcome from vasospasm (*Class I, Level A recommendation*).[20,21]
4. Prophylactic anticonvulsant therapy **MAY** be considered in the immediate posthemorrhage period (*Class III, Level B recommendation*).[20,21,25]
5. Early surgery **IS** recommended for most patients (*Class II, Level B recommendation*).[20,21]

Recommendations for transfer

1. Early referral to high-volume centers with cerebrovascular surgeons and endovascular services **IS** recommended (*Class II, Level B recommendation*).[20,21,27]

85% of spontaneous SAH. (**See Table 2.**) Approximately 80% to 85% of aneurysms are in the anterior circulation, and the rest are in the posterior circulation. Aneurysms of the spinal arteries can also result in SAH.[37] A minority of cases are due to nonaneurysmal perimesencephalic hemorrhage (**Figure 2**), a benign form of SAH defined by a specific pattern of extravasated blood around the midbrain with normal angiogram, and other rare nonaneurysmal causes.[36,38,39] (**See Table 2.**)

Figure 2. Perimesencephalic Hemorrhage On Noncontrast Head Computed Tomography



A characteristic pattern of hyperdense blood anterior to the midbrain is seen. No aneurysm was found on follow-up angiogram. (Reprinted with permission from Lisa Thomas, MD.)

Table 2. Causes Of Spontaneous Subarachnoid Hemorrhage

- Ruptured aneurysm (85%)
- Nonaneurysmal causes (15%)
  - Perimesencephalic hemorrhage (10% of total)
  - Other rare causes (5% of total)
    - Tumor
    - Coagulopathy
    - Venous sinus thrombosis
    - Arterial dissection
    - Vascular lesion
      - Arteriovenous malformation, dural arteriovenous fistula, amyloid angiopathy, others
    - Vasculitis
      - Behcet's disease, Churg-Strauss, Wegener's granulomatosis, others
    - Sickle cell disease
    - Mycotic aneurysm
    - Drugs
      - Cocaine abuse, anticoagulants

Pathophysiology Of Aneurysms

Although once thought to be congenital, retrospective studies of patients undergoing aneurysm surgery found that aneurysms are very rare in children and even rarer in neonates.[40] Previous theories of congenital muscle wall weakness have been disproved because equal gaps in muscle wall have been found in patients with and without aneurysms.[41] The current theory is that aneurysms develop gradually over one's lifetime due to acquired arterial wall changes, with a prevalence of 2.3% in an average adult without specific risk factors, based on a systematic review of 23 studies including over 56,000 patients. Lowest rates were reported in retrospective autopsy studies (0.4%), and highest rates were found in prospective angiography studies (6%).[42] Factors that increase the risk of aneurysm formation and SAH are listed in **Table 3** and were recently reviewed.[30]

What Leads To Aneurysm Rupture?

Rupture risk increases with size, and based on a systematic review of 9 prospective studies following

Table 3. Risk Factors For Subarachnoid Hemorrhage

Feature	Relative Risk	95% Confidence Interval	Reference(s)
Autosomal dominant polycystic kidney disease	4.4	2.7-7.2	42,43
Familial predisposition	4.0	2.7-6.0	42,44,45
Hypertension	2.5	2.0-3.1	46-48
Atherosclerosis	2.3	1.7-3.1	42
Smoking	2.2	1.3-3.6	46-49
Excessive alcohol use (> 150 grams/week)	2.1	1.5-2.8	47
Age > 85	1.61	1.24-2.07	28
Oral contraceptive Use*	Controversial		47,50,51
Connective tissue disorders¥	Insufficient data		52,53

**Oral contraceptives were found to have a minimal increase in the risk of subarachnoid hemorrhage, risk ratio of 1.42 (95% confidence interval, 1.12-1.80), based on a 1998 meta-analysis,[51] but this was not replicated by a newer 2005 meta-analysis with more stringent study inclusion criteria.[50]*

¥Ehlers-Danlos type IV, Marfan's syndrome, and neurofibromatosis type I are some examples of connective tissue disorders that have been reported to be associated with subarachnoid hemorrhage, but data are limited to reviews[52,53] and small case series.

aneurysms for 3907 patient-years, most aneurysms do not rupture. Whereas aneurysms < 10 mm in size have an annual risk of rupture of 0.7%, larger aneurysms are 5 times more likely to rupture.[42,54] The exact mechanisms leading to aneurysm rupture are still unclear. A common belief is that aneurysm rupture may be precipitated by activity. Increases in arterial blood pressure (BP) due to valsalva maneuvers or physical exertion in activities ranging from sneezing to sexual intercourse are associated with only a small subset (< one-third) of patients with SAH.[55-58] In fact, a recent large systematic review concluded that there is insufficient evidence to link exertion to aneurysm rupture.[30]

Grading Severity Of SAH

There are multiple clinical scales to assess the extent of rupture and severity of SAH. (**See Table 4.**) The 2 most widely used scales are the Hunt and Hess[59] and the World Federation of Neurological Surgeons.[60] Higher numbers in both scales correlate well with worse patient outcomes. A third scale, the Fisher classification,[61] uses quantity of blood on CT appearance to predict the risk of symptomatic cerebral vasospasm, a dreaded complication of SAH. The grading systems should be used in conjunction with the patient's overall condition and characteristics of the ruptured aneurysm, such as location and size, to grade severity and approach to treatment.

Table 4. Subarachnoid Hemorrhage Grading Scales

A. Hunt And Hess Severity Scale

Grade 1- Asymptomatic, mild headache
Grade 2- Moderate to severe headache, nuchal rigidity, no focal deficit other than cranial nerve palsy
Grade 3- Mild mental status change (drowsy or confused), mild focal neurologic deficit
Grade 4- Stupor or moderate to severe hemiparesis
Grade 5- Comatose or decerebrate rigidity

B. World Federation Of Neurological Surgeons

Grade 1- Glasgow Coma Scale 15, no motor deficit
Grade 2- Glasgow Coma Scale 13-14, no motor deficit
Grade 3- Glasgow Coma Scale 13-14, motor deficit present
Grade 4- Glasgow Coma Scale 7-12, motor deficit may be present or absent
Grade 5- Glasgow Coma Scale 3-6, motor deficit may be present or absent

C. Fisher Scale (Computed Tomography Appearance)

Group 1- No blood
Group 2- Diffuse deposits of subarachnoid hemorrhage blood, no clots, no layers of blood > 1 mm
Group 3- Local clots or vertical layers of blood ≥1 mm thickness
Group 4- Diffuse or no subarachnoid hemorrhage, but intracerebral or intraventricular clot

Differential Diagnosis

Up to 10% to 16% of patients presenting with sudden-onset, intense headache or "thunderclap" headache and normal neurologic function on physi-

Table 5. Differential Diagnosis Of Sudden-Onset Headache

"Cannot Miss" Causes	Suggestive History And Physical Findings
Subarachnoid hemorrhage	Abrupt headache, stiff neck, third nerve palsy (see text and **Table 7**)
Hypertensive emergencies	Severe (usually chronic) hypertension, may have papilledema and other signs of end-organ damage
Cervical or cranial artery dissections	Neck pain, abrupt onset, variable presence of neurological deficit
Idiopathic intracranial hypertension (pseudotumor cerebri)	Obese, female patient, papilledema, possible sixth nerve palsy
Cerebral venous and dural sinus thrombosis	Hypercoagulable state of any type
Meningitis or encephalitis	Fever, stiff neck, altered mental status, seizure
Temporal arteritis	Usually age > 50 years, symptoms of polymyalgia rheumatica, abnormal scalp vessels on examination
Acute narrow angle closure glaucoma	Painful red eye with midposition pupil and corneal edema
Spontaneous intracranial hypotension	Headache varies with position: worse when upright, relieved when supine
Carbon monoxide poisoning	Cluster of cases, winter season
Acute stroke (hemorrhagic or ischemic)	Abrupt onset and focal neurological deficit conforming to an arterial territory
Pituitary apoplexy	Visual acuity or field abnormalities, known pituitary tumor
Mass lesions 1. Tumor 2. Abscess 3. Parameningeal infection 4. Intracranial hematoma 5. Colloid cyst of third ventricle	Any neurological finding, focal or generalized
Common Benign Causes	
A. Migraine B. Tension-type headache C. Coital headache D. Exertional headache E. Benign-cough headache F. Sinusitis G. Segmental reversible vasospasm (Call-Fleming syndrome) H. Other causes	

cal examination will have SAH.[5-10] For the remainder, the differential diagnosis is quite broad and includes both benign and serious etiologies, as listed in **Table 5.**[7,33] The emergency clinician must rapidly narrow the diagnosis with further history, physical examination, and/or diagnostic testing.

In studies of misdiagnosis, all of the diagnoses on the list are sometimes found. Although there are no convincing data to suggest which are missed more commonly, diseases that often present with a severe headache but associated with a normal neurological examination (eg, cervical artery dissection or cerebral venous sinus thrombosis) are more easily missed. Furthermore, some of these same problems are associated with a normal noncontrast brain CT scan. Identifying and managing SAH still remain challenging and will be further discussed in greater detail.

