Head Injury

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Abstract: Head injury/traumatic brain injury (TBI) is the most prevalent etiology of morbidity as well as mortality all round the world, with colossal financial consequences on the healthcare system. The World Health Organization estimated that ten million people experience a head injury per annum. Head injury patients experience long-term cognitive as well as functional problems and medical illnesses like epilepsy, which necessitate long-period or lifelong personal medical and supportive assistance. The common causes of head injury include falls from height, road traffic accidents (RTAs), recreation and sports accidents, physical assault, firearm attacks and accidents, and explosions. TBIs may be mild, moderate, and severe, requiring proper emergency clinical and radiological assessment for appropriate emergency, urgent, or routine management, including ICU management and surgery. This chapter will briefly discuss the principles of the management of TBI patients with regard to issues ranging from emergency management to complication management.

Abbreviations

ABG	arterial blood gas	AEDs	anti epileptic drugs
ABI	ankle–brachial index	ASDH	acute subdural hematoma
APTT	activated partial thromboplastin time	AVF	arterio-venous fistula
ATLS	advanced trauma life support	CBV	cerebral blood volume
CBF	cerebral blood flow	CN	cranial nerve
CCF	carotico-cavernous fistula	CSF	cerebrospinal fluid
CSDH	chronic subdural hematoma	CT	computed tomography
CPP	cerebral perfusion pressure	DC	decompressive craniotomy
CTA	computed tomography angiogram	DVT	deep venous thrombosis
DAI	diffuse axonal injury	EEG	Electroencephalogram
DTI	diffusion tensor imaging	GCS	Glasgow coma scale
EDH	extra-/epidural hematoma	HDU	high-dependency unit
ESS	endoscopic sinus surgery	ICP	intracranial pressure
GRE	gradient echo	INR	international normalized ratio
ICH	intracranial hematoma	MMA	middle meningeal artery
ICU	intensive care unit	PBI	penetrating brain injury
LOC	loss of consciousness	PHIS	post-traumatic head injury syndrome
MRA	magnetic resonance angiogram	PTA	post-traumatic amnesia
MRI	magnetic resonance imaging	SBP	systolic blood pressure
PCA	posterior cerebral artery	SDE	subdural effusion
PPI	proton pump inhibitor	TP	tension pneumocephalus
PT	prothrombin time	TBI	traumatic brain injury
SDH	subdural hematoma	UOP	urinary output
RTA	road traffic accident	AEDs	anti epileptic drugs

1. Introduction

Traumatic brain injury (TBI) is a prevalent etiology of morbidity as well as mortality around the world, with enormous financial consequences for the healthcare system. The World Health Organization believes that ten million people suffer a head injury every year, and the Centers for Disease Control (CDC) and Prevention in the US estimate that 1.7 million people are afflicted by a head injury every year. The expanse of a head injury in the US is estimated to be more than USD 60 billion per year, including medical expenditures and costs due to loss of productivity. Patients with head injuries experience long-term cognitive as well as functional problems and medical illnesses like epilepsy, which necessitate long-period or lifelong personal medical and supportive assistance. The most common causes of head injury differ depending on the affected patient's age. Falls from height are the commonest cause of head injury among those less than 4 years old and elderly people over 75. Road traffic accidents (RTA) are the principal cause of TBI among adolescents. Other major causes of head injuries include recreation-related and sports injuries, physical assault, firearm-related incidents, and blast related injuries among military personnel (Kim and Gean 2011; Greenberg 2010; July and Wahjoepramono 2019).

2. Pathophysiology of Head Injury

The damage to the brain due to TBI is generally classified as primary or secondary. Hematomas and traumatic/diffuse axonal injury (DAI) are primary injuries that arise from direct severe impacts. Secondary brain injuries happen minutes to days following a main injury and consist of a complicated biochemical sequence that is started by the initial primary injury and leads to brain edema and herniation. This classification emphasizes that a brain injury is a continuously progressive trauma that requires appropriate medical as well as surgical treatment consisting of brain oxygenation to relive intracranial pressure (ICP), including cerebral perfusion pressure (CPP), in order to enhance patient healing and stop further trauma. Secondary insults can be triggered by primary head traumas, such as cerebral bleeding, which raises ICP. The gap between mean arterial pressure and ICP is CPP (i.e., the pressure difference pushing oxygen and nutrition supply to the cerebral tissue). CPP is a measure of cerebral blood flow. CPP will decline if the ICP rises owing to a head injury (or the systemic blood pressure lowers), and the cerebral tissue will be ischemic until the normal cerebral autoregulatory mechanism generates compensatory neurovascular vasodilation to provide enough circulation to the parenchyma of the brain. Normal neurovascular autoregulation, on the other hand, is commonly impaired in head injury patients, especially children. Autoregulation appears to be hampered at lower CPP values (under the threshold of 50–60 mm Hg), and, at these levels, the brain is prone to becoming ischemic. Complex cellular as well as biochemical pathways are initiated by ischemia, and the result is reduced glucose and oxygen supply to the cerebral tissue, aggravating brain damage. Extra calcium influx inside the cells leads to mitochondrial malfunction, cellular edema, free-radical generation, and ultimately neuronal death, the last of which is one of the pathophysiologic processes initiated by excitatory neurotransmitters (mainly glutamate) unleashed into the brain parenchyma. (Kim and Gean 2011; Greenberg 2010; July and Wahjoepramono 2019; Kirollos et al. 2019).

3. Incidence and Prevalence of Head Injury

In emerging countries, the overall injury rate is 22.1 per 1000 person years. According to statistics, one person dies from a head injury every 6 to 10 min in India.

Pedestrians, motorbike drivers, and assistants correspond to the highest rates of head injuries in Asia. The majority of people injured in RTAs are pedestrians who are considered vulnerable road users.

Every year, 50 million people are injured around the world.

In total, 1.2 million people are estimated to die due to injuries annually.

The global mortality rate is 97/1,000,000.

Every day, 70% of fatalities (8,500,000) are suffered by people under the age of 45, with 3300 deaths and 6600 catastrophic injuries. In underdeveloped nations, there has been an increase of 80% of TBI (Head Injury Foundation n.d.).

The prevalence of head injuries is high, with men accounting for the majority of cases. The sufferers are mostly illiterate and work as day laborers. The risk factors for a TBI with a difficult course include the following: high-energy injuries, bicycle accidents, RTAs in general, anticoagulant therapy, intoxication with alcohol, age greater than 60 years, and a low GSC score at the time of presentation (Kim and Gean 2011; Gururaj 2002).

4. Etiology of Head Injury

The commonest causes of head injuries are as follows:

- Car accidents, motorcycle accidents, and bicycle accidents;
- Falls from height;
- Child abuse;
- Acts of violence;
- Cycling;
- Football;
- Baseball and softball;
- Explosions and other combat injuries.

5. Risk Factors of Head Injuries

- Children, particularly newborns up to 4 years of age;
- Young adults, particularly between the ages of 15 to 24;
- Adults 60 and older;

- Male sex (any age group);
- Anticoagulant use

(https://www.mayoclinic.org/diseases-conditions/traumatic-brain-injury/symptoms-causes/syc-20378557, accessed on 7 March 2022).

