Does This Patient Have a Hemorrhagic Stroke?: Clinical Findings Distinguishing Hemorrhagic Stroke From Ischemic Stroke

Shauna Runchey; Steven McGee


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Does This Patient Have a Hemorrhagic Stroke?  
Clinical Findings Distinguishing Hemorrhagic Stroke From Ischemic Stroke

Shauna Runchey, MD, MPH  
Steven McGee, MD

**Context** The 2 fundamental subtypes of stroke are hemorrhagic stroke and ischemic stroke. Although neuroimaging is required to distinguish these subtypes, the diagnostic accuracy of bedside findings has not been systematically reviewed.

**Objective** To determine the accuracy of clinical examination in distinguishing hemorrhagic stroke from ischemic stroke.

**Data Sources** MEDLINE and EMBASE searches of English-language articles published from January 1966 to April 2010.

**Study Selection** Prospective studies of adult patients with stroke that compared initial clinical findings with accepted diagnostic standards of hemorrhagic stroke (computed tomography or autopsy).

**Data Extraction** Both authors independently appraised study quality and extracted relevant data.

**Data Synthesis** Nineteen prospective studies meeting inclusion criteria were identified (N=6438 patients; n=1528 [24%] with hemorrhage stroke). Several findings significantly increase the probability of hemorrhagic stroke: coma (likelihood ratio [LR], 6.2; 95% confidence interval [CI], 3.2-12), neck stiffness (LR, 5.0; 95% CI, 1.9-12.8), seizures accompanying the neurologic deficit (LR, 4.7; 95% CI, 1.6-14), diastolic blood pressure greater than 110 mm Hg (LR, 4.3; 95% CI, 1.4-14), vomiting (LR, 3.0; 95% CI, 1.7-5.5), and headache (LR, 2.9; 95% CI, 1.7-4.8). Other findings decrease the probability of hemorrhage: cervical bruit (LR, 0.12; 95% CI, 0.03-0.47) and prior transient ischemic attack (LR, 0.34; 95% CI, 0.18-0.65). A Siriraj score greater than 1 increases the probability of hemorrhage (LR, 0.34; 95% CI, 0.18-0.65) while a score lower than −1 decreases the probability (LR, 0.29; 95% CI, 0.23-0.37). Nonetheless, many patients with stroke lack any diagnostic finding, and 20% have Siriraj scores between 1 and −1, which are diagnostically unhelpful (LR, 0.94; 95% CI, 0.77-1.1).

**Conclusion** In patients with acute stroke, certain findings accurately increase or decrease the probability of intracranial hemorrhage, but no finding or combination of findings is definitively diagnostic in all patients, and diagnostic certainty requires neuroimaging.

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**Case 1**

A 75-year-old woman with hypertension, hyperlipidemia, and diabetes mellitus presents to the emergency department with speech difficulty and right arm weakness. Her symptoms appeared abruptly 6 hours earlier, when she awoke in the morning, but several hours passed before she could reach her daughter, who then transported her to the hospital. One year ago she was diagnosed as having a transient ischemic attack after presenting with a 10-minute episode of right-hand incoordination and numbness; subsequent neuroimaging and cardiovascular studies at that time were unrevealing. In the emergency department, her blood pressure is 140/75 mm Hg and her pulse is 70/min and regular. She is alert and oriented but struggles to generate words or name objects. She reports a mild left hemispheric headache. She exhibits right central facial paralysis and right arm weakness. Both great toes are downgoing. Do her findings suggest hemorrhagic or ischemic infarction? How accurate are these findings?

