Sensitivity of Noncontrast Cranial Computed Tomography for the Emergency Department Diagnosis of Subarachnoid Hemorrhage

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Study objective: Emergency physicians use noncontrast cranial computed tomographic (CT) imaging of headache patients to identify subarachnoid hemorrhage caused by aneurysms or arteriovenous malformations. Given sufficiently high sensitivity, CT imaging could be used as a definitive diagnostic study in these patients. The purpose of this study is to determine the sensitivity of noncontrast cranial CT in detecting all spontaneous subarachnoid hemorrhages and those caused by aneurysm or arteriovenous malformation.

Methods: This was a retrospective review performed at an urban tertiary academic emergency department (ED). Using a combination of noncontrast cranial CT radiology coding, lumbar puncture results, International Classification of Diseases, Ninth Revision discharge diagnosis, and medical record review, we identified all patients who presented to a tertiary care academic ED from August 1, 2001, to December 31, 2004, with spontaneous subarachnoid hemorrhage. We determined whether patients were diagnosed by cranial CT or lumbar puncture, the presence of headache and level of consciousness at ED presentation, and whether or not they had an aneurysm or arteriovenous malformation.

Results: We identified 149 patients who were diagnosed with spontaneous subarachnoid hemorrhage during the study period. Noncontrast cranial CT scan diagnosed 139 patients, and 10 were diagnosed with lumbar puncture. This yielded an overall CT scan sensitivity of 93% (95% confidence interval [CI] 88% to 97%). Of the 149 with subarachnoid hemorrhage, 117 (79%) had aneurysm or arteriovenous malformation; cranial CT scan demonstrated subarachnoid hemorrhage in 110 of the 117, for a sensitivity of 94% (95% CI 88% to 98%). For the 67 patients presenting with headache and normal mental status who had a subarachnoid hemorrhage and vascular lesions (either aneurysm or arteriovenous malformation), the sensitivity of cranial CT scan was 91% (95% CI 82% to 97%).

Conclusion: Noncontrast CT imaging exhibits inadequate sensitivity to serve as a sole diagnostic modality in detecting spontaneous subarachnoid hemorrhage caused by aneurysm or arteriovenous malformation. [Ann Emerg Med. 2008;51:697-703.]

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INTRODUCTION

Headache was the chief complaint of approximately 2,844,000 people presenting to US emergency departments (EDs) in 2002 and is considered the most common presenting neurologic complaint. The challenge for emergency physicians is to determine which patients with headaches have underlying pathology with the potential for death or disability, using safe, accurate, and cost-effective diagnostic techniques.

Among headache patients, approximately 1% to 4% have subarachnoid hemorrhage. If only patients with “the worst headache of their life” are considered, the percentage with subarachnoid hemorrhage is 12%. Approximately 80% of subarachnoid hemorrhages are due to ruptured intracerebral aneurysms. The incidence of subarachnoid hemorrhage as a result...
of ruptured aneurysms is estimated to be 6 to 8 per 100,000 in the United States each year, or 27,000 to 30,000 annually.2,5,6

