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Review Article

Review of aneurysmal subarachnoid hemorrhage—Focus on treatment, anesthesia, cerebral vasospasm prophylaxis, and therapy

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ABSTRACT

Aneurysmal subarachnoid hemorrhage (aSAH) is a serious and debilitating condition that leads to the development of many complications, which are followed by mortality and morbidity. As anesthesiologists, we may require to manage aSAH at various settings such as in the perioperative period or in a nonoperative setting such as the neuroradiology suite for diagnostic and therapeutic interventions. Therefore, it is important to understand the pathophysiology of aSAH and anesthetic management for operations and interventions. For decades, early brain injury and cerebral vasospasm have played major roles in the outcome following aSAH. The purpose of this article is to review recent advances and future perspectives in the treatment of aSAH, early brain injury, and cerebral vasospasm.

1. Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating disease and leads to the development of poor outcome and high mortality.[1] Endovascular or early surgical securing of aneurysm is the standard treatment procedure, and anesthetic management of these patients is precarious due to their unstable physiology and severe systemic medical complications such as neurogenic cardiac injury, neurogenic pulmonary edema (NPE), electrolyte disturbances, arrhythmia, cerebral edema, and cerebral vasospasm (CVS).

Endovascular management of intracranial aneurysms is likely to progress rapidly and has emerged as the first-line intervention for medical treatment failure patients. With the increasing use of interventional radiology techniques, familiarization with anesthetic care and procedure-related complications becomes more important. If interventional procedures fail, emergent craniotomy and clipping of aneurysm may be required. Anesthesiologists should be familiar with specific monitoring, strategies to relax the brain and maintain adequate cerebral perfusion, specific request for deliberate hyper-/hypotension, blood glucose control, and management of surgical crises of aneurysm rupture during clipping.

Delayed cerebral ischemia (DCI) as a result of CVS is the most common cause of death and disability after aSAH. Nimodipine has been shown to improve CVS in controlled trials. In addition, patients with CVS who did not response to early triple-H therapy (hypertension, hypervolemia, and hemodilution) should be considered for urgent angioplasty with intra-arterial injection of vasodilators. Finally, future perspectives on the reduction of CVS by attenuating glutamate toxicity, inflammation, oxidative stress, and early brain injury (EBI) may improve the outcome of aSAH.

2. Systemic effects of aSAH

Physiologic derangements of the cardiac, respiratory, and endocrine systems can be caused by aSAH. Here, we list some important derangements that require further evaluation and management.

2.1. Neurogenic cardiac injury and NPE

These abnormalities may due to the excessive release of catecholamines from sympathetic nerve terminals triggered by aSAH.[2–4] Marked systemic and pulmonary hypertension, cardiac

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arrhythmias, myocardial dysfunction, and NPE were easily found in these patients. Additionally, among patients suffering from cardiac events following aSAH, those with myocardial infarction and, in particular, those with a troponin level greater than 1.0 μg/L had a 10-time increased risk of death.[5] Moreover, uncontrolled prolonged heart rate elevation is associated with major adverse cardiopulmonary events and poor outcome after aSAH.[6] Myocardial dysfunction, particularly when combined with hypervolemic therapy for CVS, may exacerbate pulmonary edema.[7,8]

Pulmonary complications such as NEP, pneumonia, and acute lung injury are commonly associated with aSAH, occurring in up to 80% of patients, and a high mortality rate.[9,10]

2.2. Electrolyte disturbances

Hyponatremia is the most common metabolic derangement among aSAH patients as a result of either cerebral salt wasting syndrome or syndrome of inappropriate secretion of antidiuretic hormone and associated with poor outcome.[11,12] Etiology and treatment of hyponatremia vary in the presence of hypervolemia/euvolemia or hypovolemia.[13] Other common electrolyte disturbances include hypokalemia, hypocalcemia, and hypomagnesemia, which should be corrected carefully.[9] At the same time, hypothyroidism, cortisol deficit, and inappropriate fluid management (overload or hypovolemia) should also be considered.

2.3. Cerebral edema and CVS

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure and intracranial pressure (ICP) or central venous pressure, whichever is higher. Maintenance of an adequate CPP is a major goal in managing aSAH, but often complicated in the setting of dysfunction of blood–brain barrier (BBB) and cerebral blood flow autoregulation. The combination of cerebral edema, increased ICP (IICP), and hypovolemia increases the likelihood of CVS, which may *per se* increase ICP as a vicious cycle.

