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

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Abstract

Aneurysmal subarachnoid hemorrhage accounts for a small percentage of strokes, but it is a significant contributor to the morbidity rate. The diagnosis is challenging and has devastating consequences if it is missed. Accurate initial diagnosis and management are critical to the outcome of the disease. The emergency clinician must have a high index of suspicion and a judicious approach to evaluating the chief complaint of patients with spontaneous subarachnoid hemorrhage (ie, headache). This review evaluates the literature and current evidence, including controversies and recent American Heart Association guidelines, to support a best-practice approach to the diagnosis and treatment of patients with spontaneous subarachnoid hemorrhage.

Case Presentations

You walk into a crowded evening shift in the ED, and your first patient is a middle-aged woman clutching her head in her hands, complaining of the "worst headache of her life." You are worried about a subarachnoid hemorrhage. After treating her pain, you order a noncontrast head 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? She eventually agrees to stay for a lumbar puncture, 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 a spontaneous subarachnoid hemorrhage? After further history is obtained, you are concerned about a subarachnoid hemorrhage and you obtain a CT, which is normal. You perform a lumbar puncture, which shows some clearing of red blood cells from tube 1 to tube 4. 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 spontaneous subarachnoid hemorrhage. After calling for neurosurgical consultation, what else should you do in the ED to treat this patient?

Introduction

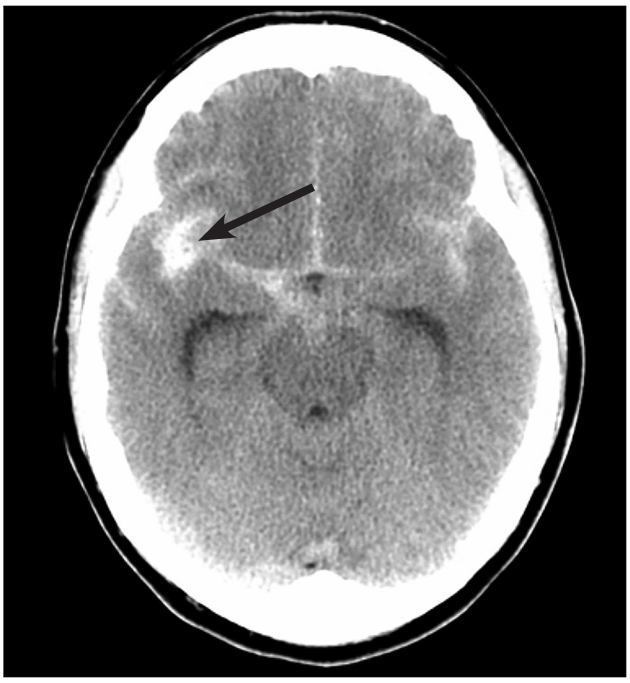
Subarachnoid hemorrhage (SAH) is the extravasation of blood into the cerebrospinal fluid (CSF). It is usually a diffuse process that results from rupture of corticomeningeal vessels and from hemorrhagic contusions of the brain.[1] Trauma is the most common cause of all SAH; however, the majority (85%) of nontraumatic, or spontaneous SAH (sSAH), the focus of this article, are related to aneurysm rupture.[2-4] Aneurysmal SAH (aSAH) and other forms of sSAH can pose diagnostic challenges in the emergency department (ED).

Distinguishing traumatic SAH from sSAH may be difficult in some cases because the trauma may have been unwitnessed; however, this distinction is important. **See Figure 1** for the typical appearance of a sSAH on noncontrast head computed tomography (CT). The emergency clinician must be able to quickly and accurately identify and categorize SAH, and should be aware of the secondary complications that affect both the central nervous system and other major organs. Initial management and treatment decisions should be made to minimize effects of the initial neurologic injury. This issue of *Emergency Medicine Practice* focuses on the diagnostic challenges, the initial management and treatment options, and some of the more severe complications of sSAH, using the best available evidence from the literature.

Critical Appraisal Of The Literature

The overall incidence of sSAH is relatively low and, therefore, the availability of high-quality evidence is limited. A literature search was performed using Ovid MEDLINE® and PubMed from 1950 to May 2014. 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 500 articles were reviewed, which provided background for further literature review. During the literature review process, the highest value was placed on 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 statements and case reports were reviewed. The Cochrane Database of Systematic Reviews and the National Guideline Clearinghouse (www.guideline.gov) were also consulted.

Figure 1. Subarachnoid Hemorrhage On Noncontrast Head Computed Tomography



Note diffuse hyperdense subarachnoid blood surrounding the basal cisterns and extending into the Sylvian fissures bilaterally (arrow).
Image courtesy of Lisa Thomas, MD.

The most relevant guidelines for emergency clinicians are the 2008 American College of Emergency Physicians (ACEP) Clinical Policy on acute headache **(see Table 1)**,[5] the 2012 American Heart Association (AHA) Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage,[6] **(see Table 2)** and the 2011 Neurocritical Care Society (NCS) Guidelines on the critical care management of patients with aSAH.[7]

Table 1. 2008 ACEP Clinical Policy On Acute Headache (Subarachnoid Hemorrhage)[5]

Recommendation	Strength of Recommendation
• Emergent head CT is the initial diagnostic study recommended in the diagnosis of any new, sudden-onset, severe headache or suspected case of SAH.[7-11]	B
• Lumbar puncture is recommended for patients with suspected SAH after negative noncontrast head CT.[9,12]	
• Angiography is not recommended in patients with sudden-onset, severe headache who have negative findings on head CT, normal opening pressure, and negative CSF findings.	
• Patients with a negative workup including negative CT and LP can be safely discharged from the ED with outpatient follow-up recommended.	
• Response to analgesia should not be used as the sole indicator to the etiology of an acute headache.	C

Abbreviations: ACEP, American College of Emergency Physicians; aSAH, aneurysmal subarachnoid hemorrhage; CSF, cerebrospinal fluid; CT, computed tomography; DSA, digital subtraction angiography; ED, emergency department; EVD, external ventricular drain; LP, lumbar puncture; SAH, subarachnoid hemorrhage.

Table 2. 2012 AHA Guidelines For Management Of Aneurysmal Subarachnoid Hemorrhage[6]

Recommendation	Classification of Recommendation
Diagnosis of Aneurysmal Subarachnoid Hemorrhage	
• aSAH is a medical emergency that is frequently misdiagnosed. A high level of suspicion for aSAH should exist in patients with acute onset of severe headache.	B
• Acute diagnostic workup should include noncontrast head CT, which, if nondiagnostic, should be followed by lumbar puncture.	
• CTA may be considered in the workup of aSAH. If an aneurysm is detected by CTA, this study may help guide the decision for type of aneurysm repair, but if CTA is inconclusive, DSA is still recommended (except possibly in the instance of classic perimesencephalic aSAH).	C
• MRI (fluid-attenuated inversion recovery, proton density, diffusion-weighted imaging, and gradient echo sequences) may be reasonable for the diagnosis of aSAH in patients with a nondiagnostic CT scan, although a negative result does not obviate the need for CSF analysis.	
Management of Aneurysmal Subarachnoid Hemorrhage	
• Between the time of aSAH symptom onset and aneurysm obliteration, blood pressure should be controlled with a titratable agent to balance the risk of stroke, hypertension-related rebleeding, and maintenance of cerebral perfusion pressure.	B
• aSAH-associated acute symptomatic hydrocephalus should be managed by cerebrospinal fluid diversion (EVD or lumbar drainage, depending on the clinical scenario).	
• For patients with an unavoidable delay in obliteration of aneurysm, a significant risk of rebleeding, and no compelling medical contraindications, short-term (< 72 h) therapy with tranexamic acid or aminocaproic acid is reasonable to reduce the risk of early aneurysm rebleeding.	
• The magnitude of blood pressure control to reduce the risk of rebleeding has not been established, but a decrease in systolic blood pressure to < 160 mm Hg is reasonable.	C
Patient Transfer	
Low-volume hospitals (eg, < 10 aSAH cases per year) should consider early transfer of patients with aSAH to high-volume centers (eg, > 35 aSAH cases per year) with experienced cerebrovascular surgeons, endovascular specialists, and multidisciplinary neurointensive care services.	B

Abbreviations: AHA, American Heart Association; aSAH, aneurysmal subarachnoid hemorrhage; CSF, cerebrospinal fluid; CT, computed tomography; DSA, digital subtraction angiography; EVD, external ventricular drain; LP, lumbar puncture; MRI, magnetic resonance imaging; SAH, subarachnoid hemorrhage.

