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Original Contributions

ANEURYSMAL SUBARACHNOID HEMORRHAGE: UPDATE FOR EMERGENCY PHYSICIANS

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□ **Abstract—Aneurysmal subarachnoid hemorrhage (SAH) is a serious cause of stroke that affects 30,000 patients in North America annually. Due to a wide spectrum of presentations, misdiagnosis of SAH has been reported to occur in a significant proportion of cases. Headache, the most common chief complaint, may be an isolated finding; the neurological examination may be normal and neck stiffness absent. Emergency physicians must decide which patients to evaluate beyond history and physical examination. This evaluation—computed tomography (CT) scanning and lumbar puncture (LP)—is straightforward, but each test has important limitations. CT sensitivity falls with time from onset of symptoms and is lower in mildly affected patients. Traumatic LP must be distinguished from true SAH. Cerebrospinal fluid analysis centers on measuring xanthochromia. Debate exists about the best method to measure it—visual inspection or spectrophotometry. An LP-first strategy is also discussed. If SAH is diagnosed, the priority shifts to specialist consultation and cerebrovascular imaging to define the offending vascular lesion. The sensitivity of CT and magnetic resonance angiography are approaching that of conventional catheter angiography. Emergency physicians must also address various management issues to treat or prevent early complications. Endovascular therapy is being increasingly used, and disposition to neurovascular centers that offer the full range of treatments leads to better patient outcomes. Emergency physicians must be expert in the diagnosis and initial stabilization of patients with SAH. Treatment in a hospital with both neurosurgical and endovascular capability is becoming the norm. © 2008 Elsevier Inc.**

Keywords—subarachnoid hemorrhage; diagnosis; lumbar puncture; cerebral angiography; xanthochromia; stroke

INTRODUCTION

Headaches, an exceedingly common complaint, are mostly caused by migraine, tension-type, and other primary headache disorders. Two percent of all Emergency Department (ED) patients have a chief complaint of headache, and of those, 2% have a serious life-, limb-, brain-, or vision-threatening condition diagnosed in the ED (Table 1) (1). One such disorder is subarachnoid hemorrhage (SAH). Although trauma is the leading cause of SAH, ruptured intracranial aneurysms account for 80% of non-traumatic cases (2,3). Of the remaining 20%, half are caused by non-aneurysmal venous "perimesencephalic" hemorrhages. The other 10% are caused by arteriovenous malformations, other vascular lesions, tumors, and other less common disorders (2–5).

One in 100 headache patients presenting to EDs have SAH (6–8). Of patients with severe, abrupt-onset head-

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Table 1. Life, Limb, Vision, or Brain-threatening Causes of Headache

Subarachnoid hemorrhage
Meningitis and encephalitis
Cervico-cranial artery dissections
Temporal arteritis
Acute narrow angle closure glaucoma
Hypertensive emergencies
Carbon monoxide poisoning
Idiopathic intracranial hypertension (pseudotumor cerebri)
Spontaneous intracranial hypotention
Cerebral venous and dural sinus thrombosis
Acute strokes: hemorrhagic or ischemic
Pituitary apoplexy
Mass lesions
Tumor
Abscess (including parameningeal infections)
Intracranial hematomas (parenchymal, subdural, epidural)
Colloid cyst of 3rd ventricle

ache and normal neurological examinations, about 10% have SAH (9–12). The initial bleed may be fatal, may result in significant neurological dysfunction, or may produce relatively minor symptoms. Because early treatment is associated with improved outcomes, timely diagnosis is critical (13). Despite a straightforward diagnostic algorithm, misdiagnosis remains common. Mildly affected patients who are most commonly misdiagnosed also have the best outcomes if correctly identified and treated (2). Misdiagnosis of SAH is an important cause of medico-legal actions against physicians (14,15).

This review updates emergency physicians on diagnostic and management issues of SAH that have emerged over the last several years.

Incidence of Aneurysms and Effects of Rupture

Intracranial aneurysms are common and are located on the large arteries of the circle of Willis and its branches. Autopsy series uncover them in 0.4–3.6% of individuals, whereas cerebral angiography documents incidental aneurysms in 3.7–6.0% of patients. Therefore, roughly 2% of all individuals harbor aneurysms (16). Approximately 80–85% of these lesions are in the anterior cerebral circulation, and the rest are in the posterior circulation (Figure 1); cerebral aneurysms are multiple in 25% of cases (17).

The reasons for aneurysmal rupture are incompletely understood. Although local hemodynamic forces may initiate aneurysmal formation, the tensile stress in the aneurysm wall may be more important in rupture. Larger aneurysm size and aspect ratio (dome size/neck size) are independently correlated with risk of rupture (18–20). Surface irregularities or multiple lobes on the aneurysm confer additive risk (21).