CVS usually develops 3–12 days after aSAH and lasts for 2 weeks on average; it affects 60–70% of patients. It frequently results in cerebral ischemia, and is the major cause of morbidity and mortality after aSAH.[14] Diagnostic modalities and treatment of CVS have been reviewed in our previous article[15] and updated in the section "Prophylaxis and therapy of CVS".

2.4. Notices for anesthesia

Hypotension and hypoxia are two of the most important insults that influence outcomes. Patients who were suffering from the two conditions should be well prepared, and the anesthesiologists explained to the family and surgeons prior to operation. In addition, postponement of the operation should be considered until the condition is improved. In view of the adverse effects, therapy of cardiac insufficiency with inotropic agents should be considered.[16–18] In the event of cardiorespiratory compromise, a full investigation, attentive monitoring, and appropriate interventions are required immediately to optimize cardiorespiratory function and allow subsequent management of aSAH. The use of prophylactic α- and β-blockade to reduce the effect of catecholamines has been a matter of debate. However, this strategy needs to be balanced against the need to maintain cerebral perfusion.[19] Patients with severe cardiovascular failure and CVS were reported to benefit from temporary intra-aortic balloon pump counterpulsation.[20,21] Patients with NPE should be treated with ventilator support having a high post end-expiratory pressure and furosemide.

3. Interventional treatment and anesthesia for ruptured cerebral aneurysms

The choice of interventional treatment following a ruptured aneurysm is either endovascularly coiling or surgically clipping. The optimal choice may not be clear and depends on the physician's judgment about age, World Federation of Neurological Surgeons grade,[22] comorbidity, aSAH onset time, and the anatomy of the aneurysm.[23]

According to the 2012 "Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Guideline for Healthcare Professionals from the AHA/ASA",[24] some important recommendations are as follows: (1) surgical clipping or endovascular coiling of the ruptured aneurysm should be performed as early as feasible in the majority of patients to reduce the rate of rebleeding after aSAH (Class I; Level of Evidence B); (2) complete obliteration of the aneurysm is recommended whenever possible (Class I; Level of Evidence B); (3) for patients with ruptured aneurysms judged to be technically amenable to both endovascular coiling and neurosurgical clipping, endovascular coiling should be considered (Class I; Level of Evidence B); and (4) microsurgical clipping may receive increased consideration in patients presenting with large (>50 mL) intraparenchymal hematomas and middle cerebral artery aneurysms. A new recommendation is that endovascular coiling may receive increased consideration in the elderly (>70 years of age), particularly in those presenting with poor-grade aSAH (World Federation of Neurological Surgeons classification IV/V) and those with aneurysms of the basilar apex (Class IIb; Level of Evidence C).

3.1. Endovascular management of cerebral aneurysms

Emerging evidence suggests that endovascular intervention may reduce the morbidity associated with surgery.[25] For poor-grade patients with brain swelling and CVS, which may deter surgeons from performing a surgery, an endovascular intervention may be preferable.[26] However, limitations of coiling exist in approximately 5–15% of cases due to morphological characteristics or location of the aneurysm.[27–30]

3.2. Radiation safety

Ionizing radiation follows the inverse square law, and the radiation exposure drops off at a rate proportional to the square of the distance from the source; therefore, activities near the patient's head should be minimized. The use of extended infusion and monitoring lines is required in patients with a difficult access to intravenous (i.v.) lines in the upper limbs once the procedure is underway.

Throughout the whole procedure, all personnel in the room should wear protective lead aprons with thyroid shields and stay as far away from the radiation source as possible. In some hospitals, there is more radiation safety with facilities for the anesthesia team to monitor the patient from a distance.

3.3. Preoperative planning and patient preparation

For i.v. sedation cases, careful padding of pressure points to obtain comfortable positioning can assist patients in tolerating a long lying motionless time. In addition to standard monitors, end tidal CO_2 sampling via nostrils by sampling line is useful for evaluation of airway pattern. Early signs of femoral artery obstruction or thromboembolism can be detected by placing a pulse oximeter probe on the side of great toe, which has femoral introducer sheath.

Invasive BP monitoring using an arterial line is suggested. Urine output measurement assists in fluid management. A significant