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Acute Epidural Hematoma: From Injury to Death

Vafa Rahimi-Movaghar[1]*, Hamid Bahmani[2], Mohsen Hajiqasemi[3]

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Abstract

Background: Acute epidural hematoma (AEDH) is a critical condition commonly resulting from trauma, particularly in young males aged 20 to 30 years. It arises from the accumulation of blood between the dura mater and the skull, leading to increased intracranial pressure (ICP) and various neurological symptoms. Early mechanisms, such as cerebrospinal fluid (CSF) shift and vasoconstriction, temporarily stabilize ICP, but as the hematoma grows, these compensatory processes fail. This failure results in a rapid progression of symptoms, including localized pain, nausea, vomiting, and, in severe cases, loss of consciousness or herniation.

Results: Clinical presentations depend on the compression location, such as the "talk and die" phenomenon in posterior fossa AEDH or motor impairments due to subfalcine herniation. Advanced stages are marked by Cushing's triad and posturing changes, indicating imminent herniation. Prompt recognition and intervention, typically surgical evacuation through craniotomy for indicated cases, are crucial for preventing mortality. Medical management focuses on controlling ICP through positioning and osmotic agents.

Conclusion: Awareness of clinical symptoms and rapid treatment are vital for improving patient outcomes in this potentially fatal disorder.

Keywords: Acute Epidural Hematoma (AEDH), Intracranial Pressure (ICP), Brain Herniation, Management

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Introduction

Acute epidural hematoma (AEDH) refers to the accumulation of blood in the space between the dura mater's external layer and the skull's inner surface, classified as an extra-axial hemorrhage (1). It is observed in about 10% of hospitalized cases of traumatic brain injury (TBI) and can result from either traumatic events or non-traumatic factors (2, 3).

Epidural hematomas are identified in approximately 2% of head injuries and contribute to up to 15% of fatal cases. They are more prevalent in males, particularly among teenagers and young adults, with an average age range of 20 to 30 years. In contrast, these hematomas are rare in individuals aged 50 to 60 and older, as age-related changes make the dura mater more tightly attached to the skull, reducing the likelihood of blood accumulation in this potential space (1, 4).

Clinical presentations of AEDH vary widely depending on the location and size of the hematoma. Symptoms may range from localized pain, nausea, and vomiting to severe neurological deficits, including motor impairments and loss of consciousness. Certain cases, such as posterior fossa

Corresponding author: Dr Vafa Rahimi-Movaghar, v_rahimi@sina.tums.ac.ir

1. Trauma and Surgery Research Center, Sina Hospital, Tehran University of Medical Sciences, Tehran, Iran
2. Students' Scientific Research Center (SSRC), Tehran University of Medical Sciences, Tehran, Iran
3. School of Medicine, Shahid-Beheshti University of Medical Sciences, Tehran, Iran

↑What is "already known" in this topic:
Acute epidural hematoma (AEDH) often affects young individuals after head trauma and can cause brain compression and herniation, leading to life-threatening complications. Elevated intracranial pressure (ICP) and rapid neurological decline necessitate prompt intervention.

→What this article adds:
As hematoma volume increases, ICP rises disproportionately due to autoregulation failure. Clinical signs like nausea, vomiting, and Cushing's triad signal critical stages, while posterior fossa hematomas can be fatal despite initial alertness. This study emphasizes early recognition, the role of ICP monitoring, and timely surgical management to improve patient outcomes.

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AEDH, may present atypically, with patients remaining alert until sudden deterioration. This variation in clinical presentation highlights the importance of clinicians maintaining a heightened level of vigilance, even when symptoms appear mild (1).

Prompt recognition and early intervention are paramount in AEDH management, as delays in diagnosis can lead to rapid progression to brain herniation and death. Advances in neuroimaging and surgical techniques have significantly improved outcomes, but the condition remains a neurological emergency requiring immediate attention (1).

Pathophysiology

According to the Monro-Kellie concept, the cranial vault is made up of three essential components: brain tissue, blood vessels, and cerebrospinal fluid (CSF). To maintain steady intracranial pressure (ICP), an increase in one component's volume must be offset by a reduction in another (5, 6).

When a traumatic brain injury occurs, such as a road traffic accident that causes a temporal bone fracture, the middle meningeal artery can be severed, resulting in an AEDH. Initially, as hematoma volume develops, ICP does not rise much because CSF is displaced from the brain to the spinal canal (compensation phase). The key mechanism for ICP stabilization during this phase is venous volume reduction, which occurs when veins are compressed due to increasing pressure, leading to blood displacement into lower-pressure areas outside the cerebral cavity. This venous contracture helps to keep ICP steady momentarily. Following this, arterial spasms can help to stabilize ICP (6, 7). However, as the hematoma grows, ICP increases linearly with volume. Eventually, autoregulation fails, and even a small volume increase can cause a significant rise in ICP (Figure 1).

The increase in ICP reduces cerebral perfusion pressure (CPP), defined as the difference between mean arterial pressure (MAP) and ICP. When ICP nears or surpasses MAP, cerebral blood flow becomes significantly impaired.

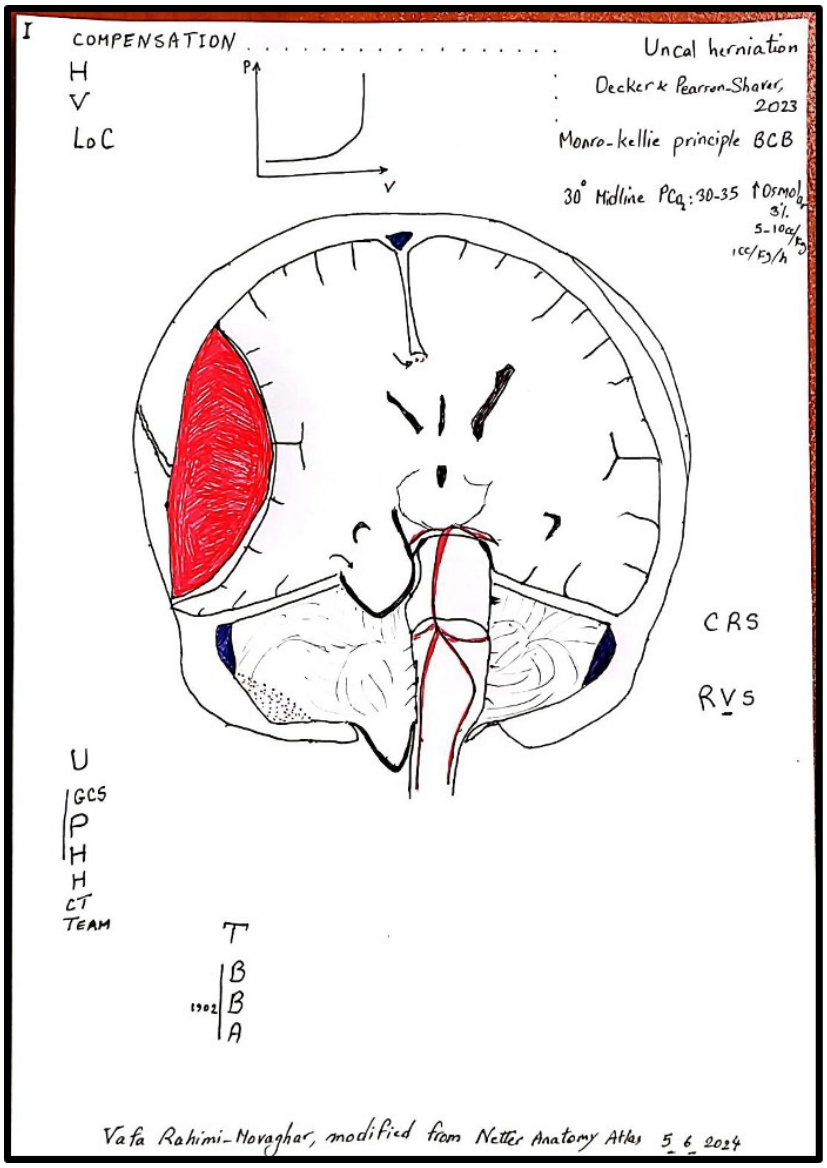

Figure 1. Pathophysiology of hematoma overcoming the autoregulation and causing rise of ICP and brain herniation. [Self-illustrated by Professor Vafa Rahimi-Movaghar inspired by Netter Anatomy Atlas]

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This ischemic cascade exacerbates neuronal damage and increases the likelihood of herniation. Additionally, as the hematoma grows, localized ischemia can develop due to the compression of adjacent brain tissue and blood vessels, further contributing to neurological decline and eventual irreversible damage (8).

