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Uncal Herniation: Emergency Recognition and Multidisciplinary Operating Room, Nursing, Laboratory, and Radiology Management

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Abstract

Background: Uncal herniation is a life-threatening neurological emergency resulting from displacement of the medial temporal lobe through the tentorial notch due to critically elevated intracranial pressure (ICP). It often arises from traumatic brain injury, intracranial hemorrhage, tumors, or diffuse cerebral edema, and can rapidly compromise brainstem function and cerebral perfusion.

Aim: To review the anatomical basis, pathophysiology, clinical presentation, diagnostic approach, and multidisciplinary management strategies for uncal herniation, emphasizing early recognition and intervention.

Methods:

This comprehensive review synthesizes current evidence and clinical guidelines on uncal herniation, including anatomical considerations, etiologic factors, epidemiology, diagnostic imaging, and emergency treatment protocols.

Results: Uncal herniation manifests with hallmark signs such as ipsilateral pupillary dilation, contralateral hemiparesis, and altered consciousness. CT imaging remains the first-line diagnostic modality for rapid identification of mass effect and midline shift. Immediate management focuses on ICP reduction through head elevation, controlled ventilation, hyperosmolar therapy (mannitol or hypertonic saline), and urgent neurosurgical intervention. Prognosis depends on timely recognition and reversal; reported reversal rates range from 50–75% when managed promptly.

Conclusion: Uncal herniation represents a final common pathway of uncontrolled intracranial hypertension and requires rapid, coordinated multidisciplinary care. Early detection and aggressive intervention are critical to prevent irreversible brainstem injury and improve survival outcomes.

Keywords: Uncal herniation, intracranial pressure, tentorial notch, traumatic brain injury, hyperosmolar therapy, neurosurgical decompression.

Introduction

The uncus is a distinctive anatomical component located on the anteromedial surface of the parahippocampal gyrus within the medial temporal lobe. This region resides in the supratentorial compartment of the cranial vault; a space separated from the infratentorial (subtentorial) compartment by the tentorium cerebelli. The tentorium cerebelli is a rigid dural reflection that functions as a critical partition within the intracranial cavity, maintaining spatial organization between the cerebral hemispheres above and the cerebellum and brainstem below. The only major communication between these compartments is the tentorial notch (incisura), an opening that accommodates essential neurovascular structures. Because this notch represents a relatively fixed anatomical boundary, it also constitutes a vulnerable site where pathological pressure gradients can precipitate brain tissue displacement. Within the tentorial notch lie structures whose integrity is indispensable for consciousness, ocular function, and cerebral perfusion. The midbrain traverses this region, and adjacent to it course the third cranial nerve (oculomotor nerve), the posterior cerebral arteries, and the superior cerebellar arteries. Posteriorly, the cerebellum occupies the infratentorial

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space and contributes to the confines of the notch. The close proximity of these structures means that even subtle shifts in tissue position can produce profound neurological deterioration. Accordingly, the tentorial notch is not merely a passive anatomical passage but a clinically critical zone where increases in intracranial pressure can translate into rapid, catastrophic compromise of brainstem function and cerebral blood flow [1][2].

Uncal herniation refers to the displacement of the medial temporal lobe—specifically the uncus—through the tentorial notch. This phenomenon is typically driven by rising intracranial pressure secondary to mass effect from intracranial hemorrhage, expanding tumors, cerebral edema, or space-occupying lesions. As intracranial pressure rises, the brain's compensatory mechanisms for maintaining intracranial compliance—such as displacement of cerebrospinal fluid into the spinal compartment and venous blood outflow—initially buffer the increase. However, when these adaptive mechanisms are exhausted, the intracranial system reaches a critical threshold at which additional volume leads to disproportionate pressure elevation. At this point, pressure gradients develop between compartments, forcing brain tissue to shift from one intracranial region to another along the path of least resistance. Clinically, uncal herniation constitutes a life-threatening neurological emergency because it indicates decompensation of intracranial compliance and imminent risk of irreversible brain injury.[1] The displaced uncus can compress the ipsilateral third cranial nerve, distort or compress the midbrain, and compromise arterial supply via the posterior cerebral and superior cerebellar arteries, leading to ischemia, altered consciousness, and rapid neurological decline. For this reason, uncal herniation is regarded as a critical endpoint of uncontrolled intracranial hypertension and requires immediate recognition and intervention to prevent fatal outcomes.[1]

Etiology

Uncal herniation is a consequence of critically elevated intracranial pressure (ICP) with the development of a compartmental pressure gradient that forces the medial temporal lobe—specifically the uncus—toward and through the tentorial notch. Accordingly, its etiologies are best understood not as a single disease entity but as a final common pathway of diverse intracranial processes that either add volume to the fixed cranial vault or disrupt normal cerebrospinal fluid and venous outflow dynamics. Because the skull is a rigid container, any significant increase in intracranial contents—whether blood, brain tissue, cerebrospinal fluid, or a mass lesion—can exceed compensatory capacity and precipitate a rapid rise in ICP. Once compensatory mechanisms fail, even small additional increases in volume may produce dramatic pressure escalation, setting the stage for tissue displacement and herniation syndromes [1][2][3].

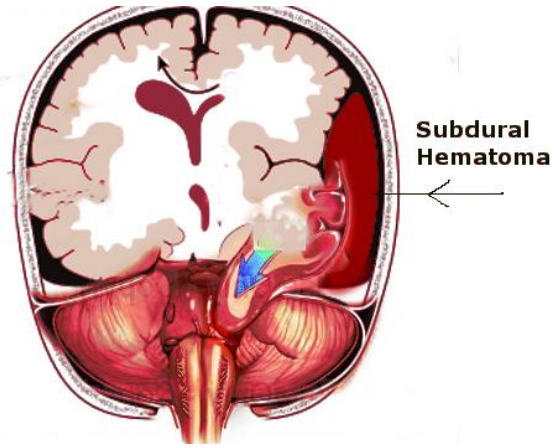

Fig. 1: Subdural Hematoma and Uncal Herniation.

