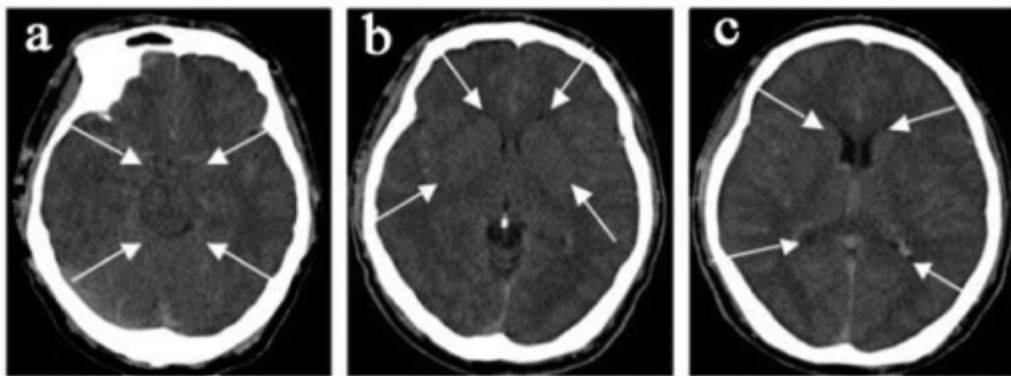


Cerebral Edema (Brain Swelling) — Causes and Treatment Guidelines

[See online here](#)

Cerebral edema describes the excess accumulation of water in the intracellular and extracellular spaces of the brain and is a common condition that usually presents as a consequence of local or systemic pathology, rather than a primary condition. In this article, you will get a great overview of the types of cerebral edema and corresponding medical treatments.



Definition

Cerebral edema is defined as the excess accumulation of water in the intracellular and extracellular spaces of the brain.

Epidemiology

Though cerebral edema is common, as it occurs as a secondary occurrence following other primary brain conditions, it is rarely reported due to the complexities associated with its diagnosis.

Pathophysiology of Cerebral Edema

Cerebral edema can occur if cells of the brain become damaged (i.e. cytotoxic edema) or if blood vessels get injured (i.e. vasogenic edema). Cellular and blood vessel damage results in the release of glutamate into the extracellular space (i.e. excitotoxicity) and entry of calcium and sodium into the cells. The sodium-potassium ATP pump pushes out some of the calcium, causing sodium accumulation within the cell. This sodium accumulation creates an osmotic gradient that pulls water into the cell, resulting in intracellular edema and cell swelling. Calcium accumulation within cells of the brain results in the activation of intracellular cytotoxic processes which can result in [apoptosis](#). Moreover, the formation of inflammatory mediators such as c-foc, c-jun, and cytokines

get activated.

Injury and ischemia of the central [nervous system](#) can also release glutamate from the intracellular space to the extracellular space, which is the cause of cerebral edema in ischemic brain injury. Direct cell injury is responsible for brain edema in [traumatic brain injury](#).

The effects of cerebral edema are a result of the process described by the Monroe-Kellie doctrine which states that an increase in one component of the cranial cavity must be followed by compensatory loss of another in equal proportions because the cranial cavity is fixed in volume, containing components in fixed proportions. The contents of the cranial cavity are:

- Brain matter making up 1400 ml
- Blood making up 150 ml
- Cerebrospinal fluid at 150 ml

Cerebral edema alters the proportions of the contents in the cranial cavity, increasing the amount of brain volume which leads to increased intracranial pressure. The body responds to this increased pressure by reducing the amount of blood flow to the brain, potentially causing further damage to the brain.

Types of Cerebral Edema

Broadly speaking, cerebral edema can be of either vasogenic origin or cytotoxic/cellular edema. However, many are the times when smaller categories of four types of cerebral edema are used in practice. They include:

Vasogenic

Vasogenic cerebral edema is caused by disruption of the blood-brain barrier, resulting in the influx of fluid and solutes into the brain. This is the most common type of brain edema and mainly affects the white matter of the brain.

Cellular edema

Cellular cerebral edema (previously known as cytotoxic cerebral edema) is characterized by cellular swelling and is seen in patients with traumatic brain and ischemic cerebral injury. Cellular cerebral edema affects the glial, neuronal, and endothelial cells and affects the gray matter of the brain. Cellular cerebral edema typically occurs within minutes after injury, whereas, vasogenic edema occurs over a period of days.

Interstitial cerebral edema

Interstitial cerebral edema is caused by the outflow of cerebrospinal fluid from the intraventricular space to the extracellular interstitial space of the brain. This type of cerebral edema is seen in patients with increased intraventricular pressure, i.e. [hydrocephalus](#). Interstitial cerebral edema may also be seen in patients with meningitis.

Osmotic cerebral edema

Osmotic cerebral edema results from abnormal levels of osmolarity in plasma and fluids within the brain. The brain plasma may have lower osmotic levels following dilution, i.e.

from SIADH secretion or increased volume intake, causing the high osmotic brain matter to pull water from the plasma leading to cerebral edema.

Etiologies of Cerebral Edema

We can summarize the causes of cerebral edema according to the following classification:

Neurological causes	<ul style="list-style-type: none">• Ischemic and hemorrhagic stroke<ul style="list-style-type: none">• Brain tumors• Meningitis• Encephalitis• Tuberculosis and toxoplasmosis.
Non-neurological causes	<ul style="list-style-type: none">• Diabetic ketoacidosis• Lactic acidosis• Malignant hypertension• Hypertensive encephalopathy<ul style="list-style-type: none">• Fulminant hepatitis• Reye's syndrome• Systemic poisoning with carbon monoxide or lead• Hyponatremia, and high altitude cerebral edema.