6. NICE Guidelines for Applying CT to Assess Head Injury

- Glasgow Coma Score (GCS) < 13 at any point;
- Focal neurological deficit;
- Suspected open, depressed, or basal skull fracture;
- Seizure;
- Vomiting, >one episode;
- Urgent CT head scan if none of the apply above but the following do:
- Age > 65;
- Coagulopathy (e.g., being on warfarin);
- Dangerous mechanism of injury (CT within 8 h);
- Antegrade amnesia > 30 min (CT within 8 h) (Nader et al. 2014; Habeeb 2017).

7. Principles of Management of Head Injury

In the neuro-intensive care unit, the current head injury management strategy focuses on preventing a cascade of secondary injuries by preserving appropriate brain perfusion. To prevent both hypoxemia and hypotension, which may increase mortality and morbidity, this technique necessitates constant neuromonitoring and blood pressure and oxygenation regulation. Patients with severe autoregulation problems rely solely on blood pressure to continue cerebral circulation to the brain tissue, which is known as "pressure passive" flow.

As a result, enough blood pressure support is essential. Patients with a severe head injury, as defined by a GCS score of 3–8 and a CT scan, have their ICP monitored. External ventricular drainage catheters are commonly used to monitor ICP, but intraparenchymal, subarachnoid, subdural, and extradural devices can also be employed. Raised ICP is linked to a worse prognosis and may indicate cerebral herniation or a potentially weakened CPP. To reduce ICP, strict procedures are utilized. Medical management, such as hyperventilation, hyperosmolar infusion, and proper sedation/analgesia, and surgical treatment, such as external CSF drainage (external ventricular drainage—EVD), cerebral hematoma removal, and, if needed, decompressive craniectomy (DC), are both options. Finally, maintaining an appropriate CPP is critical to minimize ischemic injury; again, the threshold value of CPP is 50–60 mm Hg, and brain ischemia can occur under this value (Brain Trauma Foundation et al. 2008).

7.1. Role of Imaging in Diagnosis and Treatment of Head Injury

Both the diagnosis as well as treatment of a brain injury rely heavily on imaging. Non-contrast CT scanning is the technique of choice for diagnosing head injury in the acute environment because it swiftly and accurately reveals cerebral hemorrhages that necessitate neurosurgical evacuation (Figure 1).

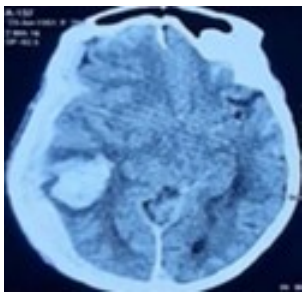


Figure 1. CT scan showing acute SDH and traumatic ICH. Source: Figure by authors.

Extradural, subdural, and subarachnoid/intraventricular hemorrhages and intra-axial brain parenchymal hemorrhages, cortical contusion, DAI, and shear injuries can be easily detected via CT. While CT scans are the gold standard for TBI imaging, magnetic resonance imaging (MRI) provides a higher diagnostic sensitivity for lesions that are not always hemorrhagic, such as cerebral contusions and non-hemorrhagic DAI (Figure 2).

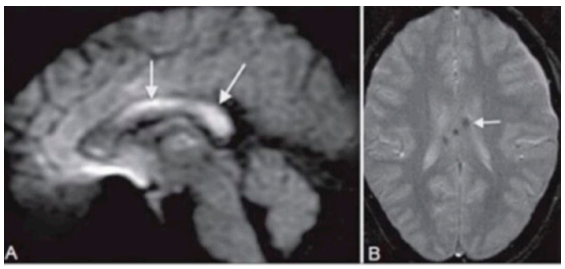


Figure 2. (**A**). MRI in sagittal view in ADC image shows the strong signal along the corpus callosum. (**B**) Axial MRI flair image showing a punctate hemorrhage in the corpus callosum. Source: Figure by authors.

Susceptibility-weighted (SW) and diffusion tensor imaging (DTI) techniques, both of which are based on MRI technology, are better for predicting outcome for brain injury patients. Non-contrast CT scans do not capture physiologic changes in brain perfusion, cerebral blood flow, and parenchymal oxygenation that are linked to the detrimental sequences following TBI that have a major impact on functional outcome. A perfusion CT scan is a neuroimaging technique that uses dynamic scanning images during i.v. contrast injection to show physiologic characteristics such as cerebral blood flow (CBF), cerebral blood volume (CBV), and mean transit time (MTT) (the time required for blood to perfuse a regional brain tissue). Normal cerebral perfusion or hyperemia (high CBF and CBV) observable via perfusion CT for head injury patients has been linked to a better clinical result, while evidence of hypoxia (low CBF and CBV) is linked to a worse clinical outcome (Kim and Gean 2011). DAI disrupts the cytoskeleton as well as axoplasmic flow, resulting in alterations in tissue diffusion that can be seen and measured via DTI. The level of white matter damage in DTI may be related to the severity of cognitive dysfunction as well as the functional result after a head injury. Another very new MRI approach that improves DAI detection sensitivity is SWI. SWI employs a three-dimensional GRE sequence that is high-resolution, velocity-compensated, and founded on both magnitude and phase data. Its sensitivity is higher than that of standard GRE sequences for the identification of hemorrhagic DAI, detecting 4–6 times more microhemorrhages than GRE sequences (Kim and Gean 2011; Qureshi et al. 2016; Badjatia et al. 2008).

7.2. Management of Head Injury/TBI Including ATLS

The American College of Surgeons created Advanced Trauma Life Support (ATLS) as a training curriculum for medical doctors in regard to the handling of acute trauma situations. The goal of ATLS is to identify and execute treatment of lethal injuries such as head injuries and polytrauma. The Golden Hour refers to the first hour following a catastrophic injury when emergency treatment is most likely to be effective. The basics of trauma management are as follows:

1. Preparation;
2. Triage;
3. Primary survey with simultaneous resuscitation;
4. Secondary survey;
5. Definitive treatment.

7.2.1. Preparation

The wearing of gloves, a gown, shoe covers, a mask, and head-covering goggles for self-protection is recommended.

7.2.2. Triage

The process of categorizing victims or mass casualties based on their need for treatment and the resources available.

7.2.3. Primary Survey (with Simultaneous Resuscitation)

The primary survey is the first and most important step in assessing patients who have experienced trauma. During this period, life-threatening injuries are detected, and resuscitation must be performed concurrently. ABCDE is a mnemonic for the order in which issues should be dealt with (Maas et al. 1997).

A. Airway Control with Cervical Spine Immobilization

1. Maintenance of airway patency:
    - Lifting of the chin or thrusting of the jaw;
    - Removal of foreign bodies, blood, and vomitus via suction;
    - Nasopharyngeal/oropharyngeal airway clearance.
2. Airway support:
    - Provision of oxygen via a nasal canula/non-breathing mask (high mask) or Ambu bag pumping.
3. Establishment of a definitive airway:
    - Endotracheal tube intubation;
    - Cricothyroidectomy;
    - Tracheostomy.
4. Imobillize the patient;
5. Apply cervical hard collar (Philadelphia collar);
6. Avoid hyperextension of the neck.

