Hydrocephalus, is it a complication or a consequence of decompressive craniectomy?

Hidrocéfalo, es una complicación o una consecuencia de la craniectomía descompresiva?

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Abstract

Decompressive craniectomy, an increasingly utilized salvage procedure, is affected by a number of complications, one of which is hydrocephalus. A thorough review of the directly and indirectly related literature was done in an attempt to elucidate the existing connections, if any, between this procedure and the complicating hydrocephalus. It became clear that a direct relationship exists between these two entities. Consequently decreasing the time in which the effects of the craniectomy interfere with the intracranial physiology, by performing an as early as possible cranioplasty, should avoid or decrease the likelihood of hydrocephalus to develop.

Key words: Hydrocephalus, Pathogenesis, Hyperosmosis, Decompression.

Resumen

La craniectomía descompresiva, un procedimiento de rescate que está siendo utilizado con frecuencia creciente, está afectada por un número de complicaciones, una de las cuales es la hidrocefalia. Aquí Se efectuó una cuidadosa revisión de la literatura relacionada directa o indirectamente con estos tópicos con el objeto de detectar posibles conexiones entre el procedimiento descompresor y la génesis de hidrocefalia. Quedó en evidencia que existe una relación directa entre ambas condiciones. Por ello, reduciendo el tiempo en el cual el proceso descompresivo interfiere con la fisiología intracraneana al efectuar una cranioplastica lo más temprana posible, debiera evitar o disminuir la posibilidad del desarrollo de hidrocefalia.

Palabras clave: Hidrocefalia, Patogenesis, Hiperosmosis, Descompresión.

Introduction

Decompressive craniectomy is currently been utilized for the management of medically refractory intracranial hypertension. Head trauma and malignant ischemic stroke are its most frequent, but not it´s only indications. With the increasing utilization of this procedure, certain complications have become evident. Among these hydrocephalus has been noted, although the frequency of its occurrence varies between the different reports because the diagnostic criteria used have not been uniform[16,23]. Moreover no consensus has been reached so far regarding the most effective modality of hydrocephalus treatment in that scenario.

Material and Methods

A thorough review of the related literature, including experimental work on the pathogenesis of hydrocephalus, was done in an attempt to detect any possible connection between the decompressive procedure and the development of hydrocephalus.

Results

Decompressive craniectomy, while been very effective in reducing intracranial pressure, in and of itself adds further difficulty when the diagnosis of hydrocephalus is arisen by the development of ventriculomegaly. That quandary cannot be resolved only by serial CT scans, because progressive

ventricular enlargement can also occur in posttraumatic brain atrophy[13]. While it has been considered by many that measuring the baseline ICP would be an important factor in deciding the need for shunt implantation[13], the coexistence of a decompressive procedure, that drastically alters the ICP, reduces the significance of that measurement. In that juncture better methods to clarify that issue would be the calculation of the pressure-volume index[19] from a lumbar computerized infusion test[2] or if a less invasive procedure would be preferable, a SPECT evaluation of temporal lobes hypoperfusion could also resolve the differential diagnosis between hydrocephalus with the possible need for shunt placement [16] and post-traumatic brain atrophy .That diagnostic difficulty seems to be the reason for the marked difference noted between various reports in reference to the frequency in which hydrocephalus develops in cases of decompressive craniectomy.

Discussion

The classical pathogenetic concept of hydrocephalus has considered it to develop either due to a blockage to the CSF circulation or to an impairment in CSF absorption. That notwithstanding, this doctrine is currently being challenged[15,18]. Furthermore, contrary to one of its basic tenets, it has been demonstrated that the ependyma is permeable to water[12].

Based on these new concepts, a novel line of investigation has proven that the intraventricular infusion of an hyperosmolar solution, in and of itself, can produce hydrocephalus by creating an osmotic gradient between the ventricular cerebrospinal fluid and the blood within the brain parenchyma[11,12,15]. That gradient could be sufficient to induce an increase in cerebrospinal fluid production, not only by choroid plexuses secretion but also through the passage of water from the brain parenchyma into the ventricles. This could occur via ion channels and aquaporin conduits, particularly aquaporin 4, which are found in the ependymal cells lining the ventricles and on the end feet of astrocytes that contact periventricular microvessels[12]. It is then reasonable to hypothesize that pathologies that cause a sustained elevation of osmotic pressure in the brain parenchyma and/or in the ventricular CSF by accumulation of macromolecules or by an impairment in macromolecular clearance, could result in hydrocephalus[8,9,14,17,22]. Various studies have demonstrated a significant increase in brain tissue osmolality and/or ventricular CSF osmolality in cases of ischemic stroke as well as in traumatic brain injury[5,9,10,14,17]. The osmotic gradient thus generated between brain parenchyma and ventricular CSF could be sufficient to result in hydrocephalus.

From a different perspective, a number of investigators working on experimental hydrocephalus and attempting to elucidate the influence of brain coverings on its development, added to their studied animals craniectomies and durotomies. While these procedures by themselves caused no significant change in the ventricular size nor in the sagittal sinus pressure[4,6,7], when those animals were submitted to a ventricular perfusion, to the determination of pressure volume index (PVI) or induced into experimental hydrocephalus, a significant reduction in the elastic properties of the brain parenchyma became evident, together with a significant increase in the ventricular distensibility, and a dramatic increase in the capacity of their ventricular system to accommodate added volume[4,6,19,20]. From all of the above, it becomes clear that a decompressive craniectomy with a duroplasty provides definite conditions that would promote the generation of hydrocephalus, without even considering potential additional factors, such as brain trauma, ischemia or other pathologies that could cause an increase in cerebral tissue or CSF osmolality and further exacerbate the proclivity for hydrocephalus.

The cerebral pathological changes induced by the hydrocephalic process, evolve at least initially, at a rapid pace and affect, in a progressive fashion the ependymal lining of the ventricles, which becomes stretched, flattened and at some locations torn, while the subependymal glial sheath gets thickened in many areas and the periventricular white matter becomes edematous and experiences axonal damage and myelin loss, leading possibly to white matter atrophy[1,3,21]. Considering that these pathological changes are, at least initially reversible, a cranioplasty done as soon as the intracranial upheaval that prompted the decompressive craniectomy has stabilized, could conceivably halt or even revert that process resolving the ventriculomegaly and improving its symptomatology either totally or partially. Moreover, with that possibility in mind, a reasonable period of time should elapse after the performance of the cranioplasty, firstly for it to be completely healed and secondly for the course of the hydrocephalus to be thoroughly evaluated so that a clear determination can be made regarding the need for shunt implantation.

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