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Improvement of Diffuse Axonal Damage and Sinking Flap Syndrome Post Craneoplasty

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1. Abstract

The trephined, craniotomized, or sunken flap syndrome encompasses neurological manifestations associated with skin flap depression and is distinguished from post-traumatic syndrome by its reversibility with repair treatment of the cranial defect. Coma is not a common form of presentation. We report a case of atypical presentation in a 35-year-old man with a history of decompressive craniectomy, who presented with profound neurological deterioration attributable to trephination syndrome, which reverted after cranioplasty. Cerebrovascular, metabolic, hydrodynamic disorders of the cerebrospinal fluid and hyper dynamism of brain structures are involved in the pathophysiology of the syndrome. The therapeutic gold standard is cranioplasty. The syndrome of the trephined, craniotomized or sunken flap syndrome is commonly referred as neurological manifestations associated to skin flap depression and reversible after craneoplasty, which allows its differentiation from post-traumatic syndrome. We present the case of a male patient, 35 years old, with history of decompressive craniectomy. He evolved with sudden neurological worsening associated to syndrome of the trephined and recovery after craneoplasty. Physiopathology of the syndrome involves cerebrovascular, metabolic, and cerebrospinal fluid hydrodynamic disturbances as well as parenchymal hyperdynamic mechanisms.

2. Introduction

Cranioencephalic Trauma (TBI), according to the National Head Injury Foundation, is defined as "damage to the brain, of a non-degenerative nature, caused by an external force, which can produce a decrease or alteration of the state of consciousness, resulting in an impaired functioning of cognitive and physical abilities". The damage occurs mainly in physical and cognitive capacities, such as attention, perception, memory, problem solving, understanding,

as well as falls, physical assaults, and traffic accidents. The latter, with the highest incidence, in 42% of the cases [1]. In Mexico, it is the third cause of death with an index of 38.8 per 100,000 inhabitants and with the highest incidence in men between 15 and 45 years old [2]. The TBI classification is carried out considering the level of consciousness measured according to the "Glasgow Coma Scale" (GCS). The GSC assesses three types of response independently: ocular, verbal and motor. A patient is in a coma when the score resulting from the sum of the different responses is less than 9 [3]. Based on this scale we differentiate:

among others. The most common causes of a TBI are accidents at work, at home, outdoors, or while playing sports.

- Mild TBI: GCS 15-14
- Moderate TBI: GCS 13-9
- Severe TBI: GCS <9
- **Mild TBI:** it is the most frequent, there is usually no loss of consciousness or if there is, its duration is usually limited to the minutes after the contusion.

- **Moderate TBI:** the period of loss of consciousness is greater than 30 minutes but does not exceed one day and the period in which the patient who suffers from it has difficulty learning new information is less than a week.
- **Severe TBI:** in this type of trauma, the period of loss of consciousness is greater than one day and / or the period in which the patient who suffers it has difficulty learning new information is greater than one week [4]. TBI can be divided into penetrating and non-penetrating injuries, the latter in turn being subdivided into focal or diffuse injuries (Figure 1) [5].

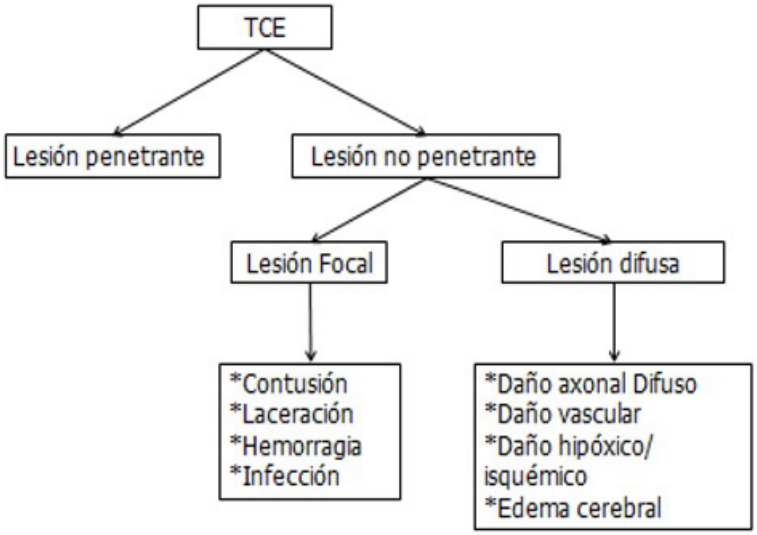

Figure 1

- **Focal Injury Includes:** contusions, which usually affect the cerebral cortex and are caused by a direct mechanism against the impact area, or by an indirect one, which by backlash affects the opposite side of the trauma, the most frequent locations are the frontal lobes and temporary. Lacerations occur when the injury is so severe that it involves the leptomeninges. Bleeding can develop after a period and spread into the subarachnoid space, forming bruises. Local infections are complications from contamination, generally bacterial [6, 7].