B. Breathing and Ventilation

Look for the conditions that impair ventilation, such as the following:

- Tension pneumothorax;
- Massive hemothorax;
- An open pneumothorax;
- A flail chest segment;
- Pulmonary contusion;
- Rib fracture (single or multiple);
- Cardiac tamponade.

If a chest X-ray or HRCT of the chest reveals a hemothorax or tension pneumothorax, then emergency large-caliber tube thoracostomy/water seal drainage of the chest must employed.

Adjuncts to primary survey:

- Hemodynamic monitoring (blood pressure, cardiac rate, and rhythm);
- ECG monitoring;
- Foley catheter placement for urinary output monitoring and to obtain evidence of a possible urethral injury (per-urethral bleeding, perineal hematoma);
- Naso-gastric tube insertion and prevention of gastric dilatation and gastric content regurgitation;
- Respiratory function monitoring with respiratory rate, pulse oximetry, and capnography.
- Analysis of arterial blood gas (ABG);
- CBC, serum electrolytes, blood glucose, serum creatinine, PT, APTT, INR, blood grouping, and cross matching.

Radiographic investigation:

- X-ray of the skull b/v, cervical spine, abdomen, pelvis, and long bones according to the clinical situation;
- CT scan of the head along with the cervical spine;
- HRCT OF CHEST when the chest X-ray presents results that are a cause of concern;
- Ultrasound of the abdomen (Nader et al. 2014; CRASH Trial Collaborators 2004).

C. Circulation Preservation with Bleeding Control

Hemorrhage is the primary etiology of avoidable post-injury mortality. Hypovolemia is induced by significant traumatic hemorrhages. A prolonged shock state leads to multi-organ failure and cell death.

1. Clinical signs of shock:
    - Altered mental status.
    - Pale, cold, and clammy slins. Delayed capillary refill (>3 s).
    - Pulse: rapid (heart rate > 100), thread pulse, or peripherally unpalpable.
    - Arterial hypotension (systolic blood pressure < 120 mm hg).
    - Decreased urinary output (UOP < 0.5 mL/kg/h).
2. Source of bleeding:
    - Scalp, skull, or contused brain tissue;
    - Chest;
    - Abdomen;
    - Pelvis;
    - Long-bone fracture.

3. General management of shock:

Stop bleeding by applying direct pressure, a crepe bandage on the patient's head, and compression or ligation of the superficial temporal artery.

Occult bleeding, either from the pelvis or the long bones, needs to be addressed quickly.

Two large-bore intravenous lines have to be administered, and crystalloid saline must be given.

Urgent blood grouping and cross-matching and rapid blood transfusion are needed to improve the patient's hemodynamic status (Nader et al. 2014; Kalangu et al. 2009).

D. Disability/Neurologic Assessment

The Glasgow Coma Scale (Table 1) is used to conduct a basic neurological examination during the primary survey. The level of consciousness (LOC) of the patient; pupil size, equality, and reaction; lateralizing symptoms; and the level of spinal cord injury are all determined in this way. The Glasgow Coma Scale is a rapid measure for determining state of consciousness and is a predictive factor of patient outcome. A change in LOC necessitates a reassessment of a patient's oxygenation, perfusion, and ventilation. Hypoglycemia and substances such as alcohol might affect one's degree of awareness. If these factors are ruled out, any changes in degree of consciousness are assumed to be the result of traumatic brain damage until proven otherwise.

Table 1. GCS scale (Glasgow coma scale).

	1	2	3	4	5	6
Visual	Eyes closed	Eyes open in response to sharp stimuli	Eyes open in response to sounds	Eyes open without induced stimuli		
Motor	No movement	Movement in response to sharp stimuli	Muscle flexion In response to sharp stimuli	Muscle flexion and bodily movement	Ability to localize touch	Appears to have normal Movement
Verbal	No sounds	Low-intensity sounds	Incoherent words	Understandable words are spoken	Normal conversation	

Highest score—15; lowest score—03. Source: Table adapted from Teasdale and Jennett (1974), used with permission.

E. Exposure (Full) and Environmental Control

The patient should be totally exposed by cutting off their clothes and under garments. Examine them for other signs of injury.

Logroll the patient to examine their back.

Prevent hypothermia by applying a warm blanket and warm IV fluid.

7.2.4. Secondary Survey

The secondary survey is started after the primary survey has been completed and the patient has been adequately resuscitated. It includes the following measures:

- History taking, including the site of the trauma, the nature of the trauma, and its sequelae;
- Complete physical examination (head to-toe) identifying all anatomic injuries;
- Ascertainment of allergy history;
- Medication history, particularly with respect to cardiac anticoagulants such as Ecosprin/clopidogril and diabetic medications
- Determination of medical/pregnancy history;
- Ascertainment of the time when the patient's last meal was eaten.

7.2.5. Definitive Care

According to the clinical and other data, the patient is shifted to the ICU, HDU, or operation theatre for definitive treatment.

All clinical and radiological data are collected for definite surgical treatment or ICU management (Kalangu et al. 2009; Kamel et al. 2011; Cruz 1998).

7.3. Management of Increased ICP

7.3.1. Elevation of Head End of the Bed

- A 30° or reverse Trendelenburg will reduce ICP;
- Set the patient's head as well as neck in a neutral position that improves venous drainage of the brain;
- Avoid compression of internal jugular veins with tight C-collars or fixation of the endotracheal tube.

7.3.2. Osmotic Diuresis

Therapies involving the use of either mannitol or hypertonic saline decrease brain swelling and edema, hence decreasing ICP, which saves some time for preparing the patient for definitive surgery.

Mannitol (Muizelaar et al. 1984):

- Used if the patient's SBP is more than ninety mmHg;
- 20% mannitol, with a bolus dose of 0.25–1 gm/kg, is used as fast infusion over 15–20 min;
- It decreases ICP within thirty minutes, and the duration of this function is 6–8 h;
- Monitor the input/output to continue euvolemia during possible diuresis, and use normal saline to substitute the volume;
- Do not use mannitol as a continuous infusion, as it passes the BBB after long-term infusion and increases cerebral edema.

Hypertonic saline can be more efficacious than mannitol, and it is currently considered a standard agent of care (Kamel et al. 2011).

- Most researchers utilized a 250 mL i.v. bolus of 7.5% saline with dextran;
- A 250 mL initial bolus dose of 3% NaCl solution will decrease ICP and can be administered through a peripheral i.v. channel;
- The expected sodium level is 145–155 mmol/dL;
- Hypertonic saline has a greater osmotic gradient and is less permeable through the BBB than 20% mannitol.

7.4. Seizure Control

Seizures are immediately managed with benzodiazepines and antiepileptic drugs (AEDs), and commonly injections of Phenytoin or Fosphenytoin are administered.

Seizure prophylaxis decreases the severity of seizures, though it does not improve long-term result.

