TRANSTENTORIAL HERNIATION

Authored by

mohammad looti

October 22, 2025

RECOMMENDED CITATION

mohammad looti (2025). *TRANSTENTORIAL HERNIATION*. PSYCHOLOGICAL SCALES. Retrieved from https://scales.arabpsychology.com/?p=54145

TRANSTENTORIAL HERNIATION

Primary Disciplinary Field(s): Neurology, Neurosurgery, Critical Care Medicine, Pathology

1. Core Definition

Transtentorial herniation is a critical neurosurgical emergency defined as the pathological displacement of brain parenchyma across the tentorium cerebelli, specifically through the aperture known as the tentorial notch or incisura. This life-threatening phenomenon occurs when severely elevated intracranial pressure (ICP)--often resulting from a rapidly expanding mass lesion such as a large hematoma or tumor--creates a powerful pressure gradient between the supratentorial and infratentorial compartments of the skull. The resulting force drives the brain tissue medially and downward.

The core mechanism of transtentorial herniation involves the dispositioning of the medial aspect of the temporal lobe, or the brain's deep hemispheric structures, medially and downward via the tentorial notch. This downward displacement leads to significant compression and distortion of the vital structures housed within the incisura, most notably the midbrain and the diencephalon. The specific anatomical structures displaced depend on the subtype of herniation, but the result is always a dangerous disruption of brainstem function.

This process is clinically significant because the displacement elicits severe dispositioning of the midbrain both horizontally and vertically, causing ischemia and direct structural damage to the crucial ascending reticular activating system (ARAS) responsible for consciousness, as well as critical cardiovascular and respiratory centers. The term uncal herniation is commonly referenced as a specific type of transtentorial herniation, referring to the specific prolapse of the uncus, the innermost part of the temporal lobe, making it the most frequent and acutely recognized subtype. This rapid sequence of events, if not immediately reversed, might cause death.

2. Pathophysiology and Mechanism

The initial event leading to transtentorial herniation is the uncontrolled rise of intracranial pressure (ICP), exceeding the compensatory reserves available within the rigid skull (governed by the Monro-Kellie doctrine). As the volume of one cranial component (blood, cerebrospinal fluid, or brain tissue) increases excessively, the brain attempts to shift away from the area of high pressure, following paths of least resistance. The tentorial notch, a natural opening in the dura mater separating the cerebellum from the cerebrum, becomes the primary escape route for the supratentorial contents.

During the herniation process, the displacement of the midbrain results in severe mechanical stress. This pressure often causes stretching and compression of the penetrating arteries originating from the basilar artery, leading to secondary ischemic injury in the brainstem, known as Duret hemorrhages. Furthermore, the lateral displacement can force the cerebral peduncle of the midbrain against the sharp edge of the tentorium on the opposite side of the initial mass lesion. This counter-intuitive finding, known as the Kernohan's notch phenomenon, results in ipsilateral (same side as the mass) hemiparesis, confusing the localization of the lesion.

A hallmark pathophysiological consequence is the early compression of the third cranial nerve (CN III) as it traverses the space near the herniating temporal lobe. CN III carries parasympathetic fibers responsible for pupil constriction. Compression of these superficial parasympathetic fibers causes the pupil on the side of the herniation (ipsilateral) to dilate and become fixed (unreactive to light), a critical and often early clinical sign indicating impending brainstem failure. This sequence underscores the immediate need for intervention to prevent irreversible damage to the brainstem and subsequent cardiovascular or respiratory arrest.

3. Types of Transtentorial Herniation

While transtentorial herniation is often used broadly, it encompasses two primary subtypes distinguished by the specific brain structures displaced and the direction of movement, which dictate the sequence of clinical deterioration. These are lateral transtentorial herniation (Uncal) and central transtentorial herniation. Understanding this distinction is crucial for interpreting imaging and predicting patient decline.

Uncal (Lateral) Herniation: This is the most common form and is characterized by the medial and downward displacement of the uncus and the adjacent medial temporal lobe gyrus. It typically results from a rapidly expanding lateral mass lesion, such as a temporal lobe epidural hematoma. Key features include early ipsilateral CN III palsy, resulting in the classic unilateral fixed and dilated pupil. The compression of the posterior cerebral artery (PCA) against the tentorium often leads to ipsilateral occipital lobe infarction, resulting in contralateral visual field defects.

Central Transtentorial Herniation: This form occurs when a symmetrically enlarging lesion or generalized severe cerebral edema--such as diffuse anoxic injury or bilateral frontal masses--pushes the entire diencephalon and midbrain directly downward through the tentorial notch. Unlike the uncal type, central herniation tends to be slower and symmetrical, resulting in gradual neurological deterioration. Clinical signs include small, reactive pupils (due to early stretching of sympathetic pathways) that eventually become fixed and mid-position, followed rapidly by deep coma and progressive loss of brainstem reflexes. This vertical displacement strains the central structures, leading quickly to irreversible damage.

4. Etiology and Precursors

Transtentorial herniation is not a primary disease but a life-threatening complication of severe space-occupying lesions or diffuse cerebral swelling that elevate intracranial pressure beyond physiological limits. The precursors are diverse but share the common feature of creating a significant supratentorial mass effect.