**Case 2**

A 62-year-old man presents to the emergency department with new-onset left arm and leg weakness. While working in his workshop 2 hours earlier, he experienced abrupt onset of a severe headache. Within 10 to 15 minutes, he had difficulty holding tools in his left hand.
Clinicians must consider hemorrhage due to hemorrhage may be as small as 25% to 60%.2,3 In the United States, new or recurrent stroke affects about 795,000 individuals each year and represents the third leading cause of death.1 The 2 fundamental subtypes of stroke are hemorrhagic stroke (ie, either intracerebral or subarachnoid hemorrhage) and ischemic stroke (ie, infarction from thrombosis or embolism). In the United States, 87% of these vascular strokes are ischemic and 13% are hemorrhagic (10% are intracerebral hemorrhage; 3% are subarachnoid hemorrhage).1 Although hospital-based surveys from other countries suggest that the percentage due to hemorrhage may be as high as 25% to 60%,2,3 clinicians must rapidly distinguish these 2 vascular subtypes because they have distinct etiologies, prognoses, and treatments.

Herein, we assume that the clinical diagnosis of stroke has already been made, a subject extensively reviewed in a prior Rational Clinical Examination article.4 Classic descriptions of stroke suggest that some clinical features may differentiate these subtypes: headache, neck stiffness, vomiting, and coma are more common in hemorrhagic stroke whereas previous transient ischémic attacks, atrial fibrillation, and atherosclerosis risk factors are more common in ischemic stroke. Clinicians frequently examine patients with these findings in mind to form a clinical impression of whether hemorrhage or ischemia is more likely. Even so, all patients with new strokes must undergo immediate computed tomography (CT) of the head, the best test that rapidly distinguishes intracerebral hemorrhage from ischemia.

In patients presenting early enough to be considered for thrombolytic therapy, the clinical priorities are urgent CT imaging, stabilization of the patient, identification of stroke mimics (eg, migraine, seizures, hypoglycemia) and contraindications to thrombolysis, and measurement of the neurologic deficit.2 In such patients, a clinician's impression of hemorrhage vs ischemia is secondary to these more pressing matters. Nonetheless, 3 of 4 patients presenting to emergency departments with stroke are already beyond the narrow time window for thrombolysis,6 and in these patients the clinical overall impression may be helpful while awaiting the CT results. The clinical impression also is essential when CT is not immediately available.2,3 Finally, clinical impression is even important in patients receiving thrombolitics. According to treatment guidelines, the infusion should be immediately discontinued (and imaging repeated) if severe headache, acute hypertension, nausea, or vomiting develop,7 a statement implying that these symptoms and signs suggest hemorrhage.

The diagnostic accuracy of clinical findings in distinguishing hemorrhagic and ischemic stroke has not been systematically reviewed. In this article, we summarize the diagnostic accuracy of all available clinical information that is easily obtainable to clinicians at the bedside—initial patient interview, physical examination, and basic laboratory tests. In addition, we review the diagnostic accuracy of different stroke scores that combine clinical information to predict the risk of hemorrhagic infarction.

**Physiologic Origins of Symptoms and Signs**
Both cerebral hemorrhage and infarction cause abrupt dysfunction of neurologic tissue, leading to neurologic deficits such as hemiparesis, hemisensory loss, aphasia, ophthalmoplegia, and visual field cuts. Cerebral hemorrhage, in contrast, also causes leakage of blood into the brain, displacing and compressing adjacent tissue, increasing intracranial pressure, and eventually dissecting into the ventricles and subarachnoid space (FIGURE). Consequently, hemorrhage may cause additional symptoms beyond neurologic deficits, such as severe headache (from increased intracranial pressure or meningeal irritation), progressive deterioration after stroke onset (from continued bleeding), vomiting (from increased intracranial pressure), neck stiffness (from meningeal irritation), bilateral Babinski signs (from enlargement of the hemorrhage beyond the distribution of a single vessel), and coma (from bilateral cerebral dysfunction or uncal herniation).

**METHODS**

**Literature Search Strategy**
One author (S.R.) searched PubMed (1970 to April 2010), using MEDLINE, and EMBASE (1988 to April 2010), using OVID, to identify English-language studies that evaluated the diagnostic accuracy of clinical history and physical signs for detecting intracerebral hemorrhage. The specific search strategy for PubMed was (Stroke/diagnosis[Mesh] OR Intracranial Embolism and Thrombosis/diagnosis[Mesh]) AND Intracranial Hemorrhages/diagnosis[Mesh]) and for EMBASE was (exp *Stroke/et, di [Etiology, Diagnosis] or exp Brain Hemorrhage/et, di [Etiology, Diagnosis] or exp Brain Infarction/et, di [Etiology, Diagnosis] and diagnostic accuracy/ or diagnostic value/). Additional articles were identified from liberal use of the search tool “all related articles” in PubMed and examination of bibliographies of selected articles.

