Revista Mexicana de Anestesiología

PROFESORES EXTRANJEROS
Vol. 32. Supl. 1, Abril-Junio 2009
pp S168-S171

# Anesthesia and subarachnoid hemorrhage

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## EPIDEMIOLOGY AND CLINICAL PRESENTATION

Intracranial aneurysms are saccular dilatations that most commonly occur at bifurcation points of the major intracranial cerebral vessels. The prevalence in North America is estimated to be 2,000 per 100,000 (1:50!!) with an annual incidence of subarachnoid hemorrhage (SAH) from rupture of 12 per 100,000 although the recently published International Study of Unruptured Intracranial Aneurysms(ISUIA) found a much lower incidence of rupture especially for small lesions. SAH occurs most frequently between the ages of 40-60 and slightly more commonly in women.

At the moment of rupture there is free communication between the artery and the subarachnoid space resulting in (local) intracranial pressure (ICP) equal to blood pressure. This results in sudden severe headache and (temporary) loss of consciousness. The blood spreads through the subarachnoid space resulting in meningism, headache and hydrocephalus, either from obstruction of CSF reabsorption or clot in the ventricle. Neurological injury presents as depressed consciousness and focal neurological signs. Isolated cranial nerve palsy may reflect a neuropraxia from «jet impact» injury from the blood at systemic pressures. Clinical diagnosis is confirmed by CT scan, MRI and four vessel angiography.

## OUTCOME AND COMPLICATIONS

Approximately one third die or are severely disabled at the time of the initial bleed and of the remaining patients only one third are «functional» survivors. This occurs in spite of an operative mortality of less than 10% in experienced hands and reflects the severity of the non-operative complications, primarily re-bleeding and vasospasm.

*Rebleeding:* The risk of rebleeding without surgery is 30-50% in the first two weeks with a mortality > 50%. The major impetus for early surgery is the prevention of rebleeding. In addition, it may reduce the likelihood of developing vasospasm by removing blood in the subarachnoid space, allow safer administration of more aggressive therapy for vasospasm, reduce the risk of developing medical complications and reduce costs by reducing hospital stay.

*Hydrocephalus:* Occurs in 15-20% of patients from communicating or obstructive hydrocephalus.

*Vasospasm:* Vasospasm remains a major cause of morbidity and mortality. Angiographic evidence of vasospasm occurs in up to 60% of patients but is seen clinically in only half. The clinical features often begin slowly with depressed consciousness (a reflection of global cerebral hypoperfusion) and later, focal neurological signs. Vasospasm is related to the volume and location of subarachnoid blood and the clinical grade of the patient. The exact etiology remains uncertain but is related to oxyhemoglobin and its breakdown products. Transcranial Doppler (TCD) is a useful bedside adjunct in the diagnosis of vasospasm. With the onset of vasospasm there is an increase in the blood flow velocity to > 120 cm/s which subsequently decreases as the spasm progresses. Nimodipine is the standard (prophylactic) drug in the management of vasospasm. There is no evidence that nimodipine angiographically relieves vasospasm and its mechanism of action may reflect a «brain protective» mechanism. Nimodipine or similar drugs will make patients prone to hypotension if they are hypovolemic and especially at the induction of anesthesia.

Currently, the most effective treatment available is hypervolemic-hypertensive-hemodilution («Triple H» therapy). The goals of this treatment are to increase cardiac output, improve the rheological characteristics of the blood and to increase cerebral perfusion pressure (CPP). Triple H therapy has been shown to reverse ischemic neurological deficits associated with vasospasm in up to 70% of patients. Other treatments include angioplasty and intra-arterial verapamil or papaverine.

## SYSTEMIC EFFECTS OF SAH

*Cardiovascular:* SAH causes a massive sympathetic discharge at the time of bleeding which results in hypertension and may cause myocardial dysfunction, ST segment changes, rhythm disturbances and neurogenic pulmonary edema. These cardio-pulmonary effects are associated with intense intra-myocardial release of catecholamines resulting in focal calcium over load which may result in cell necrosis. EKG changes occur in > 50% of patients and rhythm disturbances can occur in up to 90%. The type of EKG change, except Q waves or elevated ST segments, does not usually indicate underlying myocardial dysfunction. There is however a relationship between the extent of the neurological injury and the cardiac injury. The appropriate management of cardiac injury in SAH is poorly defined and does not usually influence anesthetic management other than occasionally requiring more invasive cardiac monitoring.

*Intravascular fluid volume & electrolytes:* Many with SAH have a reduction of their intravascular volume from bed rest, diuresis, and stress. Electrolyte abnormalities especially hyponatremia, hypokalemia, and hypocalcemia are frequent and may need correction. Hyponatremia occurs in approximately 30% and may be due to the Cerebral Salt Wasting (CSW) syndrome or Syndrome of Inappropriate ADH secretion (SIADH). CSW is caused by secretion of brain and atrial natriuretic hormone and is associated with extracellular volume depletion and thus should be treated with fluid loading with normal saline and very occasionally hypertonic saline. SIADH reflects an excess of water and theoretically should be treated by fluid depletion but in SAH it is better to keep intravascular volume high so salt containing IV solutions are usually used.

