Aneurysmal Subarachnoid Hemorrhage
Unanswered Questions

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KEYWORDS

• Subarachnoid hemorrhage • Hydrocephalus • Aneurysm • Vasospasm • Delayed cerebral ischemia

KEY POINTS

- Under the right circumstances, a good-quality computed tomography scan obtained within 6 hours of headache onset can exclude subarachnoid hemorrhage with a very high degree of confidence.
- Antifibrinolytics and antihypertensives should be considered to reduce the risk of early rebleeding, but their value is only supported by low level of evidence. Meanwhile, there is no evidence to support the use of prophylactic anticonvulsants and it is possible that anticonvulsant administration may be associated with worse outcomes.
- Modern treatment of ruptured aneurysms should be individualized and, ideally, conducted in centers with endovascular and surgical expertise.
- The neurologic examination remains the optimal tool for the diagnosis of delayed cerebral ischemia. Pharmacologic hemodynamic augmentation and endovascular therapy can reverse symptoms of delayed cerebral ischemia, though these interventions are not supported by controlled studies.
- Hydrocephalus is a very common and under-recognized complication. Aggressive control of intracranial pressure might improve microcirculatory flow. There are no validated criteria to help identify those patients who will need permanent cerebrospinal fluid diversion.

INTRODUCTION

When a patient with aneurysmal subarachnoid hemorrhage (aSAH) survives the initial rupture and reaches the intensive care unit without major apparent parenchymal damage, neurosurgeons and neurointensivists are confronted with a major responsibility: If everything goes right, that patient may regain full function; conversely, any mistakes can have devastating consequences for his or her future. Despite scientific progress in this field, the management of aSAH remains in many aspects an art. Expertise and clinical judgment often make the difference between a great outcome and a very poor one.[1] In this article, the authors highlight several questions for which the answers remain elusive, myths are too often perpetuated, or gaps in knowledge are filled variable heuristic approaches.[2]

IS LUMBAR PUNCTURE NECESSARY IN PATIENTS WITH SUDDEN HEADACHE AND NEGATIVE COMPUTED TOMOGRAPHY SCAN?

Traditional teaching has been that all patients with an acute headache, particularly if very

Disclosure Statement: A.A. Rabinstein has no pertinent disclosures. G. Lanzino is a consultant for Medtronic.
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Neurosurg Clin N Am 29 (2018) 255–262
https://doi.org/10.1016/j.nec.2018.01.001

sudden (thunderclap), should have a lumbar puncture to examine for xanthochromia of the cerebrospinal fluid (CSF) if the suspicion of aSAH remained present even after a computed tomography (CT) scan did not show subarachnoid blood.[3,4] The value of the recommendation to proceed with lumbar puncture in these instances depends on the false-negative ratio of CT scans. This ratio was not that low with early CT scanners, but technological advances have achieved a remarkable reduction in this underdiagnosis.

Contemporary studies demonstrate that the sensitivity of CT scans for the identification of aSAH is extremely high when the scan is performed within 6 hours of the headache onset. A meta-analysis of available studies concluded that a CT scan within 6 hours has a sensitivity of 98.7% and a specificity of 99.9% for the detection of SAH. The pooled likelihood ratio of a negative CT on this meta-analysis was 0.010 (95% confidence intervals, 0.003–0.034), meaning that only 1 in 1000 cases of SAH could be missed by an early CT scan alone.[5]

However, before throwing away the spinal needle, it is crucial that we keep in mind certain caveats. These data only apply to patients in whom the CT scan is done unambiguously within 6 hours of the headache onset and who do not have other neurologic symptoms or signs, such as neck pain, loss of consciousness, or meningismus. Also, the data only apply to good-quality images (not affected by motion artifacts or other technical limitations) with cuts 5 mm or less from the base of the skull, obtained with a modern CT scanner (third generation or newer), and interpreted by a reader experienced in neuroimaging who is aware of the clinical suspicion of SAH. When these conditions are not strictly met, it remains most prudent to proceed with a lumbar puncture to fully exclude SAH.[5]

WHAT IS THE VALUE OF ANTIFIBRINOLYTICS AND TREATMENT OF ACUTE HYPERTENSION TO REDUCE THE RISK OF EARLY REBLEEDING?

Decades ago, antifibrinolytics were used routinely in the management of aSAH until randomized trials showed that their benefit in reducing the risk of early rebleeding was negated by an increase in the incidence of delayed ischemic complications. Yet, those trials used antifibrinolytics for many days, including the vasospasm period, as they were conducted when early aneurysm treatment was not considered the standard of care.[6] More recently, antifibrinolytics have made a comeback. One single-center randomized controlled trial and some observational data support their administration for up to 72 hours from aneurysm rupture.[7] As a result, considering a short course of an antifibrinolytic is recommended by most current management guidelines; yet, the strength of these recommendations is moderate to weak.[4,8]

In the authors' practice, they use intravenous tranexamic acid 1 g every 6 hours, following the protocol of the only trial that showed a benefit in clinical outcomes with the use of an antifibrinolytic drug.[7] The first dose is given in the emergency department or during helicopter transportation. They stop the medication as soon as the aneurysm is secured. The authors try to avoid its administration very shortly before angiogram or surgery, and they never use it for more than 72 hours from symptom onset, which means they do not start it in patients presenting late. Acknowledging that the supporting evidence is weak, the authors' decision to use an antifibrinolytic is based on the fact that there is fairly convincing proof that it can reduce early aneurysm rebleeding and the risk of ischemia should be much lower if only very few doses are administered. (The authors typically just give 1 or 2 doses because of their fast approach to aneurysm treatment.)

The evidence for hypertension treatment is even more scant. Observational studies have shown an association of severe acute hypertension with aneurysm rebleeding, but these data do not allow discrimination of causality versus reverse causality.[9,10] In other words, these studies cannot establish conclusively if the hypertension contributed to the aneurysm rerupture, was a surrogate marker of the severity of the SAH, or actually resulted from the rebleeding. Current guidelines favor blood pressure reduction when the hypertension exceeds a mean arterial pressure of 110 mm Hg or a systolic blood pressure of 160 to 180 mm Hg.[4,8,11] The decision of how aggressively to treat the hypertension should incorporate considerations such as whether patients have signs of acute pulmonary edema, which can be a manifestation of neurogenic cardiopulmonary injury, or have clinical or radiological signs of intracranial hypertension.

At the end, the only therapeutic intervention that can eliminate the risk of rebleeding is the obliteration of the ruptured aneurysm. Thus, all early efforts should be concentrated on stabilizing patients so that aneurysm treatment can be performed promptly and safely.[12]