Etiology And Pathophysiology

Incidence

The international incidence of SAH varies significantly according to geographic region, ranging from 2 to 27 cases per 100,000 people per year, with an international aggregate of approximately 10 per 100,000 people per year, which is close to the United States incidence.[13-15] Modifiable risk factors include cigarette smoking, hypertension, heavy alcohol use, cocaine abuse, caffeine use, and smokeless nicotine uptake; only hypertension and smoking are covered extensively in the literature.[16-19] Nonmodifiable causes include family history of aSAH in a first-degree relative, genetic disorders (eg, autosomal dominant polycystic kidney, sickle cell disease, Ehlers-Danlos syndrome, and Alpha-1-antitrypsin), and female sex.[3,20-22] The risk posed by female sex varies according to parity, age of menarche, and menopause. Women who are primiparous, menopausal, or who began menstruation early have been found to have a higher risk of aSAH.[3,23-26]

Etiology

In a retrospective study comparing 99 traumatic SAH patients with 114 aSAH patients, traumatic cases were noted to have more diffuse hemorrhage, earlier resolution of bleeding, and decreased rates of delayed complication,[1] illustrating mechanistic differences in the onset and evolution of aSAH and the need for distinct management. Owing to the poorer prognosis of aSAH and the need for surgical or endovascular repair of the underlying aneurysm, ambiguous traumatic cases should receive additional imaging to rule out aneurysm as the cause. Rupture of aneurysms located on intracranial vessels at the base of the brain account for 85% of sSAH cases. The majority of aneurysms are in the anterior circulation, with the remainder in the posterior circulation. Aneurysms of the spinal arteries or other vascular malformations in the spinal cord can also result in sSAH.[20]

A minority of sSAH cases (10%) are categorized as nonaneurysmal perimesencephalic hemorrhage, a distinct form of sSAH with unclear etiology defined by a specific pattern of extravasated blood around the midbrain with normal angiogram. **(See Figure 2.)** Venous anomalies are thought to contribute to the pathophysiology.[27,28] Patients with this form of SAH have a better prognosis collectively, less devastating presentations, and fewer clinical complications when compared with aSAH patients during their initial hospital course.

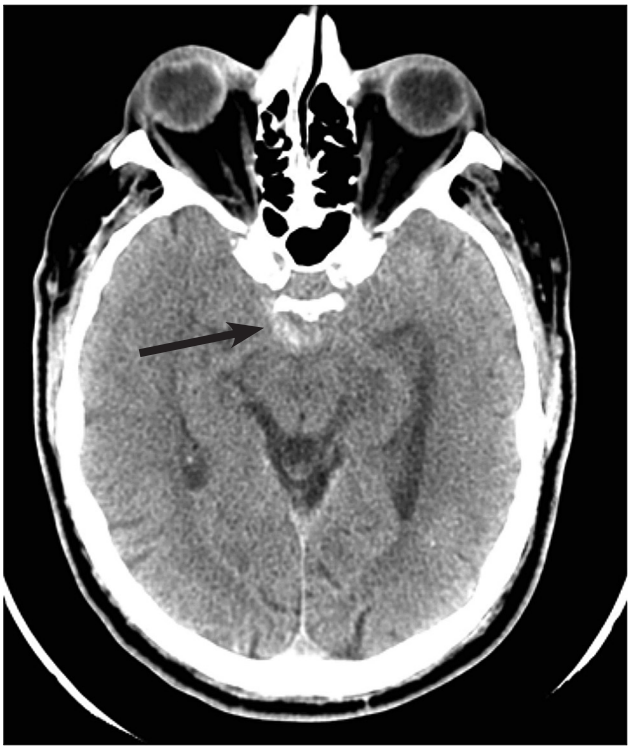
Digital subtraction angiography (DSA) is considered the diagnostic test of choice.[29] Angiogram-negative SAH with a diffuse hemorrhage pattern may have more significant morbidity, despite the failure to identify an aneurysm (due to thrombosis or large hemorrhage volume).[30,31] Noncontrast CT scan is not sufficient to identify these patients even when there are characteristic perimesencephalic bleeding patterns.[32] These patients should all get a DSA. Other causes of sSAH are listed in **Figure 3, page 5**.[4,33,34]

Reversible cerebral vasoconstriction syndrome (RCVS) is a rare but increasingly identified etiology of sSAH. RCVS represents a heterogeneous group of small vessel vasculopathy that can result in ischemic or hemorrhagic stroke.

Pathophysiology Of Aneurysms

It was previously thought that congenital defects in the tunica media at arterial bifurcations were the source of aneurysm formation. This has since been refuted and current beliefs hold that the mechanism for aneurysm formation is acquired, and that aneurysms develop gradually over one's lifetime due to interactions with modifiable and genetic factors.[35,36] In a systematic review of 23 studies that included over 56,000 patients, the prevalence of unruptured aneurysms in the general population was 2.3%. The rates varied from 0.4% in autopsy studies to 6% in angiographic studies.[37] Polycystic kidney disease, familial predisposition, hypertension, atherosclerosis, smoking, excessive alcohol consumption (> 150

Figure 2. Perimesencephalic Hemorrhage On Noncontrast Head Computed Tomography



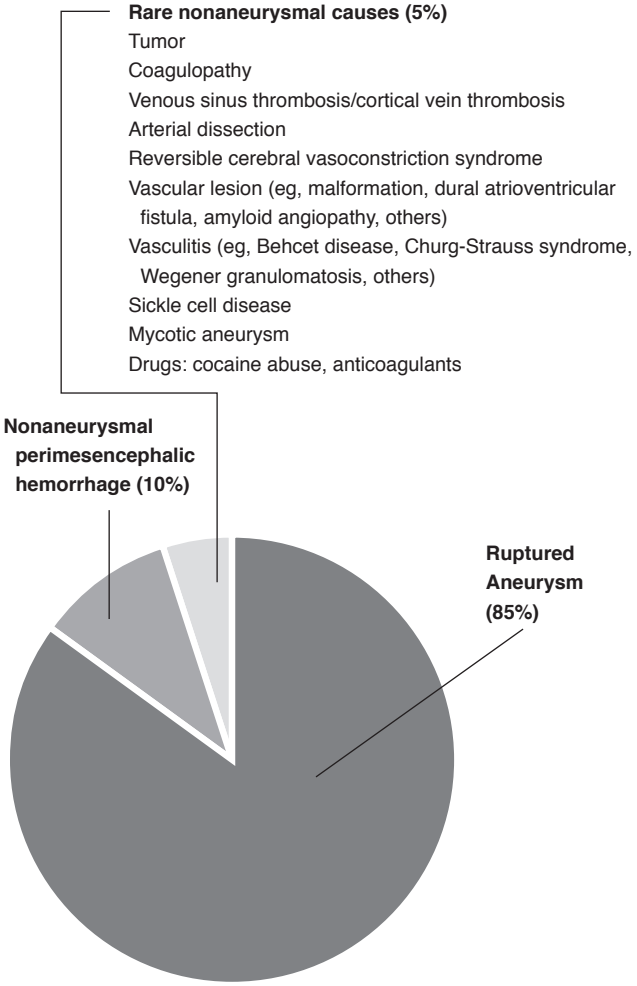
A characteristic pattern of hyperdense blood anterior to the midbrain is seen (arrow). No aneurysm was found on follow-up angiogram. Image courtesy of Lisa Thomas, MD.

g/wk), age > 85 years, oral contraceptive use, and connective tissue disorders have all been associated with aSAH.

An understanding of the pathophysiology associated with aneurysm rupture is still evolving. Clinical predictive markers for aneurysm rupture include size, location, and previous history of an aneurysm.[38] There are some limited data emerging that identify the role of inflammatory markers[39] and genetic factors such as specific polymorphisms in the endothelial nitric oxide synthase gene (found more often in patients who rupture).[40] Genetic factors have been shown to influence the location, multiplicity, and size of the aneurysms.[41] Factors such as female sex and white non-Hispanic race significantly contributed to the presence of multiple intracranial aneurysms, when compared with those without genetic predispositions.[41] Rupture risk increases with the size of the aneurysm and, based on a systematic review of 9 prospective studies following aneurysms for 3907 patient-years, most aneurysms never rupture. Aneurysms < 10 mm in size have an annual risk of rupture of 0.7%;[19] larger aneurysms are 5 times more likely to rupture.[37,42] Some risk factors predispose patients to ruptured aneurysms at a particular location. Within the subset of patients with aSAH, younger patients are more likely to have aneurysms of the middle cerebral artery, women are more likely to present with posterior communicating artery aneurysms, and alcoholics are more likely to present with basilar artery aneurysms.[43]

While it is commonly thought that Valsalva maneuvers or physical exertion lead to aneurysm rupture, this is only associated with a small subset of patients.[44-47] A recent large systematic review concluded that there is insufficient evidence to link exertion to aneurysm rupture.[48]

Figure 3. Causes Of Spontaneous Subarachnoid Hemorrhage



Subarachnoid Hemorrhage Clinical Severity Scales

There are multiple clinical scales to assess the severity of aSAH. **(See Table 3, page 6.)** The 2 most widely used are the Hunt and Hess scale[49] and the World Federation of Neurological Surgeons (WFNS) scale,[50] with the latter primarily used in the research community. The Hunt and Hess and WFNS scales correlate with mortality; the higher the score, the higher the inpatient mortality. The WFNS scale has a higher interobserver correlation but is still less utilized clinically.[51]

A third scale, the Fisher Grade, uses quantity of blood on CT appearance to predict the risk of symptomatic cerebral vasospasm, which is currently a relatively common complication of aSAH.[52] The original Fisher Grade was modified by Frontera et al to more accurately reflect the contribution of intraventricular hemorrhage.[53] Newer scales are being developed with the goal of using hemorrhage volume to more accurately predict vasospasm than the Fisher Grade.[54-56]

Differential Diagnosis

Headache is the primary complaint of patients who present to the ED with sSAH. It is a relatively common complaint that represents approximately 2% of all ED visits, with only 1% to 3% of these complaints attributable to sSAH.[57-60] Although sudden severe headache is the classic presentation, patients with a SAH may present with a vague headache and a normal neurologic examination.[61-66] Several reports suggest that the misdiagnosis rate on initial ED visit is as high as 5%.[67-71] While the case fatality rate has been gradually decreasing by approximately 0.5% per year[72] as management techniques have improved, patients with missed or delayed diagnoses have worse outcomes.[68-70,73] Overall, SAH mortality is approximately 40% at 1 week, with 10% to 15% of deaths occurring prehospital and 25% occurring within 24 hours of initial bleeding.[72,74-76]

As many as 10% to 16% of patients presenting with sudden-onset, intense, severe headaches that peak in about 60 seconds (sometimes referred to as "thunderclap" headaches) and normal neurologic function on physical examination will have sSAH.[61-66] The differential diagnosis is quite broad and includes both benign and serious etiologies, as listed in **Table 4**.[2,63] The emergency clinician must rapidly narrow the diagnosis with further history, physical examination, or diagnostic testing.

