Review article

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Perioperative management of aneurysmal subarachnoid hemorrhage

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Subarachnoid hemorrhage (SAH) secondary to ruptured intracranial aneurysms is one of the devastating diseases to treat in neurosurgery with high morbidity and mortality. Successful management of patients with spontaneous SAH involves a collective teamwork of neurosurgeons, neurologists, radiologists, anesthesiologists, intensivists and nurses. Once the patient is scheduled for surgery the role of the anesthesiologists becomes increasingly important and successful outcome from surgery depends on expedious perioperative care of the patient from the anesthesiologists apart from good surgical clipping. In this article, the authors review the management of aneurysmal SAH with emphasis on perioperative care.

Key Words: anesthesia, aneurysm, craniotomy, subarachnoid hemorrhage

Intracranial aneurysms are diverticuli arising from vessels of the circle of Willis - particularly on the anterior and posterior communicating arteries, the bifurcation of the middle cerebral artery, and the bifurcation of the internal carotid artery. Every year approximately 30,000 people suffer from SAH in the United States,[16] and roughly 80% of them have ruptured saccular aneurysms.[7] Subarachnoid hemorrhage (SAH) occurring from a ruptured saccular aneurysm accounts for 6 to 8 % of all strokes and is associated with a mortality rate of 40 to 50 %.[3,11] Mortality following the first bleed is 43 %. 35 % of the survivors die within a year if surgery is not done. Mortality following the first rebleed is 64 % and that from the second rebleed is 96 %.[11] These figures underscore the importance of early diagnosis and surgery.

Various high risk factors for SAH (positive family history, smoking, hypertension and heavy alcohol intake) have been identified in the large series.[14,20] The most common presenting symptoms are severe headache (85%) with or without loss of consciousness (45%).[10] The cause of both the initial headache and loss of consciousness is an acute increase in intracranial pressure (ICP).[21] If ICP does not rapidly normalize, the patient dies or is severely disabled. Nausea and vomiting, photophobia, fever, meningismus, and focal neurologic deficits are also common following SAH.

There are various classification systems to accurately grade patients with SAH at the time of admission. These classification systems are designed to have prognostic significance and help in the decision making process. Tables 1 and 2 show two commonly used clinical systems to grade the SAH- Hunt-Hess,[12] and World Federation of Neurological Surgeons (WFNS),[6] systems respectively. WFNS grading system is based on the Glasgow coma scale[19] and presence or absence of motor deficit.

Once the diagnosis of SAH is suspected, based on the clinical features, the investigation of choice is the plain computerized tomography (CT) scan of the head.[20] A diagnostic lumbar puncture should be performed in patients with a strong clinical suspicion for SAH and a negative or equivocal CT scan. Once the diagnosis is confirmed the patient should be aggressivly managed right from the emergency department and should preferably be admitted into the intensive care unit. These patients should have adequate intravenous fluid, analgesics (e.g. codeine),

laxatives and antiepileptic drugs for seizure prophylaxis and close management of water and

Grade	Criteria	Perioperative mortality
I	Asymptomatic or minimal headache or slight nuchal rigidity	0-5%
II	Moderate to severe headache, marked nuchal rigidity, no neurological deficit other than cranial nerve palsy	2-10%
III	Drowsiness or confusion, mild focal deficit	10-15%
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances	60-70%
V	Deep coma, decerebrate rigidity, moribund appearance	70-100%

Table 1. Hunt-Hess system of grading the subarachnoid hemorrhage.

electrolytes and acid base status. At all cost, one should strive to avoid hyperthermia, hyponatremia and hypovolemia and hypotension in this group of patients, as all of them have shown to have adverse outcome. The next step in the management is to get a cerebral angiogram to confirm the presence of aneurysm and define its morphology from the operative point of view, and to detect presence or abscess of vasospasm. Here good communication between the surgeon and the radiologist can not be overemphasized. Once the angiogram shows an aneurysm amenable to surgery, the anesthesiologist comes into play. It has been shown by various studies that early surgery lowers the morbidity and mortality in patients with aneurysmal SAH.[13] A prospective study offering surgery within 3 days of admission is currently underway at the division of Neurosurgery, Tribhuvan University Teaching Hospital (*Nepalese SAH evaluation study at TUTH, proposal, www.neuroscienceformum.org.np*) and the completion of the study will hopefully provide prospective data in this regard in our community.

