

Cerebral edema

State of increased brain volume as a result of increased in water contents (intracellular & or extracellular).

Types:

1) vasogenic edema : most common type.

Cause == break down of BBB (increased permeability of capillaries with incompetence of tight junctions) as occur in trauma, tumor , abscess , late stages of infarction.

The edema is more marked in white matter than gray matter.



2) cytotoxic edema :

Cause== hypoxia of neural tissue causing failure of ATP-dependent sodium pump promoting accumulation of intracellular sodium with subsequent flow of water into the cell to maintain osmotic equilibrium as occur in early stage of infarction & water intoxication.

It affects all types of cells (endothelial , astrocytes ,neurons) so can be seen in both gray & white matter.

3) interstitial edema:

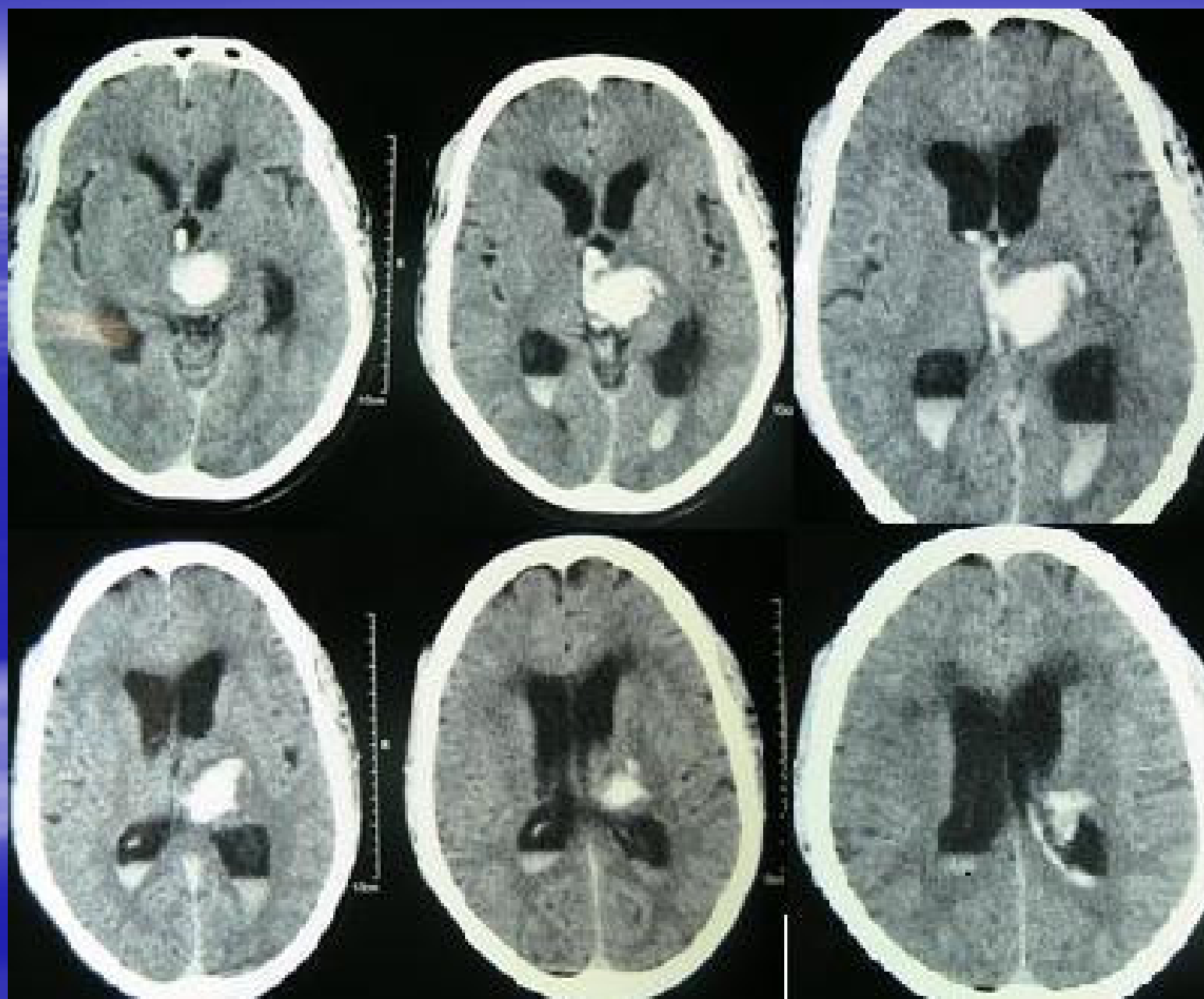
Cause== transduction of CSF in obstructive hydrocephaly.

On CT or MRI of brain , it's seen as periventricular low density because of retrograde transependymal flow of CSF into interstitial space of the white matter.

It indicates active hydrocephaly that need surgical therapy.

4) hydrostatic edema:

Cause== direct transmission of pressure to the cerebral capillaries with transduction of fluid into the extracellular space from the capillaries as occur in acute ,malignant hypertension.



Treatment of cerebral edema

- 1) surgical removal of offending lesion.*
- 2) control of arterial blood pressure.*
- 3) corticosteroid.*
- 4) osmotherapy*

Brain herniation

- *State of brain shifting across structures within the skull due to very high ICP.*
- *The brain is supported by 2 major dural folds (falx cerebri & tentorium cerebelli) that prevents undue movement of the brain within the cranial cavity*

Classification

a) supratentorial herniation

1) uncal : most dramatic & most common type seen clinically.

Cause== middle cranial fossa lesion (acute EDH,SDH, temporal lobe contusion , temporal lobe neoplasm)

C/F:

1) progressive impaired consciousness.

2)ipsilateral dilated pupil.

3) contralateral weakness.

4) ipsilateral weakness (Kernohan's notch) ,the brain stem is pressed against opposite tentorial edge

2) cingulated (subfalcine):

Cause== focal supratentorial mass lesion with progressive local pressure causing cingulate gyrus to herniated under the falx to the opposite site

The anterior cerebral artery may be compressed by the tight, sharp edge of the falx cerebri

There are no clinical signs or symptoms specific to a cingulate herniation

3) central (transtentorial):

Downward displacement of diencephalons & midbrain centrally through the tentorial incisura.

Cause== mass lesion in the frontal , parietal or occipital areas, also bilateral mass lesion as in bilateral subdural hematomas.

C/F:

- 1) disturbed level f consciousness.**
- 2) bilateral small size reactive pupils.**
- 3) Cheyne-stokes respiration.**
- 4) vertical gaze palsy.**

4) transcalvarial herniation

Herniation of the brain through a fracture or a surgical site e.g. craniectomy.

b infratentorial herniation)

1) upward cerebellar:

Posterior fossa lesion can cause the cerebellum to move up through the tentorial notch.

2) downward cerebellar (tonsillar):

Cause==

- 1) acute expansion of posterior fossa lesion.***
- 2) lumbar puncture in a patient with a mass lesion within the cranial cavity.***

The cerebellar tonsil herniates through the foramen magnum into the upper spinal canal, compressing the medulla.

C/F== cardiorespiratory impairment of medullary compression

- 1) hypertension.***
- 2) high pulse pressure.***
- 3) Cheyne-Stokes respiration.***
- 4) impaired consciousness.***
- 5) neck stiffness.***
- 6) opisthotonus.***
- 7) decorticate or decerebrate posture.***