Many clinicians use noncontrast computed tomography (CT) imaging to evaluate patients they consider at risk for subarachnoid hemorrhage and reserve lumbar puncture for those with negative CT study results. Numerous publications have documented the utility of noncontrast cranial CT in demonstrating subarachnoid blood. It is a feature of the natural history of subarachnoid hemorrhage that blood diffuses away from the site of a transient bleed, which precludes detection by CT scan in some cases. When subarachnoid hemorrhage is simulated with phantoms and mixtures of artificial cerebrospinal fluid and blood, a hematocrit level greater than 27% is denser on CT scan than normal cortex is. Above a hematocrit of 22%, the mixture is more hyperintense than the normal cortex on magnetic resonance fluid-attenuated inversion recovery images.7 Studies done in the early 1980s demonstrated a sensitivity of 92% on the day of rupture, 86% 1 day later, and 76% on day 3, decreasing to 50% on day 5.8 More recent studies have shown a sensitivity of approximately 93% within the first 24 hours9 and between 98% and 100% before 12 hours.3,10 Despite this relatively good sensitivity, multiple authors and several guidelines have recommended that, if the CT result is negative, patients undergo lumbar puncture to detect the remaining cases of subarachnoid hemorrhage with a negative CT result because of the risks associated with an undetected subarachnoid hemorrhage caused by a cerebral aneurysm.2,4,11-13 Despite these recommendations, a lumbar puncture may not always performed, even in academic centers.3 Lumbar puncture is associated with a high rate of both postdural puncture headache (up to 30%) and back pain (35%),14 and many of these patients return for medical treatment. Both conditions are usually self-limited. Occasionally, post-LP headaches are unremitting with conservative treatment and require treatment with a “blood patch” by an anesthesiologist.

There have been reports of iatrogenic meningitis, cranial nerve palsies, and epidural and subdural hematomas caused by LP.14,15

CT scan technology has been rapidly evolving with the use of multidetector scanners and more sophisticated image processing software. Many of the frequently quoted studies on the sensitivity of CT in subarachnoid hemorrhage were published 10 or more years ago.9,10 A more recent study concluded that fifth-generation CT scanners are probably more sensitive than earlier scanners at detecting subarachnoid hemorrhage; however, only 6 patients had subarachnoid hemorrhage in this series.16

CT scan has never been evaluated specifically for the detection of subarachnoid hemorrhage as a result of arteriovenous malformation or aneurysm, the most important causes of subarachnoid hemorrhage. The purpose of this study is to further define the sensitivity of multidetector cranial CT in diagnosing subarachnoid hemorrhage in ED patients with various clinical presentations and among the subgroup of patients with arteriovenous vascular malformations and vascular aneurysms. Our hypothesis was that noncontrast multidetector cranial CT had adequate sensitivity (>99%) to rule out spontaneous aneurysmal subarachnoid hemorrhage so that a lumbar puncture was not necessary in patients with negative CT results.

MATERIALS AND METHODS

Study Design and Setting

The study was performed using a retrospective review of medical records from a tertiary academic medical center with an annual ED census of approximately 40,000 visits. A 4-slice 4-detector GE Light Speed Scanner (GE Healthcare, Chalfont St. Giles, Buckinghamshire, UK) was used at the time of the study. We do not know the specific type of scanner used at all transferring hospitals; however, a 4-slice 4-detector scanner was used at the 4 hospitals that transferred patients who had a negative CT scan result and were diagnosed by lumbar puncture. The study focused on capturing all patients diagnosed with subarachnoid hemorrhage in the ED during the 41 months between August 1, 2001, and December 31, 2004.

Selection of Participants

To evaluate our hypothesis, we identified all ED patients diagnosed with subarachnoid hemorrhage using noncontrast cranial CT, cerebrospinal fluid laboratory results, and discharge International Classification of Diseases, Ninth Revision (ICD-9) codes. We obtained from medical center data management services 3 databases for the study period: (1) all ED patients who had had a noncontrast cranial CT, including the radiology diagnostic coding; (2) all patients who had cerebrospinal fluid sent to the laboratory from the ED, including the cell count results of these cerebrospinal fluid studies (tube number, color of cerebrospinal fluid supernate, and RBC and WBC counts); and (3) all patients with discharge diagnoses ICD-9 codes for spontaneous subarachnoid hemorrhage.
(430) or cerebral aneurysm (437.3). From these data sets, we identified all patients who presented to the ED with a spontaneous subarachnoid hemorrhage that was diagnosed by CT or lumbar puncture and whether they had a cerebral aneurysm or arteriovenous malformation.