7.4.1. Risk Factors for Post-Traumatic Seizures

- GCS < 10 (initially);
- Contusion of the cerebral cortex;
- Depressed fracture of the skull
- Subdural hematoma (SDH) and/or epidural hematoma (EDH);
- Subarachnoid hemorrhage (SAH) or intra cerebral hematoma;
- Penetrating wound of the head;
- Seizure within first 24 h of TBI;
- Treat any clinically obvious and definite seizures assessed via EEG;
- Consider a prophylactic anti-convulsant for TBI patients with any of the risk factors previously mentioned (Wikem 2019).

7.4.2. Use of Phenytoin or Fosphenytion First-Line Agent According to BTF Guidelines

- The loading dose (Phenytoin equivalent) is 20 mg/kg i.v. and then 100 mg i.v. q8 h for 7 days;
- Assay serum levels to achieve and maintain therapeutic serum levels;
- Levetriacetam may be utilized as an alternative;
- Administer a 20 mg/kg load i.v., followed by 1000 mg i.v. q12 h for 7 days;
- Levetiracetam may have less common but severe adverse effects analogous to those induced by phenytoin (Cruz 1998; Bullock et al. 2006).

7.4.3. Bring Down the Metabolic Rate

- Apply enough sedative and analgesia;
- Prevent hyperthermia and treat fever vigorously.

7.4.4. Barbiturate Coma

- Is used for raised ICP resistance to maximum medical as well as surgical treatment;
- Solely for patients with stable hemodynamics;
- Administer the following:
  - Pentobarbitone (10 mg/kg) over 30 min;
  - Followed by 5 mg/kg/h for 3 h;
  - Then, 1 mg/kg/h.

7.5. Other Critical Care Measures

- Cushing's ulcers (Stress ulcer) prevention with H2 blocker/PPI and sucralfate.
- DVT prevention by using sequential compression devices (SCDs), with no anticoagulation.
- Glycemic control is good, although tight maintenance is not recommended.
- Steroids, particularly methylprednisolone, are contraindicated for head injuries (CRASH Trial Collaborators 2004). Dexamethasone administration is not commonly practiced by any hospitals and institutions in head injury cases. However, some surgeons use it in some severe head injury cases either every 6 or 8 h for a short period of time.
- Routine paralysis is not advised.
- There is a greater risk of pneumonia as well as ICU length of stay (Cruz 1998; Bullock et al. 2006).

7.6. Tetanus Prophylaxis

- Tetanus-prone wound: >6 h after damage, avulsion: >1 cm deep, or crush-type injury: devitalized, polluted, or ischemic tissue;
- If the wound is tetanus-prone, provide 250 units of tetanus immune globulin intramuscularly (Bullock et al. 2006; Kalangu et al. 2009).

8. Skull and Skull Base Fractures

8.1. Classification of Skull and Skull Base Fractures

A. There are different types of skull fractures:
   - Linear skull fractures;
   - Diastatic fractured or sutural fractures:—separation of sutures due to a blunt blow on head with a blunt weapon;
   - Ping-pong fractures;
   - Growing fractures;
   - Compound/comminuted fractures:—fractures with two or more fracture lines that meet, dividing the bone into three or more fragments. When the fragment is not displaced, it resembles a spider's web or a mosaic design;
   - Depressed fractures (Figures 3 and 4):
     - A simple depressed fracture or closed fracture, that is, a fracture in which the inside of the skull is not exposed.
     - A compound depressed fracture or open fracture, in which the inside of the skull is exposed to the exterior environment. This type is commonly associated with a dural tear, which may cause meningitis and or brain abscesses.
     - A technical compound fracture, that is, a type of fracture of the air sinuses that exposes them to the environment outside the skull (Kalangu et al. 2009; Haddad and Arabi 2012).

B. Classification of fractures according to site:
   - Frontal bone fracture;
   - Temporal bone fracture;
   - Parietal bone fracture;
   - Posterior fossa fracture;
   - Orbital bone fracture;
   - Fracture of the basilar skull.

C. Findings of fracture of temporal bone:
   - Boggy temporalis muscle due to extravasation of blood;
   - A bruise posterior to the pinnae, i.e., in the mastoid area (Battle's sign), and otorrhea.

D. Findings of fracture of patrous temporal bone

- Hearing loss, CSF otorrhea, and bulging of the ear drum due to blood or CSF.
- Ipsilateral facial palsy.

E. Findings of mastoid temporal bone fracture
- Otorrhea, rhinorrhea, bulging of ear drum, Battle's sign, tinnitus, and vertigo.

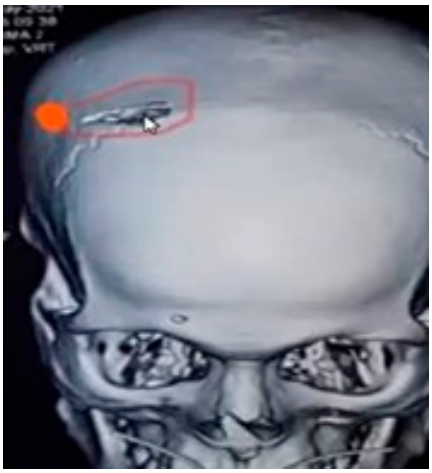


Figure 3. Three-dimensional CT image showing a depressed fracture close to the coronal suture. Source: Figure by authors.

Figure 4. A frontal depressed fracture. Source: Figure by authors.

8.2. Surgical Treatment

Surgical toileting and elevation of the depressed fracture are employed for treatment.

Generous washing with normal saline, povidone iodine, and hydrogen peroxide is required to avoid infection of the depressed fragments and underlying brain. Bone fragments are either removed or repositioned after being washed with antiseptic.

Dural tears need to repair via a direct stitch or augmented via the pericranium.

8.3. Complications of Depressed Fracture

1. Infection leads to meningitis and subsequently brain abscess formation;
2. Epilepsy—early- or late-seizure disorder;
3. CSF leak: A nasal fracture may form a connection between the subarachnoid spaces and paranasal air sinus or middle ear cavity. Rhinorrhea or otorrhea may result. Infection may lead to meningitis and its consequences.

8.4. Pond Fracture/Ping Pong Fracture

Ping pong fractures occur in newborns and young infants when their skulls are relatively flexible and robust, allowing the bone to indent without breaking. This type of fracture gets its name from its resemblance to the

depression left by a ping pong ball (Figure 5). Ping pong fractures have been reported in both accidental and non-accidental circumstances, including birth traumas. They are rarely linked to intracranial damage. Depending on the severity of the depression, it can be treated conservatively or surgically (Haddad and Arabi 2012; Khan and Banerjee 2010).

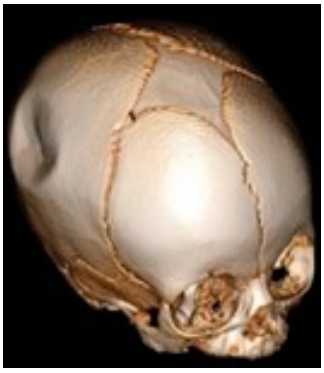


Figure 5. CT scan showing a ping-pong fracture. Source: Figure by authors.

8.5. Growing Fracture

A post-traumatic leptomeningeal cyst or growing fracture is a very rare pediatric head injury. Its formation requires a rapidly growing brain (Figure 6).