Prehospital Care

Initial prehospital care should begin with obtaining vital signs and assessing level of consciousness, including the Glasgow Coma Scale (GCS) score. Stabilization may include airway management depending on the patient's clinical status, training of the prehospital emergency care provider, and distance to the ED. A brief neurologic examination should be performed to assess for obvious focal neurologic deficit, and a formal stroke scale should be used to assess severity of symptoms.[62] Patients with focal neurological findings and suspected stroke should be rapidly transported to a hospital in accordance with local EMS protocols.[62] Time that the patient was last seen normal and time of symptom onset should be ascertained in case the patient qualifies for time-sensitive acute stroke treatment. Reversible causes of depressed mental status should be considered, such as checking a fingerstick glucose. Consider administering naloxone if opiate overdose is suspected. There are limited data specific to prehospital care of patients with SAH, since the diagnosis is rarely clear until after further evaluation in the hospital. The authors believe that since most neurologically intact patients with thunderclap headache do not have SAH or any other serious neurological disease, they do not require initial transport to a specialized center from the prehospital setting.

ED Evaluation

Whom To Evaluate?

Of the 2% of all ED patients that present with headache,[1] deciding which of these should be evaluated for SAH is particularly challenging. Someone with a classic thunderclap worst-of-life headache without a previous headache history or with focal neurologic deficits is typically a candidate for an SAH workup. However, evaluation of the alert patient with a normal neurologic examination or one with a vague change in his or her primary migraine disorder is less clear.

Of all patients who present with worst-of-life headache, most will have benign causes. Only 10% to 16% will have serious pathology such as SAH.[5-10] Thus, a thorough clinical history and physical examination is imperative in determining which patients require more rigorous diagnostic evaluation.
There are no data on how to best triage neurologically intact patients with severe, abrupt-onset headache. A more urgent triage level that puts these patients into a queue to be seen (and thus CT scanned) sooner rather than later would appear logical.

Clinical History

Seventy percent of patients with SAH present with headache alone, without focal symptoms, as reported in 1 prospective study of 137 sudden-onset headache patients.[7] This simple fact underscores the importance of properly evaluating patients with isolated, severe, sudden-onset headache. The abrupt nature of headache onset is the most distinctive clinical feature of SAH and is seen in about three-quarters of patients, according to several well-designed prospective studies.[7,9,63,64] The term "thunderclap" headache, first coined by Day and Raskin in 1986, describes an acute severe headache with rapid onset, which reaches peak intensity within seconds.[65] Although a thunderclap headache is often considered the classic description of a ruptured aneurysm, it is neither sensitive nor specific. If 10% to 15% of patients with thunderclap headache have SAH and other serious causes, it follows that 85% to 90% do not.

These latter patients—with so-called benign thunderclap headache—have excellent outcomes, and their conditions are often later diagnosed as migraine and other primary headache syndromes.[5-10] Based on prospective data of acute headache patients, the abrupt nature of symptom onset is equally seen in these benign cases and is not specific to patients with SAH. In a series of 102 acute headache patients, more than half of the patients in the SAH group as well as the benign thunderclap headache group reported symptom-onset within seconds.[64]

The International Cooperative Study on the Timing of Aneurysm Surgery, a large prospective multicenter study of 3521 patients, found a normal

Table 6. Important Historical Questions To Distinguish Subarachnoid Hemorrhage[33]

1. **Onset:** Is headache abrupt?
2. **Severity:** Is it the worst headache ever? Is it "10 out of 10"?
3. **Quality:** How does this headache compare with prior episodes? Is it distinct or unique for this patient?
4. **Associated Symptoms:** Are there focal symptoms, syncope, seizure, neck stiffness, or double vision?

level of consciousness in three-quarters of patients with SAH.[31] Transient loss or altered level of consciousness has been reported in about one-fourth of patients in other smaller prospective studies.[7,64]

Vomiting is a nonspecific feature associated with 70% of patients with SAH.[9,64] It is also seen in half of all benign cases of thunderclap headache and is quite common in migraine.[9,64] Focal symptoms, seizures, or double vision may be reported[64] and should alert the emergency clinician to consider SAH or other serious pathology, as these symptoms are rare with benign causes of headache. Neck stiffness may be another clue to more serious pathology, which was reported in 60% of 23 patients with SAH in 1 prospective study compared with only 10% of 114 benign thunderclap headache patients.[7]

Table 6 summarizes the key features that an emergency clinician may obtain from careful history taking to help identify SAH. However, there are no published prospective trials to date specifically identifying a clinical prediction model for SAH. It is important to emphasize that in patients with normal physical examinations, there are no significant features of the headache that allow reliable distinction between SAH and headaches of benign etiology.[9,64] This point is important because emergency clinicians cannot use "how well the patient looks" in estimating pretest probability of an SAH.

Physical Examination

As always, the physical examination begins with the vital signs, general appearance, and assessment of airway, breathing, and circulation (ABCs). Once the patient is stabilized, a focused physical examination should be performed that includes a relevant neurologic examination. In patients who are comatose or otherwise unable to give a cogent history, ophthalmoscopic examination may reveal an important diagnostic clue—retinal hemorrhages, which are seen in approximately 10% of all patients with SAH.[66] This finding may be the only clue to the correct diagnosis in comatose patients. Often, the physical examination is completely normal and, thus, of little help in assessing patients.[2] Abnormal physical examination findings that may be associated with SAH are listed in **Table 7**. Patterns of focal deficits sometimes suggest the location of the offending aneurysm.

Understanding A Spectrum Of Deviations From The Classic Presentation

Although the most common presentations of SAH are described above, there is no pathognomonic sign or symptom that can help to exclude or to establish the diagnosis. Headache may not even be part of the presentation at all. A recent retrospective study of over 200 patients who underwent aneurysmal clipping reported that 8% did not have any headache at onset of SAH, but instead presented with sudden onset of general malaise or isolated neck or back complaints.[72] The study has obvious limitations due to possible recall bias from retrospective data collection by mail questionnaire. The important point is that emergency clinicians must have a high index of suspicion for this condition even with atypical presentations, as SAH is not diagnosed in one-fourth of patients with this condition on initial presentation.[11-14] Consult **Table 8 (page 8)** to help identify distracting clinical scenarios in evaluating for SAH.

Table 7. Physical Examination Findings To Help Identify Subarachnoid Hemorrhage[15,35]

Finding	Likely Location Of Aneurysm
Mental status change • Seen in about one-fourth of patients[7,64,67]	Any
Meningismus • Seen in 60% of patients with subarachnoid hemorrhage[7] • Takes 3-12 hours to develop and may not be appreciated in comatose patients[68]	Any
Third nerve palsy[39,69] • 90% of patients with third nerve palsy due to aneurysm (versus other causes of third nerve palsy) have anisocoria > 2 mm[70]	Posterior communicating artery
Sixth nerve palsy[70,71] • Presents 3-14 days after onset of subarachnoid hemorrhage • Associated with higher clot burden • Gradually resolves	Any (due to increased intracranial pressure)
Bilateral leg weakness, abulia[15,35]	Anterior communicating artery
Nystagmus, ataxia, dizziness[15,35]	Posterior circulation
Hemiparesis with aphasia or neglect[15,35]	Middle cerebral artery
Subhyaloid (retinal) hemorrhage (Terson syndrome)[66] • Seen in about 10% of patients with subarachnoid hemorrhage • Associated with worse clinical grades on presentation and poorer prognosis	Any

Diagnostic Studies

Noncontrast Head CT

The initial investigative study in patients with suspected SAH is the noncontrast head CT.[19] Refer to **Figure 1** (page 3) for the appearance of a typical SAH on a CT scan. Although CT has great accuracy in detecting subarachnoid blood, the emergency clinician must understand its many limitations, as summarized in **Table 9.**

Most importantly, CT findings are time dependent. There is a decrement in identifying hemorrhage on the scan as time from symptom onset increases because blood is degraded and diluted as cerebrospinal fluid (CSF) is continuously circulated.[87] The largest prospective observational study supporting this used data collected during the 1980s from over 3500 patients with SAH on second-generation scanners presenting at various times from symptom onset; CT had a 92% sensitivity on the day of aneurysm rupture and progressively decreased to 86%, 76%, and 58% on days 1, 2, and 5 postrupture, respectively.[31] With upgrades to third-generation scanners in the 1990s, subsequent studies, although mostly retrospective and with smaller sample sizes, reported similar findings with sensitivities ranging from 90% to 98% in the first 24 hours,[8,88-90] with decreasing sensitivity even after 12 hours.[89] Newer "fifth-generation scanners" report sensitivities ranging from 93% to 100% if scanned early, within 24 hours; however, these are also based on retrospective studies and relatively small numbers of patients.[91-93]

Another important caveat in using CT to detect SAH is spectrum bias; patients with normal neurologic examinations and smaller volumes of hemorrhage may not have CT abnormalities.[31,90,94] Patients with anemia and hematocrit < 27% to 30% may have hemorrhage that is overlooked on the scan because their blood may be isodense with the brain.[94,95] Other considerations include experience of the interpreter[96] and technical factors such as quality of the scanner and artifacts of bone or motion that may limit the study.[33]

The location of the blood on CT can be useful. Blood from a ruptured aneurysm is usually located around the basal cisterns (**see Figure 1, page 3**), whereas in traumatic SAH, the blood is typically higher in the cerebral convexities or in areas of coup or contrecoup force, such as the anterior portions of the middle and frontal cranial fossae (**see Figure 3**).[15] This distinction may be especially useful in patients with SAH who may have fallen from syncope at onset. Finally, there is also an uncommon entity of spontaneous convexity SAH that is almost never aneurysmal.[97,98]

