Preoperative Management of Patients with Intra-Cranial Aneurysms

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Aneurysms of the intra-cranial vessels are congenital or acquired lesions related to a structural defect in the media of the arterial wall. They arise as sac-like outpouchings at the apices of major arterial bifurcations: 85% occur on the anterior circulation and 15% on the posterior circulation.

The flow characteristics at these bifurcations lead to progressive dilatation of the aneurysural sac. Rupture is often associated with sudden rise in blood pressure as may occur during a valsalva maneuver or physical and mental stress. It can also occur during sleep. Rupture of an aneurysm leads to leakage of blood into the subarachnoid space giving use to symptoms of severe headache, nausea, vomiting or loss of consciousness. Neurogenic pulmonary oedema may also occur.

Incidence

With an annual incidence of 11/100,000 of the population, it can be estimated that nearly 28,000 patients in North America will suffer from subarachnoid haemorrhage related to aneurysm rupture. The peak incidence is between the ages of forty and sixty years and the overall ratio of female-male is 3:2 Of these 28,000 people, 10,000 will die or be disabled as a result of the initial insult. 3,000 die rapidly without warning. Seven thousand have warning symptoms which are ignored, initially mis-diagnosed, or are referred too late for definitive therapy. Of the 18,000 available for treatment in neurosurgical units, 9,000 ultimately will die or be disabled: 3,000 from rebleeding, 3,000 from vasospasm, 1,000 from medical complications and 2,000 from surgical complications. There will remain 9,000 functional survivors of the 28,000 with subarachnoid hemmorrhage giving an overall mortality and morbidity of 70%.

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Causes of death and disability include direct effect of the hemmorrhage (intra-cranial hypertension or intra-cerebral hemmorrhage), vasospasm or medical complications.

RECURRENT HEMMORRHAGE OR REBLEEDING is the most serious immediatge threat to the patient. It is more likely to occur in the first 24-48 hours of the initial hemmorrhage and diminishes to a 3% chance per year rupture after three weeks. 20% of patients will experience rebleeding in the first 10 days; and occurence is higher among those with poor neurological status after the first subarachnoid hemmorrhage. Mortality after a rebleed is 60-78%. Vasospasm occurs between 5-7 days after a subarachnoid hemmorrhage and is unusual after two weeks.

The term vasospasm is generally understood to mean vasoconstriction of the angiographically visible blood vessels in the subarachnoid space. It may be local or diffuse and may not be symptomatic depending on the extent and severity of the process as well as the collateral circulation. The degree of spasm is related to the amount of subarachnoid blood but the actual cause is not known. The mortality rate is higher in patients who develop vasospasm after a subarachnoid hemmorrhage, with or without surgery.

The objective of preoprative management is to prevent neurological deterioration from rebleeding and vasospasm. Supportive medical therapy includes measures to prevent or treat resulting acute hydrocephalus, cardiac arrhythmias, cerebral oedema and convulsions.

Surgery is the treatment of choice in the prevention of rebleeding. The aneurysmal sac can be isolated from the circulation either by clipping or ligation. Once the aneurysmal sac is isolated, vasospasm, if and when it occurs, can be treated with volume expansion and hypertension. So far this has been the most successful therapy. Nimodipine, a calcium channel blocker which has a direct cerebral vasodilator effect, is currently under investigation for the treatment of vasospasm.

The timing of surgery in relation to the initial bleed is controversial. Advantages of early surgical intervention within the first 2 days are:-

Maximum protection against rebleeding particularly when elevation of blood pressure and volume expansion may be required to prevent vasospasm.

Once the aneurysm is secured there is no further need for anti-fibrinolytics, potent antihypertensives, sedatives, fluid restriction and bed rest to prevent rise in blood pressure and subsequent rebleeding. These measures may not only aggravate vaspspasm but can cause medical complications such as deep vein thrombosis and pulmonary embolism.

Early surgery has been shown to promote a favourable outcome and reduce morbidity and mortality.