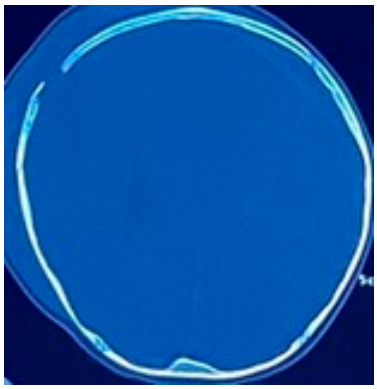


Figure 6. CT scan showing a growing skull fracture. Source: Figure by authors.

Surgical treatment: Craniotomy and closure of the dural defect. Dural augmentation from the pericranium or fascia lata is commonly required to close the defect.

8.6. Skull Base Fracture

Skull base fractures are estimated to constitute 4% of all head trauma cases.

8.6.1. Types

There are three types of skull base fractures:

1. Longitudinal fractures (the causes of these fractures are given below):
   (a) Blunt impact on the face as well as forehead or back of head;
   (b) Compression from front to back or back to front.
2. Transverse fracture—This type of fracture is caused by an impact either on the side of head or by side-to-side compression.
3. Ring fracture.

8.6.2. Clinical Manifestations

A. The frontal, ethmoidal, and sphenoidal sinuses may be involved in a fracture of the anterior skull base (because of direct contact with the chin), resulting in blood loss from the nose or mouth. CSF and

even brain matter can seep into the nose (CSF rhinorrhea) in a cribriform fracture. Bilateral preorbital ecchymosis is also known as Raccoon's eyes (black eyes) (Figure 7A).

B. A middle fossa fracture of the basi-occipital or sphenoid bone and sellae turcica may result in bleeding from the mouth.
C. Fracture of the petrous temporal bone (as a result of a direct impact posterior to the ear) may result in blood as well as CSF coming out from the ear (CSF otorrhea), and blood may pass to the oropharynx via the auditory tube or hemorrhage from the ear as a result of the rupture of the posterior branch of the middle meningeal artery.
D. A posterior fossa fracture (resulting from a direct impact to the back of the head) causes bleeding behind the mastoid process and can result in a large hematoma at the back of the neck and ecchymosis of the mastoid process (known as Battle's sign).
E. Fracture of the foramen magnum, cerebellar contusion, and edema can cause fatal cerebellar tonsillar herniation and cranial never injury (stretched or bruised) (Khan and Banerjee 2010; Joswig et al. 2016).

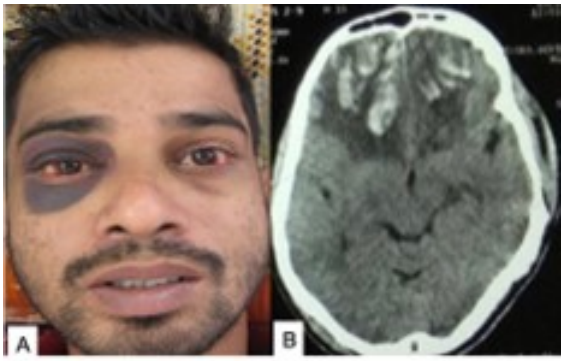


Figure 7. (**A**) Patient with black eye due tofront-basal fracture. (**B**) CT findings showing a bifrontal basal hemorrhagic contusion caused by a road traffic injury. Source: Figure by authors, used with patient's consent.

CT scanning is the most valuable diagnostic process that usually shows fractures of bone, CSF in air sinuses, and pressure in the subdural space or different parts of the intracranial cavities.

8.6.3. Treatment

Conservative treatment consisting of antibiotic administration and bed rest is the usual method of treatment for a skull base fracture.

In the case of optic nerve compression due to depressed bone fragments, surgery is required to decompress the optic nerve.

In most cases, CSF leakage ceases spontaneously in a few days. In this case, when CSF rhinorrhea persists, a lumbar drain and bed rest are options to contemplate before considering surgery to repair the dural defect either via endonasal an endoscopic approach or a bifrontal basal craniotomy. The risk of meningitis is high, and therefore it is essential to treat the patients with broad-spectrum antibiotics for at least two weeks (Khan and Banerjee 2010; Joswig et al. 2016).

9. Cerebral Injury/Brain Injury

TBI is one of the main causes of mortality and morbidity for children and young adults all over the world. Males are two times more likely to suffer a TBI than girls. Falls, automobile collisions, and violence are among the causes. A quick acceleration or deceleration within the brain box, or a complicated mix of movement and sudden impact, causes brain trauma. A multitude of events after the injury may result in a subsequent injury, in addition to the damage suffered at the time of injury.

9.1. Classification of Head (Brain) Injuries

9.1.1. According to Severity

Head injuries may be mild, moderate, or severe depending upon the affected individual's level of consciousness (Table 2).

Table 2. Severit y and level of consciousness.

Severity	GCS	PTA (Post-Traumatic Amnesia)	LOC (Loss Of Consciousness)
Mild	13–15	<1 day	0–30 min
Moderate	9–12	1 to <7 days	>30 min to <24 h
Severe	3–8	>7 days	>24 h

For children under the age of five, a somewhat different version of the GCS is employed. TBIs are classified as mild, moderate, or severe, based on patient's GCS score: Source: Authors' compilation based on data from Greenberg 2010.

Mild/Minor head injury—GCS of 13 or higher: The majority of head injuries are minor. The patient may be conscious or lose consciousness for anywhere from a few seconds to minutes at a time. Confusion, memory problems, headaches, and behavioral issues are all common symptoms. The majority of persons who present with modest head injuries will not experience any further complications. Mild head injuries can be classified into two groups: low-risk and moderate-risk. Low-risk injuries are defined as those causing mild to moderate headaches, dizziness, and nausea. After a thorough assessment, many so-affected individuals require just modest surveillance, and many do not require radiographic evaluation (CT scans). These patients may be discharged if they can be monitored by a trustworthy individual.

Moderate-risk-group patients on anticoagulant treatment, even if they have suffered minor head trauma, should be subjected to radiographic imaging (CT scan) since a slight head injury can escalate into a catastrophic injury.

Moderate head injury—GCS of 9 to 12: These patients may be released if they can be monitored by someone they can trust. Patients on anticoagulant therapy should undergo radiographic imaging (CT scan), even if they have had minimal head trauma, because even a modest head injury can lead to a catastrophic injury.

Severe head injury—GCS of 8 or lower: In this type of injury, for more than six hours, the patient will remain unconscious and in a comatose condition. Because there is a possibility of catastrophic brain damage, severe head injuries require prompt medical attention. An urgent CT scan is required to estimate the severity of the injury.

9.1.2. Types of Head (Brain) Injury According to Brain Parenchymal Injury

Concussion

The most prevalent sort of head injury is concussion. In this case, for a brief amount of time, the patient loses consciousness. A concussion occurs when the brain is jostled or shaken forcefully enough to cause it to bounce against the skull. It varies in severity from minor to severe.

Contusion

A contusion is a bruise on the brain itself. It could be a bleed or an edematous swelling of the brain (Figure 7B).