A few other major problems, which can occur in post admission period, should be preoperatively considered.

1. **Hypertension**. Hypertension in SAH is secondary, due to autonomic hyperactivity. Increase in blood pressure (BP) directly increases the transmural pressure (mean arterial pressure - ICP) and the likelihood of bleeding.[3] Hence, the BP should be carefully controlled. However it will be detrimental to lower the blood pressure drastically.

2. **Myocardial dysfunction.** Patients with SAH have an increased incidence of both disarrhythmias and other electrocardiogram (ECG) abnormalities.[15] The most frequent abnormalities are prolongation of the Q-T interval, flattened or

Grade	GCS*	Motor deficit
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present or absent
V	6-3	Present or absent

* GCS, Glasgow Coma Scale

Table 2. World Federation of Neurological Surgeons grade based on Glasgow Coma Scale and presence or absence or motor deficit

inverted T - waves, S-T segment depression and prominent U waves. The significance of these ECG changes has been debated on the majority of patients.[5] These changes do not appear to be associated with adverse neurologic or cardiac outcomes.[18]

3. **Vasospasm.** Vasospasm is a common problem. It is caused by break down products of hemoglobin, besides serotonin, histamine, catecholamines, prostaglandin, free radicals, and lipid peroxidases. The clinical syndrome is characterized by increasing drowsiness and confusion leading to stupor. Typically symptoms begin to occur 5-7 days after SAH and rarely occur after 2 weeks. Management of vasospasm includes prevention or minimizing the incidence by giving adequate fluid, use of calcium channel blockers, improving the delivery of oxygen through the spastic vessels, and dilation of spastic vessels (cerebral angioplasty, which is usually done once the aneurysm is secured). In addition, an effort should be made to protect the brain from ischemia. The regimen employed to treat vasospasm usually involves a combination of hypervolemia, haemodilution and hypertension (triple H therapy).[1] The mean arterial pressure (MAP) is increased using dopamine and the range is 20 - 30 mm Hg above the "baseline" systolic pressure. Calcium channel blockers such as nimodipine and nicardipine have shown to decrease the morbidity and mortality due to cerebral ischemia in patients with SAH.[17] Nimodipine is generally given for 21 days. However, these studies have failed to demonstrate any reduction in the incidence of vasospasm as

detected by angiography. The beneficial effect of these drugs may be due to their effect on neurons rather than on vascular smooth muscle.[2]

4. **Rebleeding.** Recurrent hemorrhage occurs within 14 days in 20-30 % of untreated patients following SAH.[13] Approaches used to decrease the risk of rebleeding include early surgical clipping, antifibrinolytic agent and blood pressure control. Antifibrinolytic drugs such as aminocaproic acid and tranexamic acid have been used in the past to inhibit the cerebrospinal fluid (CSF) fibrinolytic activity and to stabilize the aneurysmal clot and thereby decreasing the risk of rebleeding. However, theoretical benefits have not been translated into a decrease in the morbidity and mortality and these agents are no longer used in the major cerebrovascular centers in the world.

5. **Hydrocephalus.** Hydrocephalus is common (10-20%) following SAH due to impairment of CSF circulation by blood in the subarachnoid space.[8] Hydrocephalus behaves as a double-edged sword in patients who have unsecured aneurysm, which has recently ruptured. On one hand, increased ICP due to hydrocephalus is detrimental to the already sick brain; on the other, rapid release of CSF thus lowering the ICP (e.g. by ventriculostomy or during placement of lumbar subarachnoid drain before surgery) can cause rupture of the aneurysm due to the change in the transmural pressure.

Timing of surgery

Controversy exists regarding the timing of aneurysm surgery following SAH. In recent years there is a growing interest in early operation. That is, surgery within 72 hours of the SAH. This approach especially holds true in patients in good neurological grade (Grade I, II, III in the Hunt-Hess classification system). Early operation offers several advantages including prevention of rebleeding, opportunity to evacuate subarachnoid blood thus theoretically reducing the risk of vasospasm and facilitation of treatment of vasospasm if it develops by decreasing the risk of rebleeding associated with triple-H therapy. Advantages associated with delayed operative clipping (11-14 days) include resolution of the aneurysmal clot resulting in a reduced risk of intraoperative rupture and availability of more time for detailed evaluation and stabilization of other medical problems.