A principal etiologic category is the presence of an expanding space-occupying lesion. This includes intracranial tumors, abscesses, or other masses that progressively increase intracranial volume and exert focal mass effect. The risk becomes particularly pronounced when the lesion is supratentorial and located in a region capable of generating lateral or downward pressure vectors that distort medial temporal structures. Similarly, diffuse cerebral edema—whether due to traumatic brain injury, hypoxic-ischemic injury, hepatic encephalopathy, or severe metabolic derangements—can raise global ICP and contribute to herniation by reducing intracranial compliance. Intracranial hemorrhage is another major cause, including intraparenchymal hemorrhage, subarachnoid hemorrhage with associated swelling, and hemorrhagic transformation of infarction, each of which can create rapid or progressive mass effect [1][2][3]. Traumatic brain injury is among the most time-sensitive etiologies because it can produce quickly expanding extra-axial hematomas, particularly epidural and subdural hematomas. These collections can enlarge over minutes to hours, compressing the ipsilateral hemisphere and shifting the medial temporal lobe toward the tentorial notch. In severe head trauma, the combination of hematoma expansion, cerebral contusion, and secondary edema can accelerate ICP elevation and precipitate abrupt neurological decline. In the specific pathophysiology of uncal herniation, a supratentorial "driving force" generated by raised ICP propels the uncus to slide over the tentorial notch, thereby transferring tissue from one intracranial compartment to another and compressing vital structures in the process.[2] The common etiologic thread across these conditions is the creation of a significant ICP rise and a directional pressure gradient sufficient to displace medial temporal tissue, making early identification and treatment of intracranial mass effect central to prevention.[2]

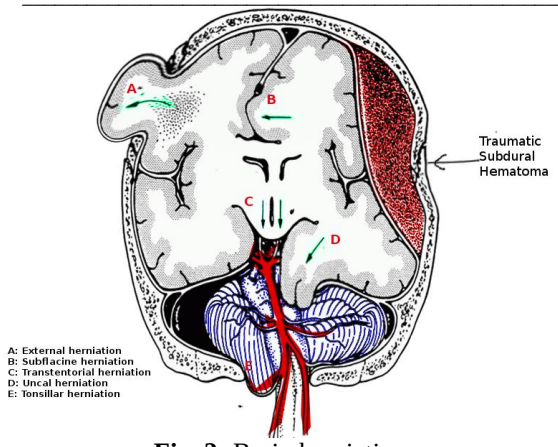

Fig. 2: Brain herniation.

Epidemiology

The true incidence of uncal herniation remains difficult to quantify with precision, largely because it is not routinely captured as a discrete epidemiologic endpoint in most surveillance systems and often occurs as a terminal or evolving complication of broader intracranial pathology. In clinical practice, uncal herniation is typically recognized in the setting of acute neurological decompensation associated with elevated intracranial pressure, and its documentation may be variably recorded as a radiographic finding, a bedside diagnosis, or part of a larger herniation syndrome. As a result, population-level estimates are inherently limited and are more reliably inferred by examining the epidemiology of high-risk conditions—most notably traumatic brain injury (TBI), intracranial hemorrhage, and space-occupying lesions—that can precipitate rapid or progressive intracranial hypertension. Because uncal herniation can develop secondary to TBI, understanding the national burden of traumatic brain injury is essential for contextualizing the size of the at-risk population. The CDC's 2015 *Report to Congress on Traumatic Brain Injury* underscores the substantial impact of injury-related events on emergency and inpatient services in the United States. The report describes approximately 30 million injury-associated emergency department visits, hospitalizations, and deaths occurring annually in the United States, highlighting the scale of trauma-related healthcare utilization and mortality. Within this broader injury landscape, TBI represents a disproportionate contributor to severe outcomes: it accounts for roughly 16% of injury-related hospitalizations and approximately one-third of injury-related deaths. This distribution is epidemiologically important because it signals that, while many injuries are minor or self-limited, a significant fraction of hospitalized and fatal injury cases involve brain injury severe enough to compromise intracranial compliance and elevate the risk of herniation syndromes [2][3]. More specifically, CDC estimates for 2010 indicate that traumatic brain injuries accounted for about 2.5 million emergency department visits, hospitalizations, and deaths in the United States, whether occurring as isolated injuries or in combination with other traumatic insults. These figures emphasize that a large number of patients enter acute care pathways each year with intracranial injury profiles that may evolve dynamically, sometimes deteriorating rapidly due to hematoma expansion, cerebral edema, or secondary injury cascades. In this context, uncal herniation should be viewed less as a common diagnosis and more as a critical, time-sensitive complication arising within a substantial pool of patients exposed to conditions capable of producing severe intracranial pressure elevation [2][3].

Pathophysiology

Uncal herniation represents a catastrophic failure of intracranial volume compensation, driven by escalating intracranial pressure and the development of pressure gradients between intracranial compartments. The intracranial vault is effectively a fixed-volume space composed of three principal components: brain parenchyma, blood (arterial and venous), and cerebrospinal fluid (CSF). The Monro–Kellie principle states that the total volume of these components remains relatively constant; therefore, an increase in one component must be offset by a compensatory reduction in one or both of the others to maintain stable intracranial pressure.[3][4] This concept is central to understanding how a localized lesion in the supratentorial compartment can evolve from a compensated state to a rapidly decompensated emergency culminating in herniation. In the early phases of intracranial volume expansion—such as from a mass lesion, hemorrhage, or progressive edema—compensatory mechanisms initially preserve pressure homeostasis. CSF is displaced from the cranial subarachnoid space into the spinal compartment, and venous blood is shunted out of the cranial vault via the dural venous sinuses.[3] These adjustments constitute the first-line buffering capacity because CSF and venous blood volumes are comparatively more compressible and more readily redistributed than brain tissue or arterial inflow. However, this compensatory reserve is finite. As the lesion enlarges or edema increases, the ability to further displace CSF and venous blood diminishes. Once these mechanisms are exhausted, incremental increases in intracranial volume produce disproportionately large rises in intracranial pressure—reflecting the steep portion of the intracranial compliance curve—and cerebral perfusion becomes increasingly threatened. As intracranial pressure continues to rise, subsequent pathophysiologic consequences include reductions in arterial blood flow and volume, impaired autoregulation, and a progressive fall in cerebral perfusion pressure. These events promote ischemia