Cerebral Laceration and Intracranial Hematoma (ICH)

These terms refer to a clot in the brain that forms under the skull. The severity of brain hematomas varies from minor to severe, and they are classified according to their origin. In injury, there is a lack of brain tissue continuity. Pia matter ruptures and subarachnoid hemorrhages are common side effects of surface lacerations. Bone shards tear the brain surface in depressed fractures.

Laceration of brain tissue is caused by any penetrating injury.

In traumatic intracerebral hematoma (ICH), there are four CT findings considered to be significant:

(1) A shift of 5 mm or more in the septum pellucidum, third ventricle, or pineal gland;
(2) Pentagonal cisterna ambient (compressed or collapsed);
(3) Effacement of peripheral sulci (ipsilaterally or diffusely);
(4) Decreased lateral ventricles (uni- or bilateral).

9.1.3. Classification of Head Injuries Depending on Site of Impact

Focal

This type occurs due to a direct overhead impact resulting in a skull fracture, contusion, EDH (extradural hematoma), acute SDH (subdural hematoma), or cerebral laceration, and an intracerebral hematoma may form.

Contusions are multiple small hemorrhages in the surface layers of the brain. They can form at the impact site and/or on the side of the head contralateral to the impact site. A 'coup' injury occurs when a lesion develops at the site of impact, while a 'contrecoup' injury occurs when a lesion occurs on the side of the brain contralateral to the site of impact. The movement of the brain within the skull cavity causes contrecoup contusions (Figures 8 and 9).

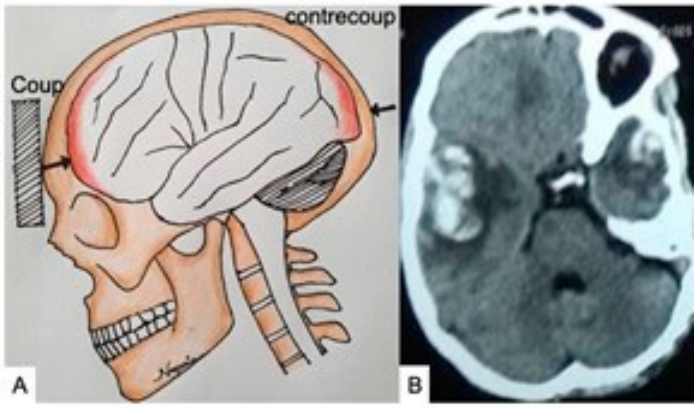


Figure 8. (**A**) Diagrammatic presentation of coup and contrecoup effect. (**B**) CT scan showing a hemorrhagic contusion in both temporal lobes as a result of coup and contrecoup effects. Source: Figure by authors.

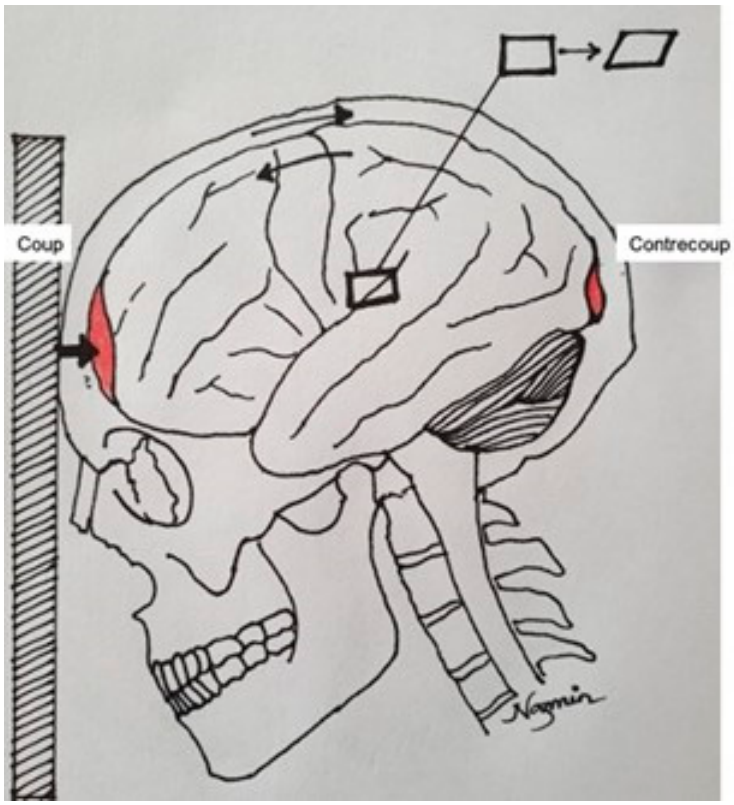


Figure 9. Diagrammatic structure of brain showing coup and contrecoup effects. Source: Figure by authors.

Diffuse Axonal Injury (DAI)

A DAI is a primary brain injury resulting from an accelerating–decelerating impact. The sudden deceleration produces a shearing force that disrupts axons as well as small vessels (Figure 9). Axonal damage results in localized transport failures in the corresponding axon, resulting in swelling and, in some cases, axonal lysis with Wallerian degeneration.

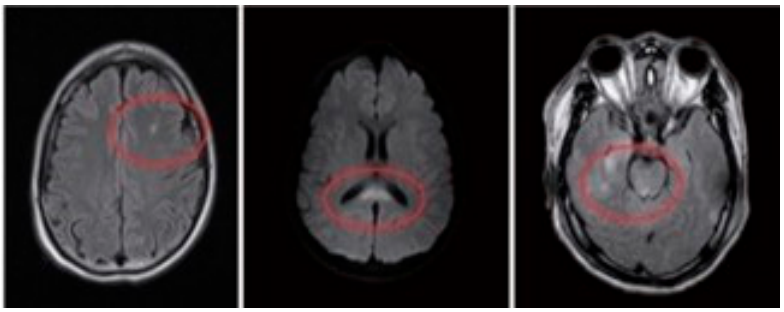
A. Grading of DAI

- Mild DAI: Coma of >6 to 24 h. Mild to severe memory loss as well as mild to moderate impairments follow.
- Moderate DAI: Coma > 24 h. Confusion and long-term amnesia are followed by mild to severe behavioral, memory, and cognitive impairments.
- Severe DAI: With flexor and extensor posturing, the coma can last months. Deficits in cognition, memory, speech, sensorimotor control, and personality are induced.

The effect of a DAI on reticular formation (without a space-occupying lesion observable via CT) induces loss of consciousness (although a DAI can be present alongside subdural or epidural hematomas).

B. Radiological features of DAI

A focal hemorrhagic or edematous lesion in the corpus callosum, parasagittal white matter, septum pellucidum, third-ventricle wall, and/or dorsolateral brainstem can be seen. These lesions are identified as hyperintense lesions in T2W MRI. Because it can identify both hemorrhagic and non-hemorrhagic lesions, MRI is markedly more sensitive than CT (Figure 10).



(**A**) (**B**) (**C**)

Figure 10. (**A**) MRI (axial image) shows small hemorrhage in frontal white matter and (**B**) hemorrhage in splenium of corpus callosum. (**C**) MRI image (axial view) showing hemorrhage in the rostral midbrain and medial temporal lobe. Source: Figure by authors.

