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# Traumatic brain contusions. Key things to know in the emergency room

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## ABSTRACT

Traumatic brain injury is one of the most important causes of morbidity and mortality worldwide. One of its presentations includes traumatic brain contusions. Brain contusions are cortical lesions of necrotic tissue and multiple petechial haemorrhages, initially perivascular, that predominantly affect the crests of the convolutions, but that can extend through the cortex and reach the subcortical white matter. These result from a collision of the brain with the adjacent inner table of the skull, without proper treatment these can evolve and cause a rapid deterioration of clinical and neurological status. The classification includes mild, moderate, and severe TBI depending on the nature of the injury and the impact on the patient's clinical status. For the initial evaluation, computed tomography is usually used, although the magnetic resonance has greater sensitivity for the detection in these patients. Treatment is guided at preventing the progression of the lesion or the development of a secondary lesion, avoiding hypotension and hypoxia and maintaining adequate cerebral perfusion. Surgery is frequently needed in severe cases to remove intracranial hematomas, provide space for the brain to swell, or place monitors to track intracranial pressure and brain oxygenation. The aim of this article is to review the most important aspects to know about brain contusion.

## INTRODUCTION

Traumatic brain injury (TBI) is a complex disease that should be understood as a public issue due to the high rates of mortality and

## Keywords

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disability attributed to it; being part of the leading causes of death in developed countries [1]. This condition encompasses an interesting spectrum that should not only consider its immediate consequences, rather it is important to assess the long-term impact also, especially those of working age [2].

The classification of the severity of the TBI is based on the clinical status, using the Glasgow Coma Scale (GCS) [3,4], knowing that a patient with mild TBI is (GCS 13-15), moderate (GCS 9-12) or severe (GCS  $\leq$ 8) [4]. Also, according to the radiological characteristics of the lesion in the computed tomography (CT) or magnetic resonance (MR), they can be classified into brain focal injuries or diffuse injury (affecting multiple brain regions); Different than diffuse injuries, the focal lesions are localized brain damage that may occur with the presence or absence of cranial opening and also present changes both intra and extra-axial [1,5]. Brain contusions are part of the focal injuries [5].

Brain contusions are one of the most common findings within the initial studies in patients with TBI, being present in up to 31% of the CT [1,6]. Contusions are found in 89% of the brains examined postmortem in patients with TBI [1]. These results from direct loading and often occurs in the absence of a generalized injury [1,7]. The most frequent locations of brain contusions are the brain regions where it collides with the irregular adjacent inner table of the skull [5], such as frontal and temporal poles, the orbitofrontal spin, the perisylvian cortex and temporal lobe cortex inferolaterally [1,8]. This injuries typically encountered with an hemorrhagic component eventually with the bleeding within a contusion which triggers local edema and ischemic changes, which leads to tissue destruction, neuronal necrosis, and ultimately cavitation and reactive gliosis around [3].

#### EPIDEMIOLOGY

Before discuss about specific numbers that allow us to discern the picture of this disease, we must remember that TBI is strongly related with traffic accidents and falls [4]; these are the most common causes of TBI of any severity which together account for over 50% of all cases [9,10]. Knowing this we could focus on prevention programs that reduce the incidence, mortality and cost. In the international frame, it has significant socio-economic implications,

in the United States (US) over 1.7 million people suffer from TBI each year [1,2,4,5]. The incidence rate of hospitalizations due to TBI has been estimated in approximately 262 cases per 100,000 people derived from a meta-analysis of 16 European countries [7,9].

For contused brain injuries associated with sports, we have an incidence rate on emergency room visits in the US of 152 cases per 100,000 people, of whom at least two-thirds where children and adults under 19 years old [9,11]. Another visibly affected population are military, according to the Brain Injury Center, US defense and veterans, more than 22,000 service members had TBI in 2015 [9]. It is widely described in the literature that actually statistics are underestimated and it is believed that if population continue along this way in the future the numbers will be markedly higher than those of today.

#### PATHOPHYSIOLOGY

Because of the complexity of this disease it can be seen that has a very diverse pathophysiology including from biomechanical forces to very specific cellular changes. However, although it has been heavily studied, main mechanism that underlies the brain contusion is not yet fully clarified [1]. When the individual receives an injury, energy transfer beyond the capacity of the brain tissue to absorb it (tensegrity) without develop consequences on the function, thereby an insult or traumatic injury is generated [12]. This insult commonly takes a short time and is known as a dynamic load including loads by direct impact and impulse without physical contact. It is essential to know the speed and the time elapsed for this insult given that these variables play an important role. It is known that an insult at high speed tends to cause more severe damage [1,7]. Accordingly, traumatic insult triggers a series of consequences directly related to the cerebral inflammatory response to, forming a contusion zone leading to brain suffering and death of the tissue [1].

