

- Worsening focal deficits may be seen in patients with localized edema, but most commonly the development or progression of edema results in diminished level of consciousness due to raised intracranial pressure (ICP).
- Herniation is the most feared consequence.
- CT scan reveals edema as an abnormal hypodense signal. When diffuse, it provokes effacement of the gray white matter junction, loss of differentiation of the lenticular nucleus, and decreased visualization of the sulci, insula, and cisterns while the presence of vasogenic edema can be inferred from the appearance of hypodensity following the course of white matter tracts.
- CT is not very helpful to distinguish vasogenic from cytotoxic edema.
- MRI shows edema as hypointense signal in T1-weighted sequences and hyperintense signal in T2-weighted and FLAIR sequences. Delineation of the spread of edema is much clearer with MRI.

- Cerebral edema is a potentially devastating complication of various acute neurologic disorders.

- Its successful treatment may save lives and preserve neurologic function.
- Cerebral edema may be comprehensively defined as a pathologic increase in the amount of total brain water content leading to an increase in brain volume.

- Edema in the brain may be topographically classified into focal or global.

- (i) Focal edema generates a pressure gradient with adjacent regions and may result in tissue shift and herniation. Examples of focal edema can be found around tumors, hematomas, and infarctions.
- (ii) Global edema diffusely affects the whole brain and, when critical, it may cause intracranial hypertension, compromise perfusion, and lead to generalized ischemia. Cardiopulmonary arrest, severe traumatic injury, and fulminant liver failure are common causes of global cerebral edema.
- A different classification based on the pathophysiologic mechanisms responsible for the production of the edema classifies it into 3 types: cytotoxic, vasogenic, and interstitial.
- (i) In cytotoxic edema, the increased water content is localized intracellularly and is due to the failure of ionic pumps that normally maintain cellular homeostasis. Ischemia and profound metabolic derangements are the most common causes. Cytotoxic edema preferentially affects gray matter
- (ii) In vasogenic edema the main problem is centered in a disruption of the blood-brain barrier, leading to increased permeability and escape of fluid from the intravascular to the extravascular, extracellular space. It accompanies tumors, inflammatory lesions, and traumatic tissue damage. Vasogenic edema tends to predominate in the white matter.
- (iii) Interstitial edema results from increased transependymal flow from the intraventricular compartment to the brain parenchyma; it typically occurs in the setting of obstructive hydrocephalus.
- the clinical manifestations of brain edema tend to be similar regardless of whether the edema is cytotoxic or vasogenic. In fact, both types frequently coexist. For example, early after an ischemic brain infarction cytotoxic edema predominates, but later vasogenic edema becomes the major cause of mass effect as the blood-brain barrier loses continuity and local inflammation develops.

clinical
manifestations

cerebral
oedema
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pathophysiology

general