and worsening cytotoxic edema, creating a self-amplifying cycle that further elevates intracranial pressure.[3] Ultimately, when the system can no longer accommodate the pressure burden, brain tissue is forced to shift along paths of least resistance through pre-existing openings in the rigid cranial partitions.[3][4] In uncal herniation, the medial temporal lobe—specifically the uncus—displaces over the tentorial notch. This displacement exerts direct mass effect on the adjacent midbrain and compresses vital structures within the tentorial region [2][3][4]. A hallmark of this syndrome is compression of the ipsilateral third cranial nerve, which courses just medial to the uncus. As the uncus herniates, it pushes against the brainstem and neighboring cranial nerves, with third nerve compression producing early and clinically significant signs related to impaired parasympathetic and somatic oculomotor function. Concurrently, distortion and compression of the brainstem can compromise reticular activating pathways and cardiorespiratory control centers, explaining the rapid progression from focal neurological findings to reduced consciousness and life-threatening instability when uncal herniation advances.[3][4]

History and Physical

Patients with impending uncal herniation typically present initially with nonspecific manifestations of rising intracranial pressure (ICP), reflecting early failure of intracranial compensatory mechanisms before overt brainstem compression becomes clinically evident. The historical features most frequently reported include progressively worsening headache, often described as diffuse and severe, accompanied by nausea and vomiting. Vomiting may be abrupt and can occur without preceding nausea, particularly as ICP rises and brainstem emetic centers are affected. Altered mental status is also common and may range from subtle confusion, irritability, and decreased attention to lethargy and progressive obtundation. Importantly, the tempo of symptom evolution depends on the underlying etiology: rapidly expanding hematomas may produce precipitous decline, whereas tumors or edema may generate a more gradual deterioration until a critical threshold is reached. Regardless of cause, any combination of headache, vomiting, and altered mentation in an at-risk context should heighten suspicion for increased ICP and potential herniation physiology. On physical examination, signs of increased ICP may be systemic, neurologic, and ocular. A classic systemic indicator of significantly elevated ICP is Cushing's triad, characterized by hypertension, bradycardia, and irregular respirations or apnea.[1] This triad reflects brainstem-mediated autonomic dysregulation and is generally a late and ominous sign, suggesting that intracranial hypertension is severe enough to compromise brainstem function. Respiratory irregularity may manifest as periods of hypoventilation, abnormal breathing patterns, or frank apnea, and should be interpreted as an urgent warning of impending decompensation. Neurologic examination must therefore be performed serially, as trends—particularly a declining level of consciousness—often provide the most sensitive indication that intracranial dynamics are worsening [2][3][4][5].

Ophthalmologic findings can provide critical supportive evidence. Papilledema is a recognized late sign of increased ICP, typically reflecting sustained elevation rather than hyperacute rises. It is identified by blurring of the optic disc margins, venous congestion, and diminished or absent venous pulsations on funduscopic examination. Although papilledema may not be present in rapidly evolving herniation, when it is observed it strongly suggests intracranial hypertension and warrants urgent evaluation. In addition to fundus findings, careful assessment of pupils and extraocular movements is essential because uncal herniation produces characteristic ocular signs through compression of the oculomotor nerve (cranial nerve III) as the uncus displaces through the tentorial notch. The cardinal clinical syndrome of uncal herniation is classically described as an acute decline in consciousness accompanied by ipsilateral pupillary dilation and contralateral hemiparesis.[1] These findings reflect simultaneous compromise of key neuroanatomical pathways: compression or displacement of ascending arousal systems within the midbrain contributes to loss of consciousness; direct pressure on the ipsilateral oculomotor nerve produces pupillary abnormalities; and distortion of the corticospinal tract leads to motor weakness on the contralateral side.[1] Clinically, the hallmark feature often emphasized is a unilateral dilated pupil (anisocoria) that is poorly reactive to light. Notably, this pupillary change may appear early and, in some patients, can precede profound impairment of consciousness or the development of contralateral motor deficits. Therefore, isolated anisocoria in an appropriate clinical context—particularly when accompanied by headache, vomiting, or subtle mental status changes—should be treated as a potential sign of impending uncal herniation rather than a benign incidental finding.[5] As herniation progresses, worsening third nerve dysfunction produces additional ocular motor abnormalities. The affected eye may develop impaired adduction, elevation, and depression, resulting in the classic "down-and-out" position caused by unopposed lateral rectus and superior oblique function. Over time, progressive midbrain compression can precipitate further decline in arousal, evolving from lethargy to stupor and coma, and can culminate in death if urgent intervention does not reverse the underlying pressure gradient. The physical examination may also evolve to show abnormal posturing, worsening respiratory patterns, and bilateral pupillary abnormalities as the

syndrome advances. Without timely management, uncal herniation can transition into more diffuse patterns of brain displacement, including progression toward central herniation, reflecting widespread failure of intracranial compliance and escalating brainstem compromise.[1]

Evaluation

The evaluation of suspected uncal herniation is fundamentally time-critical and should be organized around two parallel priorities: rapid clinical recognition based on characteristic examination findings and urgent confirmation of the underlying intracranial cause through neuroimaging. In practice, the diagnosis is initially suspected at the bedside, where evolving neurological deficits provide the earliest and most actionable indicators of impending herniation physiology. The clinician should pay particular attention to declining level of consciousness, new-onset anisocoria or a fixed dilated pupil, progressive focal motor deficits, and signs of brainstem involvement. Systemic manifestations of severe intracranial hypertension—most notably Cushing's triad of hypertension, bradycardia, and irregular respirations or apnea—are especially concerning and imply advanced derangement of intracranial compliance with imminent risk of catastrophic deterioration.[1] Because these findings may evolve quickly, evaluation should include serial neurologic examinations and continuous monitoring of vital signs to detect dynamic change [1]. While bedside assessment establishes clinical urgency, definitive evaluation requires imaging to identify the precipitating lesion and to guide emergent management. Brain imaging serves several essential purposes: it confirms whether there is mass effect and midline shift; it reveals the presence, location, and size of hemorrhage, tumor, edema, or other space-occupying pathology; and it provides anatomical detail relevant to neurosurgical decision-making. Any patient who presents Cushing's triad warrants immediate computed tomography (CT) imaging to exclude life-threatening intracranial hemorrhage, mass lesions, or established herniation.[6] In this context, imaging is not elective or confirmatory in a routine sense; it is a determinant of immediate intervention pathways, such as emergent neurosurgical consultation, decompressive surgery, or targeted reversal of anticoagulation when hemorrhage is present [1].

