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ANESTHETIC CONSIDERATIONS FOR CRANIOTOMY REPAIR OF INTRACRANIAL ANEURISMS AND TREATMENT OF SUBARACHNOID HEMORRHAGE

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Subarachnoid hemorrhage (SAH) is a common condition that is associated with significant mortality and morbidity. Early treatment is essential, yet diagnosis and management can be challenging. 85% of cases of SAH result from ruptured cerebral aneurysms, typically from berry aneurysms. At the time of the initial bleed, there is a critical reduction in cerebral blood flow (CBF) as the regional intracranial pressure (ICP) increases and approaches the systemic arterial pressure. The expanding mass effect of the hemorrhage and subsequent brain edema and hydrocephalus contributes to the acute rise in ICP. If ICP does not decrease rapidly after the initial sudden increase, death usually follows. Anesthesiologists, together with the neurosurgeon, play a vital role in the management of SAH. This article provides a brief description of the principles of anesthesia in the treatment of SAH, as well as the relevant epidemiological, pathophysiological, diagnostic and therapeutic points. The aim in anesthesia management is to prevent aneurysm rupture or rebleeding, and to avoid factors that may promote cerebral ischemia, vasospasm, cardiopulmonary dysfunction, hydrocephalus or electrolyte disturbances.

1. Epidemiology and etiology

The age adjusted incidence of subarachnoid hemorrhage (SAH) is approximately 7.5 to 12.1 cases per 100,000, though it varies between counties from 2.0 cases per 100,000 in China to 22.5 per 100,000 in Finland. 85% of cases of SAH result from ruptured cerebral aneurysms, typically berry aneurysms. 7-20% of people with SAH from an aneurysm have a family history of SAH. Other causes of SAH include trauma, vertebral and carotid dissection, spinal arteriovenous malformations, cocaine abuse, coagulation disorders, mycotic aneurysms, and sickle cell disease. SAH accounts for approximately 5-15% of all strokes, though the average age of SAH is significantly younger than other forms of stroke (50 versus 65 years of age, respectively). SAH is associated with significant mortality and morbidity. The mortality rate for SAH is up to 50% at 29 days, with 10-15% fatal before reaching a hospital. Those who survive often develop neurological or cognitive impairment.

2. Pathophysiology

The early causes of death and disability after SAH are usually the direct effect of the initial bleed. At the time of the initial bleed, there is a critical reduction in cerebral blood flow (CBF) as the regional intracranial pressure (ICP) increases and approaches the systemic arterial pressure. The expanding mass effect of the hemorrhage and subsequent brain edema and hydrocephalus contributes to the acute rise in ICP. If ICP does not decrease rapidly after the initial sudden increase, death usually follows.

Delayed complications include cerebral vasospasm, rerupture, and hydrocephalus. The persistent lack of blood flow results in cerebral vasospasm, which affects 60-70% of patients, and typically develops 3-12 days after SAH. Acute vasospasm results in swelling of perivascular astrocytes, neuronal cells, and capillary endothelium. Cerebral arteriography and Transcranial Doppler can detect cerebral vasospasm before symptoms manifest clinically 3-5 days post SAH.

Cardiac dysfunction is of major concern after SAH. The increasing ICP after SAH and subsequent injury to the posterior hypothalamus may stimulate the release of norepinephrine from the adrenal medulla and sympathetic cardiac efferent nerves, resulting in ischemic changes to the subendocardium, marked systemic and pulmonary hypertension, cardiac dysrhythmias, and neurogenic pulmonary edema.

Electrocardiogram (ECG) abnormalities such as increased QT interval, QT depression, T-wave inversions, U waves, ST depressions and arrhythmias are common but do not necessarily correlate with a poor outcome. Increased levels of serum cardiac troponin and creatine kinase MB isoenzymes have also been reported, which reflect myocardial cell injury and are associated with poor outcome.