Clinical Presentation

The clinical presentations are the result of the dynamic interplay between hematoma expansion, ICP elevation, and regional brain compression, which collectively determine the patient's neurological status. The initial symptom of an AEDH is typically localized pain at the trauma site caused by direct injury to the skull and underlying tissues. As ICP begins to rise, a more diffuse headache emerges, often accompanied by nausea and vomiting. These symptoms are believed to result from the stimulation of the vomiting center located in the reticular formation of the lateral medulla. This stimulation occurs due to the increased pressure transmitted through the brainstem structures (9, 10).

Variations in consciousness levels depend heavily on the location and size of the hematoma. For instance, compression of the diencephalon can lead to a gradual decrease in consciousness as reticular activating system function is impaired. Conversely, hematomas in the posterior fossa may cause direct brainstem compression, leading to life-threatening conditions without significant alterations in consciousness initially, a phenomenon described as the "talk and die" syndrome (1). Additionally, uncal herniation resulting from temporal lobe compression can cause progressive consciousness deterioration due to midbrain involvement, often accompanied by ipsilateral pupil dilation and contralateral hemiparesis.

Advanced Stages and Herniation

Cushing's triad, described by Harvey Williams Cushing in 1901, occurs as a late sign of increased ICP and impending herniation. It includes bradycardia, irregular respirations, and widened pulse pressure (9).

Subfalcine herniation, also called cingulate herniation, is the most frequent form of brain herniation. It is characterized by the inward shift of the cingulate gyrus beneath the falx cerebri, driven by uneven or unilateral pressure in the hemispheres. This often leads to compression of the anterior cerebral artery (ACA), which can cause ischemia and contralateral lower limb weakness. Symptoms may include acute urinary retention and frontal lobe dysfunction, such as loss of initiative and behavioral changes (11).

Uncal herniation takes place when the uncus, the medial portion of the temporal lobe, shifts downward and inward beneath the tentorial edge. This results in compression of the ipsilateral oculomotor nerve, causing pupil dilation and ptosis. In advanced stages, the midbrain may be compressed against the tentorial edge, leading to contralateral hemiparesis (Kernohan's notch phenomenon) and, eventually, bilateral posturing (decerebrate or decorticate) as the brainstem becomes involved (11).

A central herniation is characterized by the downward movement of the thalamus, midbrain, and pons through the opening of the tentorial notch. Early signs include small but reactive pupils, irregular breathing, and behavioral changes. Progression leads to fixed mid-position pupils, loss of oculocephalic reflexes, and decerebrate posturing due to brainstem ischemia. Radiographic findings may include effacement of basal cisterns and Duret hemorrhages, which are slit-like midbrain hemorrhages caused by stretching of perforating vessels (111, 12).

Tonsillar herniation occurs when the cerebellar tonsils are displaced downward through the foramen magnum, exerting pressure on the medulla. This leads to cardiorespiratory instability, pinpoint pupils, and eventual respiratory arrest. It may occur in isolation or as a result of downward transtentorial herniation. Obstructive hydrocephalus caused by fourth ventricle compression may further exacerbate the condition (11).

An upward herniation is less common and occurs when a posterior fossa lesion forces the cerebellar vermis and hemispheres upward through the tentorial notch. This can compress the midbrain and superior cerebellar artery, leading to ischemia in cerebellar and midbrain structures. Clinical signs include vertical gaze palsy and hydrocephalus due to aqueductal compression (11).

Stages of Central Herniation

Early diencephalic stage (reversible): This stage involves dysfunction of the ascending arousal system in the diencephalon, primarily caused by compression of small penetrating vessels, particularly those supplying the thalamus and hypothalamus. Clinical signs include agitation, drowsiness, small but reactive pupils, and preserved oculocephalic reflexes. Breathing abnormalities such as deep sighs or yawns may be noted (11, 12). Management at this stage is targeted at reducing ICP and preserving CPP, hyperosmolar therapy (e.g., mannitol or hypertonic saline), head elevation, and CSF drainage via ventriculostomy could be done. ICP monitoring can also guide therapeutic adjustments (12).

Late diencephalic stage: With further compression of thalamic and hypothalamic structures, ischemia and necrosis develop, leading to deeper coma. Pupils may become irregular, and decorticate posturing arises due to loss of inhibition of the red nucleus. This stage marks the transition to irreversible damage if untreated (11, 12). Surgical interventions, such as decompressive craniectomy, are often necessary to reverse mass effects and prevent further progression at this stage. High-dose barbiturates may be considered for refractory ICP management (12).