Traumatic Mass Lesions: Acute subdural hematomas (SDH) and epidural hematomas (EDH) are the most frequent immediate causes, particularly when they expand rapidly, pushing adjacent structures toward the tentorial notch.
Intracerebral Hemorrhage: Large, spontaneous hemorrhages within the cerebral hemispheres, such as those caused by uncontrolled hypertension or arteriovenous malformations, can rapidly increase localized pressure and initiate herniation.
Neoplasms: Fast-growing brain tumors, especially high-grade gliomas or large metastatic lesions, exert pressure on surrounding structures. Although generally slower in onset than acute bleeds, they frequently lead to central or uncal herniation as they grow past critical size thresholds.
Cerebral Edema: Generalized brain swelling resulting from major ischemic strokes, severe infections (encephalitis), or traumatic brain injury (TBI) can cause diffuse pressure elevation, typically leading to central herniation.
Hydrocephalus: Acute obstructive hydrocephalus, where the flow of cerebrospinal fluid (CSF) is blocked, causing rapid ventricular expansion, can increase pressure dramatically and lead to downward displacement of the deep brain structures.

5. Clinical Manifestations

The clinical presentation of transtentorial herniation follows a predictable and rapidly progressive downward trajectory, reflecting the caudal compression of the brainstem. Early recognition of these signs is paramount for patient survival, as minutes often separate reversible injury from brain death.

The earliest and most reliable sign of impending uncal herniation is the disruption of the ipsilateral oculomotor nerve (CN III), manifesting as a fixed, dilated pupil. This is often accompanied by decreasing level of consciousness, frequently measured by a precipitous drop in the Glasgow Coma Scale (GCS) score. General symptoms of severe ICP elevation, such as headache, intractable vomiting, and papilledema (swelling of the optic nerve head), usually precede the acute herniation event, though the mass effect itself accelerates the decline.

As brainstem compression progresses to the midbrain and pons, critical vital signs are affected. The phenomenon known as Cushing's triad--characterized by bradycardia (slow heart rate), systolic hypertension (widening pulse pressure), and irregular respiration--is a classic, late-stage indicator of severe brainstem compromise and profound ICP elevation. Motor signs also progress from hemiparesis to abnormal posturing: decorticate posturing (flexion of arms and extension of legs, indicating damage above the red nucleus) gives way to the more ominous decerebrate posturing (extension of all four limbs, indicating damage at the level of the midbrain/pons).

In the final stages, respiratory patterns become chaotic (e.g., Cheyne-Stokes respiration or ataxic breathing), reflecting failure of the medullary respiratory centers. All brainstem reflexes are lost--including pupillary, corneal, and vestibular-ocular reflexes--and the patient descends into deep, unresponsive coma. At this point, fixed, mid-position pupils and apnea are typically observed, indicating impending or actual brain death. This irreversible sequence highlights why transtentorial herniation represents the final, catastrophic stage of uncontrolled intracranial hypertension.

6. Diagnosis and Imaging

Diagnosis of transtentorial herniation is primarily a clinical one, based on the rapid neurological decline and the presence of pupillary changes in a patient with a known or suspected intracranial mass. However, definitive confirmation and identification of the underlying cause rely entirely on rapid neuroimaging.

The initial diagnostic procedure of choice is a non-contrast Computed Tomography (CT) scan of the head, due to its speed and availability in emergency settings. CT imaging clearly identifies the underlying lesion (e.g., hematoma, tumor, hydrocephalus) and, crucially, visualizes the displacement of brain structures. Key radiological signs of transtentorial herniation include effacement (obliteration) of the perimesencephalic and suprasellar cisterns, distortion of the midbrain, and the visualization of the medial temporal lobe (uncus) projecting into the tentorial notch space.

While Magnetic Resonance Imaging (MRI) offers superior detail regarding soft tissue and chronic changes, it is typically reserved for stable patients or for planning definitive surgery, as the time required for MRI acquisition often precludes its use in the acute, life-threatening phase of transtentorial herniation. The speed of diagnosis is critical; any delay in obtaining imaging for a patient showing signs of herniation is unacceptable, as irreversible damage occurs within minutes.

7. Therapeutic Management

Management of transtentorial herniation is a dual-pronged approach, focusing simultaneously on immediate emergency intervention to lower intracranial pressure and definitive surgical or medical treatment to address the underlying causative pathology.

The initial emergency goal is rapid reduction of ICP to reverse the mechanical compression on the brainstem. This involves several critical steps: maintaining adequate oxygenation and ventilation (often via intubation and mechanical ventilation), elevating the head of the bed to promote venous drainage, and utilizing hyperosmolar therapy. Medications such as Mannitol or hypertonic saline are administered intravenously to draw fluid out of the brain parenchyma and reduce cerebral edema acutely. If necessary, transient hyperventilation can be used to induce cerebral vasoconstriction, thereby temporarily reducing cerebral blood volume, although this must be carefully monitored to avoid secondary ischemia.

Definitive treatment requires addressing the mass lesion itself. If the cause is a surgically accessible lesion, such as an acute epidural or subdural hematoma, immediate neurosurgical evacuation is mandatory. For mass lesions resulting from tumors or inaccessible hemorrhages, measures such as external ventricular drainage (EVD) may be used to drain cerebrospinal fluid and temporarily relieve pressure. In cases of severe, refractory edema, a decompressive craniectomy--where a portion of the skull is temporarily removed to allow the swollen brain room to expand--may be necessary as a life-saving measure to prevent catastrophic herniation.

Further Reading

Transtentorial Herniation (Wikipedia)
Uncal Herniation (Wikipedia)
Intracranial Pressure (Wikipedia)
Kernohan's Notch (Wikipedia)