Table 8. Associated Findings That May Distract From Diagnosis Of Subarachnoid Hemorrhage[33]

1. **Relief with pain medications**
   Headache may improve spontaneously or with analgesics.[73-78]
2. **Absence of headache**[72]
3. **Nonspecific viral symptoms**
   Combination of headache with fever, neck pain, nausea, or vomiting may be prematurely attributed to a viral syndrome.[12,16,79,80]
4. **Musculoskeletal pain**
   Prominent neck pain may be attributed to arthritis or musculoskeletal diagnoses.[12,16,79,80]
5. **Altered behavior/mental status**
   Subarachnoid hemorrhage may present with delirium, acute confusional state, or psychosis.[81,82]
6. **Head injury from syncope**
   Transient loss of consciousness due to aneurysm rupture may lead to a focus on resulting traumatic head injury, thus disguising the preceding spontaneous subarachnoid hemorrhage.[83] Subarachnoid blood from aneurysms is typically around the basal cisterns (**Figure 1**), whereas that from trauma tends to be higher in the convexities (**Figure 3**) or in areas of coup and contrecoup forces.[15]
7. **Cardiac abnormalities**
   Electrocardiogram abnormalities and elevated cardiac markers may be present.[12,16,79,80,84,85]
8. **Concomitant hypertension**
   Excessive focus on blood pressure may lead to a diagnosis of hypertensive urgency or emergency.[12,13,16,79,80]
9. **Cardiac arrest**
   Subarachnoid hemorrhage may be associated with cardiac arrest (reported in 3.6% of patients in 1 retrospective study).[86]

LP

If findings on noncontrast CT are positive for SAH, then the emergency clinician may shift the focus from diagnosis to treatment, and LP need not be performed. However, in all patients with suspected SAH with a negative noncontrast CT, LP is recommended.[19,20]

Evidence Supporting The Need For LP After Negative Noncontrast CT

Given the life-threatening nature of this condition and the fact that CT is not 100% sensitive for

Table 9. Limitations Of Computed Tomography[33]

1. **Time:** Sensitivity decreases as time increases from symptom onset.
2. **Volume:** Small volume bleeds may not be detected by computed tomography.
3. **Interpreter experience:** Less experienced radiologists or emergency clinicians/general practitioners may have decreased sensitivity compared with experienced neuroradiologists.
4. **Technology:** Modern scanners with thinner cuts without motion artifact will have greater likelihood of identifying subarachnoid hemorrhage compared with older scanners with thicker cuts or cases with motion artifact.
5. **Anemia:** Patients who have a hematocrit < 30% may have a computed tomography that is falsely negative due to isodense blood.

Table 10. Limitations Of Lumbar Puncture[33]

1. Clinicians may be falsely reassured if the computed tomography is negative, equivocal, or suboptimal.
2. Xanthochromia may be absent very early (< 12 hours) and very late (> 2 weeks).
3. Accurate diagnosis of xanthochromia may be difficult.
4. Distinguishing traumatic tap from true subarachnoid hemorrhage may be challenging.
5. Red blood cell count is diluted by circulating cerebrospinal fluid and is affected by the timing of lumbar puncture.
6. There is no guideline for the number of red blood cells required to diagnose subarachnoid hemorrhage.

SAH, LP is typically recommended to detect small amounts of blood that may have been missed by CT. In addition, LP may diagnose or suggest other conditions that cannot be detected by CT, such as benign intracranial hypertension, cerebral venous sinus thrombosis, spontaneous intracranial hypotension, and meningitis.[19] However, in several reports, only 25% to 50% of patients with CT-negative worst-of-life headache actually had LP performed by emergency clinicians.[8,99,100] Although patients and emergency clinicians may defer LP for various reasons, such as time, benign patient general appearance, patient discomfort, and refusal because of fear of complications, there is clear evidence that supports the value of LP as the next step in diagnosis.[8,10,63,90,92,101,102]

The LP is especially critical in alert, neurologically normal patients with sudden-onset, severe headache. Alert patients with SAH are more likely to have negative CT scans than are patients with neurologic deficits.[31,89] This is the population that poses a particular challenge for emergency clinicians but also has the greatest potential for benefit if detected early.

The best evidence supporting the value of LP comes from a recent prospective study of 592 neurologically intact ED patients with acute headache; of the 61 patients with SAH, 6 (10%) cases were diagnosed on the basis of positive CSF results after a normal cranial CT.[10] Another prospective study of 107 similar adult ED patients found a diagnosis of SAH for 18 patients, of which 2 cases were missed by CT (2% miss rate but 95% confidence interval [CI] up to 8.8%).[8] Furthermore, an older prospective study of 27 patients with acute headache found 9 with SAH, of which 5 were missed by CT.[63] Additional retrospective studies, each with more than 100 patients with SAH, found 2% to 7% of cases were missed by CT but picked up by LP and confirmed by angiography.[90,92] In an in-depth evidence-based analysis of these studies, a neurologically normal patient with a negative head CT could still have as much as a 7% chance of having an SAH.[101] This posttest probability is relatively high, confirming the importance of following the head CT with an LP.

Table 11. Other Causes Of Xanthochromia

1. Jaundice (total bilirubin > 10-15 mg/dL)
2. Increased cerebrospinal fluid protein (> 150 mg/dL)
3. Rifampin
4. Excess dietary carotenoids

Interpreting The LP

Even if the decision to perform LP is clear, interpretation of CSF results can still be challenging. See **Table 10** for limitations of using LP in the diagnosis of SAH. There are no set criteria for a positive LP in the diagnosis of SAH. When blood-stained CSF is obtained, the emergency clinician must decide whether this result is due to a traumatic tap, which occurs in 10% to 15% of patients,[103,104] or due to true intracranial bleeding.

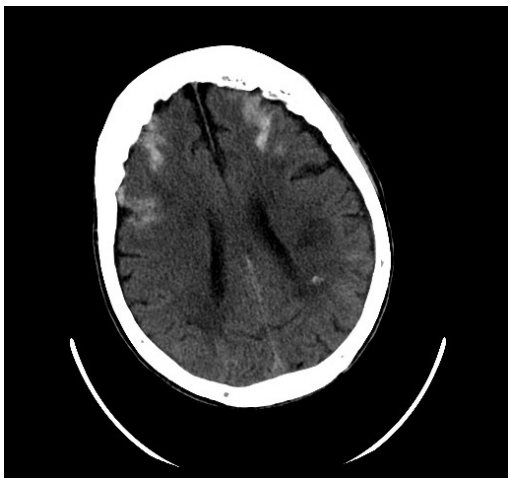
Opening Pressure

Opening pressure may be elevated, > 20 cm H_20, in 60% of patients with SAH.[105] The CSF pressure should be obtained in patients undergoing LP in the ED, if possible, as elevated pressures may be seen in cerebral venous thrombosis or idiopathic intracranial hypertension, and low pressures may be seen in spontaneous intracranial hypotension, leading to alternative diagnosis and management.[106-108]

RBC Count Analysis

It is usual practice to collect 4 serial tubes of CSF

Figure 3. Traumatic Subarachnoid Hemorrhage On Noncontrast Head Computed Tomography



Axial noncontrast computed tomography of a patient with a clear-cut history of head injury without prodromal headache is shown. In contrast to the pattern of the aneurysmal hemorrhage seen within the basal cisterns in Figure 1, subarachnoid hemorrhage due to trauma appears as hyperdensities in the convexities. No aneurysm was found on angiogram. (Reprinted with permission from Jonathan Edlow, MD.)

and assess for constancy of red cells. Regardless of the number of RBCs in the first 3 tubes, if there are 0 RBCs in the final tube, this indicates a traumatic tap, and the emergency clinician can confidently conclude that the CSF is normal. On the other hand, it is generally accepted that the persistence of constant numbers of RBCs from tube 1 to tube 4, usually in the thousands, is abnormal and may be indicative of SAH.[18,109] Interpretation becomes ambiguous when there is "clearing of red cells," or a fall in RBCs in serial tubes.[110] This can occur in both cases of aneurysmal hemorrhage as well as in traumatic tap.[111] Consider the case of an LP where 3000 RBCs are present in tube 1 and 400 RBCs in tube 4. There is no absolute cutoff for the threshold for RBCs to make a diagnosis of SAH, and there are no clear data showing average numbers of RBCs in patients with SAH. However, aneurysm rupture has been anecdotally reported with even "a few hundred" cells, although this is thought to be rare.[112,113] Thus, based on the latter CSF cell count result, an aneurysmal hemorrhage can be neither confirmed nor excluded, and further testing must be considered. (**See the "Xanthochromia" section.**) Although no specific guidelines exist for this situation, additional diagnostic steps may include performing computed tomographic angiography (CTA)[114,115] or magnetic resonance angiography (MRA)[116,117] or obtaining conventional angiography in consultation with a neurosurgeon.

