EXTRADURAL HEMORRHAGE

Authored by
mohammad looti

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1. Core Definition and Nomenclature

An **extradural hemorrhage** (EDH), medically interchangeable with the term **epidural hematoma**, constitutes a severe and time-sensitive form of traumatic brain injury. This condition is defined by the rapid accumulation of blood in the potential space situated between the inner surface of the cranium (skull bone) and the outermost protective membrane covering the brain, officially known as the **dura mater**. The location--superior to the dura--is critical to its pathophysiology, as the dura mater is typically adherent to the skull, requiring significant trauma to separate it and create the space where the hemorrhage develops. This internal bleeding is almost exclusively the result of a high-impact trauma, which frequently leads to a skull fracture that lacerates underlying blood vessels.

The clinical urgency associated with EDH stems from the primary source of the bleeding. Unlike other forms of intracranial hemorrhage which may involve slower venous leakage, the vast majority of EDHs are due to ruptured cranial arteries, which subsequently leak blood at high systemic pressure into the surrounding extradural area. This rapid influx of blood creates a mass effect that elevates the intracranial pressure (ICP) dramatically, compressing the underlying brain tissue and compromising cerebral blood flow. If this compression is not relieved promptly, the condition becomes fatal, underscoring the absolute necessity for rapid diagnosis and intervention as highlighted in fundamental medical texts.

Accurate classification distinguishes EDH from subdural hematomas (SDH), subarachnoid hemorrhages (SAH), and intraparenchymal hemorrhages (ICH). While all are serious forms of intracranial bleeding, the arterial origin and restrictive anatomical location of EDH--which causes its characteristic biconvex shape on imaging--dictate a unique and often quicker course of clinical deterioration. Although EDH constitutes only a small percentage (around 1-3%) of all head injuries, its high mortality rate if untreated makes it disproportionately significant in emergency medicine protocols.

2. Pathophysiology and Mechanism of Injury

The critical mechanism leading to an extradural hemorrhage is a forceful blow to the head, typically blunt trauma, often involving the temporal or temporoparietal regions. This traumatic force frequently causes a skull fracture that crosses the trajectory of the main dural blood vessels. The most commonly injured vessel, implicated in roughly 80-90% of supratentorial EDHs, is the **middle meningeal artery** (MMA) or its branches. The MMA runs within grooves on the inner surface of the temporal bone, making it highly vulnerable to shearing forces or direct injury when the bone fractures.

Because the bleeding is arterial, the blood collects rapidly, forcefully dissecting the dura mater away from the inner skull surface. The volume of blood accumulation often proceeds exponentially, quickly surpassing the brain's ability to compensate through displacement of cerebrospinal fluid (CSF) or venous blood. As the hematoma expands, it creates a steep pressure gradient, leading to two major consequences: first, global ischemia due to decreased cerebral perfusion pressure (CPP); and second, localized pressure causing displacement of vital neurological structures.

The physical constraints of the cranial vault are central to the EDH's presentation. The dura mater is firmly affixed at the cranial sutures; this strong attachment typically restricts the lateral spread of the hematoma, forcing it inward and giving it the characteristic lens-like or biconvex appearance observed on imaging. This shape signifies high focal pressure exerted on the underlying brain, which can swiftly result in the catastrophic complication of **uncal herniation**, where the medial temporal lobe is pushed over the tentorium cerebelli, compressing the midbrain and brainstem.

3. Clinical Presentation and Symptoms

The classic clinical picture of EDH, though present in only about one-third of cases, involves a profound sequence of consciousness changes, starting with a brief loss of consciousness immediately following the impact, followed by a period of relative neurological clarity known as the **lucid interval**, and culminating in rapid, irreversible neurological deterioration. During the lucid interval, the patient may appear deceptively stable, often complaining only of a severe headache, nausea, or localized head pain, while the arterial hematoma silently expands.

The onset of the secondary phase marks the failure of intracranial compensation, indicated by escalating intracranial pressure. Key symptoms include a worsening headache that becomes intractable, projectile vomiting, and increasing somnolence progressing to stupor and coma. Focal signs are critical indicators: the development of hemiparesis (weakness on one side of the body opposite the injury) and, most ominously, ipsilateral pupillary dilation. This fixed and dilated pupil results from the compression of the oculomotor nerve (CN III) as the temporal lobe begins to herniate, signaling imminent brainstem compromise.

Clinical presentation varies significantly between age groups. In older patients, chronic atrophy provides a slightly larger buffer space, potentially slowing clinical decline. Conversely, in infants and young children, whose skulls are more compliant (due to unfused sutures), the hematoma may expand silently without immediate severe ICP spikes, sometimes presenting simply as pallor, irritability, or generalized seizures, before sudden and catastrophic collapse. Regardless of the patient's initial status, any suspicion of EDH mandates immediate, high-priority investigation.

