Review Article
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Severe head injury and its therapeutic approach Bibliographic review

Abstract

Severe head trauma refers to an injury to the cranial,encephalic and/or meningeal structures resulting in a Glasgow Coma Scale score of 8 points or less. According to the World Health Organization, this condition causes over 5 million deaths per year. In Ecuador, in 2015, the National Institute of Statistics and Census recorded 5,768 deaths caused by this condition. The management of this condition can be either clinical or surgical depending on the patient's needs. Currently, there is controversy surrounding the therapeutic methods used to manage it, and it has been the subject of study for several years. This research presents a bibliographic review with a descriptive approach, providing useful concepts in this area of constant evolution, addressing fundamental topics such as its causes, classifications, and therapeutics. The documentation used emphasizes the use of standardized strategies and staggered management of intensive treatment for neurocritical patients, emphasizing the active implementation depending on the clinical and imaging findings. This approach has been shown to reduce mortality and improve the clinical outcome of patients.

Keywords: trauma, hypertension, craniectomy, neuromonitoring, solutions, glasgow

Volume 12 Issue 1 - 2023

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Received: November 09, 2022 | **Published:** February 07, 2023

Introduction

Traumatic brain injury represents one of the leading causes of death worldwide. According to the World Health Organization, it causes more than 5 million deaths per year worldwide. This condition can occur due to multiple mechanisms, being traffic accidents the main cause of injury, accounting for up to 70% of reported cases, followed by violent events and/or falls from their own height.[1] According to reports from the National Institute of Statistics and Census (INEC), in Ecuador, in 2015, 106,751 hospital admissions were reported due to trauma, of which 5,768 deaths were secondary to severe head trauma.[2]

Over time, a better understanding of the pathophysiology of traumatic brain injury has been achieved. Multiple clinical studies have shown that neurological damage does not stop with the impact, but it can evolve in the hours or days following the trauma, causing secondary brain damage and increasing morbidity and mortality in these patients.[1]

Severe traumatic brain injury represents 10% of hospital admissions for head trauma and is considered a continuous challenge for public health due to the high hospital costs associated with its management, which requires multifactorial medical attention. The outcome of these patients varies according to the correct management provided in hospital settings. A study conducted by the International Mission for Prognosis and Clinical Trial Design (IMPACT), which included 9,205 patients, of which 82% were severe traumatic brain injury patients, revealed that secondary mortality has been decreasing over the years due to the actions taken by healthcare personnel, significantly influencing patient survival. This has led various authors to expand their knowledge of the management of this condition and validate the use of procedures in pre-hospital settings that improve the patient's prognosis. Optimal management is carried out according to the severity of the injury, with pre-established criteria by the Brain Trauma Foundation and various Neurosurgery societies, although some of them, due to the lack of evidence or the absence of trained capacity, have fallen into disuse.[2]

This pathology represents a problem for public health due to its high morbidity and mortality rate and functional limitations in its survivors. The burden of these health problems is considered greater in low- and middle-income countries, where information available on the problem is scarce, due to the absence of organized clinical information records. This study presents an update on knowledge about the diagnosis and treatment of this condition, which should be schematic, early, and effective by healthcare personnel, in order to prevent, in relevant cases, injuries, sequelae, and death in patients who suffer from it, generating evidence-based recommendations within a patient safety framework.

Methodology

The research project carried out corresponds to a bibliographical review that uses indexed scientific information as primary sources and critiques from databases such as PUBMED, SCIELO, and GOOGLE SCHOLAR. Keywords were used in the search engines of the aforementioned databases, such as "Severe traumatic brain injury," "Intracranial hypertension," "Decompressive craniectomy," "Neuromonitoring," "Osmolar solutions," "Glasgow Coma Scale," "Mechanical ventilation," and "Glasgow Outcome Score," translated into English for a greater reach of reliable information. The title, author, abstract, and results of previous systematic reviews, cross-sectional studies, and case analyses were analyzed to determine the benefits and effectiveness of therapeutic methods used in the management of traumatic brain injury based on evidence, as well as patient outcomes.

