Chapter 1
Acute Extra-Axial Hematoma

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Clinical Scenario

A 23-year-old male patient is brought into the trauma bay by ground ambulance from the scene of a motor vehicle crash. He was an unbelted driver who was ejected from the vehicle that had struck a guard rail at approximately 65 miles/h and flipped multiple times. The paramedics report that at the scene, he was not opening his eyes, exhibited extensor posturing, and was making guttural sounds. His GCS prior to resuscitation was therefore E1/V2/M2 = 5. He was unable to protect his airway, so he was intubated in the field without any drugs being required. This was approximately 30 min prior to arrival.

On examination, a large frontal scalp laceration with active venous bleeding is noted, as well as multiple ecchymoses and abrasions scattered over the face, head, neck, upper extremities, torso, and lower extremities. He has bilateral hemotympanum and blood per nares. There are bilateral breath sounds but rhonchi are noted. The abdomen is soft and non-distended. There is a right mid-thigh deformity and massive swelling. On neurological examination, the patient does not open his eyes. He is intubated. He exhibits decerebrate posturing on the right side, and he localizes to pain on the left. His GCS is therefore E1/V1T/M5 = 7T. His left pupil is 5 mm and sluggishly reactive, while the right pupil is 3 mm and briskly reactive. He has bilateral corneal reflexes, and gag reflex is intact.

Laboratory values include hematocrit 32.2%, hemoglobin 10.9 g/dL, pO2 175 mmHg, pCO2 35 mmHg, lactate 2.8 mmol/L, base deficit −5.6, glucose 190 mg/dL, serum Na 142 mEq/L, PT 14.0 s, INR 1.6, PTT 75 s.

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1.1 History and Physical Examination

Any patient presenting with polytrauma must undergo a rapid primary trauma survey, including the "ABCs" of airway, breathing, and circulation first and foremost. Avoidance of hypoxia and hypotension in severe traumatic brain injury patients is paramount, as any single incidence of each negatively impacts outcome, and the presence of both portends an even worse prognosis [1]. Active bleeding should be staved with compression or rapid closure.

Modern concepts of the primary trauma survey also include additional elements, called "ABCDE," with "D" standing for disability or neurological status. The letter "E" stands for exposure and environmental control. Clothing is cut away and removed to allow for a thorough external examination for injuries. Attention then is turned toward keeping the patient from becoming hypothermic by controlling the environment (ambient temperature, warming blankets, etc.).

Key to the assessment of any neurologically injured patient is the rapid evaluation of consciousness and neurological status. The post-resuscitation Glasgow Coma Scale score [2] shown in Table 1.1 is a predictor of prognosis in patients with acute traumatic brain injury (TBI) and should be obtained on all patients presenting with evidence of TBI. The best response in each category is scored. This scale is commonly used to assess progress over time and can change rapidly, especially in the face of expanding extra-axial lesions, so obtaining an accurate post-resuscitation score is a critical element of the exam in the trauma bay.

Speech and orientation are not testable in comatose patients or even sometimes in responsive, but intubated patients. However, basic levels of responsiveness should be noted, such as looking toward an examiner's voice, nodding yes or no, following commands that are either simple (one step) or complex (multi-step or requiring awareness of numbers or lateralizing right-left). Occasionally, only subtle changes in these findings will signal precipitous decline due to herniation from mass lesions like extra-axial hematomas.

Table 1.1 The Glasgow Outcome Score is calculated based upon the patient's best response in each category. 1-I is the author's own modification to differentiate an intubated patient from a patient who has already had a tracheostomy; historically 1-T has been used for any type of artificial airway or "tube"

Eye opening		Motor		Verbal response	
None	1	None	1	None	1
To pain/pressure	2	Decerebrate posturing	2	Incomprehensible sounds	2
To speech/sound	3	Decorticate posturing	3	Inappropriate words	3
Spontaneous	4	Withdraws from pain	4	Regular speech but confused	4
Not testable	NT	Localizes pain	5	Regular speech and oriented × 3	5
		Follows commands	6	Not testable	NT
		Not testable	NT	Intubated	1-I
				Tracheostomized	1-T