In the emergent setting, a non-contrast cranial CT scan is generally the preferred first-line imaging modality. CT is favored because it can be obtained rapidly, is widely available in acute care environments, and is highly effective for detecting acute intracranial hemorrhage, substantial mass effect, hydrocephalus, and signs of herniation.[1] This speed is clinically decisive, as delays in diagnosis and source control—particularly for rapidly expanding epidural or subdural hematomas—can translate into irreversible brainstem injury. By contrast, magnetic resonance imaging (MRI), although superior for certain soft-tissue characterizations and subtle parenchymal abnormalities, is less practical for immediate assessment in unstable patients because it typically requires longer acquisition times, may be less accessible after hours, and can be contraindicated or logistically difficult in patients requiring intensive monitoring or life-support equipment. For these reasons, CT remains the imaging backbone for acute evaluation when uncal herniation is suspected. Ultimately, the evaluation process should be viewed as an integrated emergency workflow: prompt recognition of high-risk clinical patterns triggers immediate CT-based confirmation, which in turn enables definitive treatment planning and rapid escalation of care.[1][6]

Treatment / Management

When clinical signs of uncal herniation emerge, management must proceed as an immediate neurological emergency aimed at rapidly lowering intracranial pressure (ICP) and reversing the compartmental pressure gradient that is driving tissue displacement. Conceptually, the clinician's goal is to restore intracranial compliance by reducing the volume of one or more intracranial components—brain tissue, blood, or cerebrospinal fluid—consistent with the Monro–Kellie framework.[4] Because neurological deterioration can accelerate within minutes, effective care requires simultaneous stabilization of airway, ventilation, and circulation while initiating targeted ICP-lowering interventions and mobilizing urgent neurosurgical support. In practice, a "brain code" approach is often adopted in neurocritical care: a structured, stepwise algorithm that prioritizes immediate, high-yield actions while preparing for definitive source control of the underlying lesion. Early noninvasive measures are directed at optimizing venous outflow and minimizing secondary increases in intracranial volume. Elevating the head of the bed to approximately 30 degrees and maintaining the head in a midline position reduce jugular venous obstruction and improve cerebral venous drainage, thereby lowering ICP without sacrificing tissue integrity.[1] These measures are foundational because they can be implemented instantly, do not require specialized equipment, and can be maintained throughout resuscitation and transport. In parallel, clinicians should avoid iatrogenic contributors to intracranial hypertension such as hypoxemia, hypercapnia, agitation, and pain, each of which can increase cerebral blood flow or raise intrathoracic pressure and impede venous return. Although not always listed in brief summaries, adequate analgesia and sedation are frequently necessary in the critically ill patient to prevent coughing, ventilator

dyssynchrony, and sympathetic surges that worsen ICP, while also enabling controlled ventilation and safe imaging [1][2][3][4].

Hyperventilation is an important temporizing intervention specifically reserved for life-threatening intracranial hypertension and suspected herniation physiology. Its mechanism is physiologically direct: by decreasing arterial carbon dioxide tension, cerebral vasoconstriction is induced, reducing cerebral blood volume and thereby lowering ICP.[3][7]3 Clinically, this is achieved by increasing minute ventilation (via tidal volume or respiratory rate) to a target $PaCO_2$ of approximately 30–35 mmHg.[3][7][3] However, the same vasoconstrictive effect that reduces ICP can also decrease cerebral blood flow and risk ischemia if used indiscriminately or for prolonged periods. For this reason, major guideline frameworks emphasize that hyperventilation should be used as a short-term bridge in cases of acute neurological deterioration or herniation and should be avoided as routine "prophylaxis," particularly early after traumatic brain injury when cerebral blood flow may already be compromised. In the context of uncal herniation, hyperventilation is therefore best understood as a rapid, reversible maneuver intended to buy time while definitive therapies—hyperosmolar agents and surgical decompression—are initiated. Hyperosmolar therapy constitutes a cornerstone of emergent ICP reduction in suspected herniation and in sustained intracranial hypertension, commonly operationalized at ICP values exceeding 20 mmHg or when clinical signs of brain herniation are present.[1][8][1] Hyperosmolar agents lower ICP primarily by increasing serum osmolality, promoting osmotic movement of water out of edematous brain tissue, and in some cases improving rheology and microcirculatory flow. Mannitol, typically administered as a bolus dose of 0.5–1 g/kg, remains widely used for ICP crises and can be repeated at intervals (for example, every four hours as needed) when intracranial pressures remain above threshold, while monitoring osmolar parameters and renal function.[1][8][1] Many clinical protocols emphasize limiting excessive osmotic load—often operationalized by monitoring serum osmolality or the osmolar gap—to reduce the risk of adverse effects such as hypovolemia, electrolyte derangements, and kidney injury; the text provided specifies maintaining the osmolar gap less than 20 mOsm.[1][8][1] Contemporary critical care reviews similarly describe 0.5–1 g/kg as an initial emergency dose range for mannitol in ICP crises [3][4][5][6][7][8].