**Methods of Measurement and Data Collection and Processing**

From the ED cranial CT data set, we identified all patients diagnosed in the ED with a spontaneous subarachnoid hemorrhage by noncontrast cranial CT. To identify patients with a subarachnoid hemorrhage diagnosed by lumbar puncture, we identified all patients that had both cranial CT and cerebrospinal fluid sent to the laboratory from the ED within 24 hours of each other. From this subgroup, we identified all patients with cranial CT negative for intracranial blood per the initial radiology report and cerebrospinal fluid suggestive of intracranial bleeding, defined as greater than 200 RBCs or xanthochromic supernate. Traumatic taps, defined as having RBC counts decreasing greater than 30%, were excluded. Only patients with a clinical diagnosis of subarachnoid hemorrhage were included. If the patient had a neurosurgical procedure within 10 days of the ED visit, abnormal cerebrospinal fluid results were attributed to surgery.

Two abstractors independently reviewed the medical record of each of these patients, including the ED records, results of all neuroimaging studies, whether the patient was admitted, the neurosurgical admission history, and physical examination and discharge summary to confirm the diagnosis of subarachnoid hemorrhage. We classified patients with subarachnoid hemorrhage as presenting with headache and normal level of consciousness, with headache and abnormal level of consciousness, or with abnormal level of consciousness without headache. Headache was defined as present if it was included as the primary complaint in the triage note or the history of present illness. Level of consciousness was defined as normal if the Glasgow Coma Scale (GCS) score was 15 and the patient was described as awake and alert. All other patients were classified as having altered mental status. For instances in which disagreement occurred, a third party arbitrated and consensus was used. For patients who were transferred, we used the results of diagnostic studies at the transferring ED to classify patients as cranial CT negative, lumbar puncture positive if this was the case. For transferred patients, we also defined the presenting clinical complaint and level of consciousness from the initial presentation at the transferring ED. Key variables were age; sex; date of visit; ED-to-ED transfer; presence of headache; level of consciousness; history of aneurysm or arteriovenous malformation; results of all neuroimaging studies, including noncontrast cranial CT, CT angiography, magnetic resonance angiography, and cerebral angiography; ED lumbar puncture results; and ED or discharge diagnosis (subarachnoid hemorrhage, traumatic subarachnoid hemorrhage, no subarachnoid hemorrhage). Two of the authors (RLB and LJB) did practice runs to refine the data collection instrument and criteria for clinical classification before formal data abstraction. The investigators met and agreed about the definition of cases and classification criteria before beginning final data abstraction. The key clinical variables were defined by using a set of precise operational definitions of important variables, including subarachnoid hemorrhage, traumatic subarachnoid hemorrhage, normal level of consciousness, presence of headache, and presence of aneurysm or arteriovenous malformation. Missing data were coded as missing, except that a history of aneurysm or arteriovenous malformation was presumed to be absent if not noted anywhere in ED record, admission history and physical or discharge summary. There were meetings of three of the investigators (RLB, WRM, and LJB) to develop and review coding rules. The reviewers were not blinded to the purpose of the study.

All patients were classified as having a spontaneous subarachnoid hemorrhage, traumatic subarachnoid hemorrhage, aneurysm, or arteriovenous malformation independently by 2 investigators (RLB and LJB). Interater reliability was tested using the κ statistic. Discrepancies in classification were arbitrated by a third investigator (WRM).

**Primary Data Analysis**

Having identified all patients presenting to the ED with subarachnoid hemorrhage by noncontrast cranial CT or LP and knowing whether or not these patients had a cerebral aneurysm or arteriovenous malformation, we calculated a point estimate and associated 95% confidence intervals (CIs) for the sensitivity of cranial CT to identify all patients with subarachnoid hemorrhage and patients with subarachnoid hemorrhage as a result of cerebral aneurysm or arteriovenous malformation. We also determined the sensitivity of cranial CT scan as a function of presenting complaints: headache and normal mental status, headache and altered mental status, and altered mental status without history of headache. We used Stata 9.0 (StataCorp, College Station, TX) for data management and to perform these calculations.