Epidemiology

Severe traumatic brain injury accounts for 10% of hospital admissions secondary to traumatic brain injuries (Charry, Caceres, Salazar, Lopez, & Solano, 2018). In Ecuador, according to reports from the National Institute of Statistics and Census (INEC), in 2015 there were 106,751 hospital admissions due to trauma, from which 5,768 deaths were secondary to severe head trauma. In addition, a study carried out at the Eugenio Espejo Hospital in Quito, from

January 2017 to March 2018, with 410 patients within its study, established that 25.6% of patients admitted during that period had severe head trauma. The most affected age group was between 20-45 years old and there was a male/female ratio of 7:1. Additionally, traffic accidents were identified as the main cause of this pathology, accounting for 37.8% of the cases reported.[2]

Pathophysiology

In traumatic brain injury, the primary damage refers to the injury caused by the traumatic event itself. The secondary damage is triggered as a pathophysiological mechanism by the metabolic, hemodynamic, and electrolytic alteration that increases the neurological injury after the trauma, being this the main event that needs to be prevented. On the other hand, tertiary injury corresponds to complex neurochemical and pathophysiological processes related to possible positive feedback between them, beginning immediately after the trauma and which can continue in the following hours and days. After the trauma, a state of anaerobiosis is established, which is characterized by an acidic environment with the release of neurotoxic substances causing neuronal death either by direct effect or by cellular apoptosis.

Classification

Traumatic brain injury, in general, has different classification systems. Among the most useful for clinical practice are the classifications according to the Glasgow Coma Scale, the mechanism of injury, anatomical, and radiological.

I. Glasgow Coma Scale: Patients with a score of 8 or less points (Table 1).

II. Mechanism of injury: Impact-acceleration, impact-deceleration, acceleration-deceleration, shearing, penetrating, rotation.

III. Anatomical: Epicranial, cranial and intracranial lesions.

IV. Radiological: Focal and diffuse lesions.

Diagnosis

The diagnosis of this pathology is made through a detailed history of the trauma, clinical presentation, and complementary exams.

I. History: Detailed anamnesis, recognition of the mechanism of injury that caused the injury.

II. Clinical: Glasgow Coma Scale score of 8 or less, as well as the presence of post-traumatic seizures, post-traumatic amnesia, neurological focal signs, or signs of skull fractures, among others.

III. Radiological: CT is the Gold Standard for diagnose this pathology, and its severity is established according to the Marshall classification (Table 2).[3]

Table 1 Glassglow Coma Scale[1]

Glassgow Scale.		
Eye response or eyelid opening	**verbal response**	**motor response**
Without eye opening (1)	No verbal response (1)	No motor response (1)
To the Painful stimulus (2)	Incomprehensible or guttural sounds (2)	Abnormal response in extension or decerebration (2)
To Auditory stimulus (3)	Words out of context (inappropriate responses) (3)	Abnormal response in flexion or decortication (3)
spontaneous (4)	Disorientation in any of the 3 spheres (confused) (4)	Withdraws before nociceptive or painful stimuli (4)
	Oriented in 3 spheres (5)	Locate nociceptive or painful stimuli (5)
		Obeys orders or makes spontaneous movements (6)

Table 2 Tomographic classification of Marshall or the Traumatic Coma Data Bank[1]

Marshall classification.					
Category	**Definition**	**Cisterns**	**DLM**	**Mass Type Lesion**	**Mortality**
Diffuse Lesion I	Invisible intracranial pathology	normal	None	None	9.6%
Diffuse Lesion II	Cisterns present with a deviation from the midline of 0-5 mm and/or: presence of dense lesions of high or mixed density no larger than 25 cc. It may include bone fragments or foreign bodies.	presents	0-5mm	None > 25cc	13.5%
Diffuse Lesion III (Edema)	Compressed or absent cisterns with midline deviation between 0-5 mm; There are no high or mixed density lesions larger than 25 cc.	Compressed or Absent	0-5mm	None > 25cc	3. 4%
Diffuse IV Lesion	Midline deviation > 5 mm. High or mixed density lesions no larger than 25 cc.	Compressed or Absent	> 5mm	None > 25cc	56.2%
Evacuated Mass	Any surgically evacuated lesion.			None > 25cc	38.8%
Non-Evacuated Mass Type Injury	High or mixed density lesion greater than 25 cc not evacuated.			Mass > 25cc	52.8%
***MDL: Deviation from the midline.**					