Midbrain-upper pons stage: Increased pressure on the midbrain causes vascular compromise, including Duret hemorrhages, which are visible as slit-like hemorrhages on imaging. Pupils become fixed at mid-position, and decerebrate posturing replaces the decorticate posturing as the rubrospinal tract becomes dysfunctional. Hyperventilation and loss of oculocephalic reflexes may also occur (11, 12).

Lower pons-medullary stage: This terminal stage involves compression of the brainstem's cardiorespiratory centers, leading to ataxic respirations, fixed mid-position pupils, and generalized flaccidity. Without intervention, this results in death. If the previous interventions cannot restore viable brain function, The management for the last

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two stages is often palliative measures (11, 12).

Compression and infarction of the posterior cerebral artery can cause occipital lobe infarction, leading to homonymous hemianopia, diagnosable via CT or MRI, especially in the recovery phase if it occurs.

Management

The management of AEDH is guided by the patient's neurological status, imaging findings, and evidence-based guidelines. Treatment approaches include non-surgical and surgical interventions, along with ICP monitoring when indicated.

Non-surgical treatment: Suitable for specific AEDH cases where the hematoma volume is under 30 cm^3, thickness does not exceed 15 mm, midline shift is below 5 mm, and the Glasgow Coma Scale (GCS) score is greater than eight without focal neurological impairments (13). Key components include:

Close neurological observation: Serial neurological assessments and repeat CT scans are essential to monitor for hematoma progression.

Medical management of ICP: Hyperosmolar therapy, such as mannitol or hypertonic saline, is used to control ICP in patients with mild intracranial hypertension. Prophylactic hyperventilation is avoided to prevent ischemia.

Supportive care: Maintaining normovolemia, preventing hypoxia, and controlling blood pressure are critical to prevent secondary brain injury (12, 14).

Surgical treatment: Indicated if the hematoma size is greater than 30 cm^3 regardless of GCS score, AEDH with thickness greater than 15 mm or more than 5 mm midline shift even in stable patients, patients with GCS less than eight and anisocoria, and progressive decline in GCS, anisocoria, or new focal deficits.

Surgical options include craniotomy, which is preferred for a complete evacuation of the hematoma, and Burr Hole evacuation, which is used in emergencies where rapid intervention is needed, and craniotomy is not immediately available. The timing of surgery is also important, and rapid evacuation (< 2 hours of neurological deterioration) is critical to improving outcomes (12, 13).

Role of ICP monitoring: ICP monitoring is an adjunctive tool for patients with severe TBI, especially those with GCS ≤ 8, abnormal CT findings, or evidence of raised ICP. It helps guide therapeutic decision-making (ICP thresholds > 20 mmHg warrant aggressive intervention), outcome prediction (persistent ICP elevation is associated with poor outcomes), and ICP control (Allows titration of medical or surgical therapies to maintain CPP > 60 mmHg) (8, 14).

Conclusion

AEDH remains a critical neurosurgical emergency that requires timely diagnosis and management to prevent severe morbidity and mortality. Advances in imaging, surgical techniques, and intensive care have improved outcomes, yet challenges persist in ensuring rapid intervention, especially in resource-limited settings.

Future research should focus on the development of minimally invasive surgical techniques, enhanced imaging modalities for early detection, and biomarkers to predict disease progression. Studies exploring the long-term outcomes of patients with AEDH, as well as the integration of artificial intelligence in clinical decision-making, are also warranted. Additionally, investigating the role of advanced neuroprotective therapies in mitigating secondary brain injury could provide new avenues for improving patient care.

Equally important is the continuous education of clinicians on the latest guidelines and advancements in AEDH management. Regular training programs, workshops, and access to updated protocols can empower healthcare providers to make informed, evidence-based decisions, ensuring optimal patient outcomes. Ongoing research and education are essential to addressing gaps in knowledge and improving care standards for this life-threatening condition.

Authors' Contributions

Dr. Rahimi-Movaghar designed, supervised, contributed to writing the manuscript, and sketched the figure; Dr. Bahmani collected the data and contributed to writing the manuscript; Dr. Hajiqasemi contributed to data collection, writing, editing, submitting, and revising the manuscript.

Ethical Considerations

An ethics statement is not applicable to this article.

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Conflict of Interests

The authors declare that they have no competing interests.

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