Last, it is important to recognize that the timing of the LP affects the RBC count just as it does CT scan sensitivity — and for the same reason. With circulation of the CSF, the RBCs will diminish with time from symptom onset, sometimes clearing completely within 48 hours after the bleed.[105]

Xanthochromia

Xanthochromia, meaning literally "yellow color," is produced by the breakdown of hemoglobin in the CSF into pigmented byproducts including oxyhemoglobin, methemoglobin, and bilirubin. The first 2 pigments may form in vitro. However, the enzyme-dependent breakdown of heme to bilirubin only occurs in vivo and requires time to develop,[118] thus the presence of xanthochromia is highly suggestive of SAH, and the patient should have continued evaluation.[33,113,119] Traumatic tap is unlikely in these cases because experimental studies of artificially added RBCs to clear CSF and purposeful traumatic taps have demonstrated the absence of bilirubin and xanthochromia.[118,120] False positives may occur because of other apparent causes of xanthochromia,[121] which are listed in **Table 11**.

Xanthochromia may take up to 12 hours to develop and lasts at least 2 weeks; several studies have shown that only a small fraction (20%) of patients with SAH who were tapped within 6 hours had visual xanthochromia, but **all** who were tapped between 12 hours to 2 weeks had xanthochromia.[105,122] Some authors have recommended waiting for 12 hours from symptom onset to obtain an LP.[68,105] However, multiple studies have shown that in the absence of xanthochromia, all of these early cases will show bloody CSF with abundant RBCs.[105,122,123] Thus, finding normal CSF, even in the first few hours, successfully excludes SAH. For all of these reasons, the authors of 1 review recommend not delaying LP, but instead accepting either RBCs or xanthochromia as a positive finding of SAH.[15]

Assessing Xanthochromia: Visual Analysis Versus Spectrophotometry

The best way to assess xanthochromia remains a topic of continued debate. Some researchers claim spectrophotometry is superior to visual inspection[122]—as supported by a peripheral finding of 32 CSF samples with xanthochromia by spectrophotometry in a larger study of 231 patients with cerebrovascular injuries on CT scans, only half were identified by the naked eye[124]—and 1 guideline formally recommends this as the preferred method for CSF analysis.[125] However, visual xanthochromia is a simple valuable test for detecting aneurysmal bleeding; 1 retrospective study of about 150 patients reported a fairly high sensitivity of 93%, specificity of 95%, positive predictive value of 72%, and negative predictive value of 99% when compared with conventional angiography.[109] Furthermore, a recent study directly comparing the ability of 51 emergency clinicians and 51 students to identify xanthochromia by the visual method found sensitivities of 100% and 99%, respectively, compared with spectrophotometry,[126] indicating a minimal difference in detecting abnormal CSF between the two methods.

Regardless, the proponents for spectrophotometry cite a perfect sensitivity of 100%.[122,127] This must be weighed against the poor specificity of spectrophotometry, ranging from 29% to 75% in a prospective and retrospective study, each with more than 200 patients,[127,128] and high false positive rate (18 of 20 patients) in another prospective study.[8] Relying on spectrophotometry could lead to increased angiography, exposing patients with false positive CSF results to unnecessary risk.[128] From a practical standpoint, spectrophotometry is not available in most (> 99%) emergency laboratories in North America.[119] The standard method of assessing visual xanthochromia involves rapidly centrifuging the last tube of CSF and comparing it with an identical tube filled with an equal volume of water against a white background. Xanthochromia is identified by a yellowish hue in the CSF tube compared with the water control.[33] Once again, timing is important; xanthochromia will disappear after 2 weeks.[105]

In the absence of xanthochromia, there is no current approach to CSF analysis that can definitively distinguish between true hemorrhagic and traumatic bloodstained CSF. This dilemma may be reduced by

Table 12. Laboratory Studies For Subarachnoid Hemorrhage

1. Chemistry panel
2. Complete blood count
3. Prothrombin time/partial thromboplastin time (PT/PTT)
4. Blood typing and screening
5. Troponin

the use of fluoroscopic guidance for LP,[104] but this is not often readily available in the ED. In addition, direct imaging of the cerebral vessels in conjunction with a neurosurgical consultation can confirm the presence of an aneurysm.[114,115,129]

Laboratory Testing

See Table 12 for laboratory studies that may be helpful in cases of suspected SAH.

ECG

Cardiac abnormalities are common following acute SAH. Subendocardial ischemia may result from autonomic stimulation from the brain and circulating catecholamine surge, resulting in an abnormal ECG in 50% to 100% of patients with SAH in the acute phase.[84,85,137,138] Common benign, and usually transient, ECG changes are nonspecific ST- and T-wave changes, prolonged QRS-segments, U-waves, and increased QT-intervals, but ECG changes mimicking cardiac ischemia are known to occur as well.[84,139] Positive troponin occurs in 20% to 40% of acute cases[85,140,141] and may lead to cardiopulmonary complications and worse outcomes.[85] Serious arrhythmias may occur in < 5% of patients and have been shown to be associated with worse outcomes.[84] Whereas suspected myocardial ischemia should be treated in the usual manner, SAH is considered a contraindication to thrombolytic or anticoagulant therapy.[142] Coronary angiography has been normal in several case reports of patients with SAH with ST-elevations and/or elevated troponin, highlighting a neurocardiogenic mechanism distinct from coronary thrombosis underlying this process.[143-146]

Table 13. Emergency Department Management Of Subarachnoid Hemorrhage

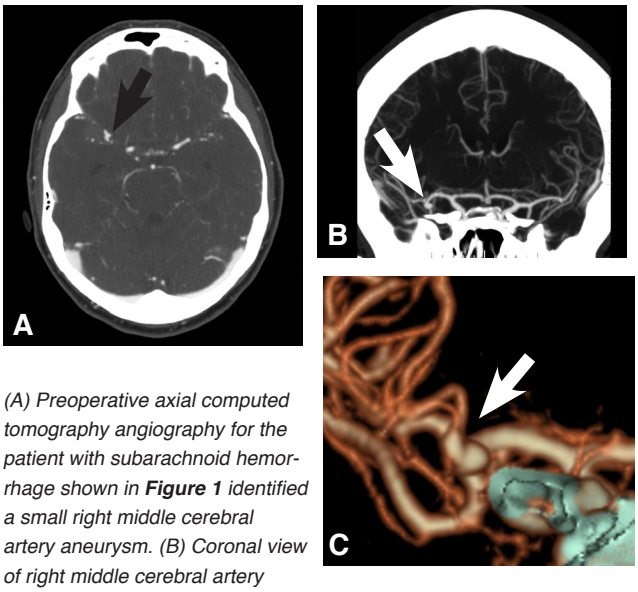
1. Page neurosurgery.
2. Obtain some form of cerebrovascular imaging in consultation with neurosurgery.
3. Order bed rest and nothing by mouth except medications.
4. Provide analgesia and sedation as needed.
5. Manage blood pressure.
   A. Discuss target blood pressure goals with consulting neurosurgical team.
   B. Commonly used intravenous agents include labetalol and nicardipine.
   C. Monitor for hypotension.
   D. Avoid nitroprusside (may increase intracranial pressure).
6. Consider seizure prophylaxis.
7. Provide nimodipine 60 mg by mouth (or by nasogastric tube if unable to swallow) every 4 hours.

Clinical Decision Making

Are Negative CT And Negative LP Sufficient To Rule Out SAH?

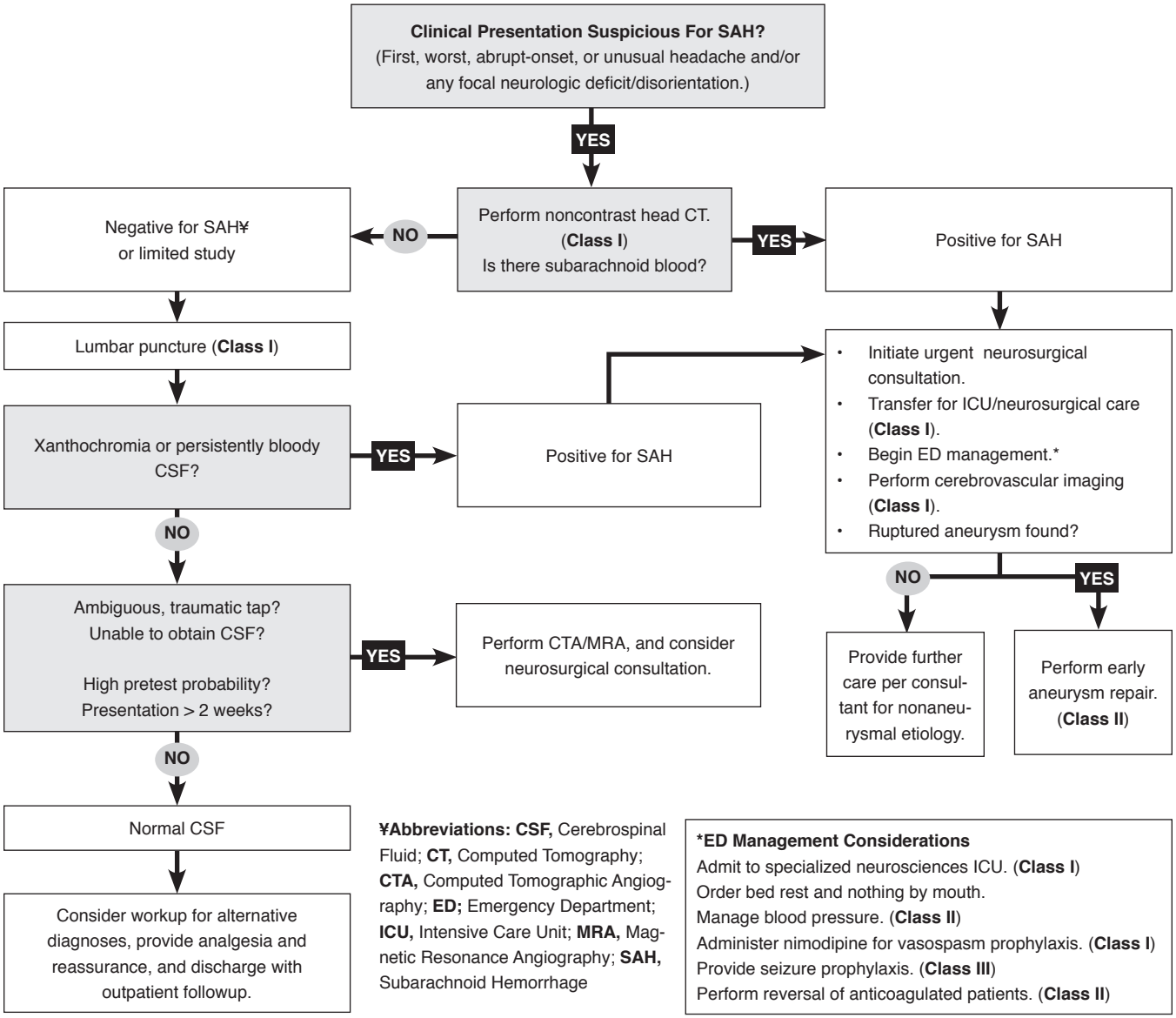
Current ACEP clinical policy states that in patients with negative CT and LP, the diagnosis of SAH can be excluded.[19] There are several well-conducted prospective studies to support this practice.[5,7,10] The largest of these was a prospective cohort study that observed 592 patients with sudden-onset headache, including 61 patients with SAH (55 diagnosed by CT, 6 by LP), for 3 years and found no cases of subsequent SAH in those patients with negative CT and LP.[10] Several smaller studies have also observed patients with "thunderclap" headache after negative CT and negative LP results and none of the patients developed SAH.[6,130,131] The largest observed clinical outcomes of 71 patients for an average of 3.3 years.[6] Furthermore, a recent systematic review that included 7 studies of patients with negative CT and LP with > 1 year follow-up found a pooled proportion of 0% with SAH.[132] Together, this body of evidence supports a standard of excluding SAH by a combination of negative CT and LP. It is important that the CSF analysis be complete and normal.

Figure 4. Detection Of Aneurysm By Computed Tomographic Angiography Of The Head



*(A) Preoperative axial computed tomography angiography for the patient with subarachnoid hemorrhage shown in **Figure 1** identified a small right middle cerebral artery aneurysm. (B) Coronal view of right middle cerebral artery aneurysm. (C) Three-dimensional reconstruction of the computed tomographic angiography better characterized the same aneurysm. Patient underwent surgical clipping for repair. (Reprinted with permission from Lisa Thomas, MD.)*

Clinical Pathway: Emergency Evaluation Of Suspected Subarachnoid Hemorrhage



¥Abbreviations: CSF, Cerebrospinal Fluid; **CT,** Computed Tomography; **CTA,** Computed Tomographic Angiography; **ED;** Emergency Department; **ICU,** Intensive Care Unit; **MRA,** Magnetic Resonance Angiography; **SAH,** Subarachnoid Hemorrhage

***ED Management Considerations**

Admit to specialized neurosciences ICU. (**Class I**)
Order bed rest and nothing by mouth.
Manage blood pressure. (**Class II**)
Administer nimodipine for vasospasm prophylaxis. (**Class I**)
Provide seizure prophylaxis. (**Class III**)
Perform reversal of anticoagulated patients. (**Class II**)

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
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- 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

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA*. 1992;268(16):2289-2295.

Role Of Primary CTA In The Diagnosis Of SAH

LP can be difficult in some patients and is associated with known risks. Most common complications include postdural puncture headache and low back pain, but other complications such as infection, bleeding, neuropathies, and, very rarely, even cerebral herniation have been reported.[133,134] CTA has the potential to be a rapid, easy, and noninvasive alternative to detect ruptured aneurysms. **See Figure 4** for the appearance of an aneurysm on CTA and refer to the **"Controversies"** section to read more about the potential role for this imaging modality in the primary diagnosis of SAH.

Role Of Primary Magnetic Resonance Imaging (MRI) In Diagnosis Of SAH

Only small studies of magnetic resonance (MR) for diagnosis of SAH exist. MRI has been shown to be better than CT in detecting subacute and chronic SAH, especially with fluid-attenuated inversion recovery (FLAIR) and T2-weighted imaging performed 4 to 14 days after SAH in 1 study of 41 patients.[116] Another recent study showed 100% sensitivity for detection of CT-proven SAH in 13 patients with acute presentations within 12 hours of symptom onset by proton density and FLAIR sequences.[117] In contrast, another study of 12 patients with CT-negative, CSF-positive SAH showed that only 2 patients who had positive FLAIR findings for SAH.[135] These were also the 2 patients that had the highest RBC count in the CSF, indicating that a minimum number of RBCs is required to cause hyperintensity on MR. Thus MR has not yet demonstrated its value as the primary diagnostic tool for acute SAH. On a practical note, this modality is of limited use in the typical ED owing to limited availability in the acute setting.[136]

Initial Management

Once a patient's acute SAH is diagnosed, emergency neurosurgical consultation should be obtained to arrange for definitive therapy for the ruptured aneurysm. In many cases, this will require transfer to a tertiary care facility. ED management should focus on close hemodynamic monitoring, supportive care, and prevention and treatment of complications. Few interventions for SAH management are unequivocally proven but rather are based on tradition and expert consensus. **Table 13** depicts a sample protocol for SAH management based on the practices of the authors' institution. Details should be discussed with the consulting neurosurgical team, as specific management practices may vary among hospitals and treating specialists and based on patient circumstances.

General Care Measures

Bed rest is recommended, although it has not been proven to improve outcomes or prevent complications.[20,21] Adequate analgesia and judicious sedation should be administered to ensure patient comfort. Serial neurologic examinations should be performed to monitor for deterioration. The airway should be managed if necessary.[20] Cardiac monitoring should be performed to evaluate for arrhythmias.[84] The head of the bed can be kept at 30º to facilitate venous drainage.[25] The patient should have nothing by mouth until surgical or endovascular treatment plan is decided.

Cerebrovascular Imaging

After the diagnosis is made, further imaging of cerebral vasculature should be obtained to identify the ruptured aneurysm in conjunction with neurosurgical consultation. Typically, this will be performed at the same center that will provide definitive management.

Cerebral digital subtraction angiography (DSA) has traditionally been the gold standard imaging technique for preoperative planning, as it accurately elucidates vascular anatomy, identifies the bleeding site, and outlines the size and location of the aneurysm.[147] Angiography may be negative in 10% to 20% of patients with SAH because of perimesencephalic hemorrhage, vasospasm, thrombosed aneurysm, or other rare causes.[148-150] The risk of complication from this procedure is considered to be very low. Two large prospective series of almost 3000 procedures[151,152] and 1 retrospective study of almost 20,000 procedures[153] report a neurologic complication risk of only 0.4% to 2.6%, of which more than half are transient.

CTA is becoming increasingly popular since it is fast and noninvasive and a growing body of evidence reports a sensitivity and specificity that is comparable to cerebral angiography.[154-160] **See Figure 4** for an example of an aneurysm detected by CTA. This has led to several prospective studies that support using only CTA for preoperative planning.[129,161-164]

Finally, MRA is available but less well studied.[165,166] The smallest aneurysm that can be detected is likely a function of the equipment available and the experience of the neuroradiologist. The choice of angiography type should be made in consultation with the neurosurgeon or interventionalist. This is important since endovascular treatment is being used with increasing frequency and involves conventional angiography and intravenous (IV) contrast. Ideally patients should be spared sequential dye loads, and in some cases, the initial diagnostic angiogram will be done at the same time as the endovascular intervention.

Monitoring And Preventing Complications

Rebleeding

Rebleeding is a significant source of morbidity and mortality, affecting up to 15% of patients within the first few hours,[167] and has a cumulative mortality risk of 40% without intervention.[168] One study of

574 patients suggested that a higher Hunt and Hess score and larger aneurysm size were associated with a higher likelihood of rebleeding.[169] Two potential strategies to reduce the occurrence of rebleeding are BP control (see next section) and antifibrinolytic therapy.

In the past, when aneurysm surgery was delayed for days to weeks from the SAH, antifibrinolytics were used to prevent rebleeding during that interval. Because this strategy often resulted in cerebral ischemia, the practice has fallen out of favor.[170] However, in the current age of early intervention, new data suggest that very short courses of antifibrinolytics may reduce rebleeding without causing ischemia.[20,171-173] Rapid diagnosis of SAH and early definitive repair probably remains the best strategy for prevention.