## TREATMENT OF ANEURYSMS

Two approaches are currently used, surgical direct clipping of the aneurysm's neck or endovascular coiling.

Surgery has been the standard treatment for the past 50 years and remains the current «gold standard».

Although endovascular approaches were attempted in Russia in the early 1970's it was not until a decade ago with the introduction of the Guglielmi detachable coils that this approach became a viable alternative. There has been a subsequent explosion of catheters and coils and more recently stents through which coils can me placed. The International Subarachnoid Aneurysm Trial (ISAT) has generated significant interest and controversy. The results including long term follow up indicate that coiling is a very reasonable treatment option for patients with suitable aneurysms. However the incidence of repeated treatments is much higher after coiling so that these patients require regular ongoing follow up angiography. Repeat treatment after surgical clipping is uncommon.

## ANESTHESIA FOR SURGERY

Anesthetic considerations for surgical and endovascular treatment are similar with obvious differences in the venue, potential blood loss and need for brain relaxation. Preoperative assessment should include neurological status, co-morbidities and complications (mentioned above). The clinical grade of the SAH correlates well with the ICP. Patients with grade I or II SAH may be assumed to have normal ICP, intact cerebral autoregulation and a normal response to hyperventilation. Patients with grade III or IV SAH will have raised ICP, impaired autoregulation and reduced $CO_2$-reactivity.

The goals of anesthetic management are to 1) control the aneurysm's transmural pressure gradient whilst 2) maintaining adequate cerebral perfusion and oxygen delivery and 3) avoiding precipitous changes in intracranial pressure. Anesthetic management should also maximize surgical exposure and reduces retraction on the brain. The aneurysm's transmural pressure gradient (TMP) is equal to the pressure within the aneurysm (arterial blood pressure) minus the pressure outside/around the aneurysm (ICP) i.e. TMP=MAP-ICP. This is the same equation that describes cerebral perfusion pressure (CPP = MAP - ICP). This highlights the dilemma of balancing adequate cerebral perfusion against the risk of potential aneurysmal rupture. Until the aneurysm is clipped blood pressure should not be allowed to rise above the preoperative baseline. Hematoma, hydrocephalus and a giant aneurysm may all serve to increase ICP. Any reductions in ICP should be gradual, at least until the dura has been opened (ICP = 0) because sudden reductions in ICP produce acute increases in the aneurysm's TMP.

All patients should have the following monitored – 5 lead EKG, intra-arterial blood pressure, pulse oximetry, capnography, and core temperature. Central venous and/or pulmonary pressures and TEE may be indicated in patients with significant heart disease or injury. EEG and/or sensory/motor evoked potentials are monitored by some. There are no prospective human trials showing a benefit to neuromonitoring.

In general, the precise choice of anesthetic drugs used is less important than adherence to the principles outlined above. Induction should be smooth. Patients face a real risk of aneurysm rupture at the time of laryngoscopy and intubation and the hypertensive response to these stimuli must be prevented. Other stimulating procedures include patient positioning and head-pin placement. All of these procedures should only be performed once adequate anesthetic depth, full muscle paralysis and control of the blood pressure are achieved. Propofol or thiopental together with an opioid and non depolarizing muscle relaxant are most commonly

used for induction. Scalp infiltration at the pin sites prior to their application is an easy way to attenuate the hemodynamic response. A TIVA technique may be preferred if ICP is very markedly elevated. Inhalational agents at <1 MAC are suitable in most patients.

Reducing brain bulk improves surgical exposure, reduces brain retraction and facilitates clipping of the aneurysm. This is usually achieved by intravenous mannitol 0.5-1 gm/kg sometimes with furosamide (10-20 mg IV). Peak mannitol effect occurs 20 minutes after infusion and the adequacy should be judged by the state of the brain and not the volume of urine. Some also use CSF drainage via lumbar catheter. Drainage should be done slowly to avoid brain shift and hemodynamic changes.

Most neurosurgeons now use «local hypotension» through temporary clipping of the proximal feeding artery rather than systemic hypotension. To maximize collateral blood flow, blood pressure should be kept in the normal range or slightly above. The only cerebral protection randomized prospective trial in this type of surgery, the International Hypothermia Aneurysm Trial (IHAST), did not demonstrate any benefit to mild (33 ºC) intraoperative hypothermia. No other so-called protective strategies have been submitted to prospective randomized trials. Despite this some centers use a variety of anesthetic based techniques. The most common is barbiturates or propofol given to achieve burst suppression.

Hypertension is a frequent occurrence with emergence from anesthesia. Mild hypertension may be of benefit in augmenting cerebral perfusion, especially in patients where there is a concern about vasospasm. Blood pressure > 20% above the patients normal level may be controlled with labetalol, esmolol or hydralazine as excessive rises in blood pressure may be associated with postoperative hemorrhage. Patients should be awake and responsive as soon as is feasible to facilitate early neurological assessment and decisions about the need for CT scanning, angiography or the initiation of Triple H therapy. Patients who have had intra-operative complications or who were grade III or IV SAH preoperatively should be returned to the ICU intubated and ventilated.

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