3. Diffuse Axonal Injury

Diffuse injury includes Diffuse Axonal Damage (DAD), diffuse vascular damage (DVD), and diffuse hypoxia / ischemia. DVD is caused by stress and traction on the capillaries of the brain parenchyma resulting in parenchymal hemorrhages. Diffuse hypoxic / ischemic damage sometimes accompanies TBI, especially in patients with intracranial hypertension [1, 4]. DAD is caused by inertial, angular, or rotational forces (acceleration-deceleration) that cause rapid movement of the skull which deforms white matter, the axonal damage pattern is described as multifocal, appears deep in the subcortical white matter and is particularly common in midline structures, including the corpus callosum [5].

The brain is relatively flexible when it is subjected to the effect of slow inertia, as it adapts to change and recovers its original physical state without being damaged; however, when this effect occurs very fast, less than 50 milliseconds, the white matter deforms considerably. Thus, factors such as the magnitude, location, duration, direction, and velocity of the force determine the severity of axonal damage [2, 5].

Axonal damage can be caused by an immediate axotomy (primary injury), which damages the structural elements of the axons, such as microtubules, and by a delayed action after minutes or hours after impact (secondary injury), in which the injury axonal is a response to the cellular cascade [5, 6].

Trauma induces focal permeability of the axolemma, which increases calcium entry, and this generates the activation of proteases, calpains and caspases, which cause the degradation of the axon cytoskeleton and its disconnection.

4. Sunken Flap Syndrome

Sunken flap syndrome corresponds to a complication of decompressive craniectomy. The pathophysiology of the syndrome is believed to be multifactorial, including hydrodynamic alterations of the cerebrospinal fluid (CSF), changes in cerebral metabolism and blood flow, pressure exerted by the atmospheric gradient and movements of the parenchyma [8]. Under normal conditions as an individual in supine position presents a CSF pressure of 15cm H2O and in an upright position a pressure of 0 at the level of the cistern and –8 cm H2O in the ventricles. This negative pressure in a craniotomized patient generates a vacuum or suction effect that is added to the positive atmospheric pressure in the opposite direction, favoring the depression of the skin flap and the underlying brain tissue (Figure 2). Likewise, this subsidence causes collapse of the subarachnoid space, contributing to the alteration of the normal dynamics of the CSF [9].

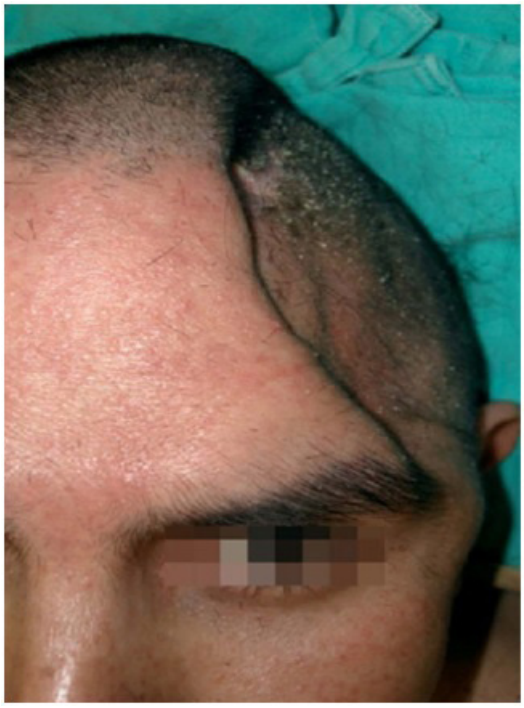

Figure 2

In craniotomized subjects, there is both a decrease in arterial flow and a decrease in venous return. The first occurs because of the increase in vascular resistance secondary to the transmission of atmospheric pressure to the cerebral blood vessels, as well as due to their distortion due to deviation of the midline structures. Venous return, for its part, is affected by focal cerebral compression at the site of subsidence. Both phenomena contribute to the decrease in plasticity of the sunken brain and to the vacuum effect on the flap. An immediate consequence of these changes corresponds to the failure of cerebral circulatory autoregulation, a mechanism by which the presentation of reperfusion injuries after cranioplasty is explained [10]. Different authors propose that the flow velocities (measured by transcranial Doppler) increase in the ipsilateral and contralateral middle cerebral and internal carotid arteries when restoring the bone defect in craniotomized patients, particularly when this gesture is performed. carried out early. Furthermore, it is observed that the flow velocities of the damaged hemispheres in craniotomized patients tend to decrease with postural changes (sitting or standing), reversing this situation with cranioplasty [11].

