Cerebral Edema

Last updated: May 8, 2019

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Etiopathophysiology

**brain edema** - brain volume↑ due to increase in ***extravascular*** brain water.

* it is general reaction to insults.

N.B. differentiate from **brain engorgement** - brain volume↑ due to increase in ***intravascular*** volume (e.g. obstruction of cerebral veins, arterial vasodilatation).

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Cerebral Edema** | | |
| **Vasogenic** | Cytotoxic | **Hydrocephalic**  **(s. interstitial)** |
| **Pathogenesis** | Capillary permeability↑ | Cellular swelling | Intraventricular fluid↑ |
| **Location** | White matter | Gray & white matter | Ventricular, white matter (periventricular) |
| **Edema fluid** | Plasma filtrate | Intracellular H2O & Na | CSF |
| **Extracellular fluid volume** | ↑ | ↓ | ↑ |
| **Contrast enhancement** | + | - | - |

**Vasogenic edema** - ***increased*** ***capillary permeability*** to macromolecules ***= BBB disruption*** (widening of tight junctions + increase in pinocytotic vesicles).

* etiology (most common type of edema!):
  1. **tumor**
  2. **abscess**, meningitis, encephalitis
  3. **stroke** (ischemia, infarction, hemorrhage)
  4. **trauma** (diffuse BBB disruption up to several hours after trauma - window of opportunity to administer cerebral protective drugs that would not penetrate intact BBB)
  5. **lead encephalopathy**.
* accumulates preferentially in **white matter** and can become very widespread.
* exception is corpus callosum (so tightly bundled that there is little extracellular space - edema does not spread readily).
* paucity of brain lymphatics impairs resorption of excess fluid.
* eventually resolves (edema fluid is reabsorbed into vascular space or ventricular system).
* BBB disruption causes CT/MRI contrast enhancement, CSF protein↑.

**Cytotoxic edema** - ***swelling of cells*** (neurons, glia, endothelial) due to ***membrane pump failure***.

* etiology:

1. ***decreased energy supply*** to brain cells (e.g. ischemia, hypoxia, trauma) → increased intracellular osmoles (Na+, lactate, H+) → rapid water entry into cells.

* even after short ischemia, brain may respond to reperfusion with severe brain edema.

1. ***plasma osmolality***↓:
   1. **osmotic disequilibrium syndromes** (in hemodialysis, diabetic ketoacidosis) - excessive *intracellular solutes* (*organic acids* in uremia; *glucose & ketone bodies* in diabetic ketoacidosis) result in excessive cellular hydration when plasma osmolality is rapidly reduced with therapy.
   2. acute dilutional hyponatremia
   3. inappropriate secretion of ADH
2. acute ***hepatic encephalopathy***, ***Reye's syndrome***.

* accumulates in **white & grey matter**.
* extracellular fluid volume is compensatory reduced!

Conditions associated with generalized edema have elements of both vasogenic and cytotoxic edema.

* both *vasogenic* and *cytotoxic* edema occur in setting of ***trauma***!
* acute ***hypoxia*** causes *cytotoxic* edema, which is followed by *vasogenic* edema as infarction develops.

**Interstitial (s. hydrocephalic) edema** (best characterized in ***obstructive hydrocephalus***) - ***CSF movement across ventricular walls***.

* accumulates in **periventricular white matter** (esp. at angles of lateral ventricles).
* volume of periventricular white matter is reduced! (after successful CSF shunting, edema is reduced, and thickness of mantle is restored)

mechanisms by which edema alters neuronal function:

1. ICP↑
2. Increased distances for nutrient diffusion (e.g. O2).
3. Lipid peroxidation in membranes

Pathology

* edematous brain is softer and appears to "overfill" cranial vault.
* **gyri** are flattened, intervening **sulci** are narrowed.
* **ventricular cavities** are compressed.
* as brain expands, *herniation* may occur. [see p. S54 >>](S54.%20Brain%20Herniation.pdf)



[Source of picture: “WebPath - The Internet Pathology Laboratory for Medical Education” (by Edward C. Klatt, MD) >>](http://library.med.utah.edu/WebPath/webpath.html)

Multiple small metastases causing cerebral edema seen at right which obscures structures:



[Source of picture: “WebPath - The Internet Pathology Laboratory for Medical Education” (by Edward C. Klatt, MD) >>](http://library.med.utah.edu/WebPath/webpath.html)

Clinical Features

- **intracranial hypertension**:

1. **Generalized brain dysfunction** (disturbances of consciousness, etc) – due to diffuse edema.
2. **Focal neurologic deficits** – due to focal edema, brain herniation.