In gradient recalled echo (GRE) and susceptibility-weighted (SW) images, acute hemorrhagic lesions show localized susceptibility and signal loss due to the paramagnetic effects of deoxyhemoglobin (SWI). When comparing 1.5 Tesla (3T) to 3 Tesla (3T), these susceptibility effects are amplified, with about two times as many hemorrhagic lesions visible at 3T. Non-hemorrhagic lesions are best seen via fluid attenuation inversion recovery (FLAIR) imaging, which makes subcortical as well as periventricular lesions stand out (in comparison to T2W images). Diffusion-weighted imaging (DWI) is also sensitive to DAI and able to detect lesions that are not visible in GRE or FLAIR images. In the acute context, DAI lesions are often hyperintense, with low apparent diffusion coefficient (ADC) values, indicating cytotoxic edema (Kim and Gean 2011; Joswig et al. 2016; Timofeev et al. 2012). Classification of DAI is shown in Table 3.

C. MRI grading of DAIs

MRI can sequentially detect punctuate micro hemorrhages in the white matter, corpus callosum, and midbrain.

Grade I: The hemorrhage is restricted to the cerebral cortex.

Grade II: The hemorrhage is restricted to the corpus callosum.

Grade III: The hemorrhage extends into the midbrain.

D. Outcome of DAI

The average amount of time it takes to regain consciousness is 20–33 days. The median time to regain awake status differs significantly for different groups. If a grade I patient takes 5 days to regain consciousness, grade

II and III patients will take 2 and 4 weeks, respectively. As the MRI grading improves, so does the length of time spent in the ICU and hospital. Finally, depending on the patient, the duration of mechanical ventilation can increase. A hemorrhage in a DAI-type lesion, especially when associated with traumatic space-occupying lesions (such as EDH, SDH, and ICH), is a poor prognosticator. Isolated DAI-type lesions that are not hemorrhagic are not linked to a poor clinical outcomes.

Table 3. Classification of diffuse axonal injuries.

Grade MRI Findings	Neuropathologic Findings	Stage
1: White matter	Microscopic axonal injury in the white matter of the hemispheres, corpus callosum, brain stem, and/or cerebellum, without hemorrhagic or necrotic lesions in the corpus callosum or superior cerebellar peduncles.	01. Traumatic lesion limited to lobar white matter or the cerebellum only.
2: Brain Stem	Microscopic or macroscopic hemorrhagic or necrotic lesion in the brain stem and corpus callosum	02. Traumatic lesion in the corpus callosum with or without lobar white matter lesion.
3: Cerebellar peduncle	Microscopic or macroscopic injury in the lobar callosum. Hemorrhagic or necrotic lesions in the dorsolateral quadrants of the rostral brain stem.	03. Traumatic lesion in the brain (dorsolateral quadrant of the brain stem and superior cerebellar peduncles) with or without lesions in white matter or the corpus.

Source: Authors' compilation based on data from Pascual and Prieto (2012).

9.1.4. Another Classification of Head Injury (Based on Mode)

Closed head injury—This type of is usually caused by a car accident, a fall, or shaking (for babies). A closed head injury is the result of rapid forward and backward movement as well as shaking of the brain parenchyma inside the brain box.

Penetrating head injury—When the head is penetrated by a sharp cutting instrument, bullet, or pellet.

9.1.5. Another Classification of Head Injury (Based on Timing)

Primary head injury—This type of injury happens at the time of the impact. It can be a focal or diffuse type of injury. EDH, SDH, and traumatic ICH are types of primary head injury.

Secondary head injury—This type refers to changes that occur over time (from hours to days) after a primary brain injury. It involves a series of neuronal, metabolic, and vascular alterations in the brain, all of which contribute to further brain tissue death. Cerebral swelling, cerebral edema, cerebral ischemia and infarct, and brain herniation occur after a primary head injury.

Cerebral Swelling

The literature is divided on whether cerebral edema (Figure 11) or hyperemia with an increased volume of blood is the root etiology of brain swelling. It is possible that both mechanisms are at work. The main mechanism of cerebral swelling, according to popular belief, is cerebral hyperemia caused by dysautoregulation with engorgement of vessels and increased CBF. Vasogenic edema occurs when the BBB is compromised, allowing extracellular water to accumulate, and cytotoxic edema occurs when cell membrane pumps fail, enabling intracellular water to leak. As evidenced by the higher water content observed in this condition, cytotoxic edema may be the primary cause of cerebral swelling among these two forms of edema. Sulcal effacement, basilar cistern compression, and flattening of the ventricular borders are all signs of cerebral edema caused by hyperemia. The attenuation and differentiation of gray and white matter are preserved. Cerebral swelling caused by vasogenic edema, on the other hand, will show up as low-attenuation patches, whereas cytotoxic edema will show up as an absence of a gray–white distinction. Diffuse cerebral swelling is more common in pediatric patients than adults after a head injury, with the prevalence of diffuse swelling being two times greater in children than in adults. Post-traumatic dysautoregulation, which causes vasodilation, hyperemia, and cerebral edema, is more common in children and young adults. When the swelling is significant, the ICP rises and the CPP drops, resulting in cerebral

infarction and brain damage. Head raising, controlled moderate hyperventilation, mannitol administration with hyperosmolar therapy, and the careful use of sedatives and analgesics to avoid pain or agitation from raised ICP are all therapeutic strategies used to fight the detrimental effects of cerebral swelling. Decompressive craniectomy (DC) can be performed to try to reduce ICP when it is unresponsive to maximal medical therapy. DC has been shown to reduce ICP and enhance functional outcomes in a number of studies. DC also improves radiologic outcomes by allowing for better viewing of basilar cisterns and a reduction in midline shift, both of which are linked to a better outcome.

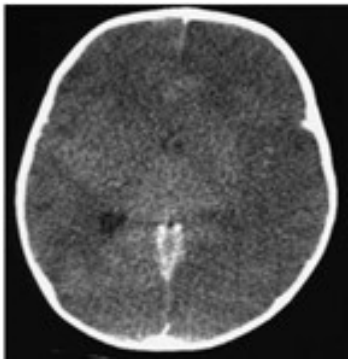


Figure 11. CT scan revealing generalized cerebral edema. Source: Figure by authors.

Cerebral Herniation

Cerebral herniations (Figure 12) are the result of an unmitigated increase in ICP. The process of the cingulate gyrus herniating below the falx cerebri is known as subfalcine herniation or midline shift. When the medial part of the temporal lobe/uncas herniates through the tentorial incisura and compresses the suprasellar cistern, this is known as uncal herniation (Kim and Gean 2011). Caudal herniation of both temporal lobes through the tentorial incisura causes descending transtentorial herniation, compressing the basilar cisterns. The cerebellum extends through the tentorial incisura and effaces the quadrigeminal cistern in upward transtentorial herniation, which happens in the opposite way. The cerebellar tonsillar protrusion into the foramen magnum is known as tonsillar herniation (Kim and Gean 2011). A dilated pupil is a clinical indication of cerebral herniation in the absence of CT imaging results. The herniated medial temporal lobe puts pressure on the same side of the third nerve, resulting in the lack of normal light reflexes exhibited by those with uncal herniation. Cerebral infarction and the Duret hemorrhage, which often occur in the anterior and paramedian midbrain/pons following fast caudal herniation, are two more serious consequences of cerebral herniation. The pathophysiology of the deleterious effects of the Duret hemorrhage is assumed to be either the rupture of the pontine perforator from the basilar artery or venous thrombosis with an infarct (Kim and Gean 2011; Quiñones-Hinojosa 2012; Gooch et al. 2009).