Asymmetry of motor and pupillary responses must be noted as potential lateralizing signs. Unilateral weakness or posturing most commonly signals the contralateral location of an extra-axial hematoma such as an epidural hematoma (EDH) or subdural hematoma (SDH). The presence of an ipsilateral pupil dilation or decrease in reactivity can further help localize a compressive lesion. A less common finding is weakness ipsilateral to a compressive extra-axial hematoma, known as the Kernohan's notch phenomenon. This is a false localizing sign with ipsilateral motor weakness caused by compression of the contralateral cerebral peduncle against the tentorium cerebelli.

Transtentorial or uncal herniation from a mass lesion is signaled initially by confusion progressing to unconsciousness, contralateral motor dysfunction, and an ipsilateral third nerve palsy. Contralateral motor dysfunction is caused by direct compression of the hemisphere by a mass lesion affecting the motor strip. Third nerve palsy is caused by compression of the third nerve against the ipsilateral tentorial incisura by the medially shifted temporal lobe uncus and is evidenced by a mydriatic, sluggishly reactive or non-reactive pupil, ptosis, and "down and out" position of the globe with the eye position pointed lateral and inferior when looking straight ahead with the unaffected eye. The pupillary abnormality occurs first due to the peripheral location of the autonomic nerve fibers within the nerve being compressed first. In the unconscious patient, the eye position and ptosis may be untestable. Altered consciousness is caused by progressive brainstem compression. As the situations progresses, the contralateral cerebral peduncle compression against the opposite tentorial incisura results in the ipsilateral motor dysfunction of the Kernohan's notch phenomenon.

An alert patient with a third nerve palsy and craniofacial trauma should raise the suspicion of a direct injury to cranial nerve (CN) III or a so-called traumatic third nerve palsy and should be distinguished from uncal compression of the third nerve.

Central or downward herniation is identified by rostral to caudal loss of neurological function progressing from headache, nausea, vomiting, and confusion to deep coma, respiratory arrest, and loss of brainstem reflexes. This progression can indicate a large extra-axial mass lesion with or without major cerebral edema, but typically is seen with significant primary brain injury and edema.

The Cushing reflex and resultant Cushing's triad are related to elevated intracranial pressure. The triad is comprised of bradycardia, hypertension, and irregular respirations (Cheyne–Stokes breathing). The pulse pressure (difference between systolic and diastolic blood pressure) is also widened. Since respiratory patterns are often masked by mechanical ventilation in intubated patients, this aspect of Cushing's triad is often obscured. This phenomenon is caused by the Cushing reflex, i.e., hypothalamic dysfunction affecting the autonomic nervous system due to poor perfusion caused by intracranial pressure elevation. Sympathetic tone is increased, resulting in increased peripheral vascular resistance and blood pressure, which then activates the parasympathetic response from carotid baroreceptors and vagal-nerve induced bradycardia.

If signs of transtentorial/uncal herniation, central/downward herniation, or Cushing's triad are present in a patient with head trauma, an extra-axial hematoma should be suspected.

Direct or vascular trauma to the limbs can affect motor responses, so any external traumatic findings should be noted. Blunt vascular injury (BVI) of the craniocerebral vasculature can also result in cerebral ischemia and unilateral motor findings, so signs of skull base fracture and neck trauma should be noted (e.g., seat belt marks, crepitus, hematoma/swelling, petechiae of the sclera, and soft tissues of the head and neck) [3, 4].

The patient should be checked for bleeding from the nares or into the oropharynx, hemotympanum, and for periorbital ecchymoses (racoon's eyes) or mastoid area ecchymoses (Battle's sign), which could signal the presence of facial fractures, injury to facial vessels, or skull base fractures. This degree of craniofacial trauma is more often associated with significant primary brain injury; therefore, SDH should be suspected if localizing signs are also present.