Hypertonic saline is an alternative (and in some centers preferred) hyperosmolar strategy, particularly when hemodynamic support is desirable because hypertonic saline expands intravascular volume while raising serum osmolality. The provided regimen describes 3% hypertonic saline boluses of 5–10 mL/kg for acute ICP elevations or signs of herniation, with the option of continuous infusion at 0.5–1.5 mL/kg/hr.[1][8][1] As with mannitol, safe administration requires close monitoring—especially of serum sodium, osmolality, acid–base status, and fluid balance—because hypertonic saline can precipitate complications such as hypernatremia, hyperchloremic acidosis (depending on formulation), and cardiopulmonary stress in susceptible patients. It is also important to recognize that dosing strategies for hypertonic saline vary across institutions (bolus-based, infusion-based, or combined) due to differences in protocols and available concentrations; thus, therapy should follow local critical care guidance while maintaining the core objective of emergent ICP reduction in herniation physiology. If clinical signs of uncal herniation fail to resolve promptly despite optimized positioning, controlled ventilation, and hyperosmolar therapy—or if imaging reveals a surgically remediable mass effect—definitive decompressive interventions must be pursued without delay.[1] At this stage, management shifts from primarily physiologic manipulation of intracranial components toward direct reduction of intracranial volume and relief of focal mass effect. Placement of an external ventricular drain (EVD) provides a mechanism for cerebrospinal fluid diversion and both monitors and treats elevated ICP when ventricular access is feasible. CSF drainage can rapidly lower ICP and may be particularly useful when hydrocephalus contributes to intracranial hypertension; however, it must be performed under appropriate neurosurgical oversight and with awareness that improper CSF removal in the presence of obstructive processes can worsen pressure gradients [1][8].

Surgical evacuation of extra-axial lesions—such as an epidural hematoma—can be life-saving because these lesions often exert substantial mass effect that is mechanically reversible once the clot is removed.[1] Similarly, resection or debulking of an intracerebral lesion may be indicated when a tumor, abscess, or hemorrhagic focus is generating the supratentorial driving pressure that displaces the uncus through the tentorial notch. In extreme and refractory cases, decompressive craniectomy (unilateral or bilateral, depending on laterality and global swelling patterns) may be required to create space for edematous brain expansion and to reduce ICP when medical therapy is insufficient. The list provided also includes "removal of brain parenchyma," which reflects rare salvage procedures (for example, lobectomy in selected circumstances) undertaken in dire situations when focal swelling or necrosis cannot otherwise be controlled; such interventions are highly specialized, carry substantial neurologic risk, and are considered only by neurosurgical teams within a comprehensive critical care setting. Across all phases of treatment, the overarching management principle is disciplined escalation: initiate rapid, low-risk interventions

immediately; use hyperventilation as a short-term bridge in life-threatening herniation; administer hyperosmolar therapy promptly when ICP is critically elevated or herniation is suspected; and move decisively to neurosurgical decompression when clinical trajectory or imaging indicates that physiologic measures alone are inadequate.[1][4] Because uncal herniation is imminently fatal without prompt reversal, these steps are implemented emergently in monitored settings and should be understood as hospital-based critical care management rather than outpatient guidance [1][4][8].

Differential Diagnosis

Because uncal herniation represents the terminal manifestation of critically elevated intracranial pressure (ICP), its clinical presentation—typically including headache, nausea, vomiting, altered mental status, and focal neurological deficits—overlaps with many other serious neurological and systemic disorders. As a result, the differential diagnosis should include any condition capable of producing intracranial pathology, metabolic encephalopathy, or infection that mimics increased ICP. Differentiating these entities from true herniation is crucial, as management priorities vary widely: some require metabolic correction, others antimicrobial therapy, and some immediate surgical intervention. Several intracranial conditions can present with signs and symptoms similar to uncal herniation. Intracranial hemorrhage—including intraparenchymal, subarachnoid, subdural, and epidural bleeds—can all elevate ICP and produce focal deficits, headache, and vomiting. Subdural or epidural hematomas, in particular, are important mimics and precipitants of herniation; they can produce rapid deterioration and unilateral pupillary dilation similar to uncal herniation. Ischemic stroke involving large arterial territories, especially the middle cerebral artery, may also lead to massive cerebral edema and subsequent herniation, though early symptoms can initially resemble transient ischemic attacks or focal deficits without raised ICP. Neoplasms—both primary and metastatic—can cause progressive intracranial hypertension through mass effect, vasogenic edema, or obstructed CSF pathways, mimicking early uncal herniation in their later stages. Traumatic brain injury (TBI) and post-concussive syndromes can lead to diffuse cerebral swelling and increased ICP, especially in patients with concurrent hemorrhages or contusions [1][4][5][6][8].

Meningitis and encephalitis often present with headache, fever, altered consciousness, and vomiting, all of which overlap with early symptoms of intracranial hypertension. These conditions, however, typically demonstrate systemic inflammatory features and cerebrospinal fluid abnormalities on lumbar puncture (when safe to perform). Systemic lupus erythematosus (SLE) and other autoimmune vasculitides can also produce neurological manifestations—such as seizures, psychosis, or headache—through mechanisms like vasculopathy or cerebral edema, occasionally resembling uncal herniation if complicated by intracranial inflammation or hemorrhage. Metabolic disorders constitute another broad category of differential diagnoses. Diabetic ketoacidosis (DKA) can cause profound dehydration, acidosis, and altered mental status, potentially leading to cerebral edema in severe or pediatric cases. While papilledema and headache may develop, the underlying mechanism differs fundamentally from the mechanical displacement seen in uncal herniation. Sepsis, through systemic inflammatory response and associated encephalopathy, may present confusion, hypotension, and tachypnea, which could mimic the altered mentation and abnormal vital signs seen in raised ICP but lack the focal neurological findings typical of herniation. Similarly, toxin exposure (e.g., carbon monoxide, alcohols, sedative-hypnotics, or heavy metals) can produce altered consciousness, nausea, or vomiting but usually lacks focal asymmetry or specific cranial nerve deficits unless accompanied by secondary cerebral injury [8].

Distinguishing uncal herniation from these differential diagnoses requires careful clinical correlation and rapid imaging. The hallmark triad of uncal herniation—acute loss of consciousness, ipsilateral pupillary dilation, and contralateral hemiparesis—reflects direct mechanical compression of the oculomotor nerve and corticospinal tract, findings rarely replicated in metabolic or infectious conditions. Likewise, systemic conditions such as DKA or sepsis produce generalized rather than lateralizing neurological signs. Neuroimaging, particularly computed tomography (CT), remains decisive in establishing diagnosis, revealing characteristic medial temporal lobe displacement and midline shift in uncal herniation, as opposed to the diffuse cortical swelling or metabolic changes seen in systemic causes. In summary, while a wide array of neurological, infectious, metabolic, and toxic conditions can present with headache, vomiting, and altered mental status, the focal, rapidly progressive, and often unilateral neurological findings—particularly pupillary changes—should immediately raise suspicion for uncal herniation. Timely differentiation through clinical acumen and emergent imaging is essential to direct appropriate and potentially life-saving management [8][9].

Prognosis

The prognosis of patients with uncal herniation is primarily determined by the speed of recognition, the immediacy and effectiveness of intracranial pressure–lowering interventions, and the reversibility of the underlying cause. In practical terms, uncal herniation is not a diagnosis that can be

managed expectantly; rather, outcome is tightly coupled to time, because progressive compression of the midbrain and adjacent neurovascular structures can rapidly become irreversible. Early diagnosis—based on vigilant neurological assessment and prompt imaging—remains one of the most decisive contributors to improved neurosurgical outcomes, as timely escalation to medical and operative decompression can prevent secondary ischemic injury and halt further displacement of intracranial contents.[5] Even when definitive treatment is available, delay increases the likelihood that brainstem dysfunction will become fixed, thereby limiting the potential for meaningful neurological recovery. The degree and duration of herniation materially influence clinical trajectory. Smaller or earlier-stage herniations may be more amenable to reversal, especially if the precipitating lesion is rapidly controlled and cerebral perfusion is restored. Conversely, advanced herniation states—characterized by profound impairment of consciousness, fixed pupillary abnormalities, and progressive posturing—are often associated with worse outcomes because they reflect more extensive brainstem compromise and greater risk of vascular compression, infarction, and hemorrhagic injury. Prognosis may be further worsened when patients present with multiple traumatic injuries or when additional complications emerge during resuscitation, such as hypoxemia, hypotension, coagulopathy, or evolving intracranial hemorrhage. As herniation increases, the physiological "window" for reversibility narrows, and the capacity to restore normal intracranial compartment relationships diminishes, making recovery increasingly difficult even with aggressive treatment. Despite these concerns, uncal herniation is not universally fatal if addressed promptly. Reversal can occur when interventions are initiated rapidly and when the underlying pathology is amenable to decompression or evacuation.[9] Reported data indicate that reversal of uncal herniation occurs in approximately 50–75% of adult patients with either traumatic brain injury or supratentorial mass lesions, emphasizing that timely and coordinated management can yield substantial salvage rates even in high-acuity presentations.[1] Long-term neurological outcomes after successful reversal may be comparatively more favorable in children than in adults, potentially reflecting differences in neuroplasticity, injury patterns, and comorbidity burden.[1] However, even when herniation is reversed, prognosis is not solely determined by survival; cognitive, behavioral, motor, and visual sequelae may persist depending on the extent of cortical and brainstem injury sustained during the herniation event. Accordingly, prognostication requires an integrated view of initial neurological status, imaging findings, response to therapy, and the broader systemic context of the patient's illness or trauma, with sustained reassessment as the acute phase evolves [1][5][9].

Complications

The complications of uncal herniation are dominated by the severe consequences of uncontrolled intracranial hypertension and progressive brainstem compression. The most catastrophic outcomes are coma and death, which occur when management strategies fail to reverse the herniation syndrome and when brainstem function deteriorates beyond recoverability. This progression is clinically understandable: the midbrain and adjacent structures within the tentorial notch are essential for consciousness, pupillary responses, and cardiorespiratory regulation, and sustained compression can disrupt these systems in a manner that becomes rapidly irreversible. Even when initial stabilization is achieved, patients may deteriorate quickly if the underlying lesion continues to expand or if secondary insults—such as hypoxia, hypercapnia, hypotension, or worsening edema—exacerbate intracranial pressure and compromise cerebral perfusion. Beyond the immediate risk of death, uncal herniation carries a high likelihood of ischemic injury due to the anatomical proximity of major arteries and the vulnerability of the brainstem and posterior circulation territories. Depending on the magnitude and direction of tissue displacement, vascular structures may be compressed or distorted, increasing the risk of brainstem or cortical ischemia. Such ischemia can produce persistent neurological deficits even after successful decompression, including impaired arousal, cranial nerve dysfunction, hemiparesis, and cognitive impairment. In addition, the mechanical effects of herniation can trigger secondary hemorrhage or infarction, and the resulting lesions may contribute to long-term disability. The clinical burden is further increased by complications related to the intensive interventions often required to reverse herniation, including risks associated with hyperosmolar therapy, mechanical ventilation, invasive monitoring, and emergent neurosurgical procedures. While these iatrogenic risks are not unique to uncal herniation, their probability increases in the context of high-acuity resuscitation and prolonged neurocritical care. A small but notable subset of complications relates to uncommon chronic forms of uncal herniation. Rare cases have been described—predominantly in pediatric populations—in which chronic uncal herniation develops in association with congenital or structural posterior fossa abnormalities, such as Dandy-Walker syndrome, or other cystic cavities that communicate with the fourth ventricle, sometimes influenced by posterior fossa shunting.[2][10] In such contexts, the pathophysiology may differ from the classic acute mass-effect scenario, and clinical presentation may be more indolent, potentially complicating recognition. These rare cases reinforce an important clinical principle: while acute uncal

herniation is typically driven by sudden or progressive intracranial hypertension, anatomic alterations and CSF dynamics can, in exceptional circumstances, produce more chronic displacement syndromes with distinct diagnostic and management challenges. Overall, the complication profile of uncal herniation underscores the necessity of immediate, decisive management in acute cases and sustained vigilance for secondary injury patterns and treatment-related adverse events throughout the neurocritical course [2][10].