**Study Selection**
Both authors independently read all articles relevant to the study question and selected those representing unique patient populations meeting the following inclusion criteria: (1) The study prospectively enrolled only patients presenting to clinicians in a hospital or emergency department with a diagnosis of stroke, defined as focal...
Figure. Explanation of Additional Symptoms of Intracerebral Hemorrhage

<table>
<thead>
<tr>
<th>Pathophysiologial events</th>
<th>Clinical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial hemorrhage</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hemiparesis</td>
</tr>
<tr>
<td><strong>Continued bleeding, compression of adjacent tissues, and increased intracranial pressure</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Headache, vomiting, Hemiplegia, Drowsiness</td>
</tr>
<tr>
<td><strong>Dissection of blood into the ventricles and extension of blood into the subarachnoid space; uncal herniation</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neck stiffness, Coma</td>
</tr>
</tbody>
</table>

In the top panel, a small hemorrhage in the right basal ganglia causes left hemiparesis and a clinical presentation indistinguishable from ischemic stroke. As intracerebral bleeding continues (middle panel), expansion of the hemorrhage exerts a mass effect on the brain, increasing intracranial pressure and causing a midline shift. Clinical findings characteristic of hemorrhagic stroke manifest, such as progressive neurological deficits, headache, and vomiting. Eventually, blood may dissect into the ventricles and extend into the subarachnoid space via the median and lateral apertures of the fourth ventricle (bottom panel), leading to neck stiffness. In severe hemorrhagic stroke, intracerebral expansion of the hemorrhage may result in coma from bilateral cerebral dysfunction or uncal herniation.
(or at times global) neurologic impairment of sudden onset and of presumed vascular origin (World Health Organization definition). 7 (2) The study used either neuroimaging (CT or magnetic resonance imaging) or autopsy to distinguish hemorrhage from ischemia (all but 1 study 8 exclusively used CT). (3) Intracranial hemorrhage referred only to intracerebral hemorrhage (studies enrolling patients with subarachnoid hemorrhage were excluded). (4) The study presented sufficient information to create 2 × 2 tables and calculate sensitivity, specificity, and likelihood ratios (LRs). Each study was assigned a quality grade similar to other articles in the Rational Clinical Examination series 6 according to whether sample sizes were adequate, cases were consecutive, and comparison with the diagnostic standard was independent (in assigning quality grades, we considered studies with ≥100 total patients and ≥30 patients with hemorrhage to be large; see eTable 1 [available at http://www.jama.com]). In all studies, patients were enrolled at the time of presentation and clinical data were collected before the criterion standard for the subtype of stroke (hemorrhage vs ischemia). No patient received thrombolytic medications. Several investigators, 8,10-18 supplied additional unpublished information from their original studies.

Statistical Analyses
Both authors independently extracted data from each study to calculate sensitivity, specificity, and positive and negative LRs using standard definitions. 19 Any differences in data entry were settled by consensus. If any cell in the 2 × 2 table had the value of zero, 0.5 was added to all cells before calculating LRs or pooled estimates. Pooled estimates were calculated using the Der-Simonian and Laird random-effects model. 20 Data are presented using LRs because they function as “diagnostic weights” that are easily translated to posttest probability of disease. 21 Excel 2007 (Microsoft, Redmond, Washington) was used for statistical analyses.

RESULTS
Study Characteristics
We retrieved 109 citations for full-text review, 19 of which met our inclusion criteria and presented data from 6438 patients worldwide with stroke (eFigure and eTable 1). The methods and enrollment of these studies varied widely. The maximal allowed interval between onset of neurologic symptoms and CT scanning was less than 3 days in 68% of studies, less than 15 days in 16%, and 15 days or more or unknown in 16%.

