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Contemporary Re-evaluation of Decompressive Craniectomy in the Treatment of Serious Cranioencephalic Trauma

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Abstract

The health complications that are responsible for most deaths from trauma worldwide are traumatic brain injury (TBI) and intracranial hypertension (ICH), refractory to neurointensive and neuroprotective management, representing a significant problem in public health. In this context, decompressive craniectomy, a technique that had fallen into disuse in the past, has regained relevance over the last decade due to its outstanding results in the management of intracranial hypertension, being used systematically in cases of severe head trauma and other causes of ICH. Given the current epidemiology of traumatic brain injury, a comprehensive review of the scientific literature is essential to understand the pathophysiological mechanisms involved in traumatic brain injury. Early identification and proper management of intracranial hypertension are crucial to prevent or minimize irreversible brain damage and improve long-term outcomes.

Keywords: *Decompressive craniectomy, Intracranial hypertension, Traumatic brain injury, Neurointensive, Neuroprotection*

1. Introduction

Traumatic brain injury (TBI) is one of the leading causes of mortality and disability worldwide. The average age of the population in developed countries increases at a great pace, although in this range the economically and economically active population remains affected, which has generated that many of the concepts and management strategies used until today become obsolete, new treatment options are reconsidered and others are resumed, as is the case of decompressive craniectomy. In addition, we are facing a multi-pathological population aging, which should make us change the approach. The World Health Organization estimates that, by 2030, TBI will be the third leading cause of morbidity and mortality worldwide. (2)

Intracranial hypertension syndrome (ICH) is manifested by an increase in the value of intracranial pressure (ICP) from the normal range, which, depending on the stage at which it is determined, causes different signs and symptoms in the patient, usually produces headaches, vomiting, papilledema, Cushing's triad and focal neurological symptoms; If not treated in time, it can lead to cephalocaudal degeneration, resulting in coma and death. According to the Monroe-Kelly law, ICP is determined by brain volume, cerebrospinal fluid (CSF), blood volume, and brain parenchyma. In this review we limit ourselves to the consideration of the traumatic etiology of this syndrome. (6)

Within the literature reviewed, there is a great variability of results. The most notorious has a temporal division of the studies, establishing a fictitious limit between those prior to 1991 and those carried out from 1997. Although those belonging to the first group combine worse results than the most recent, they are characterized by generally using retrospective studies, without a standardized methodology in their surgical technique or in conjunction with the rest of the measures applied before having resorted to CD, presenting strong heterogeneity in the groups of patients (mixing etiology, severity or even sometimes with inadequate randomization). which sometimes did not have a control group with which to establish a comparison, or resorting to CD only in very advanced stages of refractory ICH or in severely polytraumatized patients, which has made it difficult to reach conclusions and standardized protocols worldwide. [18]

The indications for performing CD have been involved in controversies, finding heterogeneity in the criteria for applying this surgery. While this is a technique that can save the lives of these patients, the high rates of disability and vegetative state reported should not be spared. Therefore, prior to the surgical resolution at the time of signing the informed consent, the type of craniotomy to be performed, its risks and complications, as well as disabling neurological sequelae and high mortality must be clearly and concisely detailed, a decision that the family will take and doubts are clarified regarding their expectations and the expected quality of life in the patient. [3]

The mortality and disability rates due to severe traumatic brain injury in Ecuador have increased exponentially over the last few years, which generates a direct and considerable increase in the health budget and in the economy of these families, which is why we developed this bibliographic review, with the aim of gathering criteria and reaching conclusions regarding the indications of decompressive craniectomy and based on Literature and professional experience in the field detail the complications of this procedure [13].

2. Materials And Methods

During the months of July and August 2022, an exhaustive descriptive literature review was carried out on decompressive craniectomy in the context of traumatic brain injury. The aim of this review was to collect and analyse the most up-to-date and relevant information related to this surgical technique.

To carry out the review, several renowned scientific databases were consulted, such as Medline, PubMed, Google Scholar, Web of Science and Clinical Key. In addition, the search was complemented with high-impact journals, including Scielo and Scopus, to ensure broad and solid coverage of the existing literature on the topic.

The search focused on articles addressing key aspects of decompressive craniectomy, including its prevalence, classification, pathophysiology, clinical manifestations, indications, complications, diagnosis, etiology and prognosis. Keywords such as "decompressive craniectomy", "CD prevalence", "classification", "pathophysiology", "clinical manifestations", "indications", "complications", "diagnosis", "CD etiology" and "prognosis" were used, both individually and in combination, to facilitate the search for relevant information.

Not only were we restricted the search to original articles, but we also included reviews of randomised controlled trials, systematic reviews and meta-analysis approaches. This methodology allowed to obtain a broad and detailed vision of decompressive craniectomy from different perspectives and levels of evidence.

Additionally, the importance of considering publications within a recent time frame was taken into account, so studies and works published in the last 5 years were selected. Clinical case presentations were also included, which allowed the review to be enriched with practical examples and relevant clinical experiences.

The review was not limited to international sources, but also explored national journals and publications. In addition, the relevance of clinical guidelines and the database of the World Health Organization (WHO) was taken into account to ensure the inclusion of updated information supported by reference institutions in the field of health.

This updated and comprehensive literature review on decompressive craniectomy in traumatic brain injury provides a detailed overview of the advances, findings and important considerations related to this crucial surgical technique in the management of traumatic brain injuries. The results obtained in this review may be very useful for health professionals, researchers and specialists interested in improving the approach and treatment of patients with traumatic brain injury.

3. Results and Discussion

Annexes

Annex 1

Table 1. Classification of the severity of Traumatic Brain Injury, according to the Glasgow Coma Scale

TEC LEVE	MODERATE ECT	SEVERE ECT
-13-15 -Alert, aware of a brief period of unconsciousness (<1min) -Headache, dizziness, fainting, nausea -Just one episode of vomiting -Difficulty concentrating -Blurred vision	-9-12 -Coma, persistent confusion -Behavioural changes -Extreme dizziness -Focal neurological signs such as hemiparesis -Increased drowsiness -Respiratory decrease -Pupillary dilation	-8 or less -Coma -Babinski positive -Affectations in pupillary size and reactivity -Hemiparesis

Note: Classification of Traumatic Brain Injury, Glasgow scale. Source: "Risk factors associated with mortality in patients with acute traumatic brain injury." Cuban Journal of Anaesthesiology and Reanimation 19.3 (2020).

Annex 2

Table 2. Marshall classification of neurotraumatic injuries

CATEGORY	DEFINITION
I. Diffuse lesion (without visible alteration)	No intracranial abnormalities visible on computerized axial tomography.
II. Diffuse Injury II	Cisterns with midline deviation 0-5mm or dense lesions >25cm3 including bone fragments or foreign bodies.
III. Diffuse Lesion III (swelling)	Compression of tanks or absence, with deviation from the midline 0-5mm. Dense lesion of >25mm
IV. Diffuse Lesion IV to. Drained mass lesions	Midline deviation >5mm Dense lesion >25mm.
b. Undrained mass lesions.	Surgically drained lesions. Dense lesions >25mm not surgically drained.

Note: Marshall classification of neurotraumatic injuries. Excerpted from Bejarano, L, Traumatic brain injury in children: relationship between tomographic findings and prognosis, Journal of Medical-Surgical Specialties, 2008.

The TCE reports an incidence rate of 579 per 100,000 person/year, the main cause reported is traffic accidents followed by falls, it is considered the main cause of disability in people between 15 and 44 years, with a predominance of the male sex in relation to sports practice, driving means of transport associated with the use of neurostimulator substances or for work reasons. [17]

Higher incidence rates are reported in third world countries, especially in Latin America; in countries with medium and low income, in relation to poor culture and road safety education; with a high range of injuries caused by traffic accidents and violence, accompanied by deficient care in health services, all these are direct factors that influence mortality from traumatic brain injury. [11]

Ecuador is considered a third world country, on mortality, the National Institute of Statistics and Census (INEC) in 2014 reported 5768 deaths determined by severe trauma. The main causes listed are: traffic accidents, falls, drowning, submersion, self-inflicted injuries and assaults in order of frequency. [11]

Traumatic brain injury is defined as all the external forces that generate a cranial injury and its contents: brain and adjacent tissues, by impact, penetration or forces of acceleration or deceleration [15].