This study was approved by the University of California Los Angeles Office for Protection of Research Subjects and met criteria for exemption from informed consent.

**RESULTS**

From 482 patients identified using the ED cranial CT, ED cerebrospinal fluid, and ICD-9 discharge diagnosis databases, we identified 149 patients presenting to the ED with spontaneous subarachnoid hemorrhage. The initial ED clinical presentation and results of diagnostic evaluation for the patients presenting to the ED with spontaneous subarachnoid hemorrhage are presented in Table 1. Patients ranged in age from 15 to 96 years, with a mean age of 53 years. Fifty-nine percent were female patients. During the 41-month study period, there were 149 patients who presented to the ED with a subarachnoid hemorrhage. Of these, 117 (79%) had vascular lesions, 112 (75%) with cerebral aneurysm and 5 (2%) with arteriovenous malformation. Eighty-seven of the 149 patients (58%) presented with headache and normal mental status; 67 (77%) of these had vascular lesions.

The relationship of initial ED clinical presentation and presence of vascular anomalies to initial cranial CT sensitivity is presented in Table 2. Noncontrast cranial CT scan identified 139 of 149
patients with subarachnoid hemorrhage, sensitivity = 93% (95% CI 88% to 97%). Noncontrast cranial CT scan identified 110 of 117 patients with subarachnoid hemorrhage and associated vascular anomaly, sensitivity = 94% (95% CI 88% to 98%). In the group of greatest interest, those with headache and normal mental status, the overall sensitivity of CT for subarachnoid hemorrhage was 90% (95% CI 81% to 95%). The sensitivity of CT for subarachnoid hemorrhage in patients with headache and normal mental status with vascular lesions was 91% (95% CI 82% to 97%). Cranial CT failed to demonstrate subarachnoid hemorrhage in only 1 of 61 patients (2%) with abnormal mental status, described below.

The clinical findings of the 10 patients with a negative CT result and a lumbar puncture suggestive of subarachnoid hemorrhage are presented in Table 3. All were ultimately diagnosed as having a subarachnoid hemorrhage. Five had cerebral aneurysms and 1 had an arteriovenous malformation. One patient, a 37-year-old woman with a history of systemic lupus who presented with headache and altered mental status (Glasgow Coma Scale score = 14), had an initially negative cranial CT result, but a subsequent CT and magnetic resonance imaging (MRI) study the day after admission were interpreted as showing a possible subarachnoid hemorrhage after the results of the lumbar puncture were known. Of the 10 subarachnoid hemorrhage patients diagnosed by lumbar puncture, 7 had symptoms for fewer than 24 hours and 4 had symptoms for fewer than 12 hours.

The proportion of subarachnoid hemorrhage due to vascular lesions was higher among patients diagnosed by CT (110/131 = 84%) than those diagnosed by lumbar puncture (7/10 = 70%). However, this difference in proportions was not statistically significant (95% CI of the difference in proportions

### Table 1. Patients with spontaneous subarachnoid hemorrhage: Initial ED clinical presentation and results of diagnostic evaluation.

<table>
<thead>
<tr>
<th>Clinical Presentation</th>
<th>Total</th>
<th>Aneurysm</th>
<th>Arteriovenous Malformation</th>
<th>No Vascular Anomaly</th>
<th>Died Before Vascular Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>149</td>
<td>112</td>
<td>5</td>
<td>24</td>
<td>8</td>
</tr>
<tr>
<td>Headache, normal mental status</td>
<td>87</td>
<td>64</td>
<td>3</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Focal seizure, normal mental status</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Headache, abnormal mental status</td>
<td>34</td>
<td>26</td>
<td>1</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal mental status, no headache</td>
<td>27</td>
<td>22</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
</tbody>
</table>

### Table 2. Spontaneous subarachnoid hemorrhage: Relationship of initial ED clinical presentation and presence of vascular anomalies to initial head CT sensitivity.