Consultations

Effective management of suspected or evolving uncal herniation requires early consultation and coordinated care from a multidisciplinary team experienced in neuroemergencies. Because herniation syndromes can progress rapidly, consultation should not be delayed until imaging confirmation if clinical findings strongly suggest impending herniation; rather, emergent specialist involvement should occur in parallel with stabilization, imaging acquisition, and initiation of intracranial pressure–lowering measures. Patients presenting with traumatic brain injury are a particularly high-risk population and should be monitored by personnel who are trained to detect early warning signs, such as evolving anisocoria, declining level of consciousness, or emerging focal neurological deficits. The core consultative services typically include neurosurgery and neurocritical care, supported by emergency medicine or trauma teams, radiology, and anesthesiology, depending on the clinical context and the anticipated need for operative intervention. Neurosurgeons are central to the pathway because definitive treatment often involves decompressive procedures, evacuation of mass lesions, placement of ventricular drains, and operative strategies tailored to the lesion driving intracranial hypertension. Early neurosurgical input can expedite decision-making regarding the urgency of surgery, the appropriateness of temporizing measures, and the selection of interventions that best match the patient's anatomy and pathology. Neuro-intensivists (or intensivists with neurocritical expertise) contribute continuous physiologic management, including ventilation targets, hemodynamic optimization to preserve cerebral perfusion pressure, sedation strategies, and monitoring protocols for intracranial pressure and neurological function. Their role is particularly important because herniation physiology is not solely an anatomic problem; it is a dynamic physiologic crisis in which systemic variables can tip the balance toward recovery or irreversible injury. Radiology consultation—especially neuroradiology when available—supports rapid interpretation of emergent neuroimaging, identification of subtle mass effect or early herniation signs, and clarification of lesion characteristics that influence surgical planning. Likewise, anesthesia and airway specialists may be needed when intubation, controlled hyperventilation, or operative transport is required, as airway management and ventilatory control are often inseparable from intracranial pressure management. In trauma settings, additional consultation with orthopedic surgery, general surgery, and interventional radiology may be required to address coexisting injuries and hemorrhage control, because systemic instability can worsen cerebral perfusion and compound neurological outcomes. Support staff—particularly nurses with neurocritical training and respiratory therapists—are also essential members of the consultative ecosystem, not merely ancillary personnel. Their continuous bedside presence allows early detection of subtle changes that precede catastrophic deterioration, such as changes in pupillary reactivity, respiratory pattern, agitation, or rising ventilatory pressures. In sum, consultations for uncal herniation should be viewed as an integrated, time-sensitive network: early neurosurgical and neurocritical involvement, rapid imaging interpretation, and coordinated operational readiness for procedures and escalation of care. This team-based structure is crucial because the margin for error is small and the tempo of clinical change is often faster than what any single discipline can safely manage in isolation [10].

Patient Education

Deterrence and prevention in the context of uncal herniation are best understood through the epidemiology of its most common precipitating conditions. Because uncal herniation frequently arises from severe intracranial hypertension caused by traumatic brain injury, hemorrhage, or mass lesions, primary prevention efforts are most effective when directed toward reducing preventable brain trauma and promoting early recognition of neurological emergencies. Public health measures that improve roadway safety, reduce high-risk behaviors, and encourage protective equipment use can meaningfully decrease the incidence of traumatic injuries that may later evolve into herniation syndromes. Appropriate precautions when operating motor vehicles—such as seatbelt use, adherence to speed limits, avoidance of impaired driving, and utilization of child restraints—remain among the most practical deterrence strategies, as motor vehicle collisions are a major driver of severe head trauma in many settings. Similarly, firearm safety practices, including secure storage, training, and adherence to safety protocols, are emphasized because firearm-related injuries represent another important cause of severe traumatic brain injury. Patient education in clinical settings should focus on recognizing warning signs that require urgent evaluation. Patients and caregivers should be informed that escalating headache, repeated vomiting, confusion, new weakness, visual changes, or abnormal pupil size can indicate serious intracranial pathology, particularly

after head injury. In post-trauma contexts, education should include explicit instructions on when to seek emergency care, emphasizing that neurological deterioration can be delayed and that a previously "stable" patient can worsen as hemorrhage expands or edema evolves. Education is also relevant for patients with known intracranial lesions (for example, tumors) who may be at risk of acute decompensation; they should understand that sudden changes in consciousness, focal neurological deficits, or severe headache represent emergency symptoms rather than routine disease fluctuations [8][9][10].

At the same time, it is important to acknowledge the limits of prevention. Many medical causes of space-occupying intracranial lesions—such as neoplasms, spontaneous intracerebral hemorrhage related to vascular pathology, or inflammatory edema—are difficult to prevent directly and may not be predictable prior to symptom onset. In these situations, secondary prevention becomes the realistic goal: encouraging timely medical evaluation, adherence to surveillance plans for known lesions, management of vascular risk factors when applicable, and rapid response to emergent symptoms. Patients with hypertension, anticoagulant use, or vascular malformations may benefit from counseling on risk modification and the importance of early assessment after neurological symptoms, because these factors can increase the likelihood or severity of intracranial bleeding. Ultimately, deterrence and education should be framed around two principles: reducing preventable head trauma and empowering patients and families to recognize urgent neurological warning signs. While not all etiologies are preventable, earlier presentation and faster access to definitive care can shorten the duration of intracranial hypertension, improve the probability of reversal, and reduce the likelihood of permanent neurological injury [10].