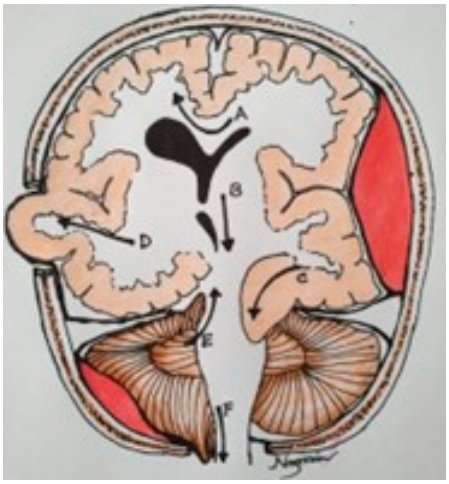


Figure 12. Schematic presentation of different types of brain herniation; A—subfalcine, B—central, C—Uncal, D—transcalvarial, E—upward cerebellar, and F—tonsillar herniation. Source: Figure by authors.

Cerebral Ischemia and Infarction

Cerebral ischemia as well as infarction affect 2% of patients who undergo a CT scan of the head for a head injury and can be caused by multiple factors. Ischemia is usually caused by a blood vessel being mechanically compressed by a brain herniation through the falx cerebri and/or tentorium. In acute subfalcine or uncal herniation, infarctions are due to a mechanical shift of the anterior cerebral artery (ACA) or posterior cerebral artery (PCA) distribution, respectively. In subfalcine herniation, the calloso-marginal branch of the ACA might be squeezed against the free border of the falx, causing infarction. The herniated medial temporal lobe may press the PCA and cause an infarction, or it can compress the anterior choroidal artery and cause an infarction of the internal capsule (the posterior limb) in uncal herniation. Vasospasm is another possible etiology of ischemia and infarction in TBIs. Extra-axial hematomas, which apply a great deal of pressure on the neighboring cortex, can also squeeze cortical veins, resulting in a venous infarction. Lastly, ischemia can occur as a result of direct arterial injury, such as blockage, dissection, or pseudoaneurysm caused by a fracture of the base of the skull.

10. EDH (Extradural Hematoma)

EDHs account for 1% of head trauma. They commonly occur in young adults, commonly following a temporo-parietal skull fracture that disrupts the MMA (middle meningeal artery), causing arterial bleeding between the dura mater and bone.

The main source of bleeding (85%) in the middle meningeal artery and the rest is either the middle meningeal vein or the dural sinus. Fracture of the temporal or frontal bone via trauma or a head fixation pin leads to an injury to the dura and dural blood vessels (main trunk, frontal branch, or parietal branch).

The main site for EDH is the pterion; the other sites are the frontal, occipital, and posterior fossa.

10.1. Presentation

Typical presentations are as follows:

1. Transient (post-traumatic) loss of consciousness (LOC) due to concussion;
2. Followed by a lucid interval for several hours (in 30–40% cases);
3. Followed by obtundation, contralateral hemiparesis, and ipsilateral pupillary dilation.

Other presentations are headache, vomiting, bradycardia, seizure, hyper reflexes, and being Babinski-sign-positive.

Contralateral hemiparesis is the most typical presentation of EDH; however, ipsilateral hemiparesis may occur due to compression of the contralateral cerebral peduncle against the tentorial free margin—the so-called Kernohan's notch phenomenon. It is a false localizing sign.

4. If both pupils are dilated, then the patient may also develop decorticated or decerebrated rigidity.

10.2. CT Appearances of EDH

Classical EDH (Figures 13 and 14) looks like a hyperdense lenticular shape adjacent to the skull. It is limited by sutures as the external dural layer is tightly attached to the sutural line.

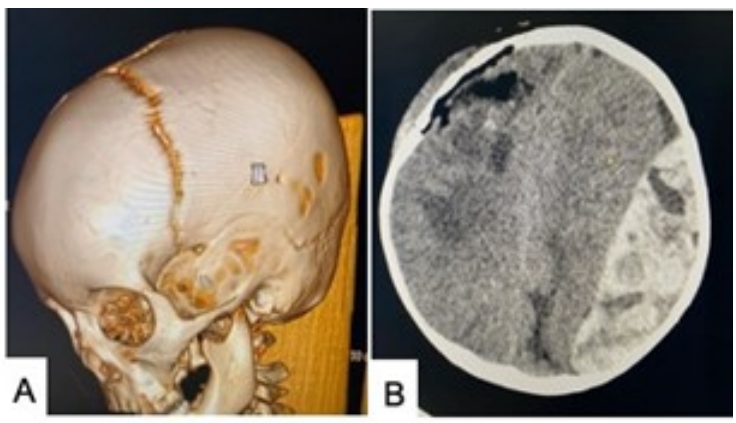


Figure 13. (**A**) A 3D CT scan showing a depressed fragment in temporal bone. (**B**) A huge EDH found in the tempero-parietal region of the same patient. Source: Figure by authors.

A total of 5–20% of EDHs are bilateral. EDHs may extend in the cranial and caudal directions with respect to the tentorium cerebelli. The hematoma volume can be calculated using the formula ABC/2 (for which the CT slice containing the greatest hemorrhage is chosen), where A represents the diameter of the hematoma on that slice, B represents the measurement taken 90° to A, and C represents the approximate number of 10 mm slices containing the hematoma (Kim and Gean 2011).

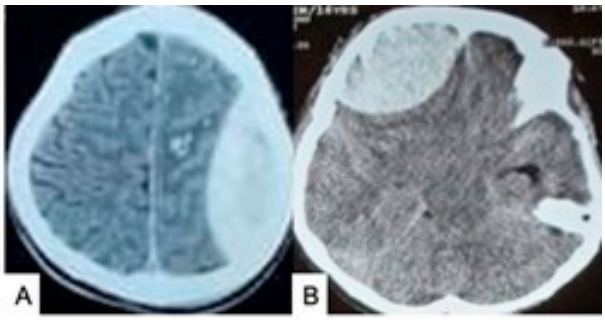


Figure 14. CT scan showing a typical extradural hematoma: (**A**) parietal convexity EDH; (**B**) frontal EDH. Source: Figure by authors.

10.3. Indications for EDH Surgery

1. Surgical evacuation is recommended for epidural hematomas (EDHs) > 30 cm^3 regardless of GCS, according to recent guidelines (Vella et al. 2017).
2. Surgery is also recommended for patients who have EDH and GCS scores less than 9, a clot thickness greater than 15 mm, a midline displacement greater than 5 mