Talk:Brain edema

An increase in the volume of brain tissue can occur due to an increase in the water content - **edema** or an increase in vascular filling - **hemodynamic swelling** (swelling).

Classification of cerebral edema:

- By range:
  - generalized;
  - Focused.
- According to pathophysiology:
  - vasogenic;
  - cytotoxic;
  - interstitial;
  - hypoosmotic;
  - hydrostatic.

Generalized cerebral edema is most often caused by hypoxia of the brain. It manifests itself clinically as a **syndrome intracranial hypertension**. Pro diagnostics, we use CT, which shows a reduction of the cerebrospinal fluid space and an erasure of the difference between gray and white matter.

Focused cerebral edema usually occurs around the focus of contusion and intracerebral hematomas. Clinically, it has focal neurological symptoms. Focal hypodensity is shown on CT.

V therapy involves antiedematous pharmacological treatment (mannitol, hypertonic NaCl solution, dexamethasone), possibly craniotomy and external decompression. Therapy focuses on addressing the cause, including addressing intracranial pressure. It is important to maintain sufficient perfusion pressure.

Causes of cerebral edema

Neurological causes:

- iCMP and intracranial hemorrhage,
- brain tumors,
- meningitis, encephalitis,
- other brain infections – TB, cysticercosis, toxoplasmosis.

Other causes:

- diabetic ketoacidosis, coma due to lactic acidosis,
- malignant hypertension, hypertensive encephalopathy,
- fulminant viral hepatitis, hepatic encephalopathy, Rey's syndrome,
- intoxication,
- hyponatremia, SIADH,
- opioid abuse,
- cerebral edema in altitude sickness.

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Types of cerebral edema

According to the pathogenesis of the disease, we distinguish several types of cerebral edema, but **the distinction is often questionable** and individual types can overlap.

Vasogenic cerebral edema

Vasogenic cerebral edema most often accompanies **tumors** and **inflammations of the brain** and is the most common type of cerebral edema. It is caused by a disorder of the blood-brain barrier (HEB). Otherwise, the tight junctions of the endothelium are loosened, allowing the transfer of proteins into the interstitium, and the fluid behind them escapes into the intercellular space. The white matter is the most damaged. It responds favorably to corticoid therapy, mainly using dexamethasone, which reduces the expression of VEGF acting proedematously by increasing the permeability of HEB. Dexamethasone is particularly advantageous due to its long half-life (dose after 4–6 hours) and minimal mineralotropic effect.

Cytotoxic cerebral edema

Cytotoxic cerebral edema arises as a result of hypoxia cells and is accompanied by a disturbance of the membrane balance. When there is a lack of oxygen, ATP runs out in a matter of seconds, the sodium pump stops and water moves **intracellularly**. Cell swelling is also potentiated by substances washed out by neutrophils or a possible bacterial infection. The most common etiological factor is trauma, but it can also occur in connection with cerebrovascular accidents. It mainly affects the gray matter of the brain.

On CT, when the whole brain is affected, the gyrification is visible and the brain is **globally hypodense**. It is treated with **mannitol** (max. 4-5 days due to the rebound phenomenon), **furosemide** or concentrated ion solutions (NaCl). As a rule, it does not respond very well to steroid treatment.

Interstitial cerebral edema


Mannitol solution 15%

Interstitial cerebral edema (*periventricular*) occurs in both obstructive and hyporesorptive hydrocephalus. It can also arise as a complication of meningitis when CSF circulation is impaired. Sodium and water pass transependymal from the ventricles into the white matter. This condition indicates **active hydrocephalus**. CT shows enlargement of the ventricular system. The arrangement of the image sometimes resembles a **smiling face**. Gradual progression can lead to a balloon-like expansion of the front corners of the lateral ventricles and III. chambers, referred to in English literature as **mickey mouse sign**.

Hypoosmotic cerebral edema


Furosemid 125mg

Hypoosmotic cerebral edema occurs in ""disorders of mineral metabolism"" (low sodium, chlorides, water poisoning, hypersecretion ADH - SIADH etc.), also in trauma**,** tumors**,** infections **or after** subarachnoid hemorrhage**. It also appears as a** postoperative complication **in procedures on the hypophysis. A warning sign in such a case can be a laboratory value of sodium in the blood < 130 mmol/l.**

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Hydrostatic cerebral edema

Arises as a result of **venous congestion** - water and small molecules escape from the vessels. There are no HEB faults.

Hemodynamic swelling of the brain - swelling

This phenomenon accompanies **brain injuries**. The cause is the loss of the autoregulatory ability of the cerebral vessels, this causes a sharp rise in intracranial pressure (ICP) within minutes, the cerebral perfusion deteriorates, which leads to the development of cytotoxic edema, in addition there is a risk of **herniation of the brain** .

It is often accompanied by **diffuse axonal damage**, acute subdural hemorrhage and contusion focus.

🔎 *For more information see Hemodynamic brain swelling.*

Intracranial hypertension

The intracranial space (1700 ml) has three compartments: brain 80% (VM – brain volume), blood in vessels 10% (VK – blood volume), cerebrospinal fluid 10% (VL – cerebrospinal fluid volume). For a closed space, the Monro-Kellie doctrine applies: VM + VK + VL = const. This means that an increase in one component must be accompanied by a decrease in another (since it is water itself and it is incompressible).

Brain Perfusion
: CPP = MAP – ICP (Cerebral Perfusion Pressure = Medium Arterial Pressure – Intracranial Pressure).

Cushing's reflex - with intracranial hypertension, we detect a **rise in BP** as a reflex effort to maintain CPP, **bradycardia** from vagus irritation, if there are also **breathing disorders** – we are talking about Cushing's Triassic. A rise in BP in intracranial hypertension is a late and alarming symptom.

🔎 *For more information see Intracranial hypertension.*

Links

External links

- Neurology for practice, indications for the use of pharmacological treatment in brain edema (https://www.neurologiepropraxi.cz/pdfs/neu/2009/01/05.pdf)

Related Articles

- Craniocerebral trauma
- Intracranial hypertension/PGS
- Cerebral Edema, HEB/Repetitor Disorders

References

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Used literature

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