Anesthetic Management

Preoperative Evaluation

Detailed evaluation of the patients should be done before surgery at the bedside. In addition to general systemic examination, the nervous system should get particular attention. Evidence of raised intracranial pressure should be elicited preoperatively, so that it can be managed expeditiously pre and intraoperatively. Optimal hydration of the patients is important as dehydration increases the viscosity of blood and can adversely affect perfusion. If the patient is a known hypertensive who had been on treatment before SAH, the dose of the antihypertensive drugs should be readjusted. Other routing preoperative 'work-up' should be done as it is done for other cases.

Premeditation

Preoperative anxiety is not a problem in patients with depressed level of consciousness (grade III, IV, V). In the awake patient, a reassuring preoperative visit will usually allay anxiety. If necessary a small dose of benzodiazepines is the best choice.

Monitoring

During induction of anesthesia heart rate, electrocardiogram (ECG), end tidal CO_2 (ET CO_2), arterial blood pressure and temperature are monitored. Central venous pressure (CVP) line is placed after induction. Usually a lumbar subarachnoid drain is put in all cases of recent SAH to decrease the ICP and to facilitate microdissection during surgery. But some surgeons do not favor the use of lumbar drain. Urinary output is monitored throughout the operation. We do not monitor electroencephalogram (EEG) and evoke potential during operation.

Induction

The goal of anesthetic management is to facilitate the operation and patient recovery while minimizing the risk of intraoperative rupture of the aneurysm. The main aim during induction is to avoid abrupt severe swings in BP and subsequently ICP. It occurs during induction and at the time of pin placement. The selection of specific drugs for induction and maintenance depends on the personal preferences of the anesthesiologist.

At our centers, we generally induce these patients with 4-5 milligram/kilogram of thiopentone sodium intravenous slowly. Then we deepen the level of anesthesia with 0.5- 1% halothane in nitrous oxide and oxygen, using a mask. Intubation is facilitated using vecuronium or pancuronium in the dose of 100microgram/kilogram. We also give intravenous lidocaine 2 milligram/kilogram, 3 minutes before intubation. Usually, laryngoscopy causes the arterial pressure to rise but in a well-controlled patient, the hypertension is unlikely to reach a dangerous level. After induction, controlled ventilation is instituted to keep $PaCO_2$ to between 30 and 35 millimeters of Hg. 20% mannitol in a dose of 1-1.5 grams /kilogram is infused at the time of induction to facilitate brain relaxation. In addition, this

agent is considered to have some brain protection from ischemia. As per the surgeon's preference intravenous antibiotic should also be given. We give 1 gram cephazoline intravenously for this purpose, repeated in 5-6 hours. Controversy exists regarding the role of steroids in the management of aneurysmal SAH and we do not give steroids to any of our patients.

Maintenance

Anesthesia is maintained using halothane, nitrous oxide and oxygen and analgesics (pethidine or morphine). During craniotomy mean arterial pressure (MAP) is maintained at 80 to 90 mmHg. Though early on, it was recommended to lower the MAP up to the time of aneurysm clipping, recent data suggests having a better outcome when the patient is kept normotensive throughout the procedure. In the event that the aneurysm prematurely ruptures, then the MAP should be lowered using sodium nitropruside. After the aneurysm is clipped, the arterial pressure is allowed to rise while the surgical site is closely observed for bleeding. There has been a great interest in recent years in the role of hypothermia, and the use of barbiturates in an effort to minimize ischemic damage to the brain particularly when the temporary clip is used during surgery.[4] However, the overall results have been largely inconclusive and it remains to the choice of the individual centers or surgeons and/or anesthetists to decide whether to use these modalities or not.

We generally prefer to keep these patients electively on the ventilator for 12 hours after surgery. If the aneurysm is unruptured or if the SAH is not recent, then after skin closure residual neuromuscular blockade is reversed with a mixture of neostigmine and atropine. Tracheal extubation is subsequently performed and the patient is carefully monitored in the intensive care unit.

Conclusions

Successful management of aneurysmal subarachnoid hemorrhage involves a dedicated team of healthcare providers familiar with the disease. The care in the perioperative period is crucial from many respects and anesthesiologists often play a pivotal role. A thorough understanding of the pathophysiology of this dreadful disease with ability to detect and manage the complications early makes a tremendous difference in the final outcome. Though perioperative anesthetic management of aneurysmal SAH is challenging, with adequate preparation it can be performed relatively safely in our set up.

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