N.B. in brain tumor, clinical signs are often caused more by surrounding edema than by tumor mass itself (so deficits maybe reversible with steroids)!

* + *rate of edema formation* is directly proportional to *severity of neurologic deficits*.
  + in chronic hydrocephalus interstitial edema manifestations are usually minor (in advanced cases - **dementia** and **gait disorder** become prominent) - CSF accumulation in extracellular space is much better tolerated than is presence of plasma in extracellular space (as in vasogenic edema).

Diagnosis

**Neuroimaging** (MRI better than CT):

* 1. ***decreased density*** of brain parenchyma (water content↑) – T1-MRI and CT signal↓, T2-MRI signal↑.
* blurring or loss of visible distinction between gray matter and white matter.

N.B. vasogenic edema spreads along white matter tracts - no grey matter involvement with preserved grey-white junction (vs. cytotoxic edema)

* 1. ***mass effect***:

1. **diffuse edema**: loss of definition of cortical sulci → bilateral compromise of ventricles → effacement of basal cisterns.
2. **focal edema** - focal mass effect.

* **MRI / CT with contrast** → brain parenchymal ***enhancement*** in vasogenic edema (BBB disruption!); no enhancement in cytotoxic edema.

**CSF** - ***protein***↑ in vasogenic edema (BBB disruption!); normal in cytotoxic edema.

**EEG**:

1. vasogenic edema – slowing.
2. interstitial edema – normal.

Treatment

* 1. **Intensive care** - patent airway, avoidance of hypoxia, maintenance of BP.

N.B. avoid salt-free fluids IV!

* 1. **Surgery** - excision / decompression of intracranial mass lesions, shunting procedures.
  2. **Pathogenetic treatment**:

Cytotoxic edema - augment CPP + increase intravascular osmolality.

Vasogenic edema – decrease hydrostatic pressure in capillaries + decrease BBB permeability.

* + 1. **glucocorticoids** (dexamethasone 10 mg IV or IM loading → 4 mg q6h maintenance; pediatric dose 1-2 mg/kg loading → 0.25 mg/kg qid maintenance) - direct effect on endothelial cell function – decreased BBB permeability – for vasogenic edema (around tumor, abscess, radiotherapy field).

Glucocorticoids dramatically and rapidly (in hours) reduce focal and general signs of brain edema around tumors!

* usual ***complications*** of steroid therapy are expected (esp. *gastric hemorrhage* - all patients receiving steroids for more than few hours should receive ranitidine or PPI!).
* not useful in cytotoxic edema (e.g. no efficacy in TBI, stroke).
* conflicting reports about efficacy in acute bacterial or tuberculous meningitis (e.g. steroids reduce deafness in infants with bacterial meningitis).
  + 1. **osmotherapy** (mannitol) – for cytotoxic edema.
* effect is ***short-lived*** (solute reaches equilibrium concentration in brain after delay of only few hours).
* parts of brain most likely to “shrink” are normal areas (e.g. regions of vasogenic edema with increased capillary permeability do not shrink\*).

\*even develop rebound edema following mannitol use because solute accumulates in edematous tissue.

* no rationale for long-term use - brain adapts to hyperosmolality with increase in intracellular osmolality.
  + 1. **drugs that reduce CSF formation** (acetazolamide, furosemide) – for interstitial edema.

Bibliography see p. S50 [>>](S50.%20GENERAL%20-%20Intracranial%20Hypertension.pdf#Bibliography)

[Viktor’s Notes℠ for the Neurosurgery Resident](http://www.neurosurgeryresident.net/)

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