Limited brainstem reflex and cranial nerve examinations can be done quickly and should include testing the pupillary reactivity and size (CN III) and the corneal reflexes (CN V) at a minimum. Breathing patterns in non-intubated or spontaneously breathing patients should be noted. The gag reflex may be tested while making certain not to dislodge any endotracheal tube (ETT) or other airway. This can be done in an intubated patient with minimal manipulation of the ETT as long as the balloon is inflated and there is no tracheal injury. Alternatively, a catheter can be inserted into the mouth to carefully touch the posterior tongue or pharynx. Deep suctioning that induces coughing is not an indicator of an intact gag reflex but rather a cough reflex. Both gag and cough reflexes are subserved by CN X with additional contribution of CN IX to the gag reflex. Oculocephalic reflexes (CN III, IV, VI, VIII) are generally not tested in comatose patients in the trauma bay due to the common co-existence of cervical spinal injuries with traumatic brain injuries [5] and because the cervical spine has not yet been cleared. Oculovestibular reflexes can be checked in cases of suspected brain death through the so-called cold caloric test.

In more responsive patients, even if intubated, extraocular movements can be tested easily to assess CN III, IV, and VI by commands to look up, down, and to each side. Nystagmus should be noted if present. Gross vision (CN II) can be assessed in responsive patients by asking them to hold up the same number of fingers they see or to report verbally if non-intubated. Blinking to visual threat is a rapid and gross test of vision that can be also used. In non-intubated patients, testing for tongue protrusion symmetry (CN XII) and gag reflex (CN IX, X) are important for potential aspiration risk. A quick check of facial symmetry can be done with smile, eyebrow raise, and/or puffing the cheeks (CN VII) and of hearing by rubbing the fingers together or snapping the fingers near each ear (CN VIII). CN VII and VIII are often directly injured in the setting of basilar skull fracture. Attempts at facial nerve assessment early are warranted to help differentiate direct nerve injury or laceration (in which case function is immediately impaired) from nerve edema (in which case onset can be gradual). Having the patient shrug the shoulders and

turn the head against resistance tests CN XI but this is often not necessary on primary survey. It is usually very difficult to test smell in the trauma bay as this requires a degree of cooperativity and access to an odorant; therefore, this is typically foregone. Facial sensation (CN V) is also typically not tested in the trauma bay setting, unless patients are responsive and non-emergent. Since the corneal reflex is subserved by CN V, this reflex represents a quick test of function of this nerve.

It is critical to obtain as many details of the mechanism of injury as possible, as well as the time of injury and potential secondary insults occurring prior to arrival. These details will inform the degree of suspicion for various injury patterns and aid in the assessment of prognostic factors. If the traumatic event was witnessed, as many details should be gleaned as possible regarding the event, such as vehicles involved, trajectory, speed, use of restraints, and the status of other victims in the case of motor vehicle-related injuries. In cases of personal assault, the objects used, the caliber of weapon if a firearm was used, and the time last seen for those "found down" should be noted. For falls, height is critical and if it is possible to ascertain, whether the fall resulted from a mechanical issue or a neurological event. Prognostic factors include time from injury to potential intervention, degree and duration of hypothermia, estimated volume of blood loss, and the occurrence of any episodes of hypoxia, hypotension, or seizure, among others. Queries should be made regarding medications and fluids administered in the field, events requiring intervention, and trends in breathing, blood pressure, and responsiveness.

It is also important to identify the most relevant aspects of the patient's medical history and obtain facts quickly from family members or friends for those patients in coma or who have been intubated. These individuals are often not available at the time of presentation so this may need to be done during the secondary, tertiary, or quaternary surveys. However, the patient's personal effects can initially be checked for documentation of medications, allergies, diabetes mellitus, hemophilia, medical implants, anticoagulation medications, or other commonly documented conditions. These identifications may be in the form of wallet card inserts or medical bracelets/jewelry. When looking for such information, care should be taken to avoid injury from broken glass, metal, needles, and other sharps or even weapons that may be obscured in clothing.

