

Cerebral edema: A major issue

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ABSTRACT

Cerebral edema is a fairly common pathophysiological entity that occurs in a variety of clinical conditions. Many of these conditions manifest themselves as medical or surgical emergencies. Cerebral edema is defined as an abnormal accumulation of water in the intra- and/or extracellular spaces of the brain. Though there has been significant progress in our understanding of the pathophysiological mechanisms

underlying cerebral edema, more effective treatment is still needed and is still in the works. The "ideal" agent for the treatment of cerebral edema—one that selectively mobilises and/or prevents the formation of edema fluid with a rapid onset and prolonged duration of action, as well as minimal side effects—remains to be discovered. We can probably expect newer agents that specifically act on the various chemical mediators involved in the pathogenesis of cerebral edema in the coming days.

Key Words: *Neurons; Hypoxia; Calcium; Sodium; Cells*

INTRODUCTION

The pathophysiology of cerebral edema is complex at the cellular level. Damaged cells swell, blood vessels leak, and blocked absorption pathways force fluid into brain tissues. Following the activation of an injury cascade, cellular and blood vessel damage occurs. The cascade starts with the release of glutamate into the extracellular space. Glutamate stimulation opens calcium and sodium entry channels on cell membranes. Membrane ATPase pumps exchange one calcium ion for three sodium ions [1]. Sodium accumulates within the cell, forming an osmotic gradient and increasing cell volume through water entry. The presence of more water causes dysfunction, but not necessarily permanent damage. Finally, hypoxia depletes the energy stores of the cells, disabling the sodium-potassium ATPase and reducing calcium exchange [2].

When the energy-dependent sodium pump in the cellular membrane fails, sodium accumulates intracellularly and water moves from the extracellular to intracellular space to maintain osmotic equilibrium. Calcium builds up inside the cell, triggering intracellular cytotoxic processes. The formation of immediate early genes such as *c-fos* and *c-jun*, as well as cytokines and other intermediary substances, initiates an inflammatory response. When microglial cells become activated, they release free radicals and proteases that contribute to the attack on cell membranes and capillaries. It is impossible for cells to recover once their membranes have been disrupted [3].

Cells are harmed by free radicals. The arachidonic acid cascade generates reactive oxygen species such as superoxide ion, hydrogen peroxide, and hydroxyl ion. The release of fatty acids such as arachidonic acid provides a source of potentially harmful molecules. Nitric Oxide (NO) is also a free radical source. NO is

produced by macrophages and activated microglial cells via the action of inducible or immunological NO synthetase (iNOS) [4].

When the Central Nervous System (CNS) is injured or ischaemic, mediators such as glutamate, free fatty acids, or high extracellular potassium compounds are released or activated, causing secondary swelling and nerve cell damage. Other substances such as histamine, arachidonic acid, and free radicals such as NO may also be considered mediators of brain edema, but evidence for each of these compounds is less clear than for BradyKinin (BK). A variety of mediators may enhance each other in a cascading fashion via various initiating reactions that may be pharmacologically inhibited. BK may be involved in the formation of edema following a cold lesion, concussive brain injury, traumatic spinal cord injury, or ischaemic brain injury [5].

The loss of membrane ionic pumps and cell swelling are two of the molecular cascades initiated by cerebral ischaemia in stroke. Secondary free radical and protease formation disrupts brain-cell membranes, causing irreversible damage.

To explain the effects of cerebral edema in layman's terms, use the Monro-Kellie hypothesis, which states that the total volume of three elements (inside the skull)—brain-1400 ml, Cerebral Spinal Fluid (CSF)-150 ml, and blood-150 ml—remains constant at all times. Because the skull is like a rigid box that cannot be stretched, increasing the volume of one of these components will force a reduction in the volume of the other components. As a result, if there is an excess of water, the volume of brain and blood inside the skull is compressed. Primary blood flow disturbances, on the other hand, cause brain edema. As the volume of the brain, blood, or CSF increases, the accommodative mechanisms fail, and Intracranial Pressure (ICP) rises exponentially [6]. A significant increase in ICP eventually results in a decrease in cerebral blood flow throughout the

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brain. The most severe form of widespread ischaemia results in brain death. Less severe but still extensive cerebral infarction can result from lower degrees of increased ICP and reduced blood flow. The numerical difference between elevated ICP and mean blood pressure within cerebral vessels, known as cerebral perfusion pressure, and the duration of its reduction are the primary determinants of cerebral damage. If these changes continue, they will result in brain herniation, which is a precursor to irreversible brain damage and death. It must be understood that, while elevated ICP is the result of significant cerebral edema, the two are not synonymous, as elevated ICP can be caused by other mechanisms as well [7].

TYPES

Klatzo classified cerebral edema into two types: vasogenic edema and cytotoxic edema. Cellular edema is preferable to cytotoxic edema; Fishman accepts these two categories but adds a third, which he calls interstitial cerebral edema. It is rare that distinct categories of edema can be distinguished; instead, there is frequently overlap between the various types of edema.

Vasogenic cerebral edema is defined as the entry of fluid and solutes into the brain via an ineffective Blood-Brain-Barrier (BBB). This is the most common type of brain edema and is caused by increased permeability of capillary endothelial cells in the white matter. The breakdown of the blood-brain barrier allows proteins to move from the intravascular space to the extracellular space via the capillary wall [8].

A cellular swelling is referred to as cellular (cytotoxic) cerebral edema. It can be seen in conditions such as head injury and hypoxia. It is caused by the swelling of brain cells, which is most likely caused by the release of toxic factors by neutrophils and/or bacteria. Cytotoxic edema occurs within minutes of an insult and is caused by swelling of glia, neurons, and endothelial cells. Cytotoxic edema primarily affects the grey matter.

In hydrocephalus, interstitial edema occurs when the outflow of CSF is obstructed and intraventricular pressure rises. As a result, sodium and water move across the ventricular wall into the paraventricular space. Interstitial cerebral edema caused by meningitis is primarily caused by obstruction of normal CSF pathways, resulting in increased resistance to CSF outflow [9].

CLINICAL FEATURE

It is critical to have a high level of suspicion. The characteristics of cerebral edema supplement and frequently complicate the clinical features of the primary underlying condition. Cerebral edema alone will not cause clinical neurological abnormalities until the ICP reaches a level that causes local ischaemia. Alteration in level of consciousness, appearance of bradycardia, rise in blood pressure, abnormal breathing patterns, evidence of extra ocular movement abnormalities, alteration and inequality of pupillary size, and extensor plantar response on the side of the lesion should all raise the possibility of cerebral edema in a given clinical setting [10].

Cerebral edema is the most common cause of neurological deterioration and death during an acute ischaemic stroke. It is present in all ischaemic strokes. Ischaemic brain edema is initially cytotoxic due to cell membrane disruptions. Later vasogenic edema develops as a result of BBB disruption. Cerebral edema typically develops soon after the onset of ischaemia and peaks between 24 and 96 hours. Typically, this is limited to the ischaemic region and has little effect on the adjacent brain. However, as it progresses, it compresses brain regions adjacent to the ischaemic zone, causing neurological deterioration [11].

A CT scan is a great technique for determining abnormalities in brain water content in vivo. On an unenhanced scan, the edema patches appear as low density. This is related to the dilution of all white matter elements. CT's anatomical specificity allows it to identify not only the presence, but also the kind of brain edema. This is useful in distinguishing the type of the underlying lesion, such as an infarction or a tumour. In general, more malignant primary brain tumours and metastatic tumours have a higher prevalence of cerebral edema, albeit the presence of brain edema does not rule out benign lesions. CT scans are a great way to monitor the clearance of cerebral edema after therapeutic intervention [12].

ICP monitoring is a critical technique for monitoring instances of cerebral edema, and it is performed routinely in all Neurology and Neurosurgery ICUs. Unfortunately, direct ICP measurement and forceful actions to offset high pressures have not delivered universally good effects, and the everyday use of ICP monitoring remains contentious despite two decades of popularity. Part of the issue might be related to the time of monitoring and effective patient selection for aggressive treatment of elevated ICP. The installation of a monitor is only justifiable if the ICP readings are to be utilised as a guide to medical therapy and the timing of surgical decompression.

SURGICAL TREATMENT

Large hemisphere infarcts with edema and life-threatening brain-shifts are occasionally treated surgically. A temporary ventriculostomy or craniectomy can help avoid further deterioration and potentially save your life. In the event of acute brain swelling after a cerebral infarction, decompressive craniectomy is a life-saving treatment that should be considered in younger patients with rapidly deteriorating neurological condition. Surgical decompression is also life-saving in massive cerebellar infarcts with cerebral edema. The excision of the lesions that cause cerebral edema leads in the disappearance of the edema. The VP shunt is extremely beneficial in situations of severe hydrocephalus.

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