BP Management

In theory, higher pressures may increase the risk of rebleeding, whereas lower pressures may compromise cerebral perfusion pressure, leading to cerebral ischemia. This balance was demonstrated by a retrospective study of 134 patients with SAH, 80 of whom had systolic BPs reduced to < 100 mm Hg.[174] While rebleeding occurred only in 15% of patients treated with antihypertensives, compared with 33% in the nontreated group, the rates of cerebral infarction were doubled (40%) in the treatment group compared with the nontreatment group (22%). Whether acute hypertension definitively increases rebleeding risk is still controversial based on conflicting results of recent studies.[167,169]

There are no conclusive data for the target BP in the management of SAH or the ideal antihypertensive agent, so these choices should be left to practices of the consulting neurosurgical team. The most recent AHA guidelines recommend that "blood pressure be monitored and controlled to balance the risk of stroke, hypertension-related bleeding, and maintenance of cerebral perfusion pressure."[20] If needed, labetalol, nicardipine, and esmolol are commonly used IV agents.[20,25] Nitroprusside should be avoided because of its tendency to increase intracranial pressure (ICP) and potential for toxicity with prolonged infusion.[20] If the patient is expected to remain in the ED for a long time, an arterial catheter may be inserted for continuous BP monitoring.[21]

Preventing Vasospasm

Cerebral vasospasm is a delayed complication that may develop from several days up to 2 weeks post-SAH, peaking 7 to 10 days after the event.[33] Vasospasm may be asymptomatic or may lead to delayed cerebral ischemic infarcts, which can cause significant morbidity related to SAH.[175] Nimodipine, a calcium channel blocker, has been shown to improve outcomes (risk ratio 0.67; 95% CI, 0.55-0.81) of death or dependence in a recent Cochrane Review of 16 trials of calcium antagonists.[176] The mechanism for this improvement is unclear. The statistical significance of this review rests heavily on the largest randomized controlled trial (RCT) of 554 patients, from which comes the standard dosing regimen.[177] Thus, it is appropriate to administer 60 mg of nimodipine orally every 4 hours, even starting in the ED.[20,21,176] If the patient is unable to swallow, the nimodipine should be crushed and given via a nasogastric tube, as there is no evidence for the efficacy of IV nimodipine.[176] Nicardipine, another calcium channel blocker, has also been shown to decrease vasospasm in a randomized trial but without any improvement in outcome.[178]

IV magnesium sulfate[176,179] and statin therapy[180-182] are alternate agents that have shown improved outcomes in a small number of prospective trials; but further study is needed, and larger trials are currently underway.[183]

Role For Seizure Prophylaxis

Less than 20% of patients have a seizure during or soon after SAH.[184] Some advocate prophylactic emergency antiepileptic drug use in all patients with SAH[185] based on older data when the rates of seizure after SAH were thought to be higher and management was often delayed. This has become controversial,[186] but there is likely a high-risk subset who may benefit from it, as those with a higher clinical grade are likely to deteriorate after a seizure.[21,187,188]

Although it is not clear why, phenytoin is commonly used but has been associated with worse outcomes, especially with prolonged use.[189,190] One pooled retrospective analysis of 4 multicenter RCTs including 3552 patients admitted with aneurysmal SAH found increased in-hospital complications and worse outcomes after antiepileptic therapy based on the GCS score, neurologic deterioration, and vasospasm.[189] Another prospective study of 527 patients with SAH found that higher serum phenytoin levels were associated with functional decline and cognitive disability at 2 weeks and 3 months.[190]
It is now common practice to give a short course (< 3 days) of an antiepileptic drug based on data showing that short courses have the same benefit of low in-hospital postoperative seizure rate (< 2%) when compared with longer courses, but without accumulating adverse effects of the drugs.[187,191] In a patient who has not already seized, discuss the administration of an antiepileptic with the accepting cerebrovascular specialist.

Hyperglycemia Management

Intensive insulin therapy in critically ill patients in the Intensive Care Unit (ICU), including those with neurovascular emergencies, has been shown to improve morbidity and mortality.[192] Many have thought that patients with SAH would similarly benefit, as hyperglycemia is known to be neurotoxic

and associated with poor outcome.[193-197] However, a recent RCT of patients with neurovascular emergencies[198] and the only existing prospective RCT of insulin therapy in postoperative patients with SAH[199] showed no such benefit. No similar studies have been done on preoperative ED patients with acute SAH and hyperglycemia.

Therefore, although hyperglycemia is common in SAH, and it is often closely managed in the ICU, it is not clear that intensive insulin therapy in the ED is of substantial benefit, and this can be deferred until the ICU. However, given the benefits of glucose control in critically ill patients in general,[192] sliding scale insulin should be considered for those patients who will have a longer stay in the ED while awaiting an ICU bed.

Acute Clinical Deterioration: Consider Repeating the Head CT

In patients who experience an early deterioration in neurological status, there are several potential causes, and repeating the CT scan is important to distinguish among them. Some, such as rebleeding or cerebral infarction, have poor prognosis; however, others, such as acute hydrocephalus or extension into the subdural space, are treatable and may not adversely affect the long-term outcomes.[33]

Definitive Aneurysm Repair: Clipping Versus Coiling

The 2 main approaches to aneurysm repair are microvascular neurosurgical clipping or endovascular coiling. Early treatment, within 72 hours, is a common approach.[185,200] The largest trial comparing clipping to coiling randomized 2143 patients suitable for both approaches and found improved outcomes for patients who underwent endovascular coiling.[201-203] However, not all aneurysms are suitable for each approach, and clipped patients had an increased risk of seizure whereas coiled patients had a slightly increased risk of rebleeding.[203] Each patient is best evaluated by a multidisciplinary team. The choice of clipping or coiling is based on a variety of factors, including anatomical characteristics of the aneurysm and expertise of the clinicians as well as the patient's clinical status, comorbidities, and preference.[204-206]

Prognosis

Even with transfer to specialized, neurosurgical ICU care in high-volume centers, in-hospital mortality after SAH is still > 30%.[207,208] Outcome in surviving patients depends on several factors, including age, grade at time of presentation, comorbidities, and perioperative complications during hospitalization.[31,209] The dreaded complication of rebleeding is the most important cause of mortality and poor outcome,[31] as up to 80% of patients with rebleeding die or remain disabled.[210] More than half of all SAH survivors have permanent neurologic disability, including deficits from rebleeding, cerebral infarction resulting from vasospasm, epilepsy, and cognitive impairment.[211]

In stark contrast to those with confirmed aneurysmal hemorrhage, patients with SAH with negative angiogram due to perimesencephalic hemorrhage have an excellent prognosis; 98% survive without deficits.[150]

Special Circumstances

Anticoagulated Patients

Some patients with SAH are taking antithrombotic medications including warfarin and antiplatelet agents at the time of presentation. For patients on warfarin, rapid reversal is recommended in the setting of intracranial bleeding with IV vitamin K and clotting factor complements.[25,212-215] Fresh frozen plasma (FFP) is most commonly used in the United States but is associated with a delay in international normalized ratio (INR) reversal in comparison with prothrombin complex concentrates (PCC), which work more rapidly but are of limited availability.[212] Recombinant Factor VIIa is an alternative that successfully reverses the INR but restores only one-fourth of the missing factors.[212] For patients taking antiplatelet agents, some have advocated platelet transfusion.[216] The risk to benefit ratio of this intervention is unclear, and this should be done in conjunction with specialty consultation.

There are very few case series that comment specifically on reversal in SAH cases,[217] but rebleeding is so common that general consensus supports reversal in the acute setting.[218,219] This is recommended regardless of the indication for anticoagulation because the risk of death from intracranial bleeding is much higher than the risk of thrombosis from conditions such as atrial fibrillation or a mechanical valve.[25,215] A recent literature review of several small retrospective series reporting reversal of warfarin-associated intracerebral hemorrhage found that reversal was safe and effective in preventing hematoma expansion without increased adverse events, even in patients with mechanical valves.[220]

Controversies

Can Primary CTA Replace LP In The Diagnosis Of SAH?

Whether CTA can be used for the primary diagnosis of SAH, which would eliminate the need for LP, is a topic of current debate. A common practice is to obtain an LP for all patients with suspected SAH after a negative noncontrast CT. However, only 25% to 50% of patients with CT-negative, worst-of-life headache actually had LP performed by emergency clinicians,[8,99,100] likely because this test is burdensome for the patient and the provider. If CTA could improve the diagnostic capability of noncontrast CT, perhaps it could reduce the need for LP.

Risk Management Pitfalls For Subarachnoid Hemorrhage

Delayed or missed diagnosis of SAH may lead to malpractice litigation for emergency clinicians.[235] Well-appearing patients with nonfocal examination findings are more likely to be misdiagnosed.[11] Most common misdiagnoses are primary headache disorders such as migraine, followed by meningitis, viral syndromes, cerebral ischemia, hypertension, cardiac abnormalities, sinusitis, musculoskeletal neck pain, and psychiatric diagnoses.[15] **See Table 8** to avoid possible distractions from diagnosing SAH and the following risk management pearls.

1. **"Even though she said her headache was worse than usual and felt different, I thought it was just a migraine, so I didn't get a head CT."**
   A head CT should be considered even in patients with a primary headache disorder, like migraine, if the characteristics of the headache are substantially different from their usual symptoms.

2. **"I thought she just had the flu."**
   Emergency clinicians should be aware of the wide spectrum of clinical symptoms that may present as SAH. Patients may have nonspecific symptoms including neck pain, myalgias, and mild headache that may be misdiagnosed as a viral syndrome. Consider working up these patients with CT and, if negative, LP.

3. **"But the patient's pain completely resolved with prochlorperazine, so I just didn't think it could be an SAH."**
   Patients with SAH may have symptoms that completely resolve with narcotic or nonnarcotic medications and sometimes even without treatment. The decision to work up a patient for SAH should not be solely influenced by response to pain medications.

