Indications for surgery

Indicaciones quirúrgicas

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Introduction

The therapeutic management of traumatic brain injury (TBI) will depend on the clinical situation of the patient, the severity of the TBI (measured by the Glasgow Coma Scale – GCS) as well as the presence of structural lesions or physiopathological changes in the brain that could have a significant impact on intracranial content.

Most TBIs are mild and are treated by neurological monitoring and/or medical treatment. However, severe TBIs with structural intracranial lesions or that involve a solution of continuity between the central nervous system and the external environment may require surgery.

To better understand the indications for surgery in TBIs, the subject will be addressed according to the different lesions or complications that may occur following TBI.

Cranial fractures

Cranial vault fractures

Most cranial vault fractures are linear and have no repercussion on the intracranial structures. However, some fractures do require surgery, such as open fractures in which there is a solution of continuity between the intracranial content and the external environment and, therefore, an important risk of infection; or depressed fractures that could cause injury to the underlying brain parenchyma, meninges or intracranial blood vessels, as well as major cosmetic defects[1]. In these cases, it is important to remove bone splinters and foreign bodies, repair the damaged structures and reconstruct the morphology of the skull, either by joining the bone fragments or, if this is not possible, through the use of titanium mesh or the application of bone cement.

Cranial base fractures

Most cranial base fractures are linear and extend through a fracture of the calvarium. Although they do not generally require surgery, the treatment of some conditions associated with fractures of this type may be necessary:

- *Cerebrospinal fluid (CSF) fistulas:* CSF fistulas increase the risk of infectious meningitis and intracranial hypotension syndrome. CSF fistulas are more common in the anterior cranial fossa (at the level of the lamina cribrosa) and through the sphenoid sinus, with rhinoliquorrhea being a characteristic sign. Fractures of the petrous bone may cause fistulas with otoliquorrhea, the leakage of CSF towards the ear. CSF fistulas can be treated conservatively through the placement of a lumbar drain or through the surgical sealing of the defect.
- *Post-traumatic facial nerve lesion:* This occurs as a result of a fracture of the petrous bone. Petrous bone fractures can be longitudinal (70-80%) or transverse (20%), the latter being the cause of most traumatic lesions of cranial paired nerves VII and VIII. Surgical treatment of the facial nerve lesion consists in nerve decompression and is performed in the event of progressive post-traumatic facial palsy or when conservative therapies have not been successful.
- *Vascular disorders:* Post-traumatic aneurysms, carotid-cavernous fistulas and vascular sections are prominent. Vascular lesions are serious complications and are generally associated with clivus fractures.

Fractures of the anterior cranial base affecting the frontal sinus are relatively frequent in contact sports. Fractures of this nature are generally accompanied by CSF fistulas and pneumocephalus. Furthermore, the presence of a connection between the upper respiratory tract and the intracranial cavity increases the risk of infection by germs from the nasal

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microbiome. Fractures affecting the frontal sinus anterior wall are treated conservatively, unless there is a major anatomical defect; while posterior wall fractures carry a greater risk of CSF fistulas and, consequently, of infection (subdural empyemas, brain or mucocele abscesses) In these cases, surgery is recommended. The operating procedure consists in the removal of the posterior wall of the frontal sinus, a process termed cranialization; subsequently mucus marsupialisation is performed towards the nasofrontal duct and, finally, the duct is plugged with sealants, muscle flaps or even fat.

Tension pneumocephalus

Pneumocephalus, also known as pneumotocele, consists in the presence of air in the intracranial cavity and generally appears in open cranial fractures or when a CSF fistula occurs. In general, there are no symptoms. Most cases are either resolved spontaneously or following oxygen therapy with FiO_2 at 100%[2]. In some cases (cranial fractures or after a craniotomy) a valve effect occurs, permitting air to enter but not to exit the intracranial compartment, leading to a sharp increase in intracranial pressure (ICP). This phenomenon is termed pneumocephalus and must be treated by surgery whenever it causes severe neurological symptoms. Surgery consists in the release of the pressurised air either through burr hole trephination or by puncturing a pre-existing closed burr hole.

Epidural haematoma

Epidural haematoma occurs in approximately 14-35% of patients with severe TBI[3]. It generally affects young patients, either because it is the age group that is most prone to suffer TBIs with a moderate-high impact (for example, sports accidents) or because the dura is more attached to the inner table of the skull than in older patients.