We identified 6 stroke scores that combined clinical findings to calculate probability of hemorrhagic stroke. 10,17,22-25 Three were excluded because they lacked extensive external validation beyond the original cohort. 22,23,25 A fourth score, the once popular Allen (Guy’s Hospital) score, was excluded because it required clinical information first available 24 hours after presentation, which is clinically unrealistic. 24 The definitions of the included stroke scores appear in Table 1.

Prevalence of Hemorrhage Stroke in Patients Presenting With Stroke
The prevalence of hemorrhagic stroke in these studies was 24% (n = 1528), with individual study prevalences ranging from 12% to 57% (random-effects summary prevalence, 29%; 95% confidence interval [CI], 24%-35%). In general, the prevalence of hemorrhage is lower in studies from the United States and Europe (summary prevalence, 15%; 95% CI, 13%-18%) than in those from Asia and Africa (summary prevalence, 34%; 95% CI, 27%-41%). Some of this difference probably reflects real differences around the world but it also may indicate referral bias (ie, in countries with more limited access to CT and thrombolytic therapy, patients thought to have hemorrhage may be more likely referred for CT). A small number of patients (range, 2.0%-2.9%) experienced a stroke mimic that was neither hemorrhagic nor ischemic (eg, attributable to tumor, subdural hematoma, or tuberculosis). 13,26,27,35

Individual Findings
Several findings significantly increase the probability of hemorrhagic stroke (LR 93% CI does not cross 1.0). In order of their LRs (the higher the value of the LR, the greater the increase in probability of hemorrhagic stroke),

Table 1. Definitions and Diagnostic Accuracy of Stroke Scores Detecting Hemorrhage: Pooled Results

<table>
<thead>
<tr>
<th>Score</th>
<th>No. of Patients</th>
<th>Hemorrhages, No. (%)</th>
<th>Definitionb</th>
<th>Threshold Values</th>
<th>LR for Hemorrhage (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Siraj stroke score 10-18</td>
<td>3439</td>
<td>1051 (31)</td>
<td>(2.5 × semicoma or 5 × coma) + (2 × vomiting) + (2 × headache within 2 h) + (0.1 × diastolic blood pressure) – (3 × ≥1 of diabetes, angina, intermittent claudication) – 12</td>
<td>&lt;1: infarction</td>
<td>0.29 (0.23-0.37)</td>
</tr>
<tr>
<td>Besson score 10,14</td>
<td>261</td>
<td>46 (18)</td>
<td>(2 × alcohol consumption) + (1.5 × plantar response both extensor + (3 × headache) + (3 × history of hypertension) – (5 × history of transient ischemic attack) – (2 × peripheral arterial disease) – (1.5 × history of hyperlipidemia) – (2.5 × atrial fibrillation on admission)</td>
<td>&lt;1: infarction</td>
<td>0.23 (0.01-5)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; LR, likelihood ratio.

See eTable 2 for results from the individual studies.

Within each set of parentheses is the number of points awarded if the criterion following the times sign is satisfied. For example, in the Siraj stroke score, (2.5 × semicoma or 5 × coma) means 2.5 points are awarded if the patient has semicoma or coma if the patient has coma.
these findings include coma (LR, 6.2; 95% CI, 3.2-12), neck stiffness (LR, 5.0; 95% CI, 1.9-12.8), seizures accompanying neurologic deficit (LR, 4.7; 95% CI, 1.6-14), diastolic blood pressure greater than 110 mm Hg (LR, 4.3; 95% CI, 1.4-14), vomiting (LR, 3.0; 95% CI, 1.7-5.5), headache (LR, 2.9; 95% CI, 1.7-4.8), and loss of consciousness (LR, 2.6; 95% CI, 1.6-4.2) (Table 2 and eTable 3). The finding of xanthochromia during lumbar puncture also greatly increases the probability of hemorrhage (LR, 15; 95% CI, 7.7-29), although this test is no longer routinely performed to identify intracranial hemorrhage. The findings of diastolic blood pressure greater than 110 mm Hg, loss of consciousness, and xanthochromia were each investigated in only a single study.

Several findings significantly decrease the probability of hemorrhagic stroke, thus increasing the probability of ischemic stroke. These findings include the presence of a cervical bruit (LR, 0.12; 95% CI, 0.03-0.47), ab

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**Table 2.** Accuracy of Findings for Diagnosing Hemorrhagic Stroke

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>No. of Patients</th>
<th>Hemorrhage, No. (%)</th>
<th>Sensitivity, % (95% CI)</th>
<th>Specificity, % (95% CI)</th>
<th>Positive LR (95% CI)</th>
<th>Negative LR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≤60 y</td>
<td>2497</td>
<td>356 (14)</td>
<td>9 (6-12)</td>
<td>93 (97-100)</td>
<td>1.6 (1.4-1.9)</td>
<td>0.93 (0.9-0.96)</td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>2947</td>
<td>577 (23)</td>
<td>34 (17-52)</td>
<td>93 (90-96)</td>
<td>3.0 (1.7-5.5)</td>
<td>0.73 (0.59-0.91)</td>
</tr>
<tr>
<td>Male</td>
<td>3974</td>
<td>708 (18)</td>
<td>46 (41-52)</td>
<td>82 (75-89)</td>
<td>2.9 (1.7-4.8)</td>
<td>0.66 (0.56-0.77)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>174</td>
<td>75 (43)</td>
<td>47 (35-58)</td>
<td>82 (74-89)</td>
<td>2.6 (1.6-4.2)</td>
<td>0.65 (0.52-0.82)</td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>887</td>
<td>109 (12)</td>
<td>44 (35-53)</td>
<td>89 (79-99)</td>
<td>0.65 (0.52-0.81)</td>
<td>1.7 (1.4-2.1)</td>
</tr>
<tr>
<td>Acute onset of deficit</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seizures accompanying neurologic deficit</td>
<td>302</td>
<td>200 (66)</td>
<td>12 (9-16)</td>
<td>93 (91-95)</td>
<td>2.0 (1.0-4.0)</td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>302</td>
<td>200 (66)</td>
<td>12 (9-16)</td>
<td>93 (91-95)</td>
<td>2.0 (1.0-4.0)</td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td>302</td>
<td>200 (66)</td>
<td>12 (9-16)</td>
<td>93 (91-95)</td>
<td>2.0 (1.0-4.0)</td>
<td></td>
</tr>
<tr>
<td>Loss of consciousness</td>
<td>302</td>
<td>200 (66)</td>
<td>12 (9-16)</td>
<td>93 (91-95)</td>
<td>2.0 (1.0-4.0)</td>
<td></td>
</tr>
<tr>
<td>Acute onset of deficit</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Physical signs</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Kernig sign, Brudzinski sign, or both</td>
<td>50</td>
<td>23 (46)</td>
<td>15 (9-29)</td>
<td>93 (93-100)</td>
<td>8.2 (4.4-150)</td>
<td>0.87 (0.73-1.0)</td>
</tr>
<tr>
<td>Level of consciousness: coma</td>
<td>1161</td>
<td>223 (19)</td>
<td>35 (19-50)</td>
<td>94 (89-99)</td>
<td>6.2 (3.2-12)</td>
<td></td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>223</td>
<td>97 (43)</td>
<td>20 (12-28)</td>
<td>97 (93-100)</td>
<td>5.0 (1.9-12.8)</td>
<td>0.83 (0.75-0.92)</td>
</tr>
<tr>
<td>Diastolic blood pressure &gt;110 mm Hg</td>
<td>50</td>
<td>23 (46)</td>
<td>15 (9-29)</td>
<td>93 (93-100)</td>
<td>8.2 (4.4-150)</td>
<td>0.87 (0.73-1.0)</td>
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<tr>
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<td>35 (19-50)</td>
<td>94 (89-99)</td>
<td>6.2 (3.2-12)</td>
<td></td>
</tr>
<tr>
<td>Plantar response: both extensor</td>
<td>370</td>
<td>106 (29)</td>
<td>16 (9-23)</td>
<td>92 (89-96)</td>
<td>1.8 (0.99-3.4)</td>
<td></td>
</tr>
<tr>
<td>Plantar response: single extensor</td>
<td>370</td>
<td>106 (29)</td>
<td>16 (9-23)</td>
<td>92 (89-96)</td>
<td>1.8 (0.99-3.4)</td>
<td></td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>3420</td>
<td>523 (15)</td>
<td>63 (25-100)</td>
<td>93 (90-96)</td>
<td>0.96 (0.9-1.0)</td>
<td>1.1 (1.0-1.2)</td>
</tr>
<tr>
<td>Plantar response: both flexor</td>
<td>370</td>
<td>106 (29)</td>
<td>16 (9-23)</td>
<td>92 (89-96)</td>
<td>1.8 (0.99-3.4)</td>
<td></td>
</tr>
<tr>
<td>Level of consciousness: alert</td>
<td>274</td>
<td>114 (42)</td>
<td>23 (15-30)</td>
<td>93 (91-94)</td>
<td>0.12 (0.03-0.47)</td>
<td>1.1 (1.0-1.1)</td>
</tr>
<tr>
<td>Cervical bruit</td>
<td>1510</td>
<td>237 (16)</td>
<td>1 (0-2)</td>
<td>93 (91-94)</td>
<td>0.12 (0.03-0.47)</td>
<td>1.1 (1.0-1.1)</td>
</tr>
<tr>
<td>Laboratory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Xanthochromia in cerebrospinal fluid</td>
<td>231</td>
<td>41 (18)</td>
<td>71 (57-85)</td>
<td>96 (92-98)</td>
<td>15 (7.7-29)</td>
<td>0.31 (0.19-0.49)</td>
</tr>
<tr>
<td>Atrial fibrillation on electrocardiogram</td>
<td>1087</td>
<td>142 (13)</td>
<td>2 (0-4)</td>
<td>93 (94-99)</td>
<td>0.19 (0.06-0.59)</td>
<td>1.2 (1.0-1.5)</td>
</tr>
<tr>
<td>Clinician’s overall impression Hemorrhage most likely diagnosis</td>
<td>300</td>
<td>111 (37)</td>
<td>76 (68-84)</td>
<td>88 (83-92)</td>
<td>6.2 (4.2-9.3)</td>
<td>0.28 (0.20-0.39)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; LR, likelihood ratio.