With late surgery, two to three weeks after a subarchnoid hemmorrhage, (1) the brain is not oedematous thus making dissection easier; and (2) the point of rupture on the dome of the aneurysm is sealed and intra-operative rupture is therefore less likely to occur. Surgical mortality with an unruptured sac is less than 1%; while overall mortality is 10% Delay of surgery, however, leaves the patient unprotected from a recurrent bleed.

Surgery is contraindicated when clinical signs and symptoms of vaspspasm (drowsiness, focal neurological deficit) are present, unless an intracranial mass effect is responsible for the neurological deterioration. Vasospasm, however, may be present angiographically without any clinical signs, surgery at this time is controversial. When surgery is postponed, anti-hypertensive drugs and anti-fibrinolytic agents are used during the preoperative period to prevent rebleeding. The anti-fibrinolytic agents are thought to preserve the aneurysmal clot by counter-acting the effect of fibrinolysins in the cerebro-spinal fluid, thus preventing rebleeding.

Anaesthesia

The goals of anaesthetic management are to prevent rupture of the aneurysm during surgery, reduce the size of the brain and at the same time maintain adequate blood flow to the vital organs.

Control of Blood Pressure

Based on Laplaces Law, the likelihood of rupture of the aneurysm is directly related to intra-aneurysmal pressure and inversely to the thickness of the wall. Since any rise in systemic pressure is directly reflected in intra-aneurysmal pressure, control of blood pressure is necessary for the prevention of rebleeding.

Control of blood pressure is started preoperatively. The anti-hypertensive agent selected depends upon the severity of the hypertension and the ongoing intracranial process. Hydralazine and sodium nitro-prusside both in-

crease intracranial pressure, so should be used with caution. Preoperative administration of Propranalol assists in the control of blood pressure, modifies baroreceptor mediated tachycardia and by its effect on renin angiotensin release limits rebound hypertension after aneurysmal clipping.

Premedication

Since these patients are heavily sedated, there is frequently no need for additional premedication. If necessary Midazolem can be given intramuscularly in doses of 5-10 mg.

Induction of Anesthesia

Induction of Anesthesia is characterized by stimulation of catecholamine release and consequent increase in blood pressure and heart rate. To modify these effects:

Vasodilator drugs may be needed for rapid predictable blood pressure control without hypotension.

All painful preoperative preparations, such as insertion of arterial and central venous pressure lines, may be deferred till a suitable depth of anaesthesia is achieved. Continuous blood pressure monitoring is possible with the aid of a non-invasive monitor -- the 'dinamap'.

Suppression of the hypertension response to laryngoscopy and intubation can be achieved by adequate depth of anaesthesia, full muscle relaxation, adrenergic Beta blockade, intravenous lidocaine prior to intubation and administration of sodium nitroprusside intravenously.

Infiltration of local anaesthetic prior to pin head holder placement and surgical incision will also help control blood pressure.

Reduction of brain volume

Reduction of brain volume is an important anaesthetic consideration in aneurysm surgery. Opening of the dura in the presence of an oedematous brain promotes ischaemia particularly to brain in tight contact with the line of the dura. Brain retraction in these circumstances causes further ischaemia due to retractor pressure. Decrease in intracranial pressure also helps in surgical dissection. Brain volume is controlled by the evacuation of cerebrospinal fluid through a lumbar puncture, the use of loop diuretics and hyperventilation. Since transmural aneurysmal pressure can be increased by a

rise in mean arterial pressure or fall in intra-cranial pressure, a rapid release of cerebrospinal fluid in the presence of hypertension can cause aneurysm rupture, it can also result in hypotension and cardiac dysrhythmias.

Hypotension

Controlled profound hypotension by releasing tension on the aneurysm wall makes dissection possible with a reduced risk of rupture. It is safe only if cerebral blood flow is sufficient to provide adequate oxygen for survival of undamaged brain tissue.