Other Issues

Uncal herniation is best conceptualized as a mechanical and physiologic emergency arising when an intracranial lesion overwhelms the compensatory capacity of the cranial vault. The intracranial compartment contains brain tissue, blood, and cerebrospinal fluid, and uncal herniation occurs when the ability of these components to compensate for rising pressure is exceeded, resulting in pathologic tissue shift through fixed dural openings. This framework is clinically useful because it highlights why seemingly different diseases—such as traumatic hematoma, tumor-related edema, or spontaneous hemorrhage—can converge on the same life-threatening endpoint when intracranial compliance is lost. A practical implication is that clinicians should treat rapidly evolving neurological deterioration as a compartment problem until proven otherwise, initiating immediate stabilization while pursuing definitive diagnosis. Several clinical pearls help distinguish uncal herniation from other causes of altered mental status. The cardinal signs are an acute loss of consciousness associated with ipsilateral pupillary dilation and contralateral hemiparesis, reflecting compression of midbrain arousal pathways, the oculomotor nerve, and corticospinal tracts. However, a key nuance is that the unilateral dilated pupil may appear early, sometimes before profound impairment in consciousness or the emergence of motor deficits. For this reason, anisocoria is inherently concerning in an appropriate clinical context, and its cause should be investigated urgently rather than dismissed as benign variation. Because time-to-intervention is prognostically decisive, clinicians should resist "watchful waiting" when anisocoria is new or unexplained in a patient with headache, vomiting, trauma history, or declining mentation [10].

Imaging choice is another crucial point. In the emergent setting, computed tomography (CT) is generally preferred over magnetic resonance imaging (MRI) because CT can be performed quickly and is more widely available in acute care environments. This is not merely a logistical preference: rapid identification of hemorrhage, mass effect, and midline shift can immediately change management by prompting surgical evacuation or decompression. MRI may provide greater soft-tissue detail in select scenarios, but its longer acquisition times and limited accessibility can delay critical decisions when minutes matter. A further high-yield concept is that uncal herniation can be reversible if recognized early and treated aggressively. This reversibility underscores the importance of rapid escalation through evidence-based intracranial pressure–lowering measures and timely neurosurgical involvement. When reversal occurs, the trajectory may shift from imminent fatality to meaningful recovery, making early recognition of the central clinical skill. Finally, clinicians should appreciate that uncal herniation is not an isolated event but can progress to more global displacement syndromes, including central herniation, if intracranial hypertension remains uncontrolled. Therefore, ongoing reassessment is mandatory even after initial improvement, and care plans should anticipate recurrence risk, evolving edema, and secondary injuries that can re-trigger dangerous pressure gradients [10][11].

Enhancing Healthcare Team Outcomes

Neurological resuscitation in uncal herniation is inherently interdisciplinary and depends on synchronized performance across multiple roles, often under severe time pressure. The most effective teams function with a shared mental model: they recognize that herniation physiology can deteriorate within minutes, that clinical signs may evolve rapidly, and that immediate actions—airway control, ventilation targets, hemodynamic stabilization, and hyperosmolar therapy—must occur in parallel with diagnostic imaging and neurosurgical mobilization.

In this setting, outcomes are not solely driven by the competence of a single physician or surgeon but by the team's capacity to communicate clearly, execute protocols reliably, and detect subtle trends before they become irreversible. Nurses are central to this process because they are the continuous patient monitors and often the first to recognize incremental but meaningful changes. Subtle deterioration in mental status, a new pupillary asymmetry, a change in motor response, or escalating blood pressure with falling heart rate may each represent early warning signs of worsening intracranial dynamics. When nurses are empowered to report these observations immediately—without hierarchical barriers—teams can initiate treatment earlier, obtain imaging faster, and shorten time to definitive decompression. This empowerment is not merely cultural; it is operationally lifesaving, because delays in recognizing neurological decline can be as harmful as delays in intervention. Respiratory therapists play an equally critical role because ventilation and airway mechanics directly influence intracranial pressure. Hypercapnia causes cerebral vasodilation and increases cerebral blood volume, raising intracranial pressure, while hypoxemia can worsen cerebral edema and secondary injury. Respiratory therapists, like nurses, should be keenly attuned to subtle changes in respiratory status, including rising airway pressures, deteriorating pulmonary compliance, or ventilator dyssynchrony, because these changes can increase intrathoracic pressure, impair venous return from the brain, raise venous pressure, and elevate PCO_2—each of which can worsen intracranial hypertension. Their expertise is vital for achieving precise ventilation targets when hyperventilation is used as a temporizing measure and for maintaining stable oxygenation and carbon dioxide control during transport and procedures. Physicians across disciplines—emergency medicine, trauma surgery, neurocritical care, anesthesiology, neurosurgery, and radiology—must communicate freely and rapidly, using concise language and closed-loop confirmation to avoid errors. Teams that perform best typically establish a predictable cadence for reassessment and planning, discussing the patient's status at least twice during critical phases and updating a shared plan that anticipates the next escalation step. When all members are encouraged to contribute observations and concerns, the team is more likely to detect early deterioration, avoid preventable delays, and align interventions with rapidly changing physiology. In uncal herniation, this coordinated team performance is not an adjunct to treatment; it is a core determinant of whether recognition, imaging, and definitive decompression occur in time to prevent coma, irreversible injury, or death [11].

Conclusion:

Uncal herniation is a catastrophic neurological event that signals the failure of intracranial compensatory mechanisms and imminent risk of death if untreated. Its pathophysiology underscores the importance of the Monro–Kellie principle, as any additional intracranial volume beyond compensatory limits precipitates dangerous pressure gradients and tissue displacement. Clinically, the syndrome progresses rapidly from nonspecific symptoms of raised ICP to hallmark findings of anisocoria, contralateral hemiparesis, and declining consciousness, reflecting direct compression of the midbrain and cranial nerve III. Management must be immediate and multidisciplinary, integrating airway stabilization, controlled hyperventilation, and hyperosmolar therapy as temporizing measures while preparing for definitive neurosurgical decompression. CT imaging remains the cornerstone for rapid diagnosis and surgical planning. Prognosis is tightly linked to time-to-intervention; early recognition and aggressive treatment can reverse herniation in up to three-quarters of cases, whereas delays often result in irreversible brainstem injury, coma, and death. Ultimately, uncal herniation should be approached as a dynamic physiologic crisis rather than a static anatomical problem. Continuous monitoring, team-based coordination, and adherence to evidence-based protocols are essential to optimize outcomes. Prevention through trauma reduction and patient education on warning signs further complements acute care strategies, reinforcing the principle that time is brain in herniation syndromes.

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