Subarachnoid Hemorrhage
An Update

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KEYWORDS

• Subarachnoid hemorrhage • Delayed cerebral ischemia • Vasospasm
• Cerebral salt wasting • Coiling

KEY POINTS

- Target euvolemia, not hypervolemia, in the management of delayed cerebral ischemia in subarachnoid hemorrhage.
- Delayed cerebral ischemia is the most prominent complication of subarachnoid hemorrhage and can occur independent of angiographic vasospasm.
- Based on large, randomized trials, initiation of statin therapy does not improve outcomes in subarachnoid hemorrhage.
- Oral nimodipine therapy remains the mainstay of neuroprotective therapy in subarachnoid hemorrhage.
- Hyponatremia is common in subarachnoid hemorrhage and is associated with longer length of stay, but not increased mortality.

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a debilitating, although uncommon, type of stroke with high morbidity, mortality, and economic impact. Occurring with an incidence between 2 and 22 per 100,000 persons per year with regional world variation, SAH accounts for about 5% of all strokes. In the United States, the incidence is about 10 per 100,000 persons per year.[1] Modern 30-day mortality is as high as 40%, and about 50% of survivors have permanent disability. Care at high-volume centers with dedicated neurointensive care units is recommended, although subspecialty expertise may be more important than clinical volume. Euvolemia, not hypervolemia, should be targeted, and the aneurysm should be secured early. Although nimodipine remains the mainstay of treatment for neurologic protection after SAH, neither statin nor

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magnesium infusions should be initiated for delayed cerebral ischemia (DCI). Cerebral vasospasm is just one component of DCI. Hyponatremia is common in SAH and is associated with longer length of stay, but not increased mortality. This article focuses on selected points of management of these critically ill patients with an emphasis on the newest understanding and care recommendations relevant to anesthesiologists and neurointensivists.

MANAGEMENT BEFORE ANEURYSM OBLITERATION

Anesthesiologists are typically consulted after diagnosis of SAH has been made. Evaluation of the patient's ability to protect the airway and hemodynamic monitoring and control are paramount in this first assessment, especially as the patient is transported to the intensive care unit or prepared for aneurysm obliteration in the angiography or operating suite (**Fig. 1**).

Neurologic examination, including mental status, is important, because anesthesia masks subtle changes. Ensuing neurologic deterioration should be expected and plans developed (**Table 1**).

An aneurysm that has caused an SAH can rebleed. Although the size of the aneurysm is the strongest predictor of rupture, rebleeding of the aneurysm may be partly attributable to uncontrolled hypertension. A titratable agent (eg, nicardipine) is preferentially used to prevent extreme hypertension, and specific blood pressure goals should be individualized based on the patient's age, cardiac and baseline blood pressure history, and aneurysm size. Hypotension should also be avoided because it may cause oligemia through compromised cerebral perfusion pressure and increase the risk and size of stroke. The Interpretation and Implementation of Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trail (INTERACT-2)[2] showed the safety of targeting systolic blood pressure less than 140 mm Hg compared with a target of less than 180 mm Hg. Similarly, The Intracerebral Haemorrhage Acutely Decreasing Arterial Pressure Trail (ICH ADAPT)[3] trial compared a

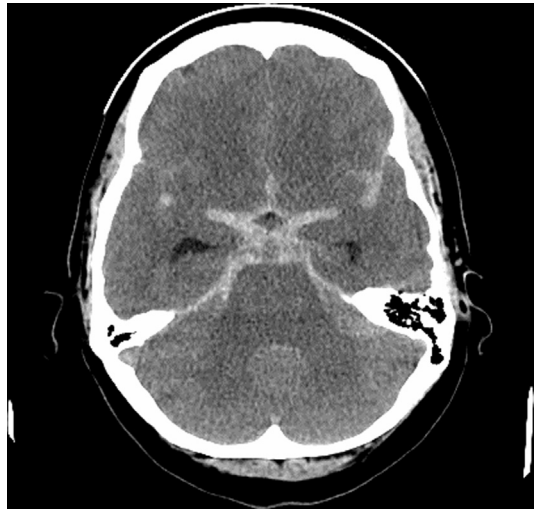

Fig. 1. Noncontrast head computed tomography (CT) showing subarachnoid blood. (*Courtesy of* Justin Fraser, MD, Lexington, KY.)