Therapeutic approach

The main objective of healthcare personnel is to prevent secondary brain injury in a brain that has suffered a traumatic event. It can be said that the basic principle of treatment for severe head trauma is that if the injured neural tissue receives optimal conditions for recovery, it can regain normal function. The hospital management of these patients is schematic and multifactorial, managed by specialized personnel in intensive care units, where good neuromonitoring can be carried out. Depending on the severity and clinical and radiological findings, the initial approach may be clinical or urgent surgical. In 100% of cases, both will be complementary, and the application of one does not suggest a detriment to the other, considering that in most cases they are steps in the same treatment process.[4]

Elevation of the headboard

Raising the head of the bed in neurocritical patients facilitates venous drainage and cerebrospinal fluid drainage, avoiding depletion of cerebral compliance and decreasing intracranial pressure levels by up to 50% as long as the patient remains euvolemic. The head of the bed should be positioned at 20-30 degrees, provided that possible spinal injuries have been ruled out.[5]

Citation: Freile EIR, Fariño DFR. Severe head injury and its therapeutic approach Bibliographic review. *MOJ Public Health.* 2023;12(1):24–28. DOI: 10.15406/mojph.2023.12.00405

Intracranial pressure monitoring

In patients with severe head trauma, increased intracranial pressure (ICP) is a significant cause of secondary brain injury and worse patient outcome. Normal ICP values for adults range from 7 to 15 mmHg and can vary with body posture, age, or clinical condition. In the fourth edition of the Brain Trauma Foundation guidelines, ICP monitoring is indicated in patients with severe traumatic brain injury (TBI) because evidence suggests that, guided by ICP, treatment can reduce early mortality. Patients must meet the following criteria: 1. Glasgow Coma Scale (GCS) score between 3-8 points after resuscitation, abnormal head CT scan according to the Marshall classification (Level II), and 2. severe TBI with a normal CT scan if two or more of the following aspects are observed at the time of admission: age over 40 years, unilateral or bilateral motor compromise, and systolic blood pressure (SBP) <90mmHg Level III.[6] An ICP threshold of 20mmHg is expected, although the latest guidelines suggest values of 22mmHg. ICP monitoring is contraindicated in awake patients or those with associated coagulopathies.[7]

However, despite the widespread use of ICP monitoring in the management of severe TBI worldwide, the multicenter BEST-TRIP study, conducted in Bolivia and Ecuador, which had two groups, one based on ICP monitoring (pressure monitoring group), and another group in which treatment was based on imaging findings, showed no significant difference in the outcome of these patients. However, it was evidenced that patients who had ICP monitoring had a shorter hospital stay.[8] Likewise, a study by Queasada and colleagues, which is part of the Guidelines for the Management of Severe Traumatic Brain Injury: 2020 Update of the Decompressive Craniectomy Recommendations, determined that there were no differences in mortality between patients managed with continuous monitoring and those managed clinically and with imaging. However, there was a reduction in the number of invasive procedures performed. Despite the lack of evidence, the study recommends continuous ICP monitoring. According to the Brain Trauma Foundation guidelines, there is insufficient evidence to support the use of continuous monitoring (Level I or Level IIA).[9]

Hyperosmolar therapy

Saline solution and mannitol are the agents used to reduce ICP. The greater efficacy of a particular agent is still under study, however, mannitol is considered the reference method at doses of 0.25-1g/kg every 4 hours.[4] On the other hand, the use of hypertonic saline (HTS) for the management of increased ICP secondary to cranial trauma has gained popularity due to the complications produced by the use of mannitol, mainly acute renal failure and rebound ICP.[10]

Dosage has been effective with 3% HTS at a rate of 0.1-2ml/kg/L, with a target between 145-155mEq/l of Na. In 2011,[11] conducted a meta-analysis of all randomized trials comparing the use of mannitol vs HTS, which found 5 trials with 112 patients, and indicated that HTS was 1.16 times more effective than mannitol in reducing ICP; both results were statistically significant, concluding that HTS is more effective, however, this meta-analysis was limited by the small number and size of eligible trials.[12]

