Traumatic brain edema

Cerebral edema can result from a combination of several pathological mechanisms associated with primary and secondary injury patterns in traumatic brain injury (TBI) [1].

For many years, vasogenic edema was accepted as the prevalent edema type following traumatic brain injury TBI.

Traumatic brain edema is predominantly cytotoxic and that water entry is modulated in part by aquaporins.

Cytotoxic edema is of decisive pathophysiological importance following traumatic brain injury TBI as it develops early and persists while blood brain barrier (BBB) integrity is gradually restored. These findings suggest that cytotoxic and vasogenic brain edema are two entities which can be targeted simultaneously or according to their temporal prevalence [2].

Treatment

Currently, there are no pharmacological treatments available for traumatically induced brain edema and the subsequent rise of intracranial pressure ICP.

see decompressive craniectomy.

Diagnosis

Non-invasive diffusion-weighted MRI (DWI) quantifies the diffusion of water in the brain associated with edema and contributes essentially to the understanding of stroke and stroke-related cerebral edema formation. The use of DWI offers the opportunity to identify the predominant edema type after TBI and, in this way, to distinguish between vasogenic and cytotoxic edema.

Outcome

As pressure within the skull increases, brain tissue displacement can lead to cerebral herniation, resulting in disability or death [3] [4] [5]

The predominant cause of death and long-term disability after traumatic brain injury TBI is brain edema [6] [7]

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