Prehospital Care

Patients with acute headache vary in presentation and severity, which can make initial assessment challenging. 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 neurological examination should be performed to assess for obvious focal neurologic deficit, and a formal stroke scale should be used to assess severity of symptoms.[77] Patients with focal neurological findings and suspected stroke

Table 3. Subarachnoid Hemorrhage Grading Scales

Hunt and Hess Severity Scale[49]

- 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

World Federation of Neurological Surgeons[50]

- Grade 1 – GCS score 15, no motor deficit
- Grade 2 – GCS score 13-14, no motor deficit
- Grade 3 – GCS score 13-14, motor deficit present
- Grade 4 – GCS score 7-12, motor deficit may be present or absent
- Grade 5 – GCS score 3-6, motor deficit may be present or absent

Fisher Scale[52] (CT Appearance)

- Group 1 – No blood
- Group 2 – Diffuse deposits of SAH 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 SAH, but intracerebral or intraventricular clot

Modified Fisher Scale[53]

- Grade 0 – No SAH; no intraventricular blood
- Grade 1 – Minimal or thin subarachnoid blood but no intraventricular blood
- Grade 2 – Minimal or thin subarachnoid blood with intraventricular blood
- Grade 3 – Thick or diffuse subarachnoid blood but no intraventricular blood
- Grade 4 – Thick or diffuse subarachnoid blood with intraventricular blood

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Scale; SAH, subarachnoid hemorrhage.

Table 4. Differential Diagnosis Of Sudden-Onset Nontraumatic Headache

Common Benign Causes

- Migraine
- Tension-type headache
- Coital headache
- Exertional headache
- Benign cough headache
- Sinusitis

"Cannot Miss" Causes	Suggestive History and Physical Findings
Subarachnoid hemorrhage	Abrupt headache, stiff neck, third nerve palsy
Hypertensive encephalopathy	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)	Obesity, female sex, papilledema, possible sixth nerve palsy
Cerebral venous and dural sinus thrombosis	Hypercoagulable state of any type, including pregnancy
Meningitis or encephalitis	Fever, stiff neck, altered mental status, seizure
Temporal arteritis/central nervous system vasculitis	Usually aged > 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 ischemic stroke	Abrupt onset and focal neurological deficit conforming to an arterial territory
Reversible vasoconstriction syndrome (Call-Fleming syndrome)	Recurrent sudden-onset headaches with nausea, vomiting, photophobia, confusion, blurred vision
Pituitary apoplexy	Visual acuity or field abnormalities, known pituitary tumor
Mass lesions, including: • Tumor • Abscess • Parameningeal infection • Intracranial hemorrhage • Colloid cyst	Any neurological finding, focal or generalized

should be transported rapidly to a hospital in accordance with local emergency medical services (EMS) protocols.[77] If the patient qualifies for time-sensitive acute stroke treatment, both the time of symptom onset and the time that the patient was last seen normal should be ascertained. Reversible causes of depressed mental status should be considered (ie, hypoglycemia and opioid ingestion/abuse). Consider administering dextrose 50% (D50) for suspected hypoglycemia and naloxone for opioid overdose. There are limited data specific to the prehospital care of patients with sSAH, 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 sSAH or any other serious neurological disease, they do not necessarily require initial transport to a comprehensive stroke center.[62,78,79]

Emergency Department Evaluation

Whom To Evaluate?

The patient presenting with the classically described acute onset of severe headache that is described as "worst ever," with a time to peak of seconds and associated nausea, vomiting, and focal deficits is typically a candidate for sSAH workup. 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. Most worst-of-life headaches treated in the ED will have benign causes. Approximately 10% to 16% will have serious pathology, including but not limited to sSAH.[61-66]

History

A thorough history should be elicited in patients who present with a suspected sSAH. The history should include timing of symptoms, any anticoagulant medications, family history of aneurysms, and social history of smoking. Seventy percent of patients referred to a hospital with SAH presented with headache alone, without focal symptoms, as reported in a study by Harling et al.[80] There is variability in the incidence of sSAH in patients with isolated headaches ranging from 11%[63] to 70%, which 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.[63,65,81,82] The term *thunderclap headache*, first described by Day and Raskin in 1986, refers to an acute severe headache with rapid onset, which reaches peak intensity within seconds.[83] Although a thunderclap headache is often considered the classic manifestation of a ruptured aneurysm, it is neither sensitive nor specific. The evaluation by Landtblom et al of 137 patients with a thunderclap headache demonstrated a 17% incidence of SAH, with the majority of patients having favorable outcomes.[63] Approximately 85% of patients with a thunderclap headache may have other etiologies. These latter patients, with so-called benign thunderclap headache, have excellent outcomes, and their conditions are often later diagnosed as migraine or other primary headache syndromes.[61-66]

The International Cooperative Study on the Timing of Aneurysm Surgery, a large prospective multicenter study of 3521 patients, found a normal level of consciousness in 75% of patients with aSAH.[84] Transient loss or alteration of consciousness has been reported in about one-fourth of patients in other smaller prospective studies.[63,82] Vomiting is a nonspecific feature associated with 70% of aSAH patients.[65,82] It is also seen in half of all benign cases of thunderclap headache and is quite common in migraine.[63,82] Focal symptoms, seizures, or double vision may be reported and should alert the emergency clinician to consider sSAH or other serious pathology, as these symptoms are rare in headaches with benign causes.[82] Neck stiffness may be another clue to more serious pathology; in 1 prospective study, it was reported in 60% of 23 patients with SAH, compared with only 10% of 114 patients with benign thunderclap headache.

Table 5 summarizes the key pieces of information that an emergency clinician should obtain during careful history taking to help identify sSAH. 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.[65,82] This point is important because emergency clinicians cannot rely upon how well the patient looks in estimating pretest probability of sSAH.[67-70] For example, one retrospective study of over 200 patients who underwent aneurysmal clipping reported that 8% did not have any headache at onset of sSAH, but instead presented with sudden onset of general malaise or isolated neck or back complaints.[85]

Table 5. Key Questions For Patients With Acute Headache

Onset	Was the onset of the headache abrupt?
Severity	Is it the worst headache ever? "10 out of 10?"
Quality	How does this headache compare with prior episodes? Is it distinct or unique for this patient?
Associated symptoms	Are there focal signs (syncope, seizure, neck stiffness, or double vision)?

Table 6 describes some distracting clinical scenarios in evaluating for sSAH.

Physical Examination

As always, the physical examination begins with assessment of the patient's vital signs and general appearance, as well as airway, breathing, and circulation. Once the patient is stabilized, a focused physical examination that includes a relevant neurologic examination should be performed. In patients who are comatose or otherwise unable to give a cogent history, ophthalmoscopic examination may reveal retinal hemorrhages, which is an important diagnostic clue seen in approximately 10% of all patients with sSAH.[86] 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.[58] Abnormal physical examination findings that may be associated with aSAH are listed in **Table 7**. Patterns of focal deficits sometimes suggest the location of the offending aneurysm.

Clinical Decision Rules

A large 2013 multicenter observational study, the Ottawa Subarachnoid Hemorrhage Rule, developed and validated a clinical decision rule for sSAH. The multicenter Canadian study included 10 hospitals and 2131 patients. The methodology involved 3 candidate rules that were assessed post hoc for interobserver reliability, sensitivity, and specificity. The primary outcome of sSAH was defined by noncontrast head CT or xanthochromia. The authors concluded that, in patients who present within 1 hour of their headache's maximal intensity, the clinical decision rule could exclude sSAH with a sensitivity of 98.5% and a specificity of 27.5%.[101] The rule is shown in **Table 8, page 9**. This rule has been criticized for its low interobserver reliability rate, and the one attempt made at external validation of the rule was unsuccessful.[102] In addition, Matloob et al looked at a retrospective application of the rule and found that it would lead to an increase in evaluation in their cohort.[103] However, the rule is instructive because it highlighted the features of the history and physical examination that may be the most useful in differentiating acute SAH.

Table 6. Associated Findings That May Distract From Diagnosis Of Subarachnoid Hemorrhage[2]

- **Relief with pain medications:** Headache may improve spontaneously or with analgesics.[87-92]
- **Absence of headache**[85]
- **Nonspecific viral symptoms:** Combination of headache with fever, neck pain, nausea, or vomiting may be incorrectly attributed to a viral syndrome.[68,73,94,96]
- **Musculoskeletal pain:** Prominent neck pain may be attributed to arthritis or musculoskeletal diagnoses.[68,73,93,94]
- **Altered behavior or mental status:** SAH may present with delirium, acute confusional state, or psychosis.[95,96]
- **Head injury from syncope:** Transient loss of consciousness due to aneurysm rupture may lead to a focus on the resulting traumatic head injury, thus disguising the preceding sSAH.[97] Subarachnoid blood from aneurysms is typically around the basal cisterns* while that from trauma tends to be higher in the convexities† or in areas of coup and contrecoup forces.[71]
- **Cardiac abnormalities:** Electrocardiogram abnormalities and elevated cardiac markers may be present.[68,73,93,94,98,99]
- **Concomitant hypertension:** Excessive focus on blood pressure may lead to diagnosis of hypertensive urgency or emergency.[68,73,93,94,98,99]
- **Cardiac arrest:** SAH may be associated with cardiac arrest (reported in 3.6% of patients in one retrospective study).[100]

*See Figure 1, page 2.
†See Figure 4, page 10.
Abbreviations: SAH, subarachnoid hemorrhage; sSAH, spontaneous subarachnoid hemorrhage.