*See eTable 3 for results from the individual studies.*
ance of xanthochromia on lumbar puncture (LR, 0.31; 95% CI, 0.19-0.49), history of transient ischemic attack (LR, 0.34; 95% CI, 0.18-0.65), peripheral artery disease (LR, 0.41; 95% CI, 0.2-0.83), and history of atrial fibrillation (LR, 0.44; 95% CI, 0.25-0.78). The finding of cerebral viral was investigated in only 1 study.

**Combinations of Findings**

The diagnostic accuracies of the Besnon score (2 studies) and the Siriraj score (13 studies) are shown in Table 1. The Siriraj score is most accurate, increasing the probability of hemorrhage when it indicates hemorrhage (LR, 5.7; 95% CI, 4.4-7.4) and decreasing it when it indicates ischemic infarction (LR, 0.29; 95% CI, 0.23-0.37). Nonetheless, in all patients presenting with stroke, 20% were classified as uncertain by the Siriraj score (LR, 0.94; 95% CI, 0.77-1.1), a diagnostically unhelpful result because it changes probability of hemorrhage little or not at all (LRs near the value of 1.0 do not change probability).

**Overall Clinical Impression**

In a single study, clinician overall impression of hemorrhage (LR, 6.2; 95% CI, 4.2-9.3) or ischemia (LR, 0.28; 95% CI, 0.20-0.39), without using explicit rules or allowing classification into an “uncertain” category, was as accurate as the Siriraj score. This suggests that these experienced clinicians are using findings not addressed in our study, combining them in nonlinear ways, or deriving greater accuracy from more thorough familiarity with the actual patients seen in these studies.

