Intracranial hypertension/PGS

Intracranial hypertension

Intracranial hypertension syndrome is a life-threatening condition. Intracranial hypertension is the main mechanism of secondary cerebral damage. The intracranial space is a tightly bounded and closed compartment by the calva, which consists of brain tissue (80%), the cerebrospinal fluid compartment (10%) and the filling of blood vessels (10%). This content remains constant and any change in one of these compartments must be associated with a compensatory change in another. Compensation mechanisms are limited. Processes that increase intracranial pressure are referred to as '*expansive intracranial processes*. English abbreviation intracranial SOL (space-occupying lesion).

Clinical symptoms of intracranial hypertension syndrome:

- **headache** - often worse lying down (especially in the morning after waking up) and when using an abdominal press, unresponsive to common analgesics
- **pain behind the eyes** or when moving the bulbs
- **vomiting** - sudden, sometimes without nausea (projectile vomiting)
- **vertigo states**
- **visual problems** - blurred vision, diplopia, loss of vision
- **focal symptoms** according to the location of the pathology
- **meningeal symptoms**
- **rise in blood pressure** and slowing of heart rate
- **disorder of consciousness** (quantitative or qualitative)

As the condition progresses, the compensatory mechanisms are exhausted, the brain is pushed into the physiological openings and '*cone symptoms* appear. Cerebral cones (herniations) have typical locations due to their anatomical arrangement.

Brain herniation

Brain herniation/PGS/diagnosis

Examination for suspected intracranial hypertension

- **CT brain *event.*** *MRI of the brain'* - sovereign method; changes in the size of the ventricular system, disappearance of subarachnoid spaces, displacement of midline structures, focal expansion.
- **Ocular fundus examination** - congestive papilla event. even with hemorrhages on the eye background.
- **EEG** - non-specific diffuse abnormalities (mostly slowing of activity).

The most common causes of intracranial hypertension syndrome

- **intracranial tumors** − primary, metastatic
- **hematomas** - spontaneous or traumatic
- **extensive ischemia**
- **hydrocephalus** - non-communicating, communicating (normotensive hydrocephalus - clinically *Hakim's triad*: spastic walking disorder, urinary incontinence, organic psychosyndrome)
- **pseudotumor cerebri**
- **diffuse brain edema** various etiologies − CNS inflammation, toxic, drug-induced, hypoxic, hypoosmolar, metabolic − metabolic ketoacidosis, hepatic encephalopathy, uremia
- **brain abscess**
- **brain trauma**

Treatment

In addition to targeted therapy, a mandatory part of the treatment of a patient with intracranial hypertension is monitoring and control of heart rate, BP, respiration, SpO< sub>2**,** ECG**,** temperatures **and** central venous pressure**, it is also appropriate to monitor ' "Intracranial pressure" (ICP sensors, ultrasound) - within the general intensive care of patients.**

The main strategy of *targeted treatment* is the effort to suppress especially the vasogenic component of brain edema by short-term *increasing blood osmolality* and creating an osmotic gradient between the vascular compartment and the interstitium (mannitol and other osmotically active substances) , in indicated cases **corticoid treatment''*,*** *mandatory hyperventilation**, neuroprotective** barbiturate coma **or** neurosurgery* - see diagram. It is also important to prevent the development of secondary cerebral damage as a result of tissue hypoperfusion (hypoxia), i.e. the **effort to maintain sufficient brain perfusion**.

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CNS Tumors

CNS Tumors