When I began practicing emergency medicine 25 years ago, a stroke patient’s arrival to the emergency department (ED) did not elicit the frenetic burst of activity that it currently does. For that matter, neither did the arrival of a heart attack patient—until the approval of thrombolytic treatment for myocardial infarction. For patients with cerebrovascular emergencies, new treatment options such as thrombolitics and pro-coagulants demand earlier and accurate diagnosis.

The spectrum of presentation of SAH ranges from an isolated headache to sudden death. Patients with obvious focal or generalized neurological deficits usually present little diagnostic difficulty because these signs usually prompt brain imaging or other diagnostic tests and neurological consultation that lead to correct diagnosis. The problem is with the other end of the spectrum.

Approximately 40% of SAH patients present with a primary complaint of headache, an extremely common symptom in EDs. The vast majority of headaches are attributable to primary disorders such as migraine or tension-type headache. Thus, front-line physicians must develop strategies to distinguish the small number of headache patients who have SAH (or other causes of life, limb, brain, or vision threatening headache) from the far larger group with benign causes.

Consider also the extensive literature on the so-called “warning leak” or “sentinel bleed.” Because we know that patients with severe, unusual and sudden-onset headache have excellent outcomes if they have normal CT scans and cerebrospinal fluid, these “leaks” and “bleeds” are likely just that: small SAHs that are not worked up and therefore not diagnosed. It is unlikely that all of these “warning” phenomena are attributable to recall bias.

In 2004, the largest single-institution series on SAH misdiagnosis by Kowalski et al was mostly consistent with previous work. The most common misdiagnoses were migraine and tension headache. Good grade (Hunt&Hess grade 1 and 2) patients were most commonly misdiagnosed. Not surprisingly, their outcomes were worse than in correctly diagnosed good-grade patients. Their major finding, that only 12% of the total (56/482) patients were misdiagnosed, is a lower figure than in all earlier studies.

Vermeulen and Schull examine the problem of misdiagnosis using different methods. They searched an administrative database from Ontario (total population 12.5 million) for all patients admitted to any hospital for nontraumatic SAH over a 3-year period. They found 1507 patients, making this by far the largest study on misdiagnosis to date. They next looked to see which of those 1507 patients had been seen in an ED during the previous 14 days. Patients who had an ED diagnosis (on the first ED visit) that was one of a predefined list of diagnoses thought to be incorrectly diagnosed SAH, were counted as initial misdiagnoses.

Their principal finding is that 5.4% of patients are misdiagnosed at a prior ED visit. At first blush, this finding would appear to halve the prior misdiagnosis rate, which was already half the rate found in pre-2000 studies. Are we that much better at correct diagnosis of SAH?

Probably not.

Most importantly, these investigators limited their study to ED-specific misdiagnosis. Although not an intrinsic weakness of the study, it does help explain the primary result. In the Kowalski study, only 43% of the total 12% misdiagnosis rate (or 5.2% of the total) was attributable to an ED-related
misdiagnosis. Previous studies also included diagnostic errors that occur in doctors’ offices (where lower overall acuity compared with an ED population makes misdiagnosis more likely), on inpatient units and patient-related delays in their totals.\textsuperscript{4,9,34}

Two potential confounders include the narrow time horizon used (14 days) and the fact that to be called a misdiagnosis the first diagnosis must have been on their prespecified list. Although the 14-day time interval between the first ED visit and the later one could artificially lower the misdiagnosis rate, when the authors extended this time period to 30 days, the misdiagnosis rate rose only by 0.2\% to 5.6\%. Likewise, using a prespecified list of incorrect diagnoses introduces the possibility of underestimation of the “misdiagnosis” rate. However, any effect on the results is likely small because the investigators generated their list of the most common incorrect diagnoses from those found in prior studies, a list that has remained remarkably constant over time.\textsuperscript{12}

Another potential confounder is that the use of more modern generations of CT scanners in this study (similar to the Kowalski study) could result in improved diagnosis compared with some of the older literature cited above.

Two other findings of the Canadian study are that (1) misdiagnosis is more likely in patients with lower acuity presentations, and (2) misdiagnosis is twice as likely in nonteaching as in teaching hospitals. This first finding is consistent with all previous data, even though ED triage scores were used in lieu of a validated clinical score (such as the Hunt&Hess scale).

The reason for the second finding is less clear. The increased misdiagnosis rate in nonteaching hospitals is not attributable to SAH volume or CT availability. This suggests the importance of specialty education because physicians with emergency medicine training are more likely to work in larger Ontario teaching EDs than in smaller nonteaching ones (Perry J, personal communication, 2006). One must consider a diagnosis to make it, and emergency physicians are systematically taught to consider “cannot miss” diagnoses such as SAH.\textsuperscript{14} Or perhaps the very interaction between supervising attending and the resident physician improves diagnostic accuracy. Another hypothesis is that physicians in teaching settings simply order more tests; if so, then we are faced with the unpleasant question of what is an “acceptable” miss rate in an age of soaring healthcare costs.

One unexpected finding in this study is that misdiagnosed patients have a lower crude mortality rate than correctly diagnosed patients—a distinctly nonintuitive result and one that is not consistent with previous work. Misdiagnosed patients likely had smaller bleeds. Thus, one explanation relates to the short mean delay to the second visit—only 2.7 days. Initially misdiagnosed patients might have returned (to be correctly diagnosed and treated) before deteriorating from complications such as rebleeding. Also, patients who died after their first ED visit (but before a second visit) would have escaped detection given the methods in this study.

Another interesting finding is that 37\% of misdiagnosed patients returned for their second visit to a different hospital. In the absence of a centralized quality assurance process or litigation, the physicians who made these erroneous diagnoses might never know that they missed a SAH. This is a crucial issue in terms of physician education.

Vermeulen and Schull’s\textsuperscript{7,8} largest series to date of missed SAH is an important contribution to the literature. However, some important questions remain. In taking a history in headache patients, what is the minimum data set physicians should collect? Which of these elements predict serious secondary causes? What are the performance characteristics of modern CT scanners?

Given the wide spectrum of presentations of SAH, we will probably never completely eliminate misdiagnosis. However, future resources ought to be spent less on study and on more education. We must educate our patients about which symptoms constitute sufficient reason to consult a physician, and we must educate physicians about the many pitfalls inherent in the diagnosis of SAH.

Disclosures

None.

References


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