5. Description of The Clinical Case

35-year-old male, with no significant personal pathological history; suffered a run over accident three years ago with severe head trauma and loss of alertness. During the initial evaluation in the hospital, a CT scan is performed showing the presence of an acute subdural hematoma in the left frontotemporal region and severe cerebral edema, in addition to presenting data of intracranial hypertension, a decompressive left front temporoparietal craniectomy is performed plus drainage of subdural hematoma acute frontotemporal plus partial temporal lobectomy and loose dura mater platy with pericranium. After the surgical procedure, he showed a notable improvement in the frontal syndrome, in addition to behavioral alterations secondary to the lesion of the frontal lobes, ability to speak without alterations, quadriparesis, limitation of movements in upper and lower extremities, sensitivity to touch and symmetrical nociceptive stimuli coordination and gait absent. His discharge was decided, however at 3 months he presented a neurological deficit and the ability to speak was symbolically degraded, it was only limited to words such as "yes" and "no", to the abnormal skull physical examination, with a scar in the front temporoparietal region, neurologically conscious with post-traumatic dementia, poor language, quadriparesis due to diffuse axonal damage, position with limbs in flexion due to ankylosis and contractures, coordination, and impaired gait. As part of the treatment, a right frontotemporal cranioplasty was decided. After the procedure, he was oriented in time, place, and person, performed movements of the lower and upper extremities without pain. Due to the health contingency due to SARS COVID-19, a second cranioplasty in the left frontotemporal region is awaiting in this hospital.

6. Discussion

In Mexico, death from trauma continues to be one of the main causes of death and disability, and injuries to the nervous system and hemorrhagic shock are the main contributors. Severe head injury is a cause of death and disability in the young, injured patient. The polytrauma patient with sequelae of head trauma constitutes a complex group of patients who may present with different types of brain injury, including diffuse axonal damage. We know that most head injuries occur in previously healthy people in general terms and, unfortunately, also many patients are left with a disability or subsequent sequela, which affects their neurological and intellectual capacities or leads to economic and family imbalances sexual or emotional [12].

Despite current technological advances there is still a very high mortality According to data from the Revest Mexicana de Neuroscience “worldwide, 1.2 million people die annually from Cranioencephalic Trauma (TBI) and between 20 and 50 million suffer non-traumatic injuries. Mortal [13].

That is why the immediate initial treatment is essential within the first hour since it will modify the prognosis of the patients. It is important to identify the symptoms of intracranial hypertension so as not to delay surgical treatment, although we do not know the time that passed between the impact and medical attention, it is an ethical and moral duty of health institutions to have the personnel and technological implements to be able to apply them promptly, there was a failure in the decision-making of the definitive treatment [14,15]. The diffuse axonal damage triggered a significant cognitive and functional deterioration during the first days after the TBI, the patient showed quadriparesis as a sequela but was neurologically stable in addition to maintaining a fluent conversation, however, three months later there was a significant decrease

in speech and neurological status, evidencing, apart from diffuse axonal damage, the sunken flap syndrome, which corresponds to a complication of decompressive craniectomy, its clinical spectrum is variable, triggering vague and nonspecific manifestations up to sudden neurological deterioration in most cases , just as our patient clinically stated. Although its incidence is unknown with certainty, it acquires importance not only because it is underdiagnosed, but also because of the possibility of reversing the symptoms with correct treatment [16]. The cornerstone of the treatment is given by the surgical repair of the defect, which requires studies of greater weight to determine the appropriate surgical time. After the cranioplasty, a notable improvement was shown in the patient, his speech was a little more fluent and he can hold a conversation for more than 7 minutes, without diverting the subject, an increase in the mobility of the lower and upper extremities was also observed [13-15].

7. Conclusion

Head trauma requires aggressive emergency treatment, to prevent or minimize irreversible damage to the nervous system, for which it is necessary to adopt a determined and organized approach that ensures optimal patient care from its initial management and during the following bases of diagnosis and definitive treatment. One case is not enough to reach definitive conclusions about the best approach to a TCS. Traumatic axonal injury is undoubtedly the most frequent variety of head injuries that predominantly affect young men and is the cause of significant disability. Prevention of traumatic brain injury is the most important pillar of medical care followed by normotensive care guided by neuromonitoring [17, 18]. Every doctor must have a good knowledge of the mechanisms of head trauma that trigger alterations whose anatomical and physiological impact can be profound and potentially fatal for the patient due to the consequences and complications that this entails. It is important to emphasize that the sequelae and complications that occur after TBI will depend, to a large extent, on the good, fast, and efficient management assigned to the patient from the moment the head injury begins. Based on the foregoing, it should be concluded that patients suffering from ADD post TBI should be evaluated early to carry out the correct therapeutic measures and thus reduce neurological sequelae. In addition to taking strict care in post-operative decompressive craniectomy patients once they have been discharged from the hospital to avoid sunken flap syndrome. Also, no less important is the prevention of motor vehicle accidents, emphasizing motorcycle drivers the use of helmets, in addition to placing important emphasis on the promotion of road safety measures among the population.

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