In most cases, the source of the haemorrhage is the lesion of the middle meningeal artery as it exits the pterion groove, as a result of a temporoparietal fracture. In this context, arterial bleeding occurs, which strips the dura away from the inner table of the skull causing an intracranial mass effect. Less than 10% of cases of epidural haematoma are located in the frontal region, occipital region or in the posterior fossa. In such cases, the source is either the lesion of the meningeal veins or the dural sinuses.

An epidural haematoma represents a surgical emergency in most cases. Epidural haematomas in children are more dangerous than in adults, given that children have a smaller intracranial volume. For this reason, it is recommended to establish a lower surgical treatment threshold for patients in the paediatric age group[4].

Following the practical recommendations with evidence level III , surgery must be performed on epidural haematomas with a volume of more than 30 cubic centimetres regardless of the patient's clinical condition, measured by the GCS, clots with a maximum thickness of more than 15 millimetres, a haematoma that causes a midline shift of more than 5 millimetres, patients with epidural haematoma evidenced by CT imaging and a score of less than 9 on the GCS, and patients with an epidural haematoma and presence of neurological focal deficits[5]. Likewise, with evidence level III, immediate surgical evacuation is strongly recommended in those cases of acute epidural haematoma with a GCS score of less than 9 and anisocoria.

The standard operating procedure for epidural haematomas consists in the performance of a craniotomy, clot evacuation, achieve appropriate haemostasis (in most cases it is necessary to clot the bleeding vessel) and prevent haemorrhagic recurrence through the placement of stay sutures on the dura mater.

For asymptomatic patients and for small epidural haematomas, conservative therapeutic management is possible[6]. In these cases, it is important to ensure neurological monitoring and the performance of serial CT scans of the brain. The use of corticosteroid drugs in a descending dose, is controversial.

Acute subdural haematoma

An acute subdural haematoma is a surgical emergency. These haematomas are frequently associated with injury in the underlying brain parenchyma and can cause symptoms, either due to the mass effect exerted by the haematoma or to the direct injury to the bran parenchyma after impact. The source of the subdural haematoma could be due to the accumulation of blood around the parenchymal laceration site or due to the rupturing of a superficial vessel as a result of the rapid acceleration-deceleration forces suffered by the brain during the movement of the head. As a result, acute subdural haematomas have a high morbidity/mortality rate, and even more so when surgery is not performed right away. In fact, it is estimated that mortality for acute subdural haematomas treated within the first four hours since the traumatic event is 30% while, after this time, mortality increases up to 90%. It should be mentioned that secondary cranioencephalic trauma caused by a motorbike accident has a worse prognosis, particularly for patients not wearing a helmet.

Chronic treatment with antithrombotic drugs, and anticoagulants in particular, considerably increases the risk of acute subdural haematoma. This is important in older athletes.

Following the practical recommendations with level of evidence III, surgery must be performed on acute subdural haematomas with a thickness of more than 10 millimetres or that cause a midline shift of more than 5 millimetres regardless of the patient's clinical condition, measured by the GCS, as well as acute subdural haematomas that have a thickness of less than 10 millimetres or a midline shift of less than 5 millimetres if the patient drops two or more points on the GCS since the time of the trauma up to the time of the medical care at the hospital, or if there are alterations in the pupil reflex (anisocoria, fixed mid pupil or bilateral mydriasis) or if the ICP is greater than 20 millimetres of mercury[7]. With level III evidence, it is recommended to monitor the ICP in all patients with acute subdural haematoma and a GCS score of less than 9[8].

Surgical procedure consists in performing a craniotomy, opening the dura mater, evacuation of the subdural clot and blood vessel haemostasis. In the event of the laceration of the cerebral cortex, it is possible to remove the contused brain parenchyma, considering the risk/benefit and possible neurological sequelae if it is an eloquent area. The dura mater can be sealed by direct suture, with flaps/plasties or, if there is considerable brain swelling, there is the possibility of not performing duroplasty or even craneoplasty. In the same way as for epidural haematomas, the placement of stay sutures on the dura mater prevents the appearance of epidural collections.

Chronic subdural haematoma

Chronic subdural haematomas have a pathophysiological mechanism that is different to that of acute subdural haematomas. Although it is likely that chronic subdural haematomas were initially acute subdural haematomas (in most cases there is a prior TBI), chronic subdural haematomas exhibit an inflammatory clotting response that leads to the formation of membranes and to the proliferation of neovessels. In most cases, when blood becomes chronic, it undergoes an enzymatic degradation process and liquefies, losing volume. The shrinkage of the clot volume leads to the tearing of small subdural neovessels, giving rise to fresh bleeding (re-bleeding) and to the development of a new subdural haematoma. This process may be repeated a number of times, creating very large subdural haematomas.