4. **"But he was just an old confused nursing home patient; I thought he had delirium from a urinary tract infection."**
   Patients with delirium or change in mental status should be carefully evaluated, as SAH may be in the differential. Studies have shown that psychiatric diagnoses and delirium are common misdiagnoses for SAH.

5. **"I thought he had had a heart attack."**
   Patients with SAH may have abnormal ECGs and/or positive cardiac markers due to effects of a catecholamine surge from brain injury. Focusing on these cardiac findings may distract the provider from diagnosing the underlying etiology, which may be SAH.

6. **"But the head CT was negative, symptoms resolved, and the patient didn't want to stay for the LP."**
   CT may be negative in 2% to 7% of patients with SAH, and sensitivity is highly time dependent. In a patient with suspected SAH, LP is required to rule out the diagnosis regardless of other circumstances.

7. **"I did not check coagulation tests and the patient did not tell me he was taking warfarin."**
   Basic laboratory tests including INR/PT and PTT should be checked in all patients with intracranial hemorrhage. Some patients may not be able to provide an accurate history. When patients on therapeutic anticoagulants are diagnosed with SAH, the clotting deficiency should be reversed quickly with IV vitamin K and clotting factor.

8. **"There were only 400 RBCs in the fourth tube and it cleared from 4000 in the first tube, so I assumed it was a traumatic tap."**
   There is no cutoff for the minimum number of RBCs required to diagnose SAH, and it has been reported with even a few hundred cells. Despite serial clearing of red cells, if there is ambiguity between traumatic tap and possible SAH, further neuroimaging and neurosurgical consultation should be obtained to rule out the diagnosis. Also, remember that the number of RBCs diminish with time after onset of headache.

9. **"I didn't transfer the patient because…"**
   Any patient whose condition is diagnosed as SAH should be transferred to a facility with neurosurgical, endovascular, and advanced neuroimaging capabilities. Data show better outcomes for patients treated quickly at these specialized centers.

10. **"I diagnosed the SAH, but the patient became disoriented all of a sudden."**
    Patients with SAH should have careful cardiorespiratory monitoring and serial neurological examinations. They are at risk for developing complications such as rebleeding, vasospasm, and hydrocephalus. Intubation may need to be performed if the patient is unable to protect her or his airway. Repeated head CT should be considered because clinical deterioration from acute hydrocephalus can be reversed with treatment.

Only 2 small studies exist[114,115] comparing CTA with noncontrast CT and LP for primary SAH diagnosis, as identified by a recent systematic review.[221] The best evidence comes from a prospective study of 116 patients in which CTA detected 6 cases of aneurysm that were not seen on noncontrast CT.[114] All these patients had positive LP results and were picked up by routine workup. However, there were 3 cases of negative CT and spinal fluid that had a positive CTA, and these were likely owing to detection of unruptured aneurysms.

Implementation of the CTA strategy may lead to unnecessary angiography or surgical risk in roughly 2% of the population harboring these incidental aneurysms.[42] Some of these incidental aneurysms may be managed conservatively with observation. Thus, discovery by CTA may not lead to additional procedural risks, but this decision involves multiple factors, including characteristics of the aneurysm, expertise of the surgeon, and preferences of the patient. Unnecessary angiography may also occur with the current use of LP in cases of equivocal CSF results or false positive xanthochromia identified by spectrophotometry.[128] However, with CTA, there may be some benefit to identifying or treating these incidental aneurysms, if they are actually causing the symptoms of presentation, as symptomatic aneurysms have 8 times the risk of rupture compared with incidental aneurysms identified without symptoms.[42] Another limitation of CTA, the risk of IV contrast to those with iodine dye allergy and renal failure, also needs to be considered.

As of 2009, although the results are promising for CTA in the diagnosis of ruptured aneurysm, the current diagnostic strategy for SAH cannot be altered at this time without larger prospective trials examining test characteristics, risks, benefits, and the cost-effectiveness of this strategy.

LP-First Strategy

Since most alert, neurologically intact patients with acute-onset headache have benign etiologies with negative workup, some experts have advocated LP as the first diagnostic test for such patients.[222] Under this rationale, patients with normal CSF results could be discharged, thus decreasing the number of CT scans performed at the cost of a minimal increase in the number of LPs. As previously described, many emergency clinicians often omit the LP,[8,99,100] and this strategy would force the LP to be completed. Although an LP-first strategy may be safe in certain patients who are neurologically normal without signs of increased ICP, removing CSF from patients with SAH with unrecognized intracranial hematoma may precipitate rebleeding or herniation; this can occur even without focal neurological findings.[223,224] Concerns about herniation have arisen from pre-CT retrospective reports of 1 sudden neurologic deterioration and death just after the procedure in a series of 129 patients with papilledema or increased ICP[225] and 1 death among 401 patients with brain tumors.[226] Also commonly cited is a case series of 74 patients with suspected intracranial hematomas, of whom 44 had LP prior to imaging.[223] All 44 were drowsy, confused, or had neurologic deficits, and 7 had clinical deterioration at the time of LP. All 7 of these patients had intracranial hematomas, but the relative contribution to the deterioration from LP versus the natural clinical course of the hematoma is unknown. Thus, adverse outcomes due to space-occupying lesions are likely much lower than depicted by the former study. Support comes from a 1988 study of 38 patients with intracranial mass seen on CT, of whom 34 had evidence of mass effect and 37 underwent LP without neurologic deterioration.[227] Overall, the risk of herniation is small even in patients with mass lesions.

Apart from alleviating concerns of herniation in patients with unknown mass lesions or signs of elevated ICP, obtaining the noncontrast CT may be valuable in identifying or excluding other conditions, such as a venous sinus thrombosis or acute paranasal sinusitis, in the differential diagnosis. There are no prospective data on safety, feasibility, or suitable patient selection for the LP-first approach, and thus CT followed by LP remains the standard order for diagnostic testing.

Role Of The "Warning" Headache

In some patients later found to have a diagnosis of SAH, a so-called "sentinel" or "warning" headache occurs days to weeks before the SAH and thus can be diagnosed only in retrospect. These episodes are sudden, severe headaches that resolve. These warning headaches occur in roughly 10% to 43% of SAH patients.[228] The etiology for this phenomenon is controversial and may be caused by minor leaking from a cerebral aneurysm before impending complete rupture,[94] misdiagnosis,[228,229] or recall bias.[229] Regardless of the explanation, observational data suggest that patients experiencing these "warning" symptoms have worse outcome. In a prospective study of over 200 patients with SAH, 17% had a sentinel headache and suffered a 10-fold higher risk of rebleeding compared with those without a sentinel headache.[230] Another, smaller retrospective study found that about 40% of patients with SAH had a "warning leak;" these patients had worse outcomes and a staggering 53% mortality.[94]

Whatever the explanation and occurrence, this phenomenon will be moot if emergency clinicians carefully evaluate patients with acute headache and pursue the diagnostic workup described in this article.

Disposition

Patients with acute SAH should be admitted to an ICU, preferably a neuroscience ICU, for continual supportive care and monitoring until definitive repair of the aneurysm.[21,27,185] Numerous studies now show that patients with SAH have better outcomes when treated in high-volume centers with specialized units and options for both surgical and endovascular repair.[207,208,231,232]

Patients can be safely transferred to these centers. One recent prospective multicenter study on emergent neurosurgical transfers, of which one-third were for SAH, found an average of 5.2 hours from time of diagnosis to patient's arrival at the neurosurgical center without significant clinical deterioration of the GCS score in 90% of patients.[233] Emergency air transportation of patients with nontraumatic acute intracranial hemorrhage has also been found to be safe and effective in facilitating early diagnosis and operative intervention.[234] Prearranged interfacility agreements may be useful for the efficient and appropriate transfer of patients to tertiary care hospitals.

Well-appearing patients with normal neurologic examinations who are suspected of having an SAH with normal CT and normal CSF analysis may be safely discharged from the ED with outpatient follow-up.[19]

Summary

When a patient presents to the ED with an abrupt-onset, unusual, or worst-ever headache, the emergency clinician must consider the diagnosis of SAH. There is a wide spectrum of complaints for patients with SAH, and atypical presentations can lead to missed diagnoses. This article summarizes the evidence supporting the diagnostic workup beginning with noncontrast CT and, if negative, LP. The practicing emergency clinician must understand the limitations of each study and, when in doubt, obtain neurosurgical consultation. Because of the potential for substantial morbidity and mortality, it is important to make the diagnosis quickly. Once the diagnosis is established, the next steps include neurosurgical consultation and transfer of patients to high-volume centers with the full range of treatment capabilities. Cerebrovascular imaging to detect the aneurysm should be arranged after discussion with the treating neurosurgeon. Emergency clinicians should address acute management issues including basic cardiorespiratory and neurologic monitoring, nimodipine to improve outcomes related to vasospasm, and supportive care.

Case Conclusion

The middle-aged woman who seemed so uncomfortable initially felt much better after receiving 1 g of acetaminophen. You and she were reassured by this, but patients with SAH can have pain that resolves spontaneously or is alleviated with medication. At this point, she wanted to go home. Regardless of the improvement in her symptoms, a patient with suspected SAH should have an LP because of the limitations of a CT scan. After a negative noncontrast CT, she could still have up to a 7% chance of having an SAH, so you did the right thing by explaining the need for her to stay for an LP. After the negative LP and CT, you appropriately sent her home with primary care follow-up for her benign headache. You can be confident about your decision since you have now reviewed the extensive prospective evidence to support that no cases of missed SAH have occurred with this strategy.

