Brain oedema

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

Classification of brain oedema:

- By scope:
  - generalized;
  - bearing.
- According to pathophysiology:
  - vasogenic;
  - cytotoxic;
  - interstitial;
  - hypo-osmotic;
  - hydrostatic.

Generalised cerebral oedema most often occurs due to hypoxia of the brain. It manifests itself clinically as an **intracranial hypertension syndrome**. For diagnostics we use CT, which shows a reduction in cerebrospinal fluid space and the elimination of the difference between gray and white matter.

Focal cerebral oedema usually occurs around the site of contusion and intracerebral hematomas. Clinically, it has focal neurological symptoms. Focal hypodension is displayed on the CT. Anti-edematous pharmacological treatment **(mannitol, dexamethasone)**, possibly craniotomy and external decompression are used in the therapy.

Causes of cerebral oedema

Neurological causes:

- iCMP and intracranial haemorrhage,
- brain tumours,
- meningitis, encephalitis,
- other brain infections - TB, cysticercosis, toxoplasmosis.

Other causes:

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

Types of brain oedema

According to the pathogenesis of the disease, we distinguish several types of cerebral oedema, but the distinction is often controversial and individual types may overlap.

Vasogenic cerebral oedema

Vasogenic cerebral oedema most often accompanies **brain tumours and inflammations** and is the most common type of cerebral oedema. It is caused by a disorder of the blood-brain barrier (BBB). Otherwise, the tight junctions of the endothelium by means of a tight junction are loosened, allowing the transfer of proteins to the interstitium and the fluid behind them escapes into the intercellular space. The white matter is the most damaged. It responds favorably to corticosteroid therapy, using mainly **dexamethasone**, which reduces the expression of pro-edema-acting VEGF 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.

Osmotic therapy (mannitol) can cause a rebound phenomenon. The drug escapes from the blood vessels into the parenchyma and the swelling worsens.

Cytotoxic cerebral oedema

Cytotoxic cerebral oedema results from cell hypoxia and is accompanied by membrane imbalance. When there is a lack of oxygen for a few seconds, ATP occurs, the sodium pump stops and the water passes **intracellularly**. The swelling of the cells is also potentiated by substances washed out by neutrophils or a possible bacterial infection. The most common etiological factor is **injury**, but it can also occur in connection with strokes. It mainly affects the gray matter of the brain.

CT scan reveals gyrification when the whole brain is affected and the brain is globally hypo-dense.

It is treated with **mannitol** (max. 4-5 days due to the rebound phenomenon), **furosemide**, or concentrated ion solutions (NaCl). It usually does not respond well to steroid treatment.

Interstitial cerebral oedema

Interstitial cerebral oedema (periventricular) occurs in both obstructive and hypo-resorbent hydrocephalus. It can also arise as a complication of meningitis in CSF circulation. Sodium and water transfer transependymally from the ventricles to the white matter. This condition indicates **active hydrocephalus**. The CT shows an extension of the ventricular system. The image sometimes resembles a **laughing face**. Gradual progression can lead to a balloon-like widening of the anterior corners of the lateral ventricles and III. chamber, referred to in English literature as the **mickey mouse sign**.

Hypo-osmotic cerebral oedema

Hypo-osmotic cerebral oedema occurs in **disorders of mineral metabolism** (low sodium, chloride, water poisoning, ADH - SIADH hyper secretion, etc.), as well as in **traumas, tumours, infections or subarachnoid haemorrhage**. It also appears as a **postoperative complication** in pituitary procedures. A laboratory sign of blood sodium <130 mmol / l may be a warning sign in this case.

Rapid fluctuations in plasma Na + can lead to myelin damage and pontine myelosis.

Hydrostatic cerebral oedema

It arises as a result of **venous congestion** - water and small molecules escape from the blood vessels. There are no BBB faults.

Hemodynamic swelling

This phenomenon accompanies **brain injuries**. The cause is a loss of auto-regulatory ability of the cerebral vessels, which in minutes causes a sharp rise in intracranial pressure (ICP), worsens cerebral perfusion, which leads to the development of cytotoxic oedema, in addition, there is a risk of **brain herniation**.

It is often accompanied by **diffuse axonal damage**, acute subdural haemorrhage and contusion deposits.

Intracranial hypertension

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

Brain perfusion: CPP = MAP - ICP (cerebral perfusion pressure = mean arterial pressure - intracranial pressure).

Cushing's reflex - in intracranial hypertension we find an **increase in blood pressure** as a reflex effort to maintain CPP, **bradycardia** from vagus irritation, if **breathing disorders** also occur - we are talking about the Cushing's Triassic. An increase in BP in intracranial hypertension is a late and alarming symptom.

See the Intracranial Hypertension page for more information.

Links

Related articles

- Intracranial hypertension/PGS

External links

1. ↑ PURI, Shri Krishna. *Cerebral edema and its management* [online]. MJAFI, ©2003. [cit. 2020-05-15]. <http://medind.nic.in/maa/t03/i4/maat03i4p326.pdf>.

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