Currently, the clinical classification of TBI is based on the altered level of consciousness, based on the Glasgow Coma scale shown in Table 1. And the tomographic classification according to the Marshall CT scale reflected in Table 2. [19]

Mild TBI is classified as an injured, conscious patient with 13 to 15 points on the Glasgow Coma Scale. They may present with a subgaleal wound or hematoma but not a fracture of the base or cranial vault, accompanied by headache, nausea and non-persistent vomiting. Most patients who suffer mild head trauma make a good recovery**.** [19]

Patients with a Glasgow Coma Scale score between 9 and 12 points, altered consciousness, focal neurological signs, progressive headache, dizziness, respiratory decline are included in moderate TBI. In the event that there is focusing neurological symptoms, the behavior will depend on the picture or the lesions found, ranging from observation and conservative treatment to surgical intervention. [1]

Severe TBI is seen in patients who have a score of less than or equal to 8 on the Glasgow Coma Scale, decreased level of consciousness, neurological signs of focality, depressed fracture, or penetrating skull injury. [16] Initial management includes prioritizing ABCDE. First the patient is stabilized, then a brain CT scan and radiological study of the cervical spine will be performed. If there are no injuries and the patient remains in a coma, he should be admitted to intensive care with measures to reduce ICP. [5]

The clinical manifestations depend on the patient's condition and the severity of the traumatic brain injury according to the Glasgow Coma Scale having a wide range of presentations such as those cited below.

- Altered level of consciousness
- Peri- and post-traumatic amnesia
- Seizures
- Immediate seizures: Appear in the first seconds or minutes after TBI
- Early seizures: Appear between the first hour and 7 days post-trauma
- Late seizures: They appear after the first week of TBI and define the existence of post-traumatic epilepsy from the second seizure (recurrence that defines the concept of epilepsy)
- Vomit
- Headache
- Difficulty concentrating
- Cephalocaudal degradation
- Comma [4]

Intracranial hypertension is the condition derived from the elevation of the pressure of the intracranial contents that exceeds the compensation mechanisms of the organism, and whose natural evolution without adequate and rapid treatment becomes irreversible brain damage and death. It is usually manifested by headache, altered level of consciousness and focal neurological deficits. Multiple situations can trigger this syndrome, and depending on the type of cerebral edema generated will require a certain therapy. ICP monitoring generally requires invasive instrumentation, and is performed in neurocritical units. In addition, in the management of these patients it is necessary to intervene in all concomitant systemic alterations, whose correct homeostasis will positively influence the prevention of complications. [3]

Monro's law - Kellie states that the intracranial content consists of 3 elements: the parenchyma, blood and cerebrospinal fluid (CSF). The parenchyma occupies a volume of 1,100 ml, and the CSF and

blood, 150 ml each. The occurrence of intracranial hypertension is based on a continent/content problem. In adults, the skull and dura mater are rigid structures, which will prevent the necessary expansion of intracranial volumes in pathological conditions. However, in the newborn, the presence of fontanelles allows a greater distension of them. When these fontanelles are closed, the cranial cavity is formed, converted into a rigid case, which protects us from mild-moderate trauma, but which in turn becomes a prepathological factor in case of need to increase the intracranial content, as occurs in severe or severe TBI. [7]

The stepwise measures for the treatment of ICP are detailed below, in Favor of the maintenance of homeostasis, steroids, diuretics, hypertonic saline or mannitol, controlled hypothermia, hyperventilation, barbiturates and decompressive craniectomy. [14]

Decompressive craniectomy has been considered the last measure for the treatment of intracranial hypertension although in recent years its early performance has decreased the incidence of neurological sequelae; CD can be divided into two types; primary and secondary, primary CD is described as a prophylactic intervention to prevent potential damage caused by cerebral edema. Secondary CD is generally performed as second-line treatment in cases refractory to medical treatment, however, it has also been used, although to a lesser extent early in patients with sustained ICH [7].

The surgical technique of decompressive craniectomy is considered a salvage technique in which an osteotomy is performed according to the intracranial lesion to be treated, followed by durotomy, evacuation of hemorrhagic lesions and duroplasty. In centres with availability of bone bank, the bone flap is preserved as long as there is no contaminated open wound or multifragmentary fracture, which if so, requires its elimination. In centres where there is no bone bank and no resources for subsequent cranioplasties, the bone flap is marsupial zed with risk of osteolysis, infection and pain of the recipient site. To reduce the incidence of these complications, it is decided to wash the flap with hydrogen peroxide, absolute desperiostización and immersion in prontosan for 20 minutes, although the abdominal area generates less pain than the anterolateral aspect of the thigh the risk of osteolysis is higher. [14]

Cranioplasty can be early from the first postoperative month, it is performed by an autologous bone implant or by a titanium mesh implant, individualized implants with PEEK or other synthetic material. [14]

Currently, the indications for performing a decompressive craniectomy are related to neurosurgical lesions that evolve with cerebral edema, which produce an endocranial hypertension syndrome with neurological deterioration of the patient and signs of cerebral herniation. [14]

Depending on the clinic and the tomographic findings, different types of craniectomies will be performed for which their indications are specified.