Table 7. Physical Examination Findings Associated With Aneurysmal Subarachnoid Hemorrhage

Finding	Likely Location of Aneurysm
Mental status change • Seen in about 25% of SAH patients[63,82,104]	Any
Meningismus • Seen in 60% of SAH patients[63] • Takes 3-12 h to develop and may not be appreciated in comatose patients[105]	Any
Third nerve palsy[34,106] • 90% of patients with third nerve palsy due to aneurysm (versus other causes) have anisocoria > 2 mm	Posterior communicating artery
Sixth nerve palsy[107,108] • Presents 3-14 days after onset of SAH • Associated with higher clot burden • Resolves gradually	Any (due to increased intracranial pressure)
Bilateral leg weakness, abulia[3,71]	Anterior communicating artery
Nystagmus, ataxia, dizziness[3,71]	Posterior circulation
Hemiparesis with aphasia or neglect[3,71]	Middle cerebral artery
Subhyaloid (retinal) hemorrhage (Terson syndrome)[86] • Seen in about 10% of SAH patients • Associated with worse clinical grades on presentation and poorer prognosis	Any

Abbreviation: SAH, subarachnoid hemorrhage.

Diagnostic Studies

There is no single test with high enough sensitivity and specificity for effectively diagnosing or ruling out sSAH. Effective diagnosis is best accomplished in a stepwise manner. See the **Clinical Pathway For Emergency Evaluation Of Suspected Subarachnoid Hemorrhage** on page 16.

Noncontrast Computed Tomography

The initial investigative study in patients with suspected sSAH is the noncontrast head CT.[5] Although CT has great accuracy in detecting subarachnoid blood, the emergency clinician must understand its limitations, as summarized in **Table 9**. Most importantly, CT findings are time-dependent. There is a decrement in the identification of hemorrhage on the scan as time from symptom onset increases because blood is degraded and diluted by the continuous circulation of CSF.[109] The largest prospective observational study supporting this finding used data collected during the 1980s from over 3500 patients presenting with sSAH at various times from symptom onset using second-generation scanners; CT had a 92% sensitivity on the day of aneurysm rupture and sensitivity progressively decreased to 86%, 76%, and 58% on days 1, 2, and 5 postrupture, respectively.[84] 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[64,110-112] and decreasing sensitivity after just 12 hours.[113] Newer fifth-generation scanners report sensitivities ranging from 93% to 100% if patients are scanned within 24 hours; however, these results are also based on retrospective studies and relatively small numbers of patients.[114-116]

Third-generation CT scanners may provide 100% sensitivity for sSAH patients presenting within 6 hours. Two retrospective studies and 1 prospective study report that CT scan alone may be sufficient in ruling out SAH in patients who present within 6 hours of ictus.[113,117,118] The largest of the studies was a prospective trial with a primary end point of developing a clinical decision rule for sSAH patients; a secondary outcome was the utility of CT scan within 6 hours of clinical symptoms.[113] There were over 3000 patients in this multicenter study, which concluded that lumbar puncture (LP) did not add to clinical decision making. The patients in this study presented with their worst headache ever; 121 of these patients underwent CT scan within 6 hours. The sensitivity and specificity for sSAH in this cohort was 100% In contrast, a case-control study designed to assess the usefulness of CT within 6 hours (as well as to validate the Ottawa SAH rule) found a 20% missed SAH diagnosis rate in their cohort.[118] The case-control methodology limited sensitivity and specificity reporting. The editorial comments to this study detail the arguments in the debate over the value of LP in negative early CT; however, limited clarity leads many experts to recommend additional evaluation with a normal CT.[119] All studies involved had significant limitations and the current data do not provide enough information to warrant a change in the recommendations. The 2012 AHA guidelines still recommend LP after negative CT scan. More compelling data may change that recommendation in the future.

In addition, there are special clinical scenarios that may impact the technical diagnostic accuracy of the CT scan. Patients with normal neurologic examinations and smaller hemorrhage volumes may not have CT evidence of sSAH.[84,112, 120] Patients with anemia and hematocrit < 30% may have hemorrhage that is overlooked on the scan because their blood may be isodense with the CSF.[120,121] Other considerations include experience of the interpreter[122] and technical factors, such as quality of the scanner and artifacts of bone or motion that may limit the study.[2]

Table 8. Ottawa Subarachnoid Hemorrhage Rules

Investigate if at least 1 high-risk variable is present:

- Age ≥ 40
- Neck pain or stiffness
- Witnessed loss of consciousness
- Onset during exertion
- Thunderclap headache (instantly peaking pain)
- Limited neck flexion on examination

For alert patients (Glasgow Coma Scale score of 15) aged >15 y with new, severe, nontraumatic headache reaching maximum intensity within 1 h. Not for patients with new neurological deficits, previous aneurysms, subarachnoid hemorrhage, brain tumors, hydrocephalus, history of recurrent headaches (≥ 3 episodes over the course of ≥ 6 months), or papilledema on fundoscopic examination.

Table 9. Limitations Of Computed Tomography[2]

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

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

The location of blood or the hemorrhage pattern on CT can be useful in predicting the presence and location of an aneurysm (whether traumatic or spontaneous) and the likelihood of an angiogram-negative SAH. Aneurysms of the anterior communicating artery, posterior communicating artery, and middle cerebral artery comprise approximately 70% of aneurysms.[76] Blood from a ruptured aneurysm is usually located around the basal cisterns **(see Figure 1, page 2)**, 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.[71] **(See Figure 4).** This distinction may be especially useful in patients with SAH who may have fallen because of syncope at onset. Finally, there is also an uncommon entity of spontaneous convexity SAH that is almost never aneurysmal.[121,123] Convexal SAH has been described in various case series with the etiologies believed to be related to reversible cerebral vasoconstriction syndrome (RCVS) in patients < 60 years of age and cerebral amyloid angiopathy in patients aged > 60 years.[124-126] This represents a cohort of nonaneurysmal SAH with diverse etiologies that may warrant different management approaches.

Lumbar Puncture

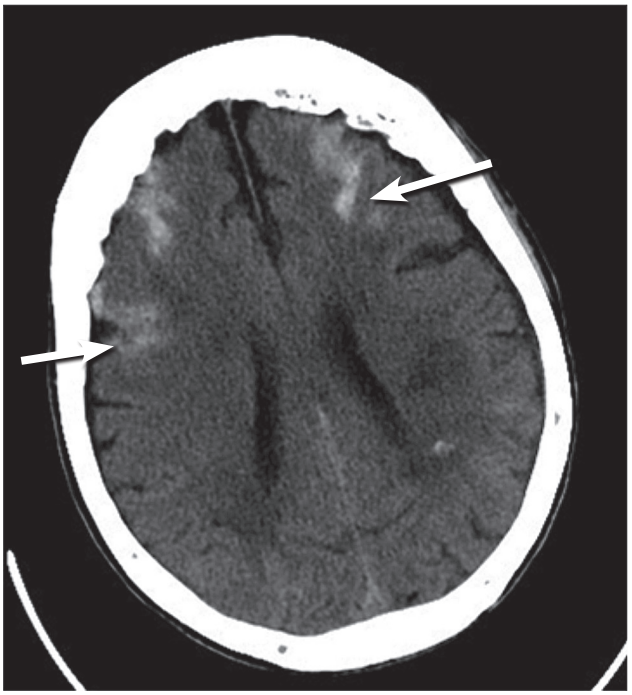
If findings on noncontrast CT are positive for sSAH, then the emergency clinician may shift the focus from diagnosis to treatment, and LP need not be performed. However, in patients with suspected sSAH with a negative noncontrast CT, LP is recommended.[5,7]

The current body of literature suggests that there may be a role for a CT-only approach in patients who present within 6 hours of headache onset. However, the data are not yet robust enough to alter the current recommendation. Given the life-threatening nature of this condition and the fact that CT is not 100% sensitive for SAH, LP is typically recommended to increase the sensitivity of the diagnostic algorithm. 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).[5] However, in several reports, only 25% to 50% of patients with CT-negative worst-of-life headache actually had LP performed by emergency clinicians.[64,127,128] Although patients and emergency clinicians may defer LP for various reasons (eg, time constraints, the patient's general appearance of good health, patient discomfort, and patient fear of complications), there is clear evidence that supports the value of LP as the next step in diagnosis.[64,66,81,112,115,129,130]

LP is especially critical in alert, neurologically normal patients with sudden-onset, severe headache. Alert patients with sSAH are more likely to have negative CT scans than patients with neurologic deficits.[84,111] This population poses a particular challenge for emergency clinicians, but it also has the greatest potential to benefit from early detection.

The best evidence supporting the value of LP comes from a 2007 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.[9] Another prospective study of 107 similar adult ED patients found a diagnosis of sSAH for 18 patients, 2 of which were missed by CT (2% miss rate but 95% confidence interval [CI] up to 8.8%).[64] Furthermore, a 1994 prospective study of 27 patients with acute headache found 9 with SAH, 5 of which were missed by CT.[81] Additional retrospective studies, each with more than 100 SAH patients, found 2% to 7% of cases were missed by CT but picked up by LP and confirmed by angiography.[112,115] 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.[129] The studies presented

Figure 4. 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 (page 2), subarachnoid hemorrhage due to trauma appears as hyperdensities in the convexities. No aneurysm was found on angiogram.
Image courtesy of Jonathan Edlow, MD.

used third-generation CT scanner technology with mean presentation times up to 60 hours from symptom onset.