SAH is frequently accompanied by electrolyte imbalances including hypokalemia, hypocalcemia, and hypomagnesaemia. Hyponatremia is a late finding in up to 56% of patients with SAH, often a result of either cerebral salt wasting syndrome or the syndrome of inappropriate secretion of antidiuretic hormone. Patients most often present with high blood

glucose levels, which have been shown to correlate with a poor outcome.

The sudden increase in regional intracranial pressure (ICP) leads to the rapid onset of a severe headache, confusion or a lowered level of consciousness. Large blood clots can cause focal neurological signs in some patients. A potential temporary loss of consciousness can predispose the patient to pulmonary aspiration of gastric contents and often may result in severe pulmonary complications. The spread of blood through the subarachnoid space is the cause of meningism, headache, and hydrocephalus. In patients who survive the initial bleed, emergent intervention is necessary to reduce the risk of rebleeding, by securing the aneurysm by surgery or endovascular coiling. The swollen and inflamed brain after SAH presents numerous technical difficulties during intervention. Neurosurgical management is complicated by the risk of rebleeding vasospasm. The incidence of rerupture is 10-30%, and carries a 60% mortality rate.

3. Preoperative evaluation

Preoperative treatment is evaluated individually, though there are a number of considerations specific to patients with SAH that are of importance. For successful anesthetic planning, one must obtain information regarding the degree of SAH and physiologic consequences that followed, as well a detailed history regarding the patient's past medical and surgical history. Close attention should also be paid to patients' medications and allergies. Due to the decline in mental status that often accompanies SAH, it is helpful to obtain relevant information from close family members.

The emergent nature of surgical intervention precludes medical optimization, therefore objective studies of cardiovascular function, including recent stress test and coronary angiography, are helpful in determining cardiac risk. ECG abnormalities are common after SAH and do not necessarily reflect underlying cardiac disease. Serum concentrations of cardiac enzymes and biomarkers should be measured and echocardiography performed in patients with a high suspicion of cardiac damage. It is important to note that surgery takes priority and should not be prevented or postponed for additional preoperative cardiac testing. Evaluation of preoperative laboratory studies, including complete blood count (CBC), chemistries, and coagulation studies, are also important prior to surgery. Neurologic status on admission is described using the Hunt and Hess or the World Federation of Neurological Surgeons (WFNS) classification, with the amount of blood on CT described using the Fisher classification.

Signs of persistent increased ICP should be sought on physical exam, though most patients will have normal ICP at the time of surgery. A comput-

ed tomography (CT) scan is done preoperatively to confirm SAH diagnosis and to determine the extent of bleeding. Radiologic studies with CT angiogram, magnetic resonance (MR) angiography, or angiography further help determine the size and location of an aneurysm and patient positioning on the operating table. In patients with hydrocephalus, review of brain imaging will often demonstrate ventricular enlargement.

4. Premedication

As with all areas of anesthetic practice, the risks and benefits of premedication must be considered in each patient individually. Particularly, in patients with SAH, the potential risks of respiratory depression associated with sedating medication (resulting in hypercapnia and subsequent increase in ICP) are weighed against the benefit of reducing the potential hypertension that accompanies anxiety (thus reducing the risk of aneurysm rupture). Although preoperative sedation is often not needed, when done it must be titrated in order to prevent hypoventilation and subsequent increases in ICP. Most conscious patients with normal ICP are sedated after SAH to prevent rebleeding, and this sedation should be continued until induction of anesthesia. Patients with persistently elevated ICP should receive little or no premedication to avoid hypercapnia.

5. Intraoperative monitoring

Standard patient monitoring generally includes 5-leads ECG, pulse oximetry, capnography, urinary output, body temperature, and neuromuscular blockade. Because of the risks associated with hypertension, an arterial catheter for hemodynamic monitoring is mandatory prior to induction. A central venous catheter is often inserted for guidance of intravascular volume, injection of cardiovascular drugs if needed, and administration of mannitol. In patients with significant cardiopulmonary disease, additional cardiovascular monitoring is often used (i.e. pulmonary artery catheterization, transesophageal echocardiography, etc.).