When an aneurysm does rupture, the intracranial pressure (ICP) rises precipitously. Cerebral perfusion may transiently cease, resulting in unconsciousness, or death, if the ICP is sufficiently high to cause irreversible structural damage or halt cerebral perfusion. The mortality rates on the

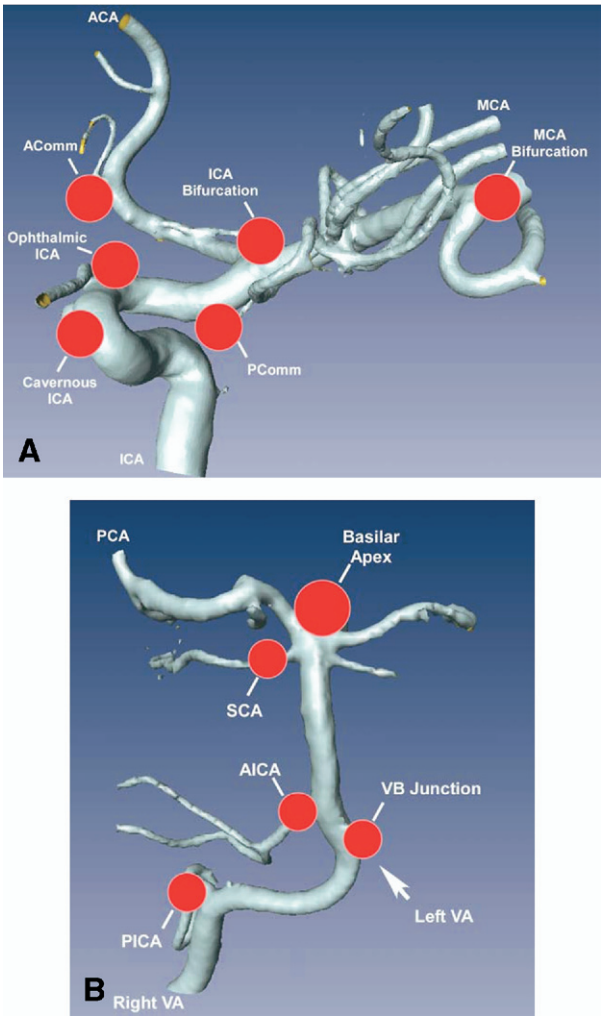

Figure 1. Cerebrovascular anatomy—Circle of Willis (shaded volume rendering images of 3-D rotational angiograms). The cerebrovascular circulation is conventionally divided into the (A) anterior circulation (internal carotid artery and its branches) and the (B) posterior circulation (the vertebral arteries and its branches). The two systems are linked up by the posterior communicating arteries, which connect the internal carotid artery and the posterior cerebral artery. The 3rd cranial nerve sits adjacent to the posterior communicating artery, which is the reason that aneurysms in this location are often associated with 3rd cranial nerve palsy. The red dots on the figure represent areas where aneurysms commonly form, typically at arterial bifurcations. ICA = internal carotid artery, PComm = posterior communicating artery, MCA = middle cerebral artery; AComm = anterior communicating artery, ACA = anterior cerebral artery; VA = vertebral artery, PICA and AICA = posterior (and anterior) inferior cerebellar artery, SCS = superior cerebellar artery; PCA = posterior cerebral artery.

Table 2. Scales for Clinical Rating of Subarachnoid Hemorrhage Patients

Hunt and Hess Scale	
Grade	Description
0	Unruptured
1	Asymptomatic, or mild H/A and slight nuchal rigidity
1a	Acute meningeal/brain reaction, but with fixed neuro deficit
2	Cranial nerve plasy, moderate to severe headache, or nuchal rigidity
3	Mild focal deficit, lethargy, or confusion
4	Stupor, moderate to severe hemiparesis, or dearly decerebrate rigidity
5	Deep coma, decerebrate rigidity, moribund appearance
	Add one grade for serious systemic disease (e.g., HTN, DM, severe atherosclerosis, COPD) or severe vasospasm on arteriography

World Federation of Neurosurgical Societies (WFNS) SAH grade		
WFNS Grade	Glasgow Coma Score	Major Focal Deficit
0 (unruptured)	—	—
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present/absent
5	3–6	Present/absent

H/A = headache; neuro = neurological; HTN = hypertension; DM = Diabetes Mellitus; COPD = chronic obstructive pulmonary disease.

first day and during the first month after hemorrhage are approximately 12% and 40%, respectively (22–27). The patient's clinical status at diagnosis is commonly measured by two clinical metrics—the Hunt and Hess (H&H) grade and the World Federation of Neurosurgical Societies (WFNS) scale (Table 2). Hunt and Hess' original article correlated clinical grade with mortality (28). Although commonly used, the H&H scale is somewhat subjective, and is associated with significant inter-observer variability (29). The more objective WFNS scale is based on the Glasgow Coma Scale (GCS) and presence or absence of motor deficits (30). Other grading scales also have been proposed but are not widely used (31,32). Their common thread is that higher scores indicate worse clinical condition and result in worse outcomes.

SAH results in hemodynamic instability, metabolic disturbances, and neurocardiogenic injury including ventricular dysfunction, cardiac enzyme leak, and electrocardiographic abnormalities (33–38).

EPIDEMIOLOGY

The incidence of SAH has not diminished over time and is roughly 10 per 100,000 of the population and more common in Blacks and Hispanics than Whites (13,39–41). Women, especially post-menopause, are more frequently affected than men (13,23,27,42). Some studies suggest a rising incidence in elderly patients, and a decreasing incidence in men (27,43).

Given that the prevalence of aneurysms is approximately 200 times higher than the annual incidence of SAH, it is clear that most aneurysms do not rupture. Peak age at rupture is 50 years (17). Important risks for SAH include heavy alcohol use, cigarette smoking, hypertension, and possibly oral contraception use (13,44–48). A positive family or past personal history of SAH also increases risk (45,49,50). Cocaine use may also increase the risk in those patients who have aneurysms (51). Other disorders associated with SAH include autosomal dominant polycystic kidney disease, Ehlers-Danlos syndrome type IV, and neurofibromatosis type 1 (52).

DIAGNOSIS

Which Patients to Evaluate?

Numerous studies over several decades document that misdiagnosis of SAH occurs approximately 25% of the time (12–50%), even in the era of ready access to cranial computed tomography (CT) scanning (53–60). These studies show three recurring, preventable reasons for misdiagnosis—failure to consider the diagnosis, failure to perform (and correctly interpret) CT scans, and failure to perform (and correctly interpret the results of) lumbar puncture (LP) (2,3). One large study found that failure to obtain a CT scan was the most common error (55). The largest and most recent report, a Canadian population-based study, found a much lower rate of ED-attributable SAH misdiagnosis (5.4%), and older studies include misdiagnosis attributable to patients, primary care physicians, and specialists (61). Over time, the misdiagnosis rate may be decreasing, but methodological differences across these studies preclude a firm conclusion (62).

The first decision emergency physicians must make when evaluating patients with headache is whether or not to pursue any diagnostic studies beyond history and physical examination. The classic history of SAH—abrupt onset of worst headache of life ("thunderclap" headache) during exercise or Valsalva, associated with transient syncope with vomiting—presents little diagnostic difficulty (63). Evaluation of headache patients with cognitive impairment, new focal abnormalities, or meningismus, is also clear cut.

Less clear is when to evaluate patients with milder symptoms and normal neurological examinations. To date, no prospective published studies help physicians know which elements of the history predict SAH in this group.

Until such data are available, we believe that four elements of the history best help identify the majority of these patients: onset, severity, quality, and associated symptoms (3,64). The onset is usually sudden and the severity is generally "worst of life." Because headaches are so common, comparing the quality of the index headache with any prior ones is important. Patients usually describe the headache from SAH as clearly different from prior headaches. Ten percent of neurologically normal patients with abrupt-onset, severe, and unusual headaches will have SAH (9–12). Half of all SAH patients present with atypical or mild features (17,28,65). Associated symptoms, such as nausea, vomiting, neck pain, and stiffness are common. However, neck stiffness (meningismus) may be absent, especially in the early hours (5,64). Headache patients over age 50 years are more likely to have SAH and other serious intracranial pathology (1,8). There are many diagnostic pitfalls to be avoided (Table 3) (2,3,64).

Many SAH patients have headaches that begin at rest or during quiet activities (66,67). Others present with symptoms such as vomiting, fever and headache, mild confusion, delirium, or severe neck pain that suggest other diagnoses such as a viral syndrome, gastroenteritis, psychiatric disorders, or neck strain (53–57,68,69). Undue focus on associated findings—abnormal electrocardiogram, dysrhythmia, head injury (from syncope) or elevated blood pressure—may divert attention from the true cause of the symptoms (64,70). The headache may improve or resolve with non-narcotic analgesics, including sumatriptan, so diagnostic significance ought not to be ascribed to improvement with medications (71–74). Headache from so-called warning (or sentinel) bleeds can remit spontaneously. On the other end of the spectrum, occasional patients with SAH who present with cardiac arrest can have excellent outcomes (75,76).

Understanding the full spectrum of presentation of SAH is the best strategy to avoid misdiagnosis (2,3,64). Once the physician decides to perform a work-up, the next steps—CT scanning and LP—are straightforward.

Table 3. Reasons for Misdiagnosis of Subarachnoid Hemorrhage

Failure to know the spectrum of presentations of subarachnoid hemorrhage
Not evaluating patients with unusual (for the patient) headaches
Is the onset abrupt?
Is the quality different from prior headaches?
Is the severity greater than prior headaches?
Are there associated symptoms that have been absent with prior headaches (such as vomiting, diplopia, syncope or seizure)?
Failure to appreciate that the headache can improve spontaneously or with non-narcotic analgesics
Over-reliance on the classic presentation with misdiagnosis of:
Viral syndrome, viral meningitis, and gastroenteritis
Migraine and tension-type headache
Sinus-related headache
Neck pain (rarely, back pain)
Psychiatric diagnoses
Focus on the secondary head injury (resulting from syncope and fall or car crash)
Focus on the electrocardiographic abnormalities
Focus on the elevated blood pressure
Lack of knowledge of presentations of the unruptured aneurysm
Failure to understand the limitations of computed tomography (CT) scans
CT scans are less sensitive with increasing time from onset of headache
CT scan can be falsely negative with small volume bleeds (spectrum bias)
Interpretation factors (expertise of physician reading the scan)
Technical factors (Have thin cuts been taken at the base of the brain? Is there motion artifact?)
CT scan can be falsely negative for blood at hematocrit level of $< 30\%$
Failure to perform lumbar puncture and correctly interpret cerebrospinal fluid findings:
Failure to do lumbar puncture in patients with negative, equivocal or sub-optimal CT scans
Failure to recognize that xanthochromia may be absent very early (< 12 h) and very late (> 2 weeks)
Failure to understand the limitations of xanthochromia measurement
Failure to properly distinguish traumatic tap from true subarachnoid hemorrhage

CT Scanning

The standard first test, unenhanced cranial CT scan (Figure 2), is highly accurate, but like all tests, possesses limitations (77). Firstly, accuracy decays with time; this is due to circulation of cerebrospinal fluid (CSF) and the resultant dilution and catabolism of the blood. Studies using third-generation scanners demonstrate sensitivities in the range of 90–98% within the first 24 h (11,78–80). One preliminary report of 913 neurologically intact patients (75 with SAH) with severe, abrupt-onset headaches found CT scan to be 92% sensitive and 100% specific; in the 305 patients scanned within 6 h of headache onset, CT scan was 100% sensitive (95% confidence interval 92–100) (81). By 3 and 7 days after the ictus, the sensitivity falls to 85% and 50%, respectively (82,83). One report using "5th generation" multi-detector CT scanners showed that no SAH case was missed; however, the study was underpowered, which yielded a lower 95% confidence interval of only 61% (84).

The second important limitation of CT is "spectrum bias." In alert and awake patients (presumably with smaller volume bleeds), the scans are less likely to show blood (80,82). These first two limitations—reduced accuracy over time and spectrum bias—are extremely im-

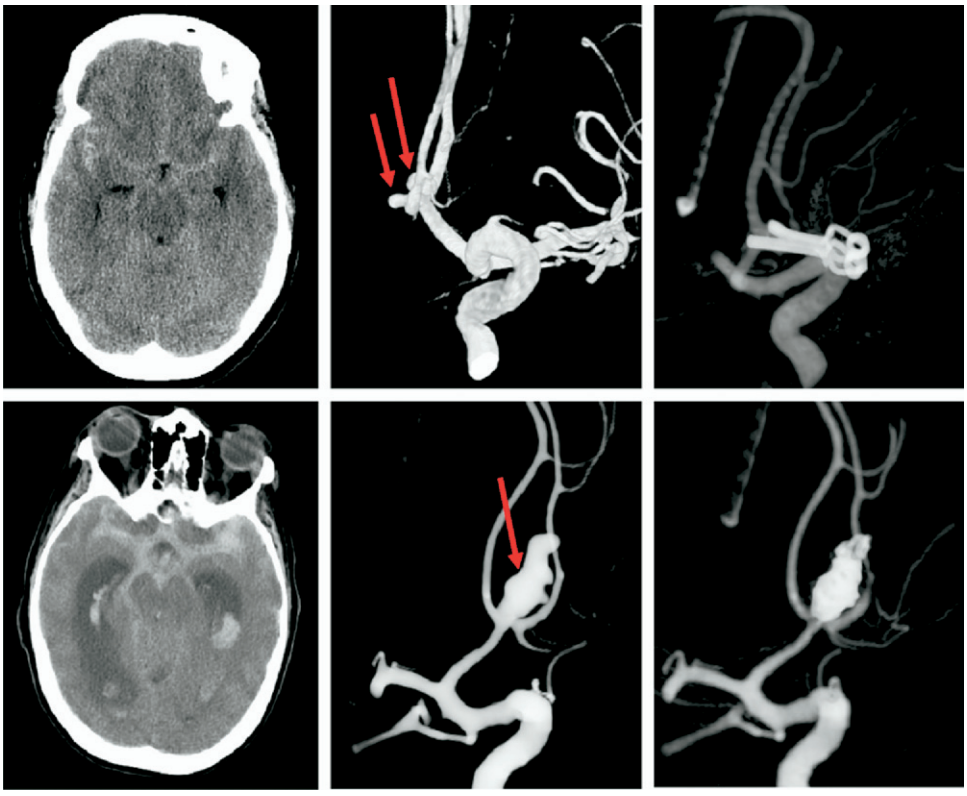

Figure 2. Imaging from patients with SAH. The top panel of three images shows a CT scan, 3-D angiogram showing two aneurysms (arrows) off the anterior communicating artery (one projecting to the left, the other projecting upwards, and partially obscured by the anterior communicating arteries also traveling upwards), and last, a post-operative angiogram with two clips obliterating both aneurysms. The bottom panel of three images shows similar images of another anterior communicating aneurysm that has been coiled, the third panel being an immediate post-operative study showing that the aneurysm is completely obliterated. The CT scan from the top panel is more subtly abnormal than the CT scan on the bottom panel, which shows much more blood.

portant for emergency physicians to understand. Thirdly, intracranial blood in anemic patients (hematocrit $< 30\%$) may appear isodense with brain and thus be more difficult to see (85). Lastly, many of these CT sensitivity studies relied on experienced neuroradiologists' interpretations, and "real world" readings by general radiologists, neurologists, or emergency physicians are less accurate (86). False-positive CT scans for SAH are unusual but have been reported in the settings of intravenous contrast neurotoxicity, purulent meningitis, spontaneous intracranial hypotension, isodense subdural hematomas, confusion with normal dural structures, and diffuse cerebral edema (87–95). Whenever meningitis is a strong possibility, intravenous antibiotics should be administered rapidly.

Until more convincing data confirm the 100% sensitivity of ultra-early or ultra-modern CT scanning in SAH, all patients being evaluated for SAH whose CT scans are normal, technically inadequate, or non-diagnostic, should undergo LP (2,77).

Lumbar Puncture

Cerebrospinal fluid (CSF) analysis also has limitations. Traumatic taps—red blood cells (RBCs) resulting from needle trauma—occur 10–15% of the time (96). Fluoroscopically guided LP may decrease this but is not routinely available (97). None of the methods of distinguishing traumatic taps from SAH is foolproof (98). Like the CT scan, CSF findings evolve over time from onset of symptoms.

Blood from ruptured aneurysms rapidly disseminates throughout the subarachnoid space, and large numbers of RBCs are present in the lumbar theca within 2–4 h (99). The development of xanthochromia, the yellowish hue resulting from hemoglobin catabolism into oxyhemoglobin, methemoglobin, and bilirubin, requires more time. Presence of xanthochromia indicates that the CSF contains blood that has undergone in vivo enzymatic degradation to bilirubin, implying true SAH (100). However, oxyhemoglobin can form in vitro, and ex-

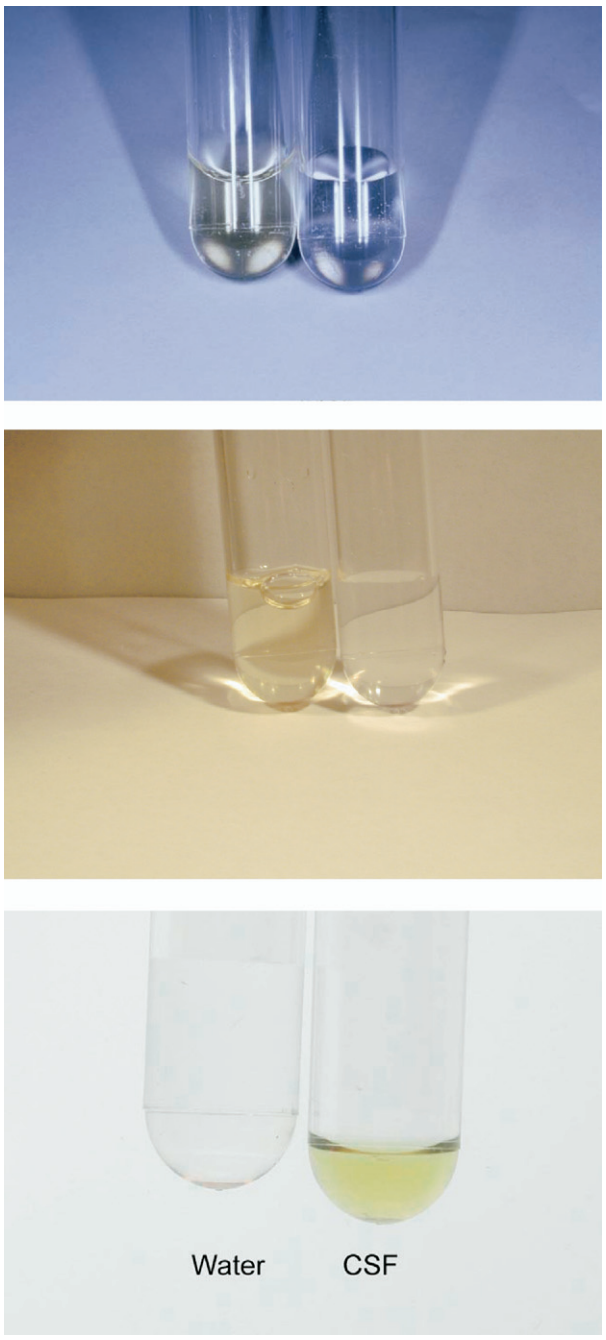

Figure 3. Xanthochromia can be measured visually or by spectrophotometry. These images show the appearance of visually measured xanthochromic cerebrospinal fluid (CSF). Technique is still important when measuring visually. Hand carry the CSF to the laboratory, rapidly centrifuge the tube, and then compare the supernatant to an identical tube filled with an equal volume of water. Note that the ambient lighting may change the ease with which one can see the color change; the top two panels are of the same specimen seen in different lighting. Some specimens (bottom panel) are more obvious than others. (This figure may be viewed in color in the web version of this article.)

perimentally, traumatic taps can result in discolored fluid from oxyhemoglobin (101).

Xanthochromia can be measured visually (Figure 3) or by spectrophotometry, the latter being clearly more sensitive (75,76,102–107). Some recommend exclusively using spectrophotometry, based on a study of 111 patients with CT-proven SAH, in which all subjects had spectrophotometrically measured xanthochromia from 12 h until 2 weeks after onset of headache (108–110). More recently, investigators studied the more clinically relevant population of CT-negative patients. Of 463 patients, CSF spectrophotometry found 2% had symptomatic aneurysms (111). Unfortunately, this retrospective chart review did not report the percentage of patients with visually measured xanthochromia, the timing of the LP, or the CSF RBC counts.

One important problem with spectrophotometry is that in series of unselected patients, false positives are very common, so that many patients without SAH will be subjected to unnecessary diagnostic testing (11,105,111,112). Furthermore, over 99% of hospital laboratories in North America measure xanthochromia visually (113). Lastly, a recent comparison between the two methods found that of CSF samples that clinicians deemed to be colorless, none had bilirubin as measured by spectrophotometry (114).

However it is assessed, xanthochromia takes time to develop. By spectrophotometry, it can take as long as 12 h; however, even by visual inspection, xanthochromia often develops earlier (99,110). Unfortunately, there are no well-performed clinical studies to allow clinicians to confidently know what the false-negative rate for xanthochromia is at specific time intervals from the bleed. To assess for xanthochromia, the CSF should be rapidly centrifuged and (in the case of spectrophotometry) stored in darkness. When measured visually, the CSF should be carefully compared to an identical tube filled with an equal volume of tap water against a white background. Spectrophotometric measurement should focus on the presence of bilirubin (111,115).

Importantly, early-presenting patients without xanthochromia will all have large amounts of RBCs in the CSF. The rare exceptions (intraparenchymal or subdural rupture or spinal block) will have positive CT scans (116–119). Two other useful, albeit imperfect, methods to distinguish traumatic taps from true bleeds are the "3-tube" test and measuring the opening pressure of the CSF (98). In the former, one looks for diminishing numbers of RBCs from the first to the last tube trending toward zero. This last detail is crucial. Older literature shows that a "decrease" (undefined) does not discriminate between traumatic tap and true SAH (120). In a more recent study using an arbitrary cutoff of a 25% reduction from tube 1 to 4, 2 of 8 patients had aneurysms (121). Thus, a simple

decrease is insufficient to exclude SAH and the last tube RBC count should approach zero. When bloody fluid is identified, wasting 2–3 mL of CSF to increase the gap between the first and last tubes improves the odds that the last tube RBC count will approach zero (98). There is no specific number of RBCs that serves as a threshold amount and the rate of RBC clearance is variable.

The opening pressure, which is elevated in two-thirds of SAH patients and is normal in traumatic taps, should also be measured (99). Elevated pressure also suggests the alternative diagnosis of cerebral venous sinus thrombosis or idiopathic intracranial hypertension, and abnormally low pressure suggests spontaneous intracranial hypotension (2,122,123).

OTHER DIAGNOSTIC ISSUES

Primary Use of CT Angiography for Diagnosis of SAH

With the increasing availability of multi-detector CT scanners, some have recommended using CT angiography (CTA) to diagnose SAH (124). Of the 116 patients, 6 (5.1%) had aneurysms (with normal CT but positive CSF findings); these patients would have been identified by the standard work-up. Three had positive CTA with normal CT and CSF, but these patients more likely had asymptomatic aneurysms with a headache of another benign cause. Like with spectrophotometry, a strategy of primary CTA for diagnosis of SAH would be expected to subject many patients to unnecessary work-ups, procedures, and angiographic contrast material.

MR for Primary Diagnosis of SAH

Magnetic resonance (MR) technology is constantly advancing and shows promise for SAH diagnosis (125). Although fluid-attenuated inversion recovery (FLAIR) and T2-graded echo MR may be better than CT for detection of chronic subarachnoid blood and possibly equivalent for intraparenchymal hemorrhage, false positives have been documented in acute SAH with both techniques (126–128). One recent study showed that all 13 patients (with positive CT scans) tested within 12 h of symptom-onset by MR (FLAIR and proton-density weighted) were MR positive (129). However, another study showed that of 12 patients with SAH (and negative CT scans), only 2 of the 12 (the 2 with the highest RBC counts) had positive FLAIR MR for SAH, showing that spectrum bias exists with MR as well (128). No large studies of MR diagnosis in unselected headache patients exist. Therefore, CT, which is quicker, cheaper, more readily available and easier to interpret, remains the diagnostic study of choice. If one uses MR, communication with the radiologist is critical to ensure acquisition of the correct sequences.

LP-first Strategy

Some have advocated an LP-first strategy in patients with severe acute headache, normal vital signs, and physical examinations (130). The rationale is that in practice, physicians evaluating patients for SAH often omit the LP after a negative CT scan (11,131). An LP-first strategy forces the LP to be done and would consume fewer resources.

An LP-first strategy may be safe, even in H&H Grade 2 and 3 patients, who have meningismus and may be drowsy (132,133). However, this practice can be dangerous, because collecting CSF in SAH patients may precipitate rebleeding or herniation from an unrecognized intracranial hematoma, which can occur in the absence of localizing neurological findings (134,135). Most of the patients who deteriorated in these latter two studies had neck stiffness and were H&H grade 1–3 (mostly 2), although one patient had no meningismus (135).

Therefore, an LP-first strategy is likely safe in carefully selected patients who are neurologically normal and without signs of elevated ICP. On the other hand, this approach would be contraindicated in patients with any kind of neurological abnormalities on examination or those with symptoms or signs of high ICP. Unfortunately, there are no clinical trials that assist clinicians in selecting patients for this approach, and the CT scan followed by LP remains the standard diagnostic sequence.

When to Stop the Work-up?

In patients with acute, severe headache and normal neurological examinations, CT scans, and CSF analysis, is further evaluation necessary? The vast majority of such patients have excellent outcomes. In one retrospective study of 71 patients, none had SAH during an average follow-up of 3.3 years (136). Furthermore, in four prospective studies (totaling 254 patients) followed for over 1 year, none had subsequent SAH or sudden death (9,137–139). This evidence strongly suggests that most patients with normal CT and CSF examinations do not require angiography. The occasional patient whose clinical presentation suggests cranial artery dissection, cerebral venous sinus thrombosis, or pituitary apoplexy may require further imaging.

"Warning" or "Sentinel" Symptoms

Some patients with SAH report unusual, severe headaches in the weeks preceding their SAH diagnosis—a phenomenon loosely described in the literature as "warning" or "sentinel" "headache" or "bleed." A recent systematic review found that such headaches occur in 10–43% of cases of SAH (140). Possible explanations for these episodes include a) initial misdiagnosis of a true SAH (either by the patient or the physician), b) recall bias of patients being admitted for a serious neurological problem, or c) pain from symptomatic but unruptured aneurysms (2,140–143). Recall bias may account for a small fraction of cases (140,144). The most likely explanation, supported by histological evidence in one case, is that most instances represent small undiagnosed SAHs (145). Data supporting this hypothesis are mixed, with one study suggesting no worse outcomes in patients returning after a "warning" event and another showing worse outcomes (142,144). Whatever the correct explanation, aggressive evaluation of patients with acute-onset severe headache should reduce the phenomenon of delayed or missed diagnosis.

Symptomatic Unruptured Aneurysms

The vast majority of unruptured aneurysms are asymptomatic, but occasional patients have thunderclap headache from intact aneurysms that are acutely expanding, dissecting, or thrombosing (2). Other symptoms of unruptured aneurysms include transient cerebral ischemia, gradual-onset headache, seizure, or mass effect (2,53,143). The classic mass lesion is a third cranial nerve palsy, an important syndrome for emergency physicians to understand (99,146,147).

INITIAL MANAGEMENT CONSIDERATIONS

Once the diagnosis of SAH is established, the priority shifts to definitive therapy, prevention of complications, and consultation with a skilled cerebrovascular specialist. This discussion between specialist and emergency physician should address several issues, including airway control, treatment of acute hydrocephalus, blood pressure control, seizure and vasospasm prophylaxis, and others (Table 4) (148,149). In this age of endovascular treatment, disposition of patients with SAH needs reevaluation because data suggest that SAH patients do better when treated in high-volume centers that offer both surgical and endovascular approaches (150–153).

Table 4. Management Issues* to be Addressed when Subarachnoid Hemorrhage is Diagnosed

Airway management
Specialist consultation and arranging an angiogram
Arrangement for transfer to a neurovascular center
Treatment of hypertension
Volume status and intravenous fluids
Seizure prophylaxis
Acute treatment of hydrocephalus, extra-axial or intracerebral hematomas
Nimodipine administration for vasospasm prophylaxis
Discuss short-term antifibrinolytics to prevent rebleeding
Cardiac telemetry
Analgesia
ICP

* When subarachnoid hemorrhage is diagnosed, rapidly consult with a cerebrovascular expert. This discussion should consider the points above on a case-by-case basis.
ICP = intracranial pressure.

Cerebrovascular Imaging

Upon diagnosis of SAH, cerebrovascular imaging is obtained as soon as possible after stabilization. A high-quality four-vessel cerebral digital subtraction angiogram (DSA) usually elucidates the etiology of the SAH. Negative angiograms occur with perimesencephalic hemorrhage, thrombosed aneurysms, or in cases of severe parent vessel spasm, which can interfere with aneurysmal filling. In the case of intracranial aneurysm, two- or newer three-dimensional angiography demonstrates the size and location of the aneurysm very well (Figure 2) (154). Modern DSA is very safe, one prospective series of nearly 3000 procedures showing a complication rate of only 1.3%, more than half of which were transient or reversible (155).

Although DSA remains the gold standard, multidetector CT angiography demonstrates high sensitivity and specificity (156–160). Four studies have evaluated a strategy of CTA-only for planning the surgical approach in large numbers of patients with SAH (161–164). Other investigators have accumulated experience with surgical planning based exclusively on MR angiography (165,166). Occasionally, both CT and MR angiography miss small aneurysms, and evidence suggests that neuroradiologists' interpretations may be superior to others (156,160,167,168). As of 2007, the choice of cerebrovascular imaging study is evolving and should be left to the discretion of the consultant.

Rebleeding

Rebleeding, an important cause of poor outcomes, occurs in 4% of patients in the first 24 h after hemorrhage, and approximately 1.5% per day for up to 2 weeks after the

initial hemorrhage, yielding a total re-hemorrhage rate of 26.5% over the first 2 weeks after the INITIAL BLEED (169–171). At one referral center, 40 of 574 (6.9%) patients rebled in the first 3 days post-ictus (172). Those who rebled had higher H&H grades, larger aneurysms, and worse outcomes. Therefore, strategies to reduce rebleeding are critical.

Although few data suggest that aggressively lowering blood pressure prevents rebleeding, most treating physicians insert an arterial catheter and use intravenous agents to maintain adequate cerebral perfusion in the patient with elevated intracranial pressure and in elderly patients with pre-existing atherosclerosis or hypertension (173). These steps are generally initiated in the intensive care unit. Also, because there is no good evidence to suggest the proper target blood pressure, this choice is generally left to the consultant. The most common antihypertensive agents used are labetolol, nicardipine, and nitroprusside. Again, there are no head-to-head clinical trials to compare these agents. Treatment of pain and anxiety may also help to reduce elevated blood pressure.

In the past, when surgery was delayed by weeks, antifibrinolytics (epsilon-aminocaproic acid and tranexamic acid) were used to reduce rebleeding. A recent review concluded that these agents were not routinely indicated because the price for the reduction in rebleeding was increased ischemic deficits from vasospasm (174). Current trends toward early aneurysm obliteration have reduced the need for long-term antifibrinolytic use. However, there has been renewed interest in using short-term antifibrinolytics from the time of diagnosis to the time of definitive treatment, even if this interval is only several hours (175). A randomized prospective multicenter trial using short-term tranexamic acid suggested that this strategy reduced rebleeding without increasing vasospasm or clinically significant cerebral ischemia (176).

Hydrocephalus

Hydrocephalus occurs in up to 33–50% of all patients with SAH, and is more likely to occur with larger volume hemorrhages. Acute (early) hydrocephalus from intraventricular blood that occludes the foramen of Monroe or Luschka, obstructing CSF outflow, occurs in approximately 20% of patients (17,177). Importantly, this cause of coma after SAH is reversible by treatment with emergent ventriculostomy.

Vasospasm and Delayed Cerebral Ischemia

Cerebral vasospasm typically develops several days after the initial SAH, peaking 7–10 days after the hemorrhage and lasting up to 2 weeks (17). The risk of developing vasospasm is related to the density of blood at the time of initial ictus (178,179). Vasospasm may be an asymptomatic angiographic phenomenon, or it may lead to symptomatic delayed cerebral ischemia (DCI), which is an important cause of morbidity after SAH. The resulting infarctions, which may be asymptomatic, may be distant from the site of the offending aneurysm or involve watershed distributions (180). Emergency physicians should be aware of this fact because some patients will present during this phase.

Prophylactic use of nimodipine improves outcomes, although the mechanism remains unclear (181). Some preliminary data suggest that intravenous magnesium sulfate may reduce DCI and poor outcomes, but further research is needed to confirm these findings (182).

If vasospasm is confirmed or suspected in the presence of neurological deterioration, "triple H" therapy (hypertension, hemodilution, and hypervolemia) may be instituted (183). Although a recent Cochrane review concluded that there is no convincing evidence supporting it, triple-H therapy is commonly used in practice (183–185). If medical therapy fails, various endovascular strategies also may be employed (186–190). These therapies underscore the advantages of a high-volume neurovascular center.

Aneurysm Obliteration

Early treatment of the ruptured aneurysm is the currently accepted strategy at most centers today. Occasionally, extenuating circumstances such as unstable medical conditions that preclude safe surgery or high-grade patients with poor prognosis may delay treatment. However, the goal in most patients is to obliterate the aneurysm within 1–3 days after the hemorrhage with either microsurgical aneurysm clipping or endovascular coil embolization (Figure 2).

Intracranial microsurgical clipping is a technique that has evolved considerably since its introduction in the 1970s with the advent of the stereoscopic high-magnification microscope. Using microsurgical techniques, the neurosurgeon opens the dura and identifies the parent vessel and the ruptured aneurysm, which is then clipped to exclude it from the circulation. The durability of successful surgical clipping is high, with a follow-up study showing aneurysm recurrence rate of 2–3% (191).

Endovascular coiling uses a micro-catheter that is threaded through a guide catheter to the origin of the ruptured aneurysm. Once inside the aneurysm, platinum coils are gently inserted into the sac in a sequential outside-in multi-layered fashion until the aneurysm is densely packed. This process relies on a critical volume

of embolization and also requires that the aneurysm inflow zone be securely occluded to deflect blood from entering the aneurysm. Although wide-necked aneurysms were initially considered poor candidates for coil embolization, newer techniques have expanded the pool of endovascular candidates (192–194).

Patients treated with coil therapy require serial monitoring and follow-up cerebrovascular imaging to detect the occasional risk of coil compaction or aneurysm recanalization, which can occur in larger size wide-necked or poorly packed aneurysms. Initial treatment yields up to 70% of patients experiencing 95–100% occlusion of the aneurysm. However, 25–30% of patients do not have complete obliteration of the aneurysm, and recanalization can occur (195).

The decision to proceed with open surgical clipping or endovascular treatment of an intracranial aneurysm after SAH rests on aneurysm-specific factors (location, size, morphology, and presence of thrombus) and patient-specific factors (age, density of SAH, patient preference, and other medical comorbidities).

The International Subarachnoid Aneurysm Trial randomized 2143 SAH patients whose aneurysms were judged to be equally suitable for surgical clipping or endovascular coiling to be treated surgically vs. endovascularly (196). Due to the selection criteria, patients were typically in good pre-treatment condition and there was a preponderance of anterior circulation aneurysms. Using a modified Rankin scale at 1 year as the outcome measure, the International Subarachnoid Aneurysm Trial demonstrated a statistically significant relative risk reduction of 22.6% and an absolute risk reduction of 6.9% in favor of endovascularly treated patients. Follow-up (1 to 7 years) revealed durability of these outcomes. The endovascularly treated patients had fewer seizures but (while low), more rebleeding (197). Similar results have been obtained in other studies (198).

Ideally, high-quality endovascular and surgical techniques will be available at a neurovascular center, which allows for the best treatment decisions to be made on a case-by-case basis (153). Whether treated endovascularly or surgically, patients are closely monitored for blood pressure, cardiac, renal, and respiratory function by a multi-disciplinary team with close neurological monitoring to detect DCI.

CONCLUSIONS

Emergency physicians must be vigilant in evaluating patients with symptoms consistent with SAH or otherwise symptomatic aneurysms. This evaluation must take place with an understanding of the limitations of the diagnostic tests used. Attention to early complications and prompt referral to centers where there are teams with cerebrovascular expertise will maximize the options available to these patients.

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ARTICLE SUMMARY

1. Why is this topic important?

Patients with aneurysmal subarachnoid hemorrhage present with a wide spectrum of signs and symptoms. Because early diagnosis and treatment leads to improved outcomes, emergency physicians must be expert in diagnosing these patients.

2. What does this study attempt to show?

This article reviews important concepts in the diagnosis of subarachnoid. Hemorrhage, including the limitations of the various commonly used tests, and also considers newer approaches. It also discusses up to date aspects of the initial stabilization and disposition of these patients.

3. What are the key findings?

Patients with subarachnoid hemorrhage can present with isolated headache and a normal neurological examination.

Non-contrast head CT scan, followed by LP (in patients with non-diagnostic scans) remain the standard diagnostic sequence.

Once hemorrhage is diagnosed, the priority shifts to identifying the offending vascular lesion and preventing early complications.

4. How is patient care impacted?

Improved diagnosis of subarachnoid hemorrhage will likely lead to improved patient outcomes and reduced physician liability.

Because there has been a shift towards endovascular therapy for ruptured aneurysms, the disposition of these patients has also shifted to centers that have both surgical and endovascular capabilities.