<table>
<thead>
<tr>
<th>Clinical Presentation</th>
<th>All Subarachnoid Hemorrhage</th>
<th>Head CT Result Positive</th>
<th>Head CT Sensitivity, % (95% CI)</th>
<th>Subarachnoid Hemorrhage and Vascular Anomaly</th>
<th>Head CT Result Positive</th>
<th>Head CT Sensitivity, % (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>149</td>
<td>139</td>
<td>93 (88–97)</td>
<td>117</td>
<td>110</td>
<td>94 (88–98)</td>
</tr>
<tr>
<td>Headache, normal mental status</td>
<td>87</td>
<td>78</td>
<td>90 (81–95)</td>
<td>67</td>
<td>61</td>
<td>91 (82–97)</td>
</tr>
<tr>
<td>Focal seizure, normal mental status</td>
<td>1</td>
<td>1</td>
<td>100 (2.5–100)</td>
<td>1</td>
<td>1</td>
<td>100 (2.5–100)</td>
</tr>
<tr>
<td>Headache, abnormal mental status</td>
<td>34</td>
<td>33</td>
<td>97 (85–100)</td>
<td>27</td>
<td>26</td>
<td>96 (81–100)</td>
</tr>
<tr>
<td>Abnormal mental status, no headache</td>
<td>27</td>
<td>27</td>
<td>100 (87–100)</td>
<td>22</td>
<td>22</td>
<td>100 (85–100)</td>
</tr>
</tbody>
</table>

### Table 3. ED patients presenting with headache and diagnosed with subarachnoid hemorrhage, with normal head CT and abnormal LP.

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Chief Complaint</th>
<th>GCS</th>
<th>Mental Status</th>
<th>Headache Duration</th>
<th>PMH*</th>
<th>CSF SUP</th>
<th>RBC Count, Tube 1</th>
<th>RBC Count, Tube 3</th>
<th>Vascular Anomaly</th>
</tr>
</thead>
<tbody>
<tr>
<td>42</td>
<td>M</td>
<td>Short LOC, then severe HA</td>
<td>15</td>
<td>Normal</td>
<td>&lt;12 h</td>
<td>None</td>
<td>na</td>
<td>70,000</td>
<td>na</td>
<td>Aneurysm</td>
</tr>
<tr>
<td>22</td>
<td>F</td>
<td>Sudden severe HA</td>
<td>15</td>
<td>Normal</td>
<td>&lt;12 h</td>
<td>None</td>
<td>xanho</td>
<td>370,000</td>
<td>86,000</td>
<td>Aneurysm</td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>Sudden severe HA, N&amp;V</td>
<td>15</td>
<td>Normal</td>
<td>&lt;12 h</td>
<td>None</td>
<td>na</td>
<td>RBCs</td>
<td>na</td>
<td>Aneurysm</td>
</tr>
<tr>
<td>79</td>
<td>F</td>
<td>Sudden severe HA</td>
<td>15</td>
<td>Normal</td>
<td>&gt;24 h</td>
<td>None</td>
<td>CC</td>
<td>93,500</td>
<td>112,000</td>
<td>Aneurysm</td>
</tr>
<tr>
<td>55</td>
<td>F</td>
<td>Sudden severe HA</td>
<td>15</td>
<td>Normal</td>
<td>3 days</td>
<td>None</td>
<td>CC</td>
<td>2,770</td>
<td>2,315</td>
<td>Aneurysm</td>
</tr>
<tr>
<td>44</td>
<td>F</td>
<td>Sudden severe HA, blurry vision</td>
<td>15</td>
<td>Normal</td>
<td>9 days</td>
<td>AVM</td>
<td>xanho</td>
<td>1,090</td>
<td>na</td>
<td>AVM</td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>Sudden severe HA</td>
<td>15</td>
<td>Normal</td>
<td>&lt;12 h</td>
<td>None</td>
<td>CC</td>
<td>16,900</td>
<td>14,000</td>
<td>No aneurysm</td>
</tr>
<tr>
<td>48</td>
<td>M</td>
<td>Severe neck pain; then severe HA</td>
<td>15</td>
<td>Normal</td>
<td>&lt;24 h</td>
<td>None</td>
<td>CC</td>
<td>710</td>
<td>700</td>
<td>No aneurysm</td>
</tr>
<tr>
<td>43</td>
<td>M</td>
<td>Sudden severe HA; then neck pain</td>
<td>15</td>
<td>Normal</td>
<td>3 days</td>
<td>None</td>
<td>CC</td>
<td>na</td>
<td>3,300</td>
<td>No aneurysm</td>
</tr>
<tr>
<td>37</td>
<td>F</td>
<td>Headache, AMS, systemic lupus</td>
<td>14</td>
<td>Drowsy</td>
<td>&lt;24 h</td>
<td>None</td>
<td>xanho</td>
<td>13,300</td>
<td>na</td>
<td>No aneurysm</td>
</tr>
</tbody>
</table>