In patients with a poor clinical grade or hydrocephalus, some type of ICP monitor, or ventricular catheter that can be used for pressure measurements, are frequently used. Cortical somatosensory-evoked potential (SSEP) and brainstem auditory-evoked potential (BAEP) can be used to monitor cerebral function. It is important to note that when neurophysiological monitoring is used, volatile anesthetics, nitrous oxide and benzodiazepines are typically avoided because they interfere with the recordings of evoked potentials. Jugular bulb oxygen monitoring can also be helpful in patients at risk for global cerebral ischemia.

7. Induction of anesthesia and airway management

During induction of anesthesia, it is important to achieve adequate depth of anesthesia while preserving cerebral oxygenation and avoiding an increase in ICP. Avoidance of hypertension, which is poorly tolerated because of its potential to elevate the aneurysm's transmural pressure and lead to rupture, is essential in patients with SAH. Hypertensive responses should be anticipated by noxious stimulation, including direct laryngoscopy, tracheal intubation, positioning the patient, placement of the patient's head in immobilizing pins, and raising the bone flap.

Induction is frequently achieved by intravenous administration of hypnotics (propofol or sodium thiopental), an opioid and a nondepolarizing neuromuscular blocking drug (e. g. vecuronium, atracurium and cisatracurium). To suppress the blood pressure responses on insertion of head pins, one should infiltrate the scalp with local anesthetic at the sites of pin placement. Opioids limit the need for higher-dose volatile anesthetics and may be useful in avoiding side effects of volatile anesthetics, such as cerebral vasodilation and increased cerebral blood flow. With their minimum alveolar concentration-sparing effects, opioids are useful adjuncts for blood pressure control during aneurysm surgery. Nitrous oxide, especially if used alone, is generally avoided because it may lead to increases in cerebral metabolic rate, cerebral blood flow, and ICP. In addition, nitrous oxide can lead to an expansion of air pockets left from a previous craniotomy within the cranium in patients who have had a recent craniotomy, which can further lead to tension pneumocephalus.

Intubation with an endotracheal tube must be accomplished quickly to reduce the expected consequences of prolonged apnea, including hypercapnia and subsequent cerebral vasodilation and increase in ICP. The anesthesiologist must be mindful that patients, especially those with poor clinical grades, with increased systemic blood pressure and prolonged apnea may quickly decompensate from a decrease in cerebral perfusion pressure. Hypertension during laryngoscopy should result in discontinuing the attempt, mask ventilating the patient, and deepening the anesthetic prior to reattempting laryngoscopy. Deepening the anesthesia can be accomplished by increasing the concentration of inspired volatile agent, or giving a bolus an intravenous agent such as propofol or remifentanyl. Additionally, esmolol (0.5 mg/kg) can be given prior to laryngoscopy to blunt the hypertensive response.

8. Maintenance of anesthesia

Regardless of the anesthetic technique used, the management of anesthesia maintenance should focus on preventing aneurysm rupture or rebleeding,

and avoiding factors that promote cerebral ischemia or vasospasm. A balanced anesthetic with continuous intravenous infusion of an opioid and either propofol or inhalation of a volatile anesthetic is used to maintain the anesthesia, however one should keep in mind the hemodynamic effects of these agents. Volatile anesthetics cause a dose-related reduction in the amplitude and increases in the latency of the cortical components of median nerve SSEP. Therefore, for both SSEP monitoring and brain relaxation, the depth of anesthesia with sevoflurane or isoflurane should not exceed 1 MAC or should be avoided. Furthermore, the inspiratory concentration of sevoflurane must not exceed 3% because of the assumed epileptogenic effect.

In certain situations, it is best to avoid inhaled anesthetics entirely. If motor evoked potentials (MEP) are monitored, which requires high signal quality, and when brain relaxation is inadequate with a volatile agent, a continuous propofol infusion is used for maintenance. Also, if there is brain edema present, the administration of any inhalation anesthetic including nitrous oxide must be discontinued and propofol should be used instead. Propofol also is advantageous in that it decreases the risk of postoperative nausea. The short-acting agent remifentanyl is the most appropriate opioid to use, because of its minimal residual effects after extubation, allowing the patient's neurological status is examined. If SSEP or MEP monitoring is employed together, muscle relaxation should be avoided. Muscle relaxation can be used though in isolated SSEP monitoring.

9. Ventilation management

In most cases, unless there is increased ICP, normoventilation is the goal of ventilating patients with SAH. In patients with elevated ICP, hypocapnic cerebral vasoconstriction is an important tool during surgery to control cerebral blood flow. Thus, surgical dissection is facilitated by and moderate hyperventilation of the patient's lungs, which help decrease brain swelling. A goal PaCO₂ of 30 to 35 is generally well-tolerated in patients undergoing treatment of SAH. The duration of the effect of hyperventilation is controversial, though the clinical benefits appear to be sustained during procedures of modest duration.

10. Fluid management and hemodynamics

Hemodynamic control is important during dissection of the aneurysm to prevent intraoperative rupture. It was once common practice to deliberate hypotension to the patient in order to decrease the risk for rupture of an intracranial aneurysm and to facilitate surgical placement of occlusive vascular clips. However, now temporary occlusive clips applied to the major feeding artery of the aneurysm can create regional hypotension without the need for systemic hypotension and its inherent risk on multiple

organ systems. Therefore, normal or even increased systemic arterial blood pressure should be introduced to facilitate perfusion through the collateral circulation, though this point is controversial. Drugs, such as thiopental may be given to provide some protection from cerebral ischemia during periods of prolonged or excessive hypotension or vascular occlusion. Occasionally, hypothermic circulatory arrest may be used for very large complex aneurysms.

The hemodynamic goal in the surgical treatment of SAH should be euvolemia to slight hypervolemia. Hypotonic solutions, including lactated Ringer solution and glucose-containing solutions should be avoided. Colloid has no proven advantage over crystalloid fluids, and hetastarch can even result in coagulopathy. Upon opening the dura, mannitol (usually at a dose of 0.5-1.0g/kg) can be administered to reduce brain water content. This effect facilitates surgical exposure, and reduces tissue trauma from surgical retraction. Mannitol is not administered prior to opening the dura, however, because sudden decreases in ICP can cause rebleeding by removing a tamponading effect on the aneurysm. An alternative to mannitol is furosemide, which can be given alone at a high dose (1mg/kg) or in combination with mannitol (0.25-1g/kg). The disadvantages of using furosemide is that alone, it reduces ICP and brain bulk less reliably and effectively as mannitol, and combination therapy can be associated with large losses of free water and electrolytes.

In the event of intraoperative aneurysm rupture, a devastating complication that can carries significant morbidity and mortality, the hemodynamic goal (regardless of size and location of the rupture) is to maintain normovolemia.

12. Emergence and postoperative considerations

The approach to awakening and extubation is similar to other craniotomies. In most cases, unless there is significant neurologic impairment, the patient should be awakened at the completion of surgery and the endotracheal tube can be removed. Careful attention should therefore be paid to the patient's ability to follow commands, the airway, oxygenation, and ventilation. A rapid awakening allows for neurological examination after the surgery before transport to the intensive care unit. If the awakening time is prolonged beyond expected elimination time of the effects of the anesthetic drugs, a CT scan should be considered to rule out a postoperative intracerebral hematoma, occlusion of a blood vessel or other causes of unconsciousness. Arterial carbon dioxide concentration should not be allowed to rise in case of a postoperative hematoma, because even mild hypercarbia can cause a marked increase in the ICP. Serum electrolyte balance is also monitored carefully and hyponatremia is considered a sign of immanent postoperative complications.