Chronic subdural haematomas are frequent in older adults and with factors predisposing to bleeding (antithrombotic drugs, chronic alcoholism, coagulopathies, etc.)[9], and are therefore infrequent in athletes. Likewise, very large chronic subdural haematomas are common in older patients and/or with brain cortical atrophy, given that there is a larger subdural space for the haematoma and they better tolerate re-bleeding until the onset of symptoms.

Surgical treatment is required for chronic subdural haematomas with a maximum thickness of more than 1 centimetre or in cases of focal neurological deficits or altered mental condition.

Post-traumatic brain haemorrhage

Post-traumatic brain haemorrhages or cerebral haemorrhagic contusion result from acute bleeding in the brain parenchyma. Post-traumatic brain haemorrhages are more frequent at locations in which the sharp deceleration forces of the head cause the brain to impact against bony protuberances (for example the frontal lobes, temporal poles and occipital lobes)[4]. A delayed post-traumatic brain haemorrhage can occasionally occur, in other words outside the first 72 hours; this is due either to blood extravasation towards the necrotic tissue, the convergence of various microhaemorrhages or to coagulopathies. It is important to perform serial imaging studies in order to detect bleeding of this type.

Following the practical recommendations with level of evidence III, post-traumatic parenchymal haemorrhages should be surgically treated when a patient has a score of 6-8 on the GCS or exhibits progressive neurological deterioration and has a volume of more than 20 cubic centimetres in the temporal lobes or greater than 50 cubic centimetres in any location, causing a midline shift of more than 5 millimetres and/or showing imaging signs of brain herniation. Surgery must also be performed on those post-traumatic brain haemorrhages causing neurological deterioration and exhibiting intracranial hypertension that is resistant to pharmacotherapy[10].

Surgical procedure consists in performing a craniotomy and the evacuation of the cerebral parenchymal haematoma. It is important to ensure good haemostasis and the application of anti-oedema measures, given that in many cases oedema is associated with the cerebral parenchymal contusion. In the event of a cerebral oedema or external cerebral herniation (through the craniotomy) decompressive craniectomy should be performed[11]. Likewise, the risk/benefit of a surgical operation should be assessed with regard to the patient's vital/functional condition, all the more so when the haemorrhage is located in eloquent areas. In these cases, it is important to assess the prior functional condition, potential comorbidities and the psychosocial situation of the patient.

Cerebral oedema (swelling)

Cerebral oedema or *swelling* appears as a result of structural damage to the nerve tissue, not only in the neuronal cells themselves but also at the hematoencephalic barrier, producing a mixed vasogenic-cytotoxic oedema. In most cases, cerebral oedema is associated with a primary cerebral lesion, and haemorrhagic contusions in particular. In other cases, a delayed oedema appears as a result of secondary lesions such as microhaemorrhages, cerebral infarctions, or due to changes in the metabolic mechanisms for the self-regulation of the brain.

The surgical indications are a point of contention. According to the practical recommendations with level of evidence III, the performance of decompressive craniectomy is recommended for patients with post-traumatic diffuse cerebral oedema and a structural lesion in the cerebral parenchyma or exhibiting intracranial hypertension that is resistant to pharmacotherapy. The procedure must be performed within the first 72 hours after the traumatic event, being more effective when performed earlier on or whenever the failure of the cerebral autoregulation mechanisms is observed through an increase in the ICP[12]. The DECRA study reported an increase in the survival of patients with severe TBI and refractory intracranial hypertension, treated through decompressive craniectomy[13]. However, the increased survival rate is closely related to an increase in neurological disability[14]. In these cases, the risk/benefit of the surgical operation must be assessed, and particularly in older patients or those with a poor baseline condition.

Post-traumatic hydrocephalus

Post-traumatic hydrocephalus consists in the excessive pathological accumulation of CSF in the cerebral ventricles following TBI. It generally appears weeks of months after TBI, particularly in the case of a severe

TBI. When the hydrocephalus appears more than 6 months, or even years, after the traumatic event, it is difficult to differentiate it from the ex-vacuo hydrocephalus secondary to a diffuse axonal injury.

Standard surgical procedure to treat post-traumatic hydrocephalus consists in a ventricular shunt placement[15]. Post-traumatic hydrocephalus must be treated when there are signs of intracranial hypertension, papilledema, the presence of ventriculomegaly and transependymal oedema in the imaging tests, high values of ICP in one or more lumbar punctures, or positive scores in the provocation test[16].

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