A meta-analysis conducted by Cochrane in 2019, which included 6 studies and 287 participants who met the inclusion criteria; 91% of them had a diagnosis of severe TBI; regarding patient outcomes, it showed that those managed with mannitol showed an improvement in their outcome by up to 47%; regarding mortality, in the same study Jagannatha and collaborators in 2016,[13] that 16.6% of patients managed with HTS died in the first six days of treatment, while only 5% of patients managed with mannitol died in the same period of time; however, due to the small sample size, it was not statistically significant. The authors concluded that, due to the lack of significant studies, the efficacy between these two osmolar agents cannot be demonstrated.

The Brain Trauma Foundation in its latest guidelines establishes that the efficacy of hyperosmolar therapy in reducing ICP after severe TBI has been demonstrated, however, there is not enough evidence to support the clinical outcome in relation to a specific agent. In the previous guideline of the Brain Trauma Foundation, with a level of evidence II and III, an increase in the incidence of HTS use for trauma management and its good outcome was established; however, due to the lack of studies, a formal recommendation cannot be made.[9]

Cerebrospinal fluid drainage

The latest guidelines from the Brain Trauma Foundation establish a lack of Level I or Level II evidence for the use of this technique, which is why it is not recommended for management.[9]

Mechanical ventilation and hyperventilation

In these patients, a tidal volume of 6-8ml/kg of ideal body weight should be used, while maintaining a PaO2 >60mmHg and a PaCO2 between 30-35mmHg for adequate ventilation management. Respiratory rate should be adjusted according to the patient's needs. One therapeutic strategy for decreasing ICP is hyperventilation. In cases where there is evidence of clinical or tomographic signs of intracranial hypertension, hyperventilation may be used for a brief period, with continuous neuromonitoring to assess adequate hemodynamics. A systematic review by Zhang et al.[14] six studies which found that both hypocapnia and hypercapnia following traumatic brain injury were associated with poor patient outcomes. Two PaCO2 levels (25 vs 35mmHg) were compared, but no significant differences in outcomes were observed.

Therapeutic hypothermia

Therapeutic hypothermia (HT) is the lowering of body temperature to values between 32-34°C. For a long time, prophylactic hypothermia has been a topic of interest in reducing tissue damage associated with traumatic brain injury (TBI), however, its benefits cannot be presumed. A meta-analysis including 23 studies conducted between 1993 and 2018 comparing TBI patients treated with TH reported that TH increases the mortality rate compared to those who did not receive this treatment. Furthermore, it was evident in a subgroup of patients with early HT (<24 hours), the increase in mortality was higher compared to those with HT after 24 hours of hospitalization.[10]

According to the guidelines Brain Trauma Foundation, there is not enough Level I or IIA evidence to support the use of this therapeutic technique.[9]

Barbiturates

Barbiturates have been used to control intracranial pressure ICP due to their effect by preventing unnecessary movements by altering vascular tone and decreasing cerebral metabolism, better coupling regional perfusion to tissue metabolic demands and inhibiting the formation of radicals from oxidative stress.[9]

A study conducted by Pérez Bárcena et al.,[15] which compared the effects of pentobarbital and thiopental to decrease ICP, concluded that thiopental is more effective in reducing refractory intracranial hypertension; however, caution is advised in its management due to its broad side effects, mainly hypotension, which occurs in 1 out of every 4 patients treated with barbiturates.[16]

Citation: Freile EIR, Fariño DFR. Severe head injury and its therapeutic approach Bibliographic review. *MOJ Public Health.* 2023;12(1):24–28. DOI: 10.15406/mojph.2023.12.00405

A recent retrospective study analyzed the use of barbiturates in patients with TBI, showing that the use of these at high doses decreases ICP up to 6%, but mostly causes hemodynamic instability. Cochrane conducted a systematic review of RCTs studying barbiturates as part of the treatment of severe TBI, which concluded in its last update in 2012 that there is not enough evidence to prove the usefulness of barbiturates in severe TBI. The Brain Trauma Foundation, in 2016, does not recommend barbiturate therapy as prophylaxis against intracranial hypertension (Level I and IIA), however, when dealing with a refractory hypertension condition, the use of barbiturates is justified, as long as hemodynamic stability is maintained.[9]