Measures to prevent vasospasm and seizures while maintaining adequate cerebral perfusion pressure should be continued during care of these patients postoperatively, with the greatest risk of vasospasm up to 14 days after the bleed. Triple-H therapy (hypertension, hypervolemia, and hemodilution) is used to treat vasospasm and increase cerebral blood flow. Other treatment options include intravenous administration of nimodipine (a calcium channel blocker), urgent balloon catheter angioplasty of the effected cerebral vessel, and intra-arterial injection of vasodilators such as papaverine.

RECOMMENDED LITERATURE

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ТҰЖЫРЫМ

Субарохноидалды қан құйылу көп жағдайда мүгедектік пен өлімге әкеліп соғатын жағдай болып табылады. Осы кезде емді ерте бастау өте маңызды, сонымен қатар осы ауыр жағдайдағы науқастарды диагностикалау мен жүргізу ауыр мәселе болып отыр. Субарохноидалды қан құйылудың 85 пайызы ми аневризмасының жарылуы нәтижесінен (әдетте қалталы аневризма)

болады. Қан құйылудың бастапқы сәттерінде ми қан айналымы күрт төмендейді және сол уақытта бас ми қысымы жылдам жоғарылап, жүйелі артериалды қан қысымымен теңеседі. Қан құйылудың өз көлемінің ұлғаюы мен мидың ісінуі күшейіп, гидроцефалюс (мидың сулану) бірігіп, бас ми ішінің қысымын жоғарылатады. Көтерілген бас ми ішінің қысымын бастапқы жоғарылау кезеңінде

тез және әсерлі емдемесе, ол әдетте мидың өліміне әкеледі. Субарохноидалды қан құйылдыуды анестезиологтар мен нейрохирургтар бірігіп емдеуі өте маңызды рөл атқарады. Беріліп отырған статьяда субарохноидалды қан құйылдыуды қарқынды емдеу мен анестезия беру барысы қысқаша түсіндіріледі. Сонымен қатар патогенезі мен диагностикалау әдістері көрсетілген. Осы науқастарға операция

жүріп жатқан кездегі анестезия жүргізудің мақсаты: аневризманың қайталанып жарылуы нәтижесінен болатын мидың ісінуі, ми ишемиясы, вазоспазм, тыныс алу мен жүрек – қан айналымы жүйесінің қызметінің бұзылуы мен су-электролит тепе-теңдігінің бұзылуына әкелетін факторлардың алдын-алып, қайта қан құйылу қауіп-қатерін төмендету.

РЕЗЮМЕ

Субарахноидальное кровоизлияние (САК) является обычным состоянием, которое сопряжено со значительной инвалидностью и смертностью. Раннее начало лечения является исключительно важным, в то время, как диагностика и ведение этих тяжёлых больных остаются серьёзной проблемой. 85% всех случаев САК являются результатом разрыва мозговых аневризм (типично мешотчатых аневризм). В момент начального кровоизлияния происходит критическое снижение мозгового кровотока, в то время, как региональное внутричерепное давление (ВЧД) резко возрастает, достигая системного артериального давления. Нарастающий объёмный эффект самого кровоизлияния и нарастающего отёка мозга и гидроцефалюс (водянка мозга) вместе способствуют нарастанию ВЧД. Если повышенное ВЧД не удаётся контролировать быстро и эффективно после

начального подъёма, обычно наступает смерть мозга. Анестезиологи вместе с нейрохирургами играют критически важную роль в лечении САК. Данная обзорная статья предлагает краткое описание принципов анестезии и интенсивной терапии САК, также как, и патогенез, и диагностику данного состояния. Цели анестезии и ведения этих больных в периоперационном периоде: снижение риска повторного кровоизлияния, как результат повторного разрыва аневризмы, избегания факторов, которые могут спровоцировать отёк мозга, ишемию мозга, вазоспазм, водянку мозга, сердечно-лёгочную дисфункцию и водно-электролитные нарушения.

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