In the current clinical scenario, information about the mechanism of injury and details regarding the scene were gleaned from communication with EMS providers. No family was available at presentation to provide details regarding the patient's past medical history or medications. The presence of extensive soft tissue injury, as well as obvious deformity of the right leg, suggests multisystem trauma. The neurologic exam, as described, suggests severe traumatic brain injury. Lateralizing findings such as pupillary and motor asymmetry raise concern for a space-occupying intracranial lesion. The presence of a large scalp laceration should raise concern for the possibility of open skull fracture with underlying intracranial pathology. The presence of blood at the nares and ear canals might suggest a skull base fracture. Once the ABCs have been satisfied and any life-threatening bleeding contained, further investigation of this patient's neurologic injury should take priority.

1.2 Differential Diagnosis

The differential diagnosis of altered consciousness or coma in a polytrauma patient such as this one includes impairment from drugs (alcohol, prescription medications, illicit drugs) and toxins. A combination of other findings points to traumatic brain injury or a compressive lesion as the cause of coma, namely, motor asymmetry, pupillary asymmetry, localizing or focal neurological deficits, and the presence of external trauma. Even so, serum and urine testing for alcohol and drug levels remains a mandatory portion of the workup, since central nervous system acting agents can mask neurological changes, can mimic the effects of TBI and cerebral edema, and can lower the seizure threshold. Hypothermia can occur even in relatively warm exterior temperatures if patients are exposed or unconscious for a significant period of time, and this can cause global depression of cerebral function as well.

A lucid interval in which there is no or minimal neurological impairment followed by a rapid decline with signs of transtentorial herniation is a "classical" presentation of an epidural hematoma. When coupled with external signs of temporal region trauma, this is the likely diagnosis, as temporal fracture associated with laceration of the middle meningeal artery can lead to a rapidly expanding epidural hematoma causing transtentorial or uncal herniation. Lucid intervals can be seen in other scenarios of brain trauma, including a rapidly expanding arterial subdural hematoma. While most subdural hematomas are venous in origin, cerebral arteries can be injured even in blunt trauma and result in SDH and rapid decline.

EDH can be seen in more posterior regions with occipital or suboccipital trauma. Common clinical scenarios include falling or jumping out of a moving car or truck bed, falling backwards off of a stool or falling backwards from a standing/walking position (such as in slipping on ice or being struck in the face during an assault). Swelling and bruising on the occiput coupled with one of these mechanisms should raise the index of suspicion for an occipital or posterior fossa extra-axial hematoma (EDH or SDH). These can arise from sheared draining cortical veins or direct trauma to the posterior draining cerebral venous sinuses, with or without skull fracture.

While both EDH and SDH may occur in isolation, without significant underlying brain injury, subdural hematomas are far more commonly associated with significant underlying brain parenchymal injuries and edema [6]. Therefore, signs of global cerebral dysfunction, multiple deficits, and bilateral posturing should raise suspicion of primary TBI with or without SDH.

Again, blunt vascular injury can mimic a unilateral extra-axial hematoma by causing hemispheric cerebral ischemia and infarction. Occasionally, BVI and TBI co-occur, obscuring the physical differential diagnosis even further.

1.3 Diagnostic Evaluation

The trauma care team should first ensure adequate oxygenation and blood pressure, and if hypothermia is present, employ a safe strategy for rewarming in order to gain the most reliable neurological examination. Serum and urine drug and alcohol tests should be done, as well as basic laboratory tests for electrolyte or glucose abnormalities that could contribute to coma, seizures, or altered sensorium. Routine tests for comatose trauma patients include arterial blood gases, comprehensive metabolic profile, complete blood count, coagulation studies, urinalysis, serum alcohol level, and urine drug toxicology tests at a minimum.

The mainstay of traumatic brain injury diagnostic imaging is computed tomography (CT) of the head without contrast. Rapid stabilization and transport to the CT scanner are a routine part of management of the comatose trauma patient. If polytrauma is obvious or the mechanism of injury suggests polytrauma, then chest, abdomen, and pelvis CTs are employed in addition to head CT. Cervical, thoracic, and lumbar spine CTs are routinely done in comatose trauma patients due to the high propensity for concurrent spinal trauma in such cases [5, 7, 8]. In isolated cranial injuries, cervical studies should be done at a minimum. Facial CTs may be required. If signs that are high-risk for BVI are present (major craniofacial fracture, neck soft tissue trauma, lateralizing examination without cause noted on brain CT, etc.), a screening CT-angiogram (CTA) of the head and neck arteries may also be done at the same time or subsequently.

EDH and SDH are hyperdense compared to brain on CT (i.e., bright white compared to gray). EDHs are typically lentiform or biconvex in shape, located near the common sources of bleeding (middle meningeal artery or venous sinuses), and usually do not cross cranial sutures due to the rigid attachment of the dura at the suture lines (Fig. 1.1). SDHs are typically crescent shaped as they follow the shape of the cranium and cerebral hemisphere, and they do cross suture lines (Fig. 1.2a–c). Supratentorial SDHs may be focal, hemispheric (layered over the surface of the hemisphere), tentorial (as blood layers onto the tentorium cerebelli), falcine (layering along one or both sides of the rigid falx cerebri), or a combination of any of these since the subdural space is essentially continuous. SDH can technically cross from the supratentorial and infratentorial space (a.k.a. posterior fossa) at the incisura of the tentorium cerebelli, but such occurrences are rare. They can also extend from the posterior fossa into the spinal canal. Isolated posterior fossa SDH can occur but is more unusual than supratentorial SDH.

Mixed density extra-axial hematomas can be seen with hyperacute and/or pulsatile bleeding into the hematoma. This is usually seen as an eddy-shaped, jet-shaped, or circular hypodensity in the center of a hyperdense hematoma. Mixed density SDH can also be seen in older patients who have had an acute hemorrhage into a subacute (isodense to brain) or chronic (hypodense to brain) subdural hematoma. In such instances, the SDH may be loculated due to the presence of multiple membranes (Fig. 1.3a, b).

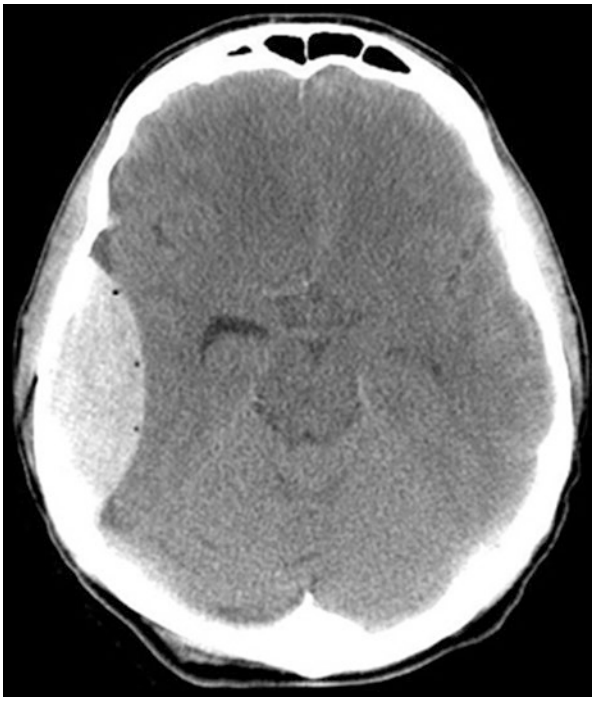

Fig. 1.1 Acute epidural hematomas are hyperdense on CT scan without contrast, are typically lentiform in shape, and do not usually cross cranial suture lines

Bone window interpretation is a key element of radiographic assessment of both TBI in general and in differentiating between EDH and SDH. Fractures involving the foramen spinosum and/or adjacent to the middle meningeal artery, or adjacent to any of the cerebral venous sinuses, or involving the orbital roof are commonly associated with the formation of EDH. Orbital roof fractures can result in more slowly developing and self-limited subfrontal EDH due to bleeding from bone fragments and bridging veins. Clival or other skull base fractures indicate significant force and are commonly associated with severe primary brain injury and SDH.