Clinical Presentation of Cerebral Edema

Cerebral edema is intrinsically asymptomatic. Symptoms start to arise once the intracranial pressure reaches a level that is sufficient to cause local ischemia.

Symptoms of increased intracranial pressure include:

- Altered level of consciousness
- Bradycardia
- Rise in blood pressure
- Abnormal breathing patterns
- Anisocoria
- Extensor plantar response on the side of the lesion

Cerebral edema is the most common cause of death in patients with acute [ischemic stroke](#). Patients with acute ischemic stroke typically have cytotoxic cerebral edema in the earlier stages but later develop vasogenic edema due to blood-brain-barrier disruption.

Diagnostic Workup for Cerebral Edema

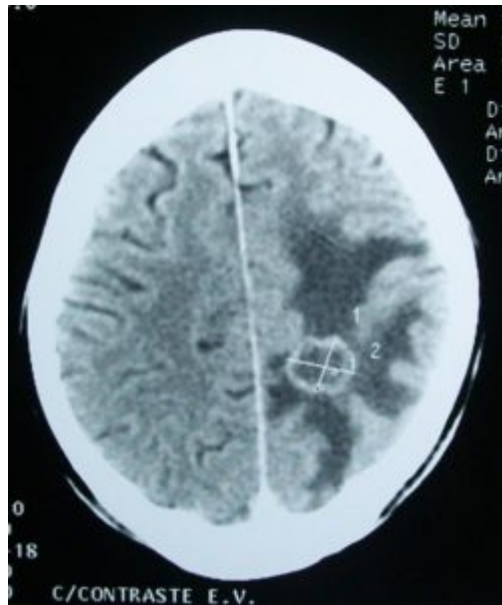
A brain [computed tomography scan](#) is an excellent imaging modality for the evaluation and exclusion of acute brain pathologies, including cerebral edema. Brain edema appears as an area of low density on unenhanced computed tomography scans of the brain.

When evaluating the brain computed tomography scan of a patient suspected of having cerebral edema, one should focus on the appearance of the white matter. Other features that suggest increased intracranial pressure include flattened gyri and narrowed sulci as well as compression of the ventricles. Brain computed tomography scans should be ordered during the follow-up period to monitor the resolution of brain edema once treatment is initiated.

Patients with epidural and intracerebral hematomas due to traumatic brain injury typically have white matter confined cerebral edema without apparent involvement of the cortex.

Magnetic resonance imaging is superior to computed tomography scans in detecting

brain abscess which can be the cause of cerebral edema in septic patients with focal neurological deficits.



[Image](#): "Edema (darker areas) surrounding a secondary brain tumor" by Bobjgalindo.
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Though intracranial pressure monitoring is indicated when cerebral edema is observed in the previous imaging studies, the routine use of intracranial pressure monitoring remains controversial. Currently, intracranial pressure monitoring should be initiated only if the management plan will be affected by the results of the monitoring.

The electroencephalogram is not useful in evaluating cerebral edema; however, it may be helpful in a subgroup of patients with an epilepsy disorder as the cause of cerebral edema, i.e. status epilepticus.

Medical Treatment of Cerebral Edema

While our understanding of cerebral edema and its pathogenesis have expanded dramatically in the last decade, treatment options are still limited.

Osmotherapy for Cerebral Edema

Osmotic therapy is the mainstay of treatment for cerebral edema. Osmotic therapy aims to decrease brain bulk and water content via osmодиuresis.

Osmotic therapy helps to:

- Draw water out of the brain
- Decrease intracranial pressure
- Increase cerebral blood flow

Mannitol is the most common osmotic agent used, which decreases the intracranial pressure dramatically by decreasing the overall water content of the brain, reducing blood volume by vasoconstriction, and reducing cerebrospinal fluid volume. Mannitol also decreases blood viscosity, improves cerebral perfusion, and may have a cytoprotective effect. The most commonly used dose of mannitol for cerebral edema is a bolus dose of 1 g/kg followed by 50 g every 2–3 hours.

Glycerol can also be administered in oral doses of 30 mL every 4–6 hours for its osmotic effects as an alternative to mannitol.

Diuretic Therapy for Cerebral Edema

[Loop diuretics](#) such as furosemide can be administered after the administration of mannitol as they prolong the osmotic effects of mannitol. The typical dose of furosemide is 0.7 mg/kg.

Corticosteroids for Cerebral Edema

Corticosteroids decrease tumor-associated vasogenic edema by decreasing capillary permeability of the disrupted blood-brain-barrier. Though corticosteroids are beneficial in patients with vasogenic cerebral edema, they are not useful in the treatment of cytotoxic cerebral edema. Therefore, corticosteroids should not be routinely administered to patients with cerebral edema due to ischemic stroke, hemorrhagic stroke, or traumatic brain injuries. Corticosteroids have been found to increase the rate of complications in patients with cytotoxic cerebral edema.

Hyperventilation for Cerebral Edema

Controlled hyperventilation is helpful in reducing raised intracranial pressure, especially in patients with cerebral edema after traumatic brain injury. If hyperventilation is used, care must be taken not to reduce the pCO₂ level below 25 mm Hg.

Surgical Treatment of Cerebral Edema

Currently, barbiturates and procaine derivatives are no longer recommended in the management of acute cerebral edema. Therefore, patients who fail to respond to the previous medical treatments of cerebral edema might need surgical intervention to decompress the swollen brain.

Patients with life-threatening brain-shifts might need a temporary ventriculostomy or a decompressive craniectomy as these procedures may be life-saving. Patients with large cerebral and cerebellar infarcts with cerebral edema should also undergo surgical decompression as a life-saving measure.

References

Jha S. Cerebral Edema and its Management. Medical Journal, Armed Forces India. 2003;59(4):326-331. doi:10.1016/S0377-1237(03)80147-8.

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