The other patient with migraine history described this episode as being worse than her usual migraines, and you were concerned because the patient said she usually sees flashing lights prior to headache onset and this time she had no aura. Diagnosing SAH in patients with primary headache disorders can be challenging, but changes in their usual pattern can be a clue. Your excellent history taking led you to work the patient up for SAH. You performed the correct diagnostic steps but initially had some question about the CSF results. Traumatic taps can occur in 10% of patients, and you found no guidelines for the minimum number of red cells needed to diagnose SAH by CSF analysis. Luckily, the laboratory technician was able to visualize xanthochromia, confirming the diagnosis. Based on these findings, you call for neurosurgical consultation and get a CTA to further assess for ruptured aneurysm. In coordination with neurosurgery, you begin acute supportive care in the ED. You place the patient on continuous monitoring and treat her pain as needed, watching for excessive BP elevations. You have frequent nursing checks to ensure no deterioration in the patient's clinical status. On CTA, she has a large aneurysm of the anterior communicating artery. She is ordered to have nothing by mouth except medications prior to definitive repair. While you are awaiting the patient's bed on the neurosciences ICU, you begin treatment with nimodipine, since you know that this calcium channel blocker improves outcomes in acute SAH. Shortly thereafter, the patient is transferred to the neurosciences ICU in stable condition to be cared for by a highly specialized team.

Cost-Effective Strategies

1. **In patients with suspected SAH, make sure to get an LP if the noncontrast CT is negative.** The cost of a missed SAH in terms of finances, clinical outcome, and possible litigation is much more than initial workup, as patients are often much sicker and have worse outcomes when they re-present after a rebleed.[12]

2. **Use the visual method to assess CSF for xanthochromia.** It is what is most widely available in U.S. laboratories and does not require a spectrophotometer. Also owing to the low specificity of spectrophotometry, false positives with that method could lead to unnecessary cost and risk of angiography or further testing.[128]

3. **Use nimodipine in confirmed cases of SAH.** It is cost-effective.[236]

4. **Transfer patients with SAH to regional hospitals with high-volume and specialized neuroscience ICU care.** It is worth the benefit in clinical outcome. Patients with SAH admitted from the ED to low-volume hospitals had 1.4 times the odds of dying in the hospital (95% CI, 1.2-1.6) as those admitted to high-volume hospitals.[208] It is also feasible and cost-effective to establish such regional centers.[237]

5. **Use teleradiology, if available, to discuss any questions about transfer patients with the nearest neurosurgical center.** Remote neuroradiology capability at neurosurgical centers may reduce costs by decreasing futile or unnecessary transfers.[238]

6. **Implementation of education programs may improve early diagnosis and outcomes of SAH.** A local teaching program to referring doctors in 1 prospective study was implemented at a low cost, reducing diagnostic error by 77% while improving overall management outcome.[239]

References

Evidence-based medicine requires a critical appraisal of the literature based on 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, are included in bold type following the reference, where available. In addition, the most informative references cited in this paper will be noted by an asterisk (*) next to the number of the reference.

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CME Questions

1. Among those ED patients with sudden-onset, severe headache and normal neurologic examinations, what percent will have subarachnoid hemorrhage?

a. 1%
b. 10%
c. 40%
d. 60%

2. Which of the following medications is given to improve outcomes related to vasospasm?
   a. Nimodipine
   b. Nitroprusside
   c. Phenytoin
   d. Aspirin

3. All of the following are proven risk factors for nontraumatic SAH EXCEPT?
   a. Smoking
   b. Polycystic kidney disease
   c. Hypertension
   d. Obesity
   e. Age > 85

4. Which symptom is pathognomonic for SAH?
   a. Sudden-onset, severe headache
   b. Vomiting
   c. Neck stiffness
   d. One symptom alone cannot be used to diagnose SAH

5. What is the first diagnostic test that should be obtained in suspected cases of SAH in the ED?
   a. LP
   b. Complete blood count
   c. Noncontrast head CT
   d. Cerebral angiography

6. How many RBCs in the CSF are required to make the diagnosis of SAH?
   a. 50
   b. 500
   c. 1000
   d. 5000
   e. No specific cutoff value exists

7. After an SAH is diagnosed in the ED, what is the next step in management?
   a. Admit to medicine
   b. Obtain a neurosurgical consultation.
   c. MRI
   d. Intubation

Physician CME Information

Date of Original Release: July 1, 2009. Date of most recent review: April 27, 2009. Termination date: July 1, 2012.

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals & Objectives: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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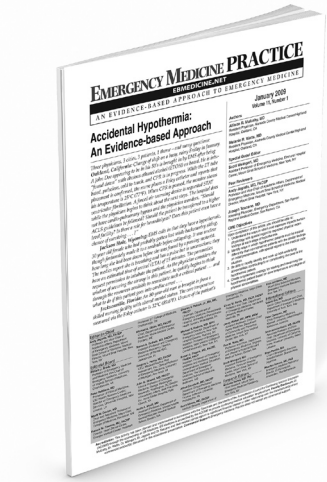

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Evidence-Based Approach To Diagnosis And Management Of Aneurysmal Subarachnoid Hemorrhage In The Emergency Department

Thomas LE, Edlow J, Goldstein JN. July 2009; Volume 11, Number 7

This issue of Emergency Medicine Practice focuses on the challenge of diagnosing and managing SAH, using the best available evidence from the literature. For a more detailed discussion of this topic, including figures and tables, clinical pathways, and other considerations not noted here, please see the complete issue at www.ebmedicine.net/topics.

Key Points	Comments
Evaluate all patients with classic "worst-of-life" sudden-onset headache as well as those with suspicious changes in usual headache history for SAH.	Only 10% to 16% of patients who present with worst-of-life headache will have serious pathology such as SAH.[5-10] A thorough clinical history and physical examination will help determine which patients require more rigorous diagnostic evaluation.
Begin the diagnosis with a noncontrast head CT. If the head CT is negative, perform LP.[8,10,19,20,63,90,92,101,102]	Both noncontrast CT and LP have limitations in interpretation. Alert patients with SAH are more likely to have negative CT scans than are patients with neurologic deficits.[31,89]
If patients suspected of having SAH have a fully negative workup, including negative CT and negative LP, they can be safely discharged.[19]	There is sufficient prospective evidence (Class II, Level B recommendation by the American College of Emergency Physicians) showing that there are no missed cases of SAH with this strategy.[19] Angiography or other additional testing is not needed, but outpatient follow-up should be arranged.
Once SAH is diagnosed, collaborate with neurosurgical colleagues and perform cerebral angiography to detect the underlying cerebral aneurysm.[20,21]	This is a Class I, Level B recommendation from the American Heart Association. Consider magnetic resonance angiography and computed tomographic angiography when conventional angiography cannot be performed in a timely fashion.[20]
Transfer patients with SAH early to a high-volume hospital with advanced endovascular, neurosurgical, and ICU services.[20,21,27]	Even with transfer to specialized, neurosurgical ICU care in high-volume centers, in-hospital mortality after SAH is still > 30%.[207,208] Outcome in surviving patients depends on several factors, including age, grade at time of presentation, comorbidities, and perioperative complications during hospitalization.[31,209]
Perform continuous cardiorespiratory monitoring, including blood pressure monitoring, in patients with SAH.	There are no set protocols for specific management of blood pressure in SAH. Elevated blood pressure should be controlled to balance the risk of stroke and rebleeding as well as to maintain cerebral perfusion pressure.[20,25] This is a Class II, Level B recommendation from the American Heart Association.[20,25]
Give oral nimodipine to patients with SAH.[20,21]	This is a Class I, Level A recommendation from the American Heart Association[20,21] and has been proven to decrease poor outcome from vasospasm. If a patient is unable to tolerate oral intake, the nimodipine should be crushed and given by nasogastric tube.
Discuss the need for anticonvulsant treatment with the treating neurosurgeons.	Prophylactic anticonvulsant therapy in the immediate posthemorrhage period is a Class III, Level B recommendation from the American Heart Association and may be considered useful in some patients.[20,21,25]
Reverse coagulopathy in anticoagulated patients by providing emergent clotting factor and vitamin K.[25,212-215]	Rapid reversal is recommended to prevent hematoma expansion, regardless of indication for anticoagulation.

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3.	[a]	[b]	[c]	[d]	[e]
4.	[a]	[b]	[c]	[d]	[e]
5.	[a]	[b]	[c]	[d]	[e]
6.	[a]	[b]	[c]	[d]	[e]
7.	[a]	[b]	[c]	[d]	[e]
8.	[a]	[b]	[c]	[d]	[e]
9.	[a]	[b]	[c]	[d]	[e]
10.	[a]	[b]	[c]	[d]	[e]
11.	[a]	[b]	[c]	[d]	[e]
12.	[a]	[b]	[c]	[d]	[e]
13.	[a]	[b]	[c]	[d]	[e]
14.	[a]	[b]	[c]	[d]	[e]
15.	[a]	[b]	[c]	[d]	[e]
16.	[a]	[b]	[c]	[d]	[e]

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