**Limitations**

One potential limitation is the methodological diversity of these studies, making it unwise to pool results, but when we reexamined the Siriraj score in various subgroups—patients receiving CT within 72 hours, studies meeting grade 1 criteria, or studies meeting grade 2 or 3 criteria—the LRs were largely unchanged (eTable 4). Also, we found no correlation between prevalence of hemorrhage in a particular study and that study’s hemorrhage LR (P = .38 by Pearson correlation) or ratio of hemorrhage LR to infarction LR, an indicator of overall discriminatory power (P = .47).

Another concern might be the definition of stroke used by most investigators, a post hoc definition by the World Health Organization requiring that neurologic deficits persist for longer than 24 hours. This raises the possibility that our conclusions may not apply to patients presenting to emergency departments with only 1 to 2 hours of symptoms. Nonetheless, we believe that the patients in these studies are similar to those with shorter duration of symptoms for the following reasons. First, the 24-hour time limit was based on an outdated and arbitrary distinction between transient ischemic attack and stroke that no longer is applicable and probably was misleading because most patients with transient ischemic attack have less than 30 minutes of symptoms and their symptoms are unwitnessed by physicians.36 Second, few patients with only 1 to 2 hours of symptoms experience complete resolution by 24 hours (unless they receive treatment). In the placebo group of the National Institute of Neurological Disorders and Stroke trial of recombinant human tissue plasminogen activator, only 2% of patients presenting with less than 3 hours of neurologic deficits had resolution of findings at 24 hours.37

**SCENARIO RESOLUTION**

**Case 1**

The patient’s findings suggest ischemic stroke. Her pretest probability of hemorrhagic stroke is 13% (in the United States), and the findings of previous transient ischemic attack, alert mental status, and absence of severe hypertension or Babinski sign decrease the probability of hemorrhagic stroke even further. Even with the presence of headache, her calculated Siriraj score is −5.5, which is test-positive for ischemic infarction (scores >−1: LR, 0.29), decreasing her probability of hemorrhagic infarction from 13% to 4%. Her CT scan reveals an infarction in the left posterior frontal lobe.

**Case 2**

The patient’s findings of headache, severe hypertension, emesis, drowsiness, and rapid neurologic progression all strongly suggest hemorrhagic stroke, and the calculated Siriraj score is 5.3, which is test-positive for hemorrhage (scores >1: LR, 5.7), a result that increases the probability of hemorrhage from 13% to 46%. In this patient, urgent CT scanning reveals an intracerebral hemorrhage centered in the right basal ganglia, with surrounding edema and shift of the midline structures to the left.

**CLINICAL BOTTOM LINE**

Other than previous transient ischemic attack, classic risk factors for cerebral atherosclerosis—such as diabetes mellitus, hyperlipidemia, cigarette smoking, and hypertension—fail to distinguish the 2 subtypes of stroke, either because the risk factor is only weakly linked to ischemic stroke (eg, in the Framingham Stroke Profile, a tool for assessing risk of stroke, diabetes or cigarette smoking increases the risk of stroke only half as much as atrial fibrillation) or because the risk factor is linked to both hemorrhage and ischemia, thus nullifying its diagnostic value (eg, hypertension is linked to both cerebral atherosclerosis—and, thus, ischemia—and intracerebral hemorrhage).

We conclude that in patients with stroke, additional clinical findings characteristic of hemorrhage—headache, vomiting, severe hypertension, neck stiffness, and coma—increase the probability of hemorrhagic stroke. Even so, many patients with hemorrhage lack any distinctive findings or are unclassifiable by stroke scores. Neither the clinical impression of experienced clinicians nor the most accurate stroke score can improve the posttest probability of hemorrhage to greater than 50%. While combinations of findings are more predictive than individual
Distinguishing Hemorrhagic and Ischemic Strokes

Findings, Diagnostic Certainty Requires Neuroimaging

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References