Traumatic brain inflammation can be summarized in three related but different phases [3,13]. Firstly, lipids and proteins of the cell membrane are degraded into smaller molecules which prompt an osmotic gradient. In the central area of the contusion, cells as neurons and glia suffer disintegrative and homogenization processes. All this results in a disbalance in osmolality passing from normally 310 milliosmoles to 370-390 milliosmoles in

the contusion area [13]. Towards 24-48 hours after the insult the second phase predominates, which is mediated by the activation of thrombin as a result of the coagulation cascade [1]. Thrombin among other inflammatory mediators act accordingly to stop the cerebral blood flow, resulting in vasogenic edema. The start of the third phase is triggered by lysis of erythrocytes in the intracerebral clot, subsequently hemoglobin breakdown leads to the activation of the complement system, and cytokines and reactive oxygen species contribute to the inflammatory process [1,12,13].

This inflammatory process also has a vascular component that causes a hypoperfusion in the tissue. Mass effect in contusion processes results in decreased cerebral blood flow by various causes. The occurrence of increased intracranial pressure due to cerebral vascular failure self-regulatory mechanisms leads to a marked decrease of the perfusion pressure and the develop of secondary injuries [3,13].

#### NEUROIMAGING

Usually, for the initial evaluation of a patient with TBI, the imaging modality of choice is usually the CT, due to its availability, cost and sensitivity for acute intracranial hemorrhage and fractures [14]. However, in recent decades there has been increasing use of MR, because this offers improved soft tissue with increased sensitivity for the detection of a wide variety of traumatic pathologies such as diffuse axonal injury and brain contusions. This increased sensitivity for TBI is particularly useful in the context of a traumatic brain contusion where initial CT image may be negative [15]. However, images with CT tracking may become apparent, because these tend to enlarge and become more visible [16].

Contusions appear as mixed density lesions on brain CT, often surrounded by areas wedge-shaped, hypodense and are in close contact with the inner surface of the skull, this lack of homogeneity often confer an appearance of "salt and pepper" [1,17]. The MR is more sensitive (93% -98%) than the CT (18% -56%) in the location of hemorrhagic and non-hemorrhagic contusions, which are more explicitly distinguished in T2 weightings, therefore, MR better detects all types and stages of intracranial hemorrhage, including contusions [15,17]. The recovery sequence by fluid attenuated inversion

(FLAIR) is superior to T1-weighted sequences and T2 weighted for detecting cerebral edema in the context of a CC [17]. Yuh *et al.* in RACK / TBI study (Clinical Research and Knowledge Transformation Traumatic Brain Injury) observed that 28% of patients with normal CT, MR showed abnormalities [18]. However, for primary evaluation of acute trauma not using MRI unless the patient continues with neurological signs and symptoms and CT proven no initial or follow-up is required no injury apparent recommended [1].

#### TREATMENT

Brain contusions become over time in space occupying lesions, as the inflammatory process and the secondary edema can elevate intracranial pressure (ICP), which may lead to deterioration of the patient's condition. In this sense, the treatment is guided to prevent progression of the lesion, reduce edema, ICP control and maintain good brain perfusion [1,19]. Some guidelines have recommended maintaining SBP at  $\geq 100$  mmHg for patients 50 to 69 years of age or  $\geq 110$  mmHg for patients aged 15 to 49 or more than 70 years [20,21], start treatment for ICP values above 22mmHg [20,21] and maintain cerebral perfusion pressure (CPP) between 60 and 70 mmHg to reduce mortality and improve outcomes [20-23].

#### Oxygenation

The guidelines recommend control ventilation and PaCO<sub>2</sub> in patients with severe TBI [23], the PaCO<sub>2</sub> should be maintained between 30 and 35mmHg and the PaO<sub>2</sub> at 100%, since it optimizes the supply of O<sub>2</sub> [13]. Hypocapnia induces cerebral vasoconstriction and cerebral ischemia [24].

#### Cerebrospinal fluid drainage

A method for reducing the ICP is the drainage of cerebrospinal fluid (CSF), with external ventricular drainage [13]. Brain Trauma Foundation's (BTF) recommends the use of CSF drainage to reduce ICP in patients with GCS <6 during the first 12 hours after injury [21].