Guidelines recommend the use of pentobarbital in boluses of 10mg/kg over 30 minutes, followed by an infusion of 5mg/kg/h for 3 hours, then a maintenance dose of 1mg/kg/h.[6]

Surgical management

Decompressive Cracientomy

Decompressive Craniectomy (DC) consists of removing a substantial portion of the skull and dura mater to increase the volume of the cranial cavity and thereby decrease intracranial pressure (ICP).[17]

There are two types of DC based on the timing of the procedure: primary, performed during the evacuation of a hematoma, and secondary, when conservative measures to control ICP have failed, which can be bifrontal, unilateral, or bilateral.[18]

Despite its widespread use in severe traumatic brain injury (TBI), it is a therapeutic measure that generates great controversy. There are surgical indications for performing this procedure. The DECRA study evaluated the efficacy of DC compared to optimized medical therapy; This research showed that DC controlled and reduced the days of mechanical ventilation and ICU stay, but did not improve patient outcomes.[19]

It is important to note that its sample size is insufficient, and another consideration is the definition of hypertension in the study (ICP >20mmHg for >15min), as most neurosurgeons do not consider performing a DC in such a situation. On the other hand, with level I evidence, the RESCUE-icp study recruited 407 patients with TBI and refractory raised ICP (>25mmHg) despite medical treatment, compared two groups, one managed surgically and the other medically (treated with barbiturates). The Glasgow Outcome Score (GOSE) was applied at 6 and 12 months to patients in the surgical and medical treatment groups. At 6 months, mortality was 26.9% and 48.9%, respectively, being higher in the medically managed group, and the GOSE scale was higher in patients who underwent DC. The outcome at 12 months showed a mortality of 30.4% (194 patients) in the DC group compared to 52% (179 patients) in the medical treatment group. The GOSE was also superior in the group that underwent DC. The authors concluded that patients who underwent DC had lower mortality and better outcomes. [20] (Table 3)

Table 3 Pathologies that require a surgical approach[9]

Surgical Indications	
Epidural Hematoma	· Lesion >30cm3 regardless of ECG. · Displacement >5mm midline. · Thickness >15mm.
Subdural Hematoma	· Lesion >10mm regardless of ECG. · Displacement >5mm midline. · Thickness >10mm.
Intraparenchymal Hematoma	· Lesion >50cm3 regardless of ECG. · Patients with a mass lesion in the parenchyma and signs of progressive neurological deterioration attributable to the lesion. · Refractory intracranial hypertension attributable to mass effect. · Frontal or temporal hematomas >20cm3 with ECG 6-8. · Displacement >5mm midline.

Conclusion

Severe traumatic brain injury is an injury to cranial, encephalic and/or meningeal structures that leads to a degradation of the state of consciousness with a Glasgow Coma Scale score of less than 8 points, in addition to the presence of neurological deficit, associated intracranial injuries and post-traumatic seizures. Its diagnosis is based on the pillars of trauma history and kinematics, clinical evaluation, and complementary laboratory, imaging, and special examinations, with CT being the main diagnostic imaging method. The therapeutic management of severe traumatic brain injury depends on the clinical status and radiological findings of the patient and can be clinical or surgical, always adapted to the patient's needs. In 100% of cases, both approaches will be complementary, and the application of one does not suggest a detriment to the other, considering that in most cases, they are steps of the same treatment process. The active, stepped and complementary application of evidence-based neurointensive clinical and surgical therapeutic measures in patients with severe traumatic brain injury has reduced their mortality.

Acknowledgments

None.

Conflicts of interest

The authors declare that there is no conflict of interest.

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Citation: Freile EIR, Fariño DFR. Severe head injury and its therapeutic approach Bibliographic review. *MOJ Public Health.* 2023;12(1):24–28. DOI: 10.15406/mojph.2023.12.00405