CT head without contrast for the patient in the current clinical scenario (Fig. 1.4a, b) reveals a left extra-axial hyperdense collection, associated with effacement of the local sulcal-gyral pattern, uncal herniation, dilatation of the contralateral temporal horn, effacement of the basal cisterns, and midline shift to the right that is disproportionate to the maximum thickness of the subdural hematoma.

1.4 Clinical Decision-Making and Next Steps

Clinical decision-making regarding extra-axial hematomas in the trauma bay centers on whether or not the patient needs to go to surgery for an emergent craniotomy. Clinical signs of herniation (transtentorial, uncal, or central/downward) and/or the presence of Cushing's triad, in conjunction with an identified causative lesion on

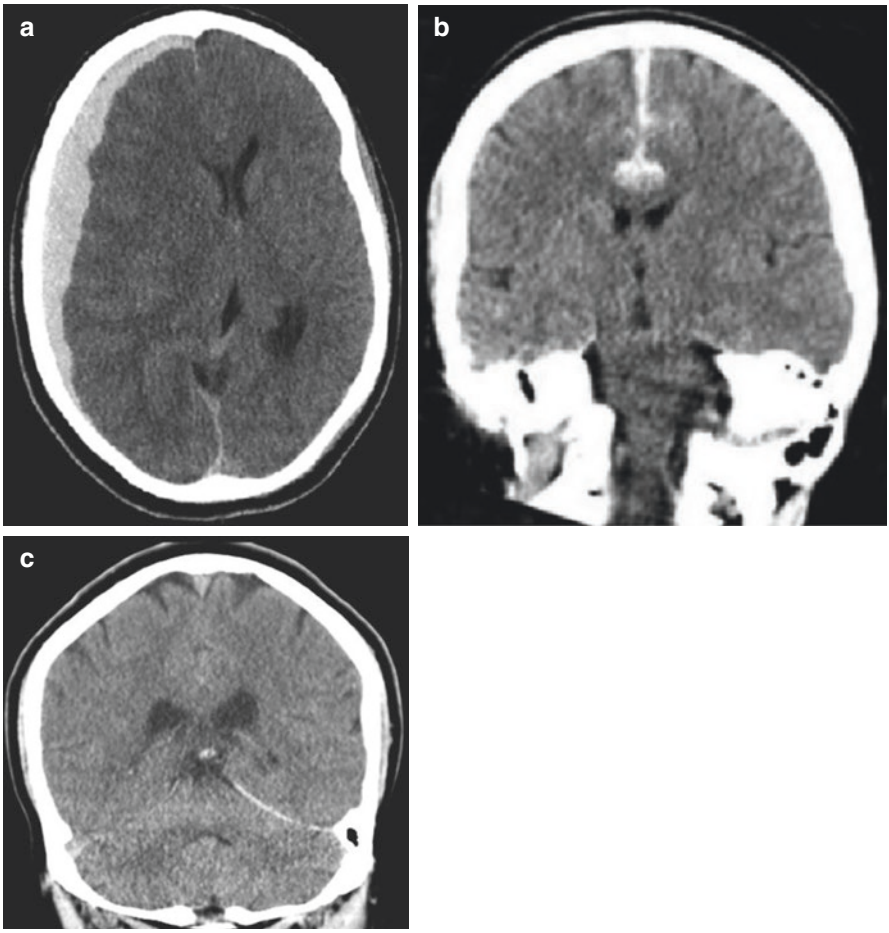

Fig. 1.2 (**a**) Acute SDH is hyperdense on CT without contrast, is typically crescent-shaped, and can cross suture lines. SDH can be located in any part of the subdural space. (**b**) Subdural blood along the anterior falx. (**c**) Subdural blood along the left tentorial leaflet

CT, mandate surgical evacuation of EDH or SDH if the patient's overall injuries appear to be survivable.