In order to assess the adequacy of cerebral blood flow, monitoring of the following parameters can be used:-

Blood pressure.

Cerebral blood flow and oxygen supply to the brain.

Electronic assessment of cerebral brain function: electroencephalogram evoked potentials.

Indirect assessment of cerebral function such as changes in respiration and autonomic nervous responses.

Assessment of other organs e.g. heart and kidneys. Since the patient's blood pressure is pharmacologically manipulated, continuous direct measurement of systemic arterial pressure is mandatory. In profound hypotension (mean arterial pressure 50 mm Hg) during aneurysm surgery), the reference point for all blood pressure monitoring must be the highest level of the cortex; because even slight elevation of the head by 10-15 cm above the heart level can reduce cerebral perfusion pressure by 10 mm Hg and thus compromise cerebral blood flow.

Before induction of hypotension, arterial oxygen tension and pH must be at adequate levels. After reduction of mean arterial pressure below 50 mm Hg, arterial blood gases should be determined at fifteen minute intervals to assure adequate oxygenation and detect the appearance of metabolic acidosis.

Electrocardiographic monitoring is routine during anaesthesia. Bradycardia is consistently seen with moderately severe cerebral ischaemia and when associated with arrhythmia it is a very ominous sign.

Specific hypotensive agents such as sodium nitroglycerine and the volatile anaesthetic agents are used to produce hypotension. Since these agents can all cause significant increase in cerebral blood volume and intra-cranial pressure, their use could be hazardous before brain volume has been decreased by diuretics and hyperventilation.

Nitroglycerine becuase of its protective action on the myocardium has an advantage over sodium nitro-prusside; further more the risks of toxicity from cyanide production are absent and tachyphylaxis has not been a problem.

Anaesthetic Agents

Selection of anaesthetic agents will depend on the effect of these agents on cerebral blood flow, intra-cranial pressure and brain metabolism. Hypnotic agents like the barbiturates Althesin and Etomidate have all been shown to reduce brain metabolism, cerebral blood flow and intra-cranial pressure. They are capable of maximizing perfusion to ischaemic areas by reducing cerebral blood flow in normal brain. This together with a reduction in intracranial pressure may explain their protectve action. Althesin is rapidly metabolised by the liver and so recovery is quicker. It can be used as a cotinuous intravenous drip. Norcotics by causing respiratory depression and a rise in arterial carbon dioxide can increase cerebral blood flow; however, with controlled hyperventilation they are unlikely to have adverse effects.

The volatile agents Halothane and Isoflurane will cause rise in intra-cranial pressure, but hyperventilation and the simultaneous administration of Althesin or barbiturate will control any adverse vasodilator effects of these agents. Halothane is a cerebral metabolic depressant: an added advantage in profound hypotension.

Nitrous oxide causes marked rise in intra-cranial pressure in patients with intra-cranial lesions causing a mass effect.

FLUID REPLACEMENT

Fluid Replacement is carefully controlled to match urine output until the aneurysm is clipped. This will help to decrease intra-cranial and systemic arterial pressure; any fluid deficit is replaced with 5% albumin and plasma after the aneurysmal clipping, as part of volume expansion to prevent vasospasm.

RECOVERY FROM ANAESTHESIA

Recovery form anaesthesia should be as smooth and controlled as induction. Hypertension and hypotension tachycardia are controlled with the appropriate anti-hypertensive agents and volume replacement.

Early awakening from anaesthesia is preferred so that post-operative neurological status can be followed. Except in the case of respiratory depression, patients are extubated in the operating room.

In the recovery room, systemic arterial pressure, ECG, and arterial blood gases and serum electrolytes are closely monitored.

Conclusion

The high mortality and morbidity from intra-cranial aneurysms demonstrate ample room for improvement of pre-operative management. Strategies of potential benefit include: improved diagnosis and more accurate recognition of antecedent symptoms and warning leaks, early referral to specialised centres, prevention of rebleeding and vasospasm, and improved surgical results through technical innovations and regionalisation.

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