Interpreting The Lumbar Puncture

Even if the decision to perform a LP is clear, interpretation of CSF results can still be challenging. **See Table 10** for limitations of using LP in the diagnosis of sSAH. 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[131,132]) or true intracranial bleeding.

Opening Pressure

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.[133-135] Opening pressure may be elevated by > 20 cm H_20 in 60% of patients with sSAH.[2] For accurate measurement of opening pressure, the patient must be in the lateral recumbent position. After initial fluid confirming location, the manometer is inserted and the column height is noted to measure the opening pressure.

Red Blood Cell Analysis

It is usual practice to collect 4 serial tubes of CSF and assess for constancy of red blood cells (RBCs). Regardless of the number of RBCs in the first 3 tubes, if there are zero RBCs in the final tube, this indicates a traumatic tap. 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 sSAH.[18,136] Interpretation becomes ambiguous when there is "clearing of red cells," or a fall in RBCs in serial tubes, without complete clearing down to 0.[137] This can occur in cases of aneurysmal hemorrhage, as well as in a traumatic tap, or even a combination of the 2.[138] Consider the case of a 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.[139] Recent analysis provides some guidance for differentiating traumatic lumbar puncture from sSAH, where the final LP tube RBC count and the percent change in RBC count were associated with sSAH.[140] In this small retrospective study, patients with more than 10,000 RBCs in the CSF were 6 times more likely than those with fewer than 100 RBCs to have SAH.[140] In a similar analysis, patients with bloody CSF and negative cerebrovascular imaging were found to be at low risk for subsequent SAH.[141] Further research to quantify and define bloody CSF may be needed. Although no specific guidelines exist for this situation, additional diagnostic steps may include performing computed tomography angiography (CTA)[142,143] or magnetic resonance angiography (MRA),[144,145] or obtaining conventional angiography in consultation with a neurosurgeon. Lastly, 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.[146]

Xanthochromia

Xanthochromia, literally meaning "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 hemoglobin to bilirubin only occurs in vivo and requires time to develop;[147] thus, the presence of xanthochromia is highly suggestive of sSAH.[2,139,148] Xanthochromia is unlikely with a traumatic tap, as experimental studies of the artificial addition of RBCs to clear CSF and purposeful traumatic taps have demonstrated the absence of bilirubin and xanthochromia.[147,149] False-positives may occur because of other apparent causes of xanthochromia,[150] which are listed in **Table 11**.

Table 10. Limitations Of Lumbar Puncture[2]

- Failure to perform LP when CT is negative, equivocal, or suboptimal.
- Xanthochromia may be absent very early (< 12 h) and very late (> 2 wk).
- Accurate diagnosis of xanthochromia can be difficult.
- Distinguishing traumatic tap from true SAH may be challenging.
- Red blood cell count is diluted by circulating CSF and is affected by timing of LP.
- There is no guideline for the number of red blood cells required to diagnose SAH.

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; LP, lumbar puncture; SAH, subarachnoid hemorrhage.

Table 11. Non-SAH Causes Of Xanthochromia

- Jaundice (total bilirubin > 10-15 mg/dL)
- Increased CSF protein (> 150 mg/dL)
- Rifampin use
- Excess dietary carotenoids

Abbreviation: CSF, cerebrospinal fluid, SAH, subarachnoid hemorrhage.

Xanthochromia may take up to 12 hours to develop and it 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 and 2 weeks had xanthochromia.[146,151] Some authors have recommended waiting 12 hours from symptom onset to obtain a LP.[57,146] However, multiple studies have shown that, in the absence of xanthochromia, all of these early cases will show bloody CSF with abundant RBCs.[146,151,152] Thus, finding normal CSF, even in the first few hours, successfully excludes sSAH. For all of these reasons, the authors of one review recommend not delaying LP but, instead, accepting either RBCs or xanthochromia as a positive finding of sSAH.[71]

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,[151,153] and 1 guideline formally recommends this as the preferred method for CSF analysis.[154] However, visual inspection is probably highly effective; a 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.[136] One 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,[155] indicating a minimal difference in detecting abnormal CSF between the 2 methods. Multiwavelength spectrophotometric analysis for xanthochromia has recently been proposed as a superior method to traditional spectrophotometry and possibly visual analysis.[156] This method is not widely available clinically and has not been tested directly; regardless, the proponents for spectrophotometry cite a perfect sensitivity of 100%.[151,157] This must be weighed against the poor specificity of spectrophotometry, which ranged from 29% to 75% in 1 prospective and 1 retrospective study (each with more than 200 patients)[157,158] and a high false-positive rate (18 of 20 patients) in another prospective study.[64] Relying on spectrophotometry could lead to increased angiography, exposing patients with false-positive CSF results to unnecessary risk.[158] From a practical standpoint, spectrophotometry is not available in most (> 99%) emergency laboratories in North America.[148] 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.[2] Once again, timing is important; xanthochromia will disappear after 2 weeks.[146] In the absence of xanthochromia, there is no current approach to CSF analysis that can definitively distinguish between true hemorrhagic and traumatic blood-stained CSF. This dilemma may be reduced by the use of fluoroscopic guidance for LP,[132] 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.[142,143,159]

Cardiopulmonary Testing

Cardiac abnormalities are common following acute sSAH. Subendocardial ischemia may result from autonomic stimulation from the brain and circulating catecholamine surge, resulting in an abnormal electrocardiogram (ECG) in 50% to 100% of patients with aSAH in the acute phase.[97,98,160,161] Common, benign, and usually transient, ECG changes are nonspecific ST wave 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.[98,162] Positive troponin occurs in 20% to 40% of acute cases[99,163,164] and may lead to cardiopulmonary complications and worse outcomes.[99] Serious arrhythmias may occur in < 5% of patients and have been shown to be associated with worse outcomes.[98] Whereas suspected myocardial ischemia should be treated in the usual manner, SAH is considered a contraindication to thrombolytic or anticoagulant therapy.[165] Coronary angiography has been normal in several case reports of patients with SAH with ST segment elevations and/or elevated troponin, highlighting a neurocardiogenic mechanism distinct from coronary thrombosis underlying this process.[166-169] Neurogenic stunned myocardium (also known as Takotsubo cardiomyopathy) has been reported in patients with aSAH. This phenomenon refers to acutely decreased left ventricular function[170-172] due to left ventricular apical akinesis or apical ballooning.[171,173] This is usually in the setting of normal coronary arteries[171] and its mechanism is believed to be a stress-induced cardiomyopathy. Patients may have an acutely depressed ejection fraction (as low as 20%). Most patients usually recover function over several weeks. This presents significant initial diagnostic challenges, as patients may have pulmonary edema and symptoms of acute cardiac ischemia.[174-176] The syndrome is considered transient and the initial diagnostic and management strategies should focus on the primary neurological insult while supporting the cardiac dysfunction.

Clinical Decision Making

Current ACEP clinical policy states that, in patients with negative CT and LP, the diagnosis of SAH can be excluded.[5] There are several well-conducted pro-

spective studies to support this practice.[61,63,66] The largest of these was a prospective cohort study that observed 592 patients with sudden-onset headache for 3 years. The study included 61 patients with sSAH (55 diagnosed by CT and 6 by LP) and found no cases of subsequent SAH in those patients with negative CT and LP.[66] Several smaller studies have also observed patients with thunderclap headache after negative CT and negative LP results and none of the patients developed sSAH.[62,80,177] The largest of these observed clinical outcomes of 71 patients for an average of 3.3 years.[62] Furthermore, a recent systematic review that included 7 studies of patients with negative CT and LP with > 1 year of follow-up found a pooled proportion of 0% with sSAH.[178] 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.

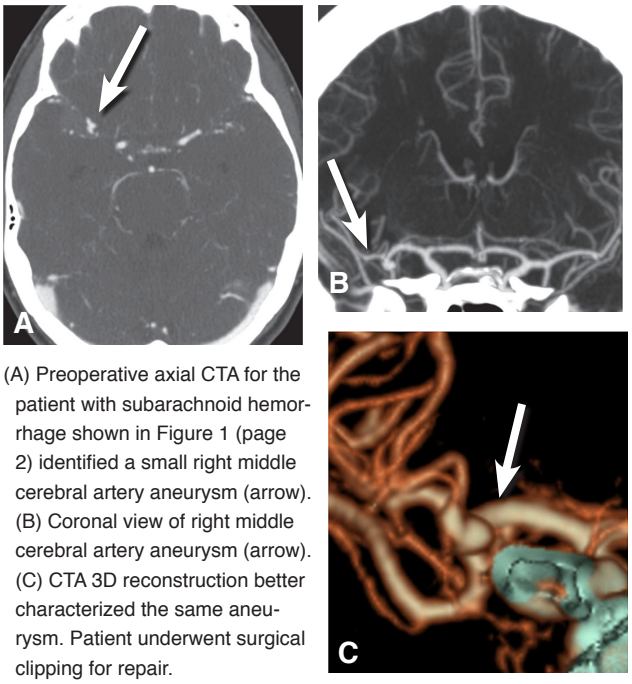
Role Of Primary Computed Tomography Angiography In Subarachnoid Hemorrhage

CTA is an emerging technology that has the diagnostic advantage of being noninvasive. The diagnostic accuracy of CTA varies widely and, when compared to the standard DSA, the sensitivity and specificity range from 77% to 100% and 87% to 100%, respectively.[76,179-185] There are very limited data to compare the CTA approach to the current standard approach. A methodological review supported a CTA approach in patients presenting within 48 hours of symptoms and with a neurologically intact examination.[186] CTA sensitivity decreases with aneurysms < 4 mm in size[181,187] and carries the risk of contrast-induced nephropathy, especially if DSA is performed after CTA.[76] **See Figure 5** for the appearance of an aneurysm on CTA. The use of CTA has not replaced the current approach but it has gained acceptance, although its utilization is center-specific and more prevalent in continued management than initial diagnosis.[188-190] CTA may be part of the algorithm in the future. This is an area of rapid technologic growth. Newer 3-dimensional 320-detector row CT scanners are now available and provide even greater detail, although they are not in widespread use.[191] The 2012 AHA guidelines recommended CTA as part of the workup for aneurysm but not as the primary diagnostic tool. In a patient diagnosed with sSAH, a nondiagnostic CTA should be followed up with a DSA (except possibly in the case of perimesencephalic hemorrhage).