Absent signs of impending or actual herniation and death, there are several other indications for surgical evacuation. Surgical guidelines [9, 10] rely upon the key elements of hematoma size/volume and degree of mass effect attributed to the extra-axial lesion. The maximal thickness of EDH and SDH should always be measured, taking care to avoid apical measurements which can be overestimated because of tangential geometry. Midline shift should always be measured at the level of the anterior commissures. Notation of the appearance of the cisterns (patent, blood-filled, compressed, or absent/obliterated) should be made. Finally, estimates of the volume of extra-axial hematoma can be made using the $A \times B \times C/2$ method [11].

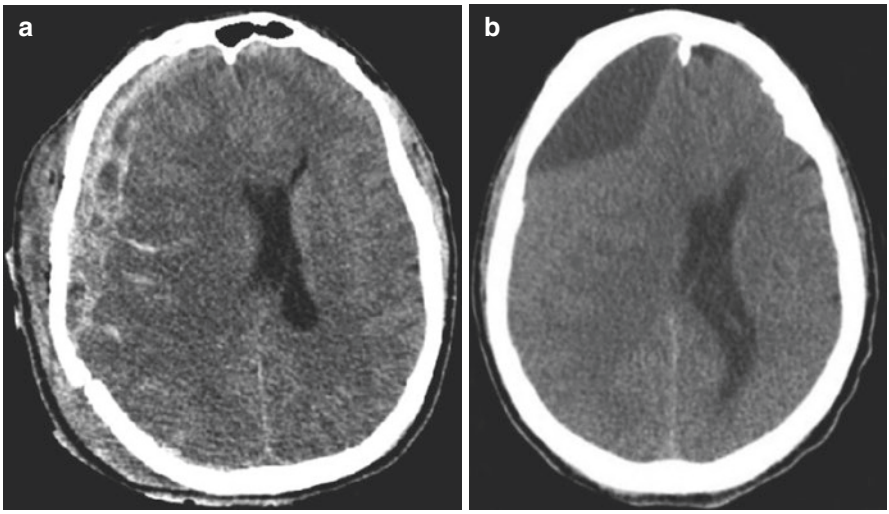

Fig. 1.3 (**a**) A hyperacute SDH is distinguished by a swirling appearance of hyper- and hypodense components within the subdural collection, whereas (**b**) an acute-on-chronic SDH may demonstrate a "hematocrit" effect reflecting the greater density of the dependent acute blood component

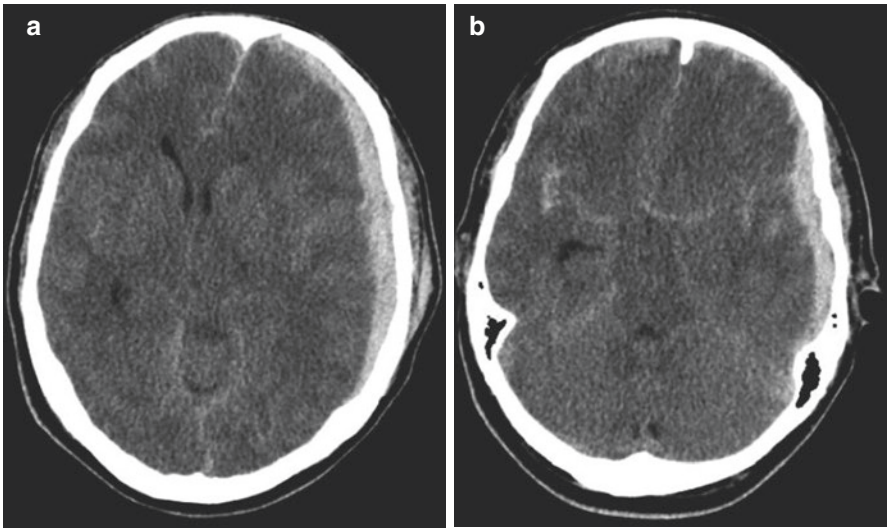

Fig. 1.4 (**a**, **b**) CT head reveals a left extra-axial hyperdense collection, associated with effacement of the local sulcal-gyral pattern, uncal herniation, effacement of the basal cisterns, and midline shift to the right that is disproportionate to the maximum thickness of the subdural hematoma. This constellation of radiographic signs, coupled with evidence of primary parenchymal injury such as contusion or edema, suggests the need for a decompressive hemicraniectomy (i.e., leaving the bone flap out) at the time of surgery