_GCS_, Glasgow Coma Scale; _CSF_, cerebrospinal fluid; _PMH*, past medical history; _LOC_, loss of consciousness; _HA_, headache; _na_, not available; _AVM_, arteriovenous malformation; _CC_, clear colorless; _AMS_, altered mental status.

*PMH of vascular malformation.
–0.15% to 0.43%). Eighty percent (87/149) of the patients with subarachnoid hemorrhage were transferred from another hospital to our university hospital ED for higher level of care. There was no significant difference in the presenting clinical condition or the proportion with vascular lesions by transfer status.

The only missing clinical data were cerebrospinal counts on tubes 3 or 4 when tube 1 had greater than 1,000 RBCs. These were not available on a total of 5 patients diagnosed as subarachnoid hemorrhage by lumbar puncture. One was diagnosed with arteriovenous malformation and 2 with cerebral aneurysm by vascular imaging. The fourth patient had an MRI/magnetic resonance angiography that demonstrated a focal area of hyperintensity involving the left mesiotemporal lobe/uncus, interpreted as possibly representing a bleed, given the patient’s lumbar puncture results and his clinical diagnosis. The fifth patient’s discharge diagnoses included subarachnoid hemorrhage.

The agreement of classification of patients as having a spontaneous subarachnoid hemorrhage for the 2 reviewers was 93% (κ=0.85; 95% CI 0.80 to 0.90). The agreement for classification of patients with subarachnoid hemorrhage as having an aneurysm was 98% (κ=0.92; 95% CI 0.84 to 1.00). The observers were able to agree on the presence or absence of a normal mental status in 88% of cases (κ=0.75; 95% CI 0.63 to 0.88).

**LIMITATIONS**

Because academic centers are often acting on a radiology resident interpretation and some community practices are acting on the emergency physician interpretation, this could result in false low sensitivity of cranial CT for subarachnoid hemorrhage. We had 1 instance of lumbar puncture diagnosis of subarachnoid hemorrhage when the initial CT was reinterpreted as subarachnoid hemorrhage after a subsequent CT demonstrated a subarachnoid hemorrhage.

We excluded the 8 of 149 patients with subarachnoid hemorrhage who died before having a diagnostic angiographic study performed to confirm aneurysm or arteriovenous malformation from the analyses of the sensitivity of CT scan in detected subarachnoid hemorrhage associated with vascular malformations. All of these patients were diagnosed as having subarachnoid hemorrhage with CT. If one postulates that because perimesencephalic hemorrhage is not associated with a poor outcome, one could presume that all of these patients had vascular lesions, which would result in a higher overall sensitivity for cranial CT in detecting subarachnoid hemorrhage associated with vascular lesions (118/125=94%). All of these patients had altered mental status (GCS range 3 to 7; median=3); therefore, this would not influence the sensitivity of cranial CT in detecting subarachnoid hemorrhage associated with vascular abnormalities in the group with headache and normal mental status.