Bifrontal decompressive craniectomy is indicated in:

- Bilateral diffuse axonal lesions, with absence of subarachnoid space, presence of compressed cisterns and lateral ventricles in cleft (bilateral Marshall III III, so there is no dislocation of midline structures).
- Patients with unilateral or bilateral Marshall VI with focal lesions located in one or both frontal lobes.
- ICP greater than 25 mm Hg, refractory first level interventions.
- Age under 65 years [9-24]

Bilateral fronto–temporo–parieto–occipital decompressive craniectomy shares indications of the preceding one and is added to patients with unilateral or bilateral Marshall VI with focal lesions extending beyond the frontal lobes of both hemispheres. [9]

Fronto–temporo–parieto–occipital decompressive hemicraniectomy is indicated when:

- Displacement of midline structures greater than 5 mm.
- No lesions of high or mixed density greater than 25 cm^3 (Marshall IV).

- Patients with high or mixed density lesion greater than 25 cm^3 not surgically evacuated (unilateral Marshall VI).
- ICP greater than 25 mmHg, refractory to first-line treatment
- Age less than 65 years. [9]

With regard to the patient's clinic and the application of the CD intervention, it is observed that complications manifest themselves later.

- Irreversible lesions located in the brainstem, or covering an area of both cerebral hemispheres and representing compatibility with life after clinical consideration.
- Hemodynamic instability of the patient associated with other complications of admission.
- Age over 65 years [9]

The surgical procedure presents complications that are classified in relation to the time of onset in:

Immediate and Mediate Complications

Hypovolemic Shock
It is an almost immediate and quite frequent complication, given the vascularity of the scalp, which tend to produce haemorrhages with significant blood losses that can lead to a very feared critical state in the patient and even death. [9-23]

Increased Cerebral Edema
It is considered a rare complication, the risk of which should not be ruled out in the early postoperative period of CD. It can be found in hyperaemic patients, as there is an increase in cerebral blood flow so it increases the probability of generating cerebral edema. This pathophysiological problem will be corrected with a therapy based on moderate or optimized hyperventilation depending on the results of the calculation of the arterio-jugular difference of oxygen or jugular oxygen saturation. On the other hand, we also consider in this classification the phenomena of necrosis by localized reperfusion and complicated hernias due to excessive or insufficient size of the flap. [3]

Increase in ICP
The sudden decrease in ICP occurs in the first hours after surgical procedure with the CD technique. It is observed that in the range of the first 24 hours there are peaks of intracranial hypertension generally related to changes in brain blood flow and increased cerebral perfusion pressure (CPP) that are usually self-limiting. In these circumstances it is counterproductive to use adjusted capelins for the immediate postoperative period, since they have a direct impact by eliminating the physiological effect expected with CD

It is important to know that the high figures of the ICP are sustained for more than 24 hours, suspicions of errors when calibrating the system, insufficient diameter of the DC, elevation of the size of the contusions, appearance of other intraencephalic lesions, acute hydrocephalus should be raised. [3]

CSF Fistulas and Subdural Hygroma
They manifest themselves within the complications of CSF dynamics, are relatively frequent, appear on the same side in which the CD has been done. They usually resolve spontaneously, and intervention for their resolution is rare, which is done by means of lumbar punctures, individual or serial, lumboperitoneal or ventriculoperitoneal shunts and drains. Leakage of external cerebrospinal fluid poses an additional risk of surgical flap infection. The initial treatment consists of postural therapy, a compression for 48 to 72 hours covering the wound, a situation that can develop complications of burn or necrosis of the flap. [3]

Late Complications

Skin flap infections and more rarely intracranial infections
They are preventable complications through the use of prophylactic antibiotic procedures in all cases of admission, due to surgical time and secondary injuries associated with trauma. In addition, cranialization of the frontal sinus and ostium isolation are also used as fundamental maneuvers for the prevention of infections. Once the diagnosis is established, the dosage of antibiotics should be

considered in relation to the antibiogram. These complications are also accompanied by dehiscence's without infection, which appear mainly in older, female patients, with malnutrition, endocrine pathology, wounds with lacerated edges or abrasions. [3-22]