Role Of Primary Magnetic Resonance Imaging In Diagnosis Of Subarachnoid Hemorrhage

Only small studies of magnetic resonance imaging (MRI) for diagnosis of SAH exist. In a study of 41 patients, MRI was 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 aSAH.[144] A 2002 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.[145] In contrast, another study of 12 patients with CT-negative, CSF-positive SAH showed that only 2 patients had positive FLAIR findings for SAH.[192] These were also the 2 patients that had the highest RBC count in the CSF, suggesting that a minimum number of RBCs is required to cause hyperintensity on magnetic resonance. MRI is of limited clinical utility in the ED setting owing to limited general availability (institutional and 24-hour availability) and the technical expertise required to interpret the study.[160] While MRI is a growing technology and several studies report higher sensitivity detection rates depending on the sequence of MRI utilized,[193-195] there are not enough clinical and feasibility data to recommend MRI as part of the routine evaluation for acute diagnosis of SAH. The 2012 AHA guidelines do recommend its possible use in patients with a nondiagnostic CT scan; however, a patient with suspected sSAH and a negative MRI still requires a LP.

Figure 5. Detection Of Aneurysm By Computed Tomography Angiography Of The Head



(A) Preoperative axial CTA for the patient with subarachnoid hemorrhage shown in Figure 1 (page 2) identified a small right middle cerebral artery aneurysm (arrow). (B) Coronal view of right middle cerebral artery aneurysm (arrow). (C) CTA 3D reconstruction better characterized the same aneurysm. Patient underwent surgical clipping for repair.

Abbreviation: CTA, computed tomography angiography.

Image courtesy of Lisa Thomas, MD.

Management

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 airway management, close hemodynamic monitoring, supportive care, and prevention and treatment of complications. The numbers of clinical interventions are limited for SAH patients; however, outcomes have been shown to be best when care is provided by multidisciplinary teams in specialized centers.[196] Few interventions for SAH management are unequivocally proven to be effective; rather, most are based on tradition and expert consensus. **Table 12** depicts a sample protocol for SAH management based on the practices outlined in the AHA and NCS guidelines. Details should be discussed with the consulting neurosurgical team, as specific management practices may vary among hospitals and treating specialists or according to patient circumstances.

General Care Measures

Bed rest is recommended, although it has not been proven to improve outcomes or prevent complications.[7,8] Adequate analgesia, antiemetics, and judicious sedation should be administered to ensure patient comfort. Serial neurological examinations should be performed to monitor for deterioration. The airway should be managed, if necessary.[7] Cardiac monitoring should be performed to evaluate for arrhythmias.[98] The head of the bed may be kept at a 30º angle to facilitate venous drainage.[9] The patient should have nothing by mouth until a 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 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.[197] Angiography may be negative in 10% to 20% of patients with SAH because of perimesencephalic hemorrhage, vasospasm, thrombosed aneurysm, or other rare causes.[198-200] The risk of complication from this procedure is considered to be very low. Two large prospective series of almost 3000 procedures[201,202] and a retrospective study of almost 20,000 procedures[203] report a 0.4% to 2.6% risk of neurologic complications, more than half of which were 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 conventional cerebral angiography.[184,185,204-208] This has led to several prospective studies that support using only CTA for preoperative planning.[159,209-212] **See Figure 5, page 13**, for an example of an aneurysm detected by CTA.

MRA is available but less well studied.[213,214] The majority of the limited data for comparison to DSA is in unruptured aneurysms, where MRA has sensitivity and specificity ranging from 69% to 99%. 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 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 one of the most devastating sources of morbidity and mortality after aSAH. The cumulative incidence of rebleeding after SAH during the first 72 hours has been estimated to range from 8% to 23%.[215] Studies have shown that 50% to 90% of rebleeding episodes occur within the first 6 hours after the primary bleed.[12,215] Predictors of rebleeding include patients with high-grade SAH, larger aneurysms, prolonged time to surgery, sentinel bleeds, and those who undergo catheter angiography. [12,215] Clinically, rebleeding may manifest as an acute or worsening headache, a decrease in the level of con-

Table 12. Emergency Department Management Of Subarachnoid Hemorrhage

- Consult neurosurgery or other cerebrovascular specialist.
- Obtain additional cerebrovascular imaging in consultation with neurosurgery.
- Patient should have nothing by mouth except medication.
- Administer analgesia and sedation as needed.
- Manage blood pressure:
  - Discuss target blood pressure goals with consulting neurosurgical team.
  - Consider commonly used intravenous agents: labetalol and nicardipine.
  - Monitor for hypotension.
- Avoid nitroprusside (may increase intracranial pressure).
- Consider short-term seizure prophylaxis (treat seizures if they have occurred).

sciousness, a loss of brainstem reflexes, posturing, respiratory arrest, or seizures. There are no robust randomized trials to guide the best management approach to preventing rebleeding. The NCS guidelines identify early repair as the primary strategy for preventing rebleeding. Judicious administration of antifibrinolytics and blood pressure management may also be beneficial.[12] In the past, when aneurysm surgery was delayed for days to weeks after 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.[216] However, in the current age of early intervention, new data suggest that very short courses of antifibrinolytics may reduce rebleeding without causing ischemia.[7,217-219] The administration of antifibrinolytics should be discussed with the accepting neurosurgical team. Rapid diagnosis of SAH followed by early definitive repair probably remains the best strategy for prevention.

Blood Pressure 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 blood pressure reduced to < 100 mm Hg.[195] While rebleeding occurred in 15% of patients treated with antihypertensives, compared with 33% in the untreated 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 a controversial subject, based on the conflicting results of recent studies.[220,221]

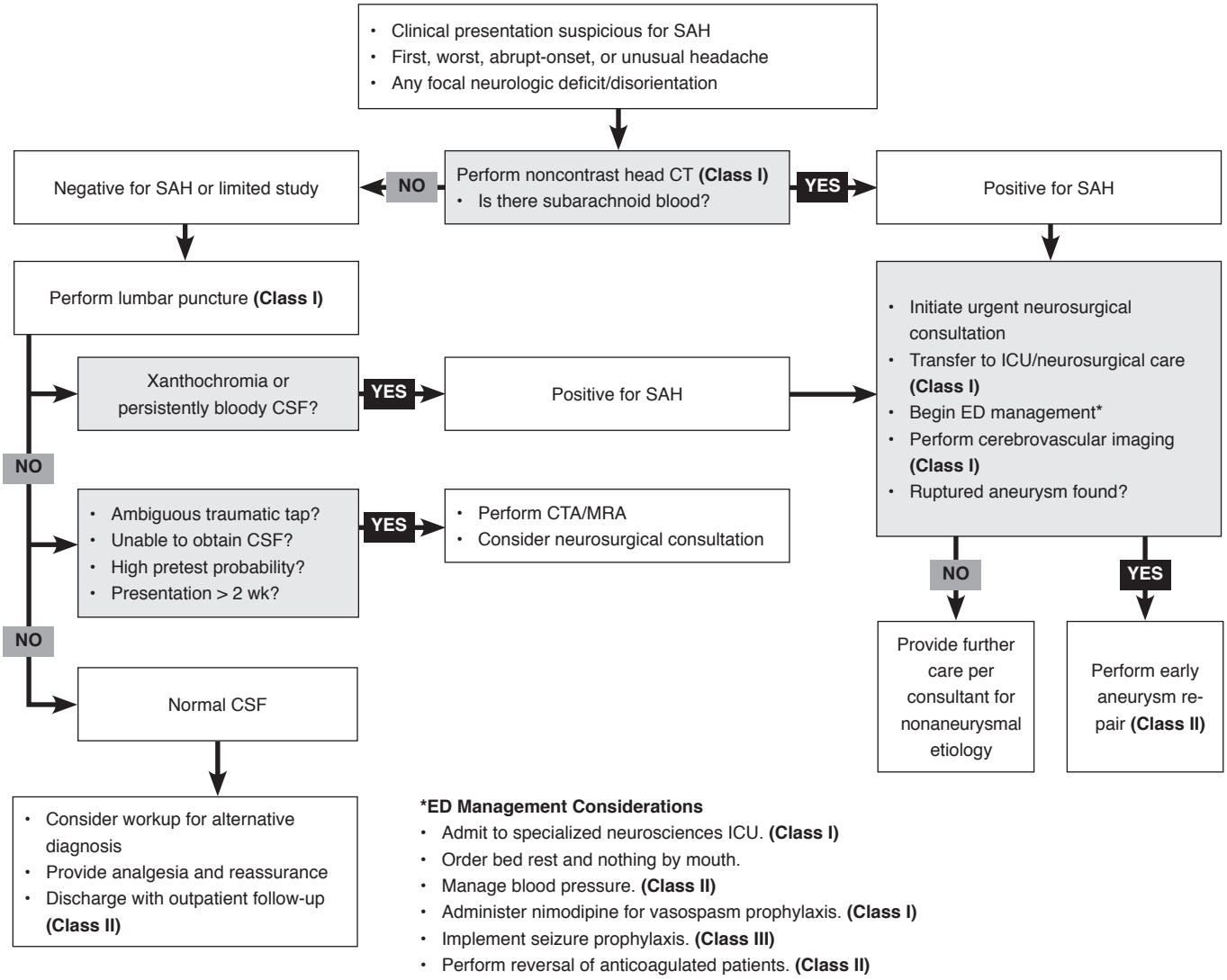
Although there are conflicting data on the causal relationship of hypertension in SAH patients, there may be some relationship between premorbid hypertension and poor prognosis and higher severity of disease on presentation.[222] Acute management, however, has not been clearly demonstrated to alter clinical outcome. There are no conclusive data pointing to a target blood pressure or the ideal antihypertensive agent in the management of SAH, so these choices should be left to the established practices of the consulting neurosurgical or neurointensive care team. The authors recommend the collaborative development and utilization of regional protocols to help guide the management of these patients at every point in the system of care. The 2012 AHA guidelines recommended that, from the time of symptom onset to obliteration of the aneurysm, the blood pressure be controlled with a titratable agent, with a goal systolic blood pressure of 160 mm Hg.[6] The guidelines note that an evidence-based goal has yet to be identified but that 160 mm Hg systolic is a reasonable target.[6] Titratable intravenous agents may include labetalol, nicardipine, clevidipine, and esmolol.[7,9] Clevidipine, an intravenous dihydropyridine with a pharmacokinetic profile similar to that of nicardipine, has not been extensively studied in SAH patients. Due to the addition of an ester linkage, it has a half-life of less than 1 minute and may therefore require less titration.[224,225] Availability of this agent may be increasing for management of acute hypertension of various etiologies. Nitroprusside should be avoided because of its tendency to increase intracranial pressure and potential for toxicity with prolonged infusion.[7] If the patient is expected to remain in the ED for a long time, an arterial catheter may be inserted to facilitate continuous blood pressure monitoring.[8]