4. Diagnostic Procedures

Timeliness is paramount in diagnosing extradural hemorrhage, making the non-contrast **Computed Tomography (CT) scan** the indispensable cornerstone of evaluation. The CT scan provides near-instantaneous, detailed cross-sectional imaging of the brain, allowing emergency physicians and neurosurgeons to assess the hematoma's size, morphology, and its impact on surrounding structures. The classic radiographic signature of EDH is a homogeneous, hyperdense (bright white) collection conforming to a biconvex or lenticular shape, tightly pressing against the inner table of the skull.

Critical measurements derived from the CT scan guide management decisions, specifically the thickness of the hematoma (measured in millimeters) and the extent of midline shift (how far the central structures of the brain have been displaced). A hematoma thickness greater than 15 mm or a midline shift exceeding 5 mm are conventional thresholds often used to mandate urgent surgical evacuation, regardless of the patient's initial Glasgow Coma Scale (GCS) score. Additionally, the CT scan is invaluable for identifying the associated skull fracture, often seen traversing the temporal bone in the region of the middle meningeal artery groove.

While CT angiography may be used to delineate the specific source of arterial bleeding pre-operatively in stable patients, and Magnetic Resonance Imaging (MRI) offers superior detail of soft tissue, these modalities are often bypassed in the acute, life-threatening EDH setting where minutes matter. The diagnosis must be made rapidly based on clinical suspicion corroborated by the immediate CT findings to facilitate rapid transfer to the operating room, ensuring that treatment is not delayed by prolonged secondary diagnostic procedures.

5. Management and Treatment Protocols

The primary goal in the management of symptomatic extradural hemorrhage is swift surgical decompression to evacuate the hematoma and control the bleeding source. Initial management follows Advanced Trauma Life Support (ATLS) protocols, focusing on airway protection (intubation if GCS is low or declining), adequate ventilation, and maintenance of hemodynamic stability. Pharmacological interventions aimed at managing impending herniation may include administering hyperosmolar therapy, such as mannitol or hypertonic saline, to transiently reduce cerebral edema and lower ICP while awaiting surgical access.

The definitive surgical intervention is a **craniotomy**. This procedure involves removing a sizable section of the skull (known as the bone flap) centered over the hematoma. Once access is established, the clotted blood is meticulously aspirated and removed, ensuring complete decompression of the underlying brain. The most crucial step is achieving hemostasis--identifying and controlling the source of hemorrhage, usually through cauterization or ligation of the torn middle meningeal artery. The dura mater is inspected, and the bone flap is subsequently replaced and secured.

In instances of extreme emergency, particularly in pre-hospital or remote settings where immediate craniotomy is impossible, a burr hole trephination (creating a small hole in the skull) may be performed to rapidly decompress the hematoma and temporize the rising ICP. However, this measure is often insufficient to fully evacuate the typically solid, arterial clot and must be followed by a formal craniotomy once the patient reaches a neurosurgical center. Post-operative care involves meticulous monitoring in the Intensive Care Unit to maintain optimal cerebral perfusion pressure and prevent common sequelae such as seizures and infection.

6. Prognosis and Complications

The prognosis following an extradural hemorrhage is critically dependent upon the patient's neurological condition immediately preceding surgical intervention. EDH is often described as having an excellent outcome potential if treated before neurological compromise becomes profound. Patients who undergo timely evacuation while maintaining an alert or minimally confused mental status (high GCS score) typically have a mortality rate below 10% and a high probability of making a good functional recovery with minimal long-term deficits.

Conversely, prognosis worsens dramatically once signs of brainstem compression--such as bilateral pupillary changes or fixed, dilated pupils--are evident, reflecting severe secondary injury. Mortality rates for patients presenting in a deep coma can exceed 50%. The key determinant of poor outcome is the duration of uncorrected high ICP and resulting global ischemia. Therefore, the long-term impact is intrinsically linked to the speed of the trauma system response.

Potential long-term complications for survivors include post-traumatic epilepsy, chronic post-concussive syndrome, cognitive impairment, or persistent focal motor deficits, particularly if there was associated underlying brain contusion. Technical complications specific to the surgical procedure include re-bleeding, development of infection (e.g., osteomyelitis or empyema), and the risk of developing hydrocephalus requiring CSF diversion. Ongoing research focuses on optimizing pre-hospital protocols to minimize the time delay between injury, diagnosis, and surgical decompression.

7. Further Reading

Epidural Hematoma (Extradural Hemorrhage) - Wikipedia
Dura Mater - Wikipedia
Middle Meningeal Artery - Wikipedia
Lucid Interval - Wikipedia
Craniotomy - Wikipedia

Brain Herniation - Wikipedia