#### Hyperosmolar therapy

Osmotherapy has been used for a long time for the management of intracranial hypertension (ICH) and still remains an important element for the management of TBI [20]. Hypertonic saline (HTS) and mannitol are commonly used effectively to reduce

ICP [25], these are able to establish an osmotic gradient between the brain and the cerebral vasculature, resulting in a net loss of water in brain tissue [26–28]. The HTS is administered in concentrations of 2% to 23.4% and usually of choice for patients requiring resuscitation volume, mannitol is administered at doses of 0.25 to 1g/kg every 4 to 6 hours [26,29]. Until recently the gold standard was considered mannitol, but is now controversial superiority of one agent over another [26], however, Rockswold et al showed that hypertonic saline had a more significant effect on ICP reduction, increased cerebral perfusion pressure, cardiac output and oxygen tension in the brain tissue than mannitol [30]. Consequently, the decision to choose one drug over the other may be based on factors such as drug availability, comfort physician to administer an unknown agent and side effects [26].

#### Anticonvulsant therapy

Patients with a GCS score  $\leq 10$ , age  $< 65$  years, chronic alcoholism and cortical contusion increased risk of seizures, which can aggravate neurological deficit, therefore these patients should be considered anticonvulsant drugs [1]. BTF recommends the use of phenytoin for seizure prevention early posttraumatic (within 7 days after injury) when the overall benefit outweighs the complications associated with treatment, but not recommended for use late seizures posttraumatic [21].

#### Surgical management

Current indications for surgery for traumatic brain contusions include a lower score on the GCS, location of the contusion, presence of neurological deterioration, increase in the volume of the lesion, appearance of the lesion in CT (increase of the midline and/or compression of the basal cistern) and increase of the ICP [31].

#### Decompressive craniectomy

The aim of decompressive craniectomy (DC) is to suppress the relationship between pressure and volume of the closed cranial cavity, with a large bone flap is removed and the dura is expanded with the help of autologous or artificial tissue [13]. There is no evidence that DC improves the outcome compared to medical therapy [32]. The DECRA trial showed that in patients with severe TBI refractory diffuse and ICH, early bifrontotemporoparietal DC decreased ICP and

length of stay in the ICU [33]. But, at six months of follow-up, 70% of patients in the craniectomy group had an unfavorable outcome compared to 51% of patients in the standard care group. In general, this trial has shown that DC reduces mortality but at the expense of a greater number of survivors with severe neurological disability and loss of independence [34].

#### Surgical evacuation

Surgical excision is best performed by brain contusions conservatively with minimal or absent trauma to the surrounding tissue, and ideally is performed through a limited incision and placed cortical optimally [1,13], but it is reported that conservative contusectomies are better combined with a decompressive craniectomy [13]. Sinha et al. A recent study showed that contusectomy or right lobectomy is useful in severe TBI with contusions [35]. The benefit of eliminating the contusion zone, include suppression and removal of necrotic and apoptotic process caused by degradation products of blood [13].

#### OUTCOME

A recent study observed that the frontal and temporal location is associated with a better surgical result than parietal and cerebellar contusions. In addition, they reported that age  $> 30$  years, a severe GCS, and a median line displacement of more than 5 mm were the greatest predictors of mortality after surgery [36]. Laccarino et al, showed similar results to the previous study, but also observed that the clinical deterioration in the first hours after the trauma and the onset or increase of the midline change in the follow-up CT images were associated with unfavorable clinical results [37].

In this order of ideas, despite what has been described, there are many points that need to be reinforced from the volume and quality of evidence, such as epidemiology, diagnosis and specific treatment in areas where there are barriers to access high-cost technological tools, to contribute to the reduction of the burden of neurological diseases, in addition to reducing the risk of morbidity, mortality and disability [38–44]. It is necessary to carry out prospective multicenter studies that allow us to know the behavior, approach and outcomes of this disease in different contexts and to design a standardized prognostic tool that has an excellent

performance, accessible and understandable to all health professionals. In the current post-pandemic phase of COVID-19, many of the efforts in disciplines other than infectious diseases have waned, so it is necessary to resume research in other areas [45,46].

## CONCLUSIONS

Brain contusions if not treated in time can have rapid and progressive in the clinical condition of the patient and neurologic effects, not only for their local ripple effects, but also the possible injury of hippocampal and brainstem. In this regard it is important to establish a proper, immediate and effective treatment because it has been shown that this improves clinical outcomes. Information about brain contusions is scarce and concepts need to be standardized to provide proper management of patients with TBI.

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