Preventing Vasospasm

Cerebral vasospasm is a delayed complication that may develop several days up to 2 weeks post aSAH, peaking 7 to 10 days after the event.[2,19] Vasospasm may be asymptomatic or may lead to delayed neurologic deterioration, which can cause significant morbidity related to aSAH.[225] Nimodipine, a calcium-channel blocker, has been shown to reduce the occurrence of secondary ischemia with a favorable trend towards reducing case fatality. In a recent Cochrane Review of 16 trials of calcium antagonists, it had a risk ratio of 0.67; 95% CI, 0.55-0.81.[226] The mechanism for this improvement is unclear but is not mediated by vasospasm and is not demonstrated in other calcium-channel–blocking agents The statistical significance of this review rests heavily on the largest randomized controlled trial of 554 patients, from which comes the standard dosing regimen.[227] It is thus appropriate to administer 60 mg of nimodipine orally every 4 hours, even starting in the ED.[7,8,226] 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 intravenous nimodipine.[226] Nimodipine should be given in hemodynamically stable patients and in consultation with the specialist team. Note that nimodipine is part of the comprehensive stroke center core measures (although not necessarily administered in the ED). Nicardipine, another calcium channel blocker, has also been shown to decrease vasospasm in a randomized trial but without any improvement in outcome.[228]

Intravenous magnesium sulfate[226,229] has shown no difference in vasospasm prophylaxis.[230] Statins have shown some promise in early literature for preventing vasospasm.[230-232] Recent larger trials have been inconclusive. Some trials have shown decreases in vasospasm incidence with no improvement in delayed cerebral ischemia or mortality.[233-235] Currently, high-dose statin therapy is being evaluated in comparison with normal-dose statin therapy.[236,237] Ultimately, the data are not conclusive enough to make firm recommendations for statin use, especially in the ED.[238]

Clinical Pathway For Emergency Evaluation Of Suspected 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)**
- Implement seizure prophylaxis. **(Class III)**
- Perform reversal of anticoagulated patients. **(Class II)**

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; CTA, computed tomography angiography; ED, emergency department; ICU, intensive care unit; MRA, magnetic resonance angiography; 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.

Seizure Prophylaxis

Less than 20% of patients have a seizure during or soon after aSAH.[239] Some advocate prophylactic emergency antiepileptic drug use in all patients with aSAH,[240] based on older data collected when the rates of seizure after aSAH were thought to be higher and management was often delayed. This has become controversial,[241] but there is likely a high-risk subset that may benefit from it, as patients with a higher clinical grade are likely to deteriorate after a seizure.[8,242,243]

Although it is not clear why, phenytoin has been associated with worse outcomes, especially with prolonged use.[244,245] Data from 4 multicenter randomized controlled trials of aSAH were pooled together to identify associations in antiepileptic drug use in aSAH. The analysis consisted of 3552 patients admitted with aSAH and found increased inhospital complications and worse outcomes after antiepileptic therapy, based on GCS score, neurologic deterioration, and vasospasm.[244] 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.[245] A review of 7000 aSAH patients given antiepileptic drugs found a 2.2% early seizure rate and 5.5% late seizure rate, with no difference between the patients who received some course of antiepileptic drugs and those who received no antiepileptic drugs.[246] Common practice may include a short course (< 3 days) of an antiepileptic drug, based on data showing that short courses have the same benefit of low inhospital postoperative seizure rate (< 2%) when compared with longer courses, without the accumulating adverse effects of the drugs.[242,247] In a patient who has not already seized, it is reasonable to defer antiepileptic drug initiation to the inpatient management service due to the high variability in antiepileptic drug use and the limited data to support efficacy in the early phase. This is best done in accordance with protocol developed at local institutions.

Acute Clinical Deterioration

In patients who experience an early deterioration in neurological status, there are several potential causes, and it is important to repeat the CT 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 long-term outcomes.[2]

Definitive Aneurysm Repair

The 2 main approaches to aneurysm repair are microvascular neurosurgical clipping or endovascular coiling. Early treatment (within 72 hours) is a common approach.[141,248] The largest trial comparing clipping to coiling randomized 2143 patients suitable for both approaches and found improved outcomes for patients who underwent endovascular coiling.[249,250] 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.[250] 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, expertise of the clinicians, and the patient's clinical status, comorbidities, and preference.[251-253]

Prognosis

Even with transfer to specialized neurosurgical intensive care in high-volume centers, inhospital mortality after SAH is still > 30%.[254,255] Historically, patients with grade 4 or 5 aSAH (Hunt and Hess) had poor outcomes, with mortality reports of 70% to 90%.[256] With improvements in management strategies, mortality is still high in patients with severe aSAH but it is significantly better than historical reports.[257] Aggressive approaches may still be warranted. Outcome in surviving patients depends on several factors, including the patient's age and comorbidities, SAH grade at time of presentation, and perioperative complications during hospitalization.[17,258] Rebleeding carries the highest mortality rate; however, early repair has decreased the incidence of rebleeding significantly.[259] In contrast to patients with confirmed aneurysmal hemorrhage, patients who have SAH that is angiogram-negative due to perimesencephalic hemorrhage have an excellent prognosis; 98% survive without deficits.[200]

Special Circumstances

Pregnant And Postpartum Women

Acute headache in pregnant or postpartum women requires additional understanding and expansion of the differential diagnosis. As pregnancy and the acute postpartum period represent hypercoagulable states, emergency clinicians should strongly consider ischemic stroke and cerebral venous thrombosis. Other etiologies more common in the peripartum period that may present with headache are reversible cerebral vasoconstriction syndrome, pre-eclampsia, and posterior reversible encephalopathy syndrome.

Anticoagulated Patients

Some patients with sSAH may be taking antithrombotic medications (including warfarin and antiplatelet agents) at the time of presentation. There are very few case series that comment specifically on reversal in SAH cases;[260] however, general consensus supports reversal in the acute setting.[261,262] There are more data in the symptomatic intracranial hemorrhage population, but these data are also limited.

In the absence of data to inform a clear path, the authors recommend that regional protocols be developed to address management of patients on anticoagulant or antiplatelet therapy who present with a SAH. There are several newer agents but fewer reversal options; the risks and benefits should be discussed and policy may be influenced by regional availability of reversal agents.

Controversies And Cutting Edge

Computed Tomography Angiography

Whether CTA can be used for the primary diagnosis of sSAH, eliminating the need for LP, is a topic of current debate. A common practice is to obtain a 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 underwent LP in the ED,[64,127,128] 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. Only 2 small studies comparing CTA with noncontrast CT and LP for primary SAH diagnosis exist.[142,143] The best evidence comes from a prospective study of 106 patients in whom CTA detected 6 cases of aneurysm that were not seen on noncontrast CT.[142] All of 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, likely owing to detection of unruptured aneurysms.