Hydrocephalus
It is considered one of the most frequent complications after performing a CD. This procedure is an identifiable risk factor for post-trauma hydrocephalus. Although the cause is not clear, it has been proposed that an increase in CSF in the subdural or subgaleal space manifests alterations in its circulation, producing hydrocephalus. [3]

Refining Syndrome
It is a condition consisting in a clinic with headaches, paresis and paralysis, vertigo, behavioural alterations, vision problems, seizures, which tends to appear in patients undergoing a CD. [3]

Bone Flap Resorption
It is one of the characteristic complications of this type of surgical intervention, with an incidence of 10% in patients once the flap has been repositioned. Situation for which, the use of a synthetic flap has been recommended. Some authors suggest that the complication is more frequent when the flap was stored in the abdomen or irradiated before its repositioning. [3]

Decompressive craniectomy is characterized by being a complex high-risk procedure, which although it offers positive results in severe cases on the patient's survival, does not reduce the risk of sequelae and disability. Its implications and its execution place it as a second-level measure. According to the evidence, it is applicable even after an increase in sequelae in patients, in terms of disabilities to vegetative states, results that should be taken into account when performing a holistic analysis. [12-19]

The death during the first seven postoperative days of these patients will depend fundamentally on the initial neurological situation, so that the early action in the indication of CD before it deteriorates can play an important role. These results are plausible with greater aggressiveness in treatment in the most severe cases [20-21]. On the other hand, we consider that they reflect the broad spectrum of variability of acute brain damage, a fact that hinders the development of studies and the generalization of the conclusions obtained. The trials carried out to date have included a small number of patients, which requires new lines of research that allow a better selection of patients for decompressive craniectomy in the different pathologies and identify useful information in clinical decision making [3].

The correct mechanics of neurotrauma is aimed at extending the decompression effect, dispensing with surgical complications, and thus facilitating its eventual resolution. The application of inappropriate decompression techniques can represent death for the patient. [10]

TBI is a public health problem, since it often represents disability in people who suffer from it due to the high morbidity and mortality that concerns it, due to the injuries it generates. Decompressive craniectomy is a practical neurosurgical strategy that allows reducing ICP when first-line measures to reduce it have failed, or when it is performed early for the evacuation of lesions in order to reduce cerebral edema. [8]

In the RESCUE ICP and DECRA studies, it was found that CD was associated with high rates of vegetative states and high disability, which again underlines the importance of good patient selection. The work presents a detailed review of the indications and complications to be taken into account when selecting a patient for this surgical procedure. [10-25]

4. Conclusion

Decompressive craniectomy is a vital intervention within the set of therapeutic measures used in the treatment of severe traumatic brain injury. Understanding the type of decompressive craniectomy performed and knowing the normal findings after this surgery are crucial aspects for proper patient management. Likewise, recognizing the main postoperative complications and their sequential presentation is essential to provide optimal care and improve clinical outcomes.

The indications of the different types of decompressive craniotomy and the possible complications that may arise should be carefully evaluated before proceeding with surgery. Proper patient selection and individualized assessment are essential to optimize results and minimize associated risks.

It has been observed that the age of the patient and the initial neurological status, measured through scores on the Glasgow Coma Scale, are determining factors in survival and in the degree of long-term sequelae. In general, younger age and better baseline neurological status have been associated with a higher chance of survival and more favourable functional recovery.

However, despite the benefits in terms of reduced mortality and increased survival, decompressive craniectomy is not without its challenges. High rates of vegetative state and significant functional neurological sequelae have been identified as complications associated with this intervention. These sequelae can have a significant impact on the patient's quality of life and require a multidisciplinary approach to rehabilitation and long-term care.

Importantly, mortality after decompressive craniectomy tends to be concentrated mainly in the first few days after surgery. Therefore, intensive care and close monitoring during this critical period are essential to prevent and address potential early complications.

In conclusion, decompressive craniectomy plays a crucial role in the treatment of severe traumatic brain injury. It is necessary to be familiar with the different types of procedures, understand the normal post-surgery findings, and recognize the main postoperative complications. Careful patient selection, consideration of age and baseline neurological status, and intensive care during the postoperative period are key to improving outcomes and reducing the important functional neurological sequelae associated with this intervention. Comprehensive and collaborative management between the medical team, therapists and caregivers is essential to optimize the prognosis and quality of life of patients undergoing decompressive craniectomy.

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