Indications for emergent evacuation of EDH via craniotomy are: (1) volume ≥30 cm^3, regardless of GCS and (2) anisocoria and coma (GCS ≤8), regardless of size. It is generally feasible to replace the bone flap after EDH evacuation.

Indications for evacuation of SDH are (1) ≥10 mm thickness OR ≥5 mm midline shift, regardless of GCS. (2) If a patient is in coma with GCS ≤8 AND the SDH maximal thickness is ≤10 mm AND midline shift is ≤5 mm, surgery may still be indicated. This is especially true if (a) the GCS has decreased by ≥2 points between injury and arrival, (b) the patient has anisocoria, (c) the patient has fixed and dilated pupils, or (d) the intracranial pressure (ICP) is >20 mmHg. When multiple types of lesions are present, these guidelines still apply, with the caveat that more aggressive action may be needed despite a smaller EDH or SDH in the face of multiple other mass lesions.

When the midline shift is proportional to the maximum thickness of the subdural hematoma, and there is relatively little parenchymal injury, the bone flap may be able to be replaced. However, if the initial CT shows that the midline shift is out of proportion (i.e., greater than) the maximum thickness of the SDH, the neurosurgeon should be prepared to perform a large hemicraniectomy incision and bone flap in anticipation of intraoperative cerebral edema outside the bounds of the cranium and the need to leave out the bone flap. Other radiographic signs suggesting this eventuality include compressed or absent cisterns, loss of sulci over the affected hemisphere, uncal herniation, or ipsilateral hemispheric contusions, hematomas, or edema, along with clinical signs of herniation or majorly elevated ICP if a monitor has been placed in the trauma bay. The patient in the current clinical scenario presents with most, if not all, of these ominous features (Fig. 1.4a, b). A primary decompressive craniectomy was performed, in conjunction with evacuation of the acute subdural hematoma.

ICP monitors (intraparenchymal or intraventricular) may be inserted emergently in the trauma bay in cases of delayed imaging due to ongoing resuscitation or CT unavailability. They can help guide surgical decision-making and medical therapeutics for cerebral edema and intracranial hypertension. For those patients whose EDH or SDH and/or clinical scenario does not meet the indications for emergent surgery, but who are in coma and harbor extra-axial lesions, ICP monitoring should be employed to provide a continuous physiological monitor of intracranial pressure and cerebral perfusion pressure (requires arterial line) that will (1) guide nonsurgical therapeutic measures for the primary brain injury, particularly in the setting of polytrauma and (2) provide an early warning of lesion expansion that would prompt surgical evacuation.

Rapid, detailed assessment of multiple history, examination, and diagnostic findings is necessary in the initial evaluation of the trauma patient suspected of harboring an extra-axial hematoma so that life- and function-saving surgery and medical management can be employed as quickly and efficiently as possible.

1.5 Clinical Pearls

- A lucid interval is often seen in the presentation of an epidural hematoma, followed by potential for rapid decline, herniation, and death.
- Contralateral motor weakness and ipsilateral third nerve palsy (evidenced by pupillary dilation) are localizing signs caused by mass effect from extra-axial hematomas.
- The Kernohan's notch phenomenon is a false localization sign; ipsilateral motor weakness is due to compression of the contralateral cerebral peduncle against the tentorium cerebelli.
- Acute subdural hematoma is frequently associated with significant underlying primary parenchymal injury.
- When the degree of midline shift is out of proportion to the thickness of an acute subdural hematoma, this is a sign of significant parenchymal injury and/or cerebral edema and decompressive hemicraniectomy may be indicated.

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