Implementation of the CTA strategy may lead to unnecessary angiography or surgical risk in the 2% of the population harboring incidental aneurysms.[37] However, many of these incidental aneurysms can 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.[158] 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.[37] Finally, the risk of intravenous contrast to patients with iodine dye allergy or renal insufficiency also needs to be considered.

As of 2014, although the results are promising for CTA in the diagnosis of ruptured aneurysm, the current diagnostic strategy for sSAH should not be altered without larger prospective trials examining test characteristics, risks, benefits, and cost-effectiveness.

Lumbar Puncture–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.[263] 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;[64,127,128] this strategy would force the LP to be completed. Although a LP-first strategy may be safe in certain patients who are neurologically normal without signs of increased intracranial pressure, removing CSF from patients with SAH with unrecognized intracranial hematoma may precipitate rebleeding or herniation; this can occur even without focal neurological findings.[264,265] Concerns about herniation have arisen from pre-CT retrospective reports of sudden neurologic deterioration and death in 1 patient just after the procedure in a series of 129 patients with papilledema or increased intracranial pressure,[266] and 1 death among 401 patients with brain tumors.[267] Also commonly cited is a case series of 74 patients with suspected intracranial hematomas, 44 of whom had LP prior to imaging.[264] 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. 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.[268] 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 intracranial pressure, 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 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 can thus 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.[269] The etiology for this phenomenon

is undetermined; it may be caused by minor leakage from a cerebral aneurysm before impending complete rupture,[120] misdiagnosis,[269,270] or recall bias.[270] Regardless of the explanation, observational data suggest that patients experiencing these warning symptoms have worse outcomes. 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.[271] 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.[120]

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

Disposition

Patients with acute SAH should be admitted to an intensive care unit, preferably a neuroscience intensive care unit, for continual supportive care and monitoring until definitive repair of the aneurysm.[9,240,272] 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. A recent meta-analysis that used data from 4 studies and included over 36,000 patients showed a reduction in inhospital mortality in patients treated in high-volume centers (odds ratio, 0.77; 95% CI, 0.60-0.97).[196]

Patients can be safely transferred to these centers. One recent prospective multicenter study on emergent neurosurgical transfers, one-third of which were for SAH, found an average of 5.2 hours from time of diagnosis to the patient's arrival at the neurosurgical center without significant clinical deterioration of the GCS score in 90% of patients.[273] Emergency air transportation of patients with spontaneous acute intracranial hemorrhage has also been found to be safe and effective in facilitating early diagnosis and operative intervention.[274] 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, normal CT, and normal CSF analysis may be safely discharged from the ED with outpatient followup.[5]

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 emergency clinician must understand the limitations of each study and, when in doubt, obtain neurosurgical consultation. Because of the potential for significant 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 Conclusions

The middle-aged woman who initially seemed so uncomfortable felt much better after receiving 1 g of acetaminophen. You were both reassured by this, but you knew that patients with SAH can have pain that resolves spontaneously or is alleviated with medication. At this point, she wanted to go home, but you explained the need for her to stay for a LP. After the negative LP and CT, you appropriately sent her home with primary care follow-up for her benign headache.

The other patient with migraine history had described this episode as being worse than her usual migraines, and you were concerned because she said she usually saw flashing lights prior to headache onset and this time she had no aura. 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. Luckily, the laboratory technician was able to visualize xanthochromia, confirming the diagnosis. Based on these findings, you called for neurosurgical consultation and got a CTA to further assess for ruptured aneurysm. In coordination with neurosurgery, you began acute supportive care in the ED. You placed the patient on continuous monitoring and treated her pain as needed, watching for excessive blood pressure elevations. You ordered frequent nursing checks to ensure no deterioration in the patient's clinical status. On CTA, she had a large aneurysm of the anterior communicating artery. She was ordered to have nothing by mouth except medications prior to definitive repair. While you were awaiting the patient's bed on the neurosciences ICU, you began treatment with nimodipine. Shortly thereafter, the patient was transferred to the neurosciences ICU in stable condition to be cared for by a highly specialized team.

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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 will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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Cost-Effective Strategies

- In patients with suspected SAH, make sure to get a LP if the noncontrast CT is negative. The financial cost, clinical outcome, and possible litigation has the potential to be much more than initial workup, as patients are often much sicker and have worse outcomes when they re-present after a rebleed.
- Use the visual method to assess CSF for xanthochromia. It is what is most widely available in United States laboratories and does not require a spectrophotometer. Also, due to the low specificity of spectrophotometry, false-positives with that method could lead to unnecessary cost and risk of angiography or further testing.
- Transfer SAH patients to regional hospitals with high-volume and specialized neuroscience intensive care unit care. 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. It is also feasible and cost-effective to establish such regional centers.[265]
- Use teleradiology, if available, to discuss any questions about transferring patients with the nearest neurosurgical center. Remote neuroradiology capability at neurosurgical centers may reduce costs by decreasing futile or unnecessary transfers.
- Implementation of education programs may improve early diagnosis and outcomes of SAH. A local teaching program to referring doctors in one prospective study was implemented at a low cost, reducing diagnostic error by 77%, while improving overall management outcome.

Risk Management Pitfalls For Subarachnoid Hemorrhage

1. **"I thought it was a migraine, so I did not order 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. **"Her symptoms were suggestive of 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, myalgia, and mild headache, which may be misdiagnosed as a viral syndrome. Consider CT and, if negative, LP.

3. **"His symptoms improved with antiemetics and analgesics."**
   Patients with SAH may have symptoms that completely resolve with pain 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. **"He was from a nursing home, delirious, and had a urinary tract infection, so I thought it was urosepsis."**
   Patients with delirium or a 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. **"Her pain was atypical and she had chest pain with an abnormal ECG."**
   Patients with SAH may have an abnormal ECG and/or positive cardiac markers due to effects of a catecholamine surge from brain injury. Focusing on these cardiac findings may distract the emergency clinician from diagnosing the underlying etiology, which may be SAH.

6. **"The head CT was negative and the patient clinically improved and wanted to go home."**
   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. However, patients' autonomy is important and should be respected as long as they are informed.

7. **"The patient did not mention taking anticoagulants, so I did not check anticoagulation tests."**
   Basic laboratory tests (including prothrombin time and partial thromboplastin time) 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 intravenous vitamin K and clotting factor.

8. **"The number of RBCs decreased from the first tube to the fourth tube so I presumed 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 a 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 diminishes with time after onset of headache.

9. **"I did not 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 and the patient was waiting to transfer when she became disoriented."**
    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 the airway. Repeated head CT should be considered, because clinical deterioration from acute hydrocephalus can be reversed with treatment.

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1. All of the following are proven risk factors for spontaneous SAH EXCEPT:
   a. Smoking
   b. Polycystic kidney disease
   c. Hypertension
   d. Obesity

2. When is a noncontrast CT most sensitive in diagnosing SAH?
   a. Within 6 hours of symptom onset
   b. 24 hours after symptom onset
   c. 2 days after symptom onset
   d. 1 week after symptom onset

3. Factors that may compromise the sensitivity of the noncontrast CT for diagnosing SAH include:
   a. Volume of hemorrhage
   b. Experience of the radiologist
   c. Thickness of CT slices
   d. Anemia
   e. All of the above

4. How many RBCs in the CSF are required to make the diagnosis of SAH?
   a. 500
   b. 1000
   c. 5000
   d. No specific cut-off value exists.

5. When is LP most sensitive in diagnosing SAH?
   a. Within 1 hour of symptom onset
   b. 6 hours after symptom onset
   c. 12 hours after symptom onset
   d. 12 days after symptom onset

6. **In cases of suspected SAH in patients with familial risk factors, what test is needed after negative noncontrast CT and negative CSF results to exclude the diagnosis?**
   a. Repeat noncontrast CT after 6 hours
   b. Cerebral angiography
   c. Transcranial Doppler
   d. MRI
   e. No further testing is needed

7. **Which of the following approaches to blood pressure management may increase mortality in hypertensive patients with SAH?**
   a. Nitroprusside for a goal blood pressure of < 110/50 mm Hg
   b. Nicardipine for a goal blood pressure of < 140/80 mm Hg
   c. Esmolol for a goal blood pressure of < 160/80 mm Hg
   d. Labetalol for a goal of reducing blood pressure by about 25%

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

9. **A neurologically intact patient has just been diagnosed with SAH based on noncontrast CT and then has a sudden change in mental status, what is the next appropriate step?**
   a. Lumbar puncture
   b. Anticonvulsant
   c. Nimodipine
   d. Repeat head CT

10. **Which etiology of spontaneous SAH has the best prognosis?**
    a. Arteriovenous malformation
    b. Perimesencephalic hemorrhage
    c. Ruptured aneurysm
    d. Cocaine abuse

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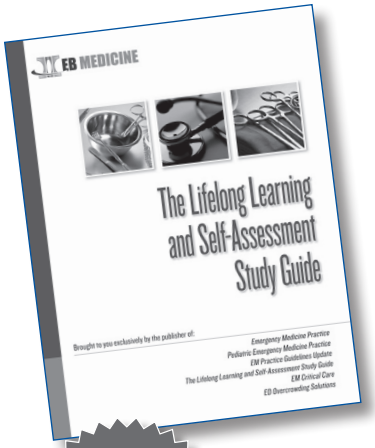

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Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common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

Objectives: Upon completion of this article, you should be able to: (1) describe both classic and atypical presentations of SAH; (2) describe the diagnostic approach to a patient suspected of having SAH; (3) identify the major limitations in interpreting the diagnostic modalities; (4) discuss general principles of acute SAH management in the emergency department and (5) identify common pitfalls in the diagnosis of SAH.

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