At our academic medical center, patients with possible subarachnoid hemorrhage are evaluated with CT and, if the CT is negative, lumbar puncture. However, if a patient refuses lumbar puncture, then none is performed. At referring hospitals, it may have been more likely that patients would be evaluated for subarachnoid hemorrhage without a lumbar puncture if the CT was negative. We did not attempt to identify ED patients who had a negative cranial CT to rule out subarachnoid hemorrhage and no lumbar puncture at our academic medical center, and we could not do this at referring hospitals. However, we did review the records of all patients who were diagnosed as having subarachnoid hemorrhage for previous ED visits for headache and found none with a negative previous CT result without lumbar puncture. The failure to perform lumbar punctures on patients with severe sudden headache who are CT-result negative will result in fewer patients being diagnosed with subarachnoid hemorrhage by lumbar puncture, resulting in a higher sensitivity of CT scan for subarachnoid hemorrhage.

**DISCUSSION**

Subarachnoid hemorrhage accounts for 3% of all strokes and 5% of total stroke deaths. Because subarachnoid hemorrhage occurs at an earlier age than other strokes, it accounts for more than 25% of potential lost years because of stroke. Prompt diagnosis and referral to surgery or angiographic intervention are important aspects in reducing patient morbidity and mortality. Unfortunately, misdiagnosis and delay in treatment occur in patients with subarachnoid hemorrhage. Kowalski et al demonstrated a missed diagnosis rate of 12%. Mayer et al reported a rate of missed subarachnoid hemorrhage in patients with headache of 25% from multiple centers in Connecticut. Between 12% and 53% of patients with subarachnoid hemorrhages are not diagnosed on their initial presentation for medical care. The failure to diagnose subarachnoid hemorrhage results in catastrophic outcomes in the affected patients. Patients with subarachnoid hemorrhage in whom the diagnosis was initially missed had an unadjusted odds ratio of 3.4 of death at 3 months and 4.7 at 12 months compared to those of similar clinical illness who were correctly diagnosed. Half of those missed had deterioration in their mental status as opposed to only 2.5% with a correct ED diagnosis. This leaves the emergency physician in a difficult position. Benign headaches greatly outnumber those caused by subarachnoid hemorrhage; migraine headache outnumbers subarachnoid hemorrhage by 50 to 1.

Spontaneous subarachnoid hemorrhage is primarily caused by 3 disorders: primarily cerebral aneurysms, arteriovenous malformations, and perimesencephalic hemorrhages. Cerebral aneurysms and arteriovenous malformations are particularly important lesions because they are potentially amenable to treatment and are associated with significant morbidity and mortality. Perimesencephalic hemorrhages are usually of limited clinical significance and do not generally require specific treatment. Consequently, clinicians have focused on identifying patients who harbor aneurysms and arteriovenous malformations as the cause of their sudden-onset severe headache. Many patients with subarachnoid hemorrhage have already sustained severe permanent brain injury by the time of their initial evaluation. Neurologic impairments make it relatively easy to identify such patients, but their injuries limit the benefit that they receive from therapeutic intervention. In contrast, patients who present with normal neurologic function, including many of those...
presenting with “sentinel bleeds,” could arguably receive the greatest benefit from the detection and management of their vascular lesions. This situation creates a diagnostic asymmetry in which greatest benefit is obtained in identifying cases in which evidence of disease is minimal and less benefit is obtained in diagnosing more obvious cases. This situation is exacerbated by CT’s ability to detect subarachnoid hemorrhage. Our data reveal that multidetector scanners have an overall sensitivity of 94% in detecting subarachnoid hemorrhage caused by arteriovenous malformation or aneurysm. We found that sensitivity was lower among patients with normal mentation, in which CT imaging failed to detect subarachnoid hemorrhage in nearly 10% of these patients who harbored arteriovenous malformations or aneurysms.

Although we set out to demonstrate that modern noncontrast CT had improved to the point at which it could exclude spontaneous subarachnoid hemorrhage without further testing, our results do not support this hypothesis. Our findings have direct implications to clinical practice and the evaluation of patients with sudden-onset severe headache. Although clinicians may elect to use CT as an initial diagnostic modality, a negative study result does not preclude the presence of an underlying subarachnoid hemorrhage caused by rupture of an aneurysm or arteriovenous malformation. Patients with negative imaging study results, particularly those with normal neurologic examination results, must undergo further evaluation to exclude such lesions. We would continue to recommend lumbar puncture in patients with a negative cranial CT result.

Our findings also raise questions about the choice of initial diagnostic modality. The majority of patients who undergo CT imaging will have negative study results and will ultimately require lumbar puncture to exclude subarachnoid hemorrhage. Patients who have normal neurologic examinations are at greatest risk of having their aneurysms and arteriovenous malformations missed by imaging and are also unlikely to have lesions that would preclude lumbar puncture. Consequently, many emergency clinicians may elect to use lumbar puncture as the initial diagnostic study in such patients and abort any further diagnostic testing for patients who have normal cerebrospinal fluid results. Lumbar puncture is considered to be 100% sensitive for the diagnosis of subarachnoid hemorrhage. There is limited evidence to support this view. Wijdicks et al followed 71 subjects with normal lumbar puncture for an average of 3.3 years to determine that there were no sequelae and concluded that if CT and cerebrospinal fluid findings are normal, cerebral angiography is not indicated. Lumbar puncture is not highly specific. Reported rates of traumatic lumbar puncture are between 10% and 20%, depending on the definition of “traumatic.” A decrease in the number of RBCs between tubes 1 and 4 has been shown to be unreliable, and blood from a traumatic tap can lyse and cause xanthochromia in the supernatant, especially if time to analysis is prolonged. There is no standard for number of RBCs considered consistent with a subarachnoid hemorrhage. Some authors have recommended admitting or observing patients for 6 to 12 hours to allow xanthochromia to appear.

The criterion standard for detection of intracranial vascular malformations, including saccular aneurysm, is cerebral angiography. This diagnostic procedure is associated with a combined permanent and transient complication rate of 1.8%. Permanent neurologic dysfunction from resulting stroke is reported to occur in 0.5%. To avoid these risks, CT angiogram is often used to detect vascular malformations. In a recent study, CT angiogram was found to be useful in the diagnosis of aneurysmal subarachnoid hemorrhage. Six of 116 patients had aneurysm found on CT angiogram, with normal CT and positive lumbar puncture; 3 had a positive CT angiogram result with normal CT and lumbar puncture findings (1 of which had a negative cerebral angiogram result). In a blinded prospective study, 40 patients with known or suspected intracranial saccular aneurysms underwent both CT angiogram and DSA. The sensitivity and specificity for CT angiogram for aneurysm were 86% and 90%, respectively. Thus, CT angiogram cannot be relied on to replace lumbar puncture in the ED diagnosis of subarachnoid hemorrhage because it lacks sensitivity and detects aneurysms that have probably not ruptured.

Until there is a significant improvement in the ability of CT imaging to detect subarachnoid blood, the most promising area in deciding who to assess for possible spontaneous subarachnoid hemorrhage and who in the face of a negative cranial CT should have further diagnostic testing is the development of a risk-scoring system. A highly accurate scoring system could help clinicians in 2 ways. First, physicians would be able to use scores to make a more informed decision about which patients should be investigated for spontaneous subarachnoid hemorrhage. These scores could also be used to assign a posttest probability for subarachnoid hemorrhage and provide physicians and patients with valuable information to guide further testing. Unfortunately, with its derivation and validation components, and the fact that we were able to identify only 149 patients in 41 months at a neurosurgical referral center, such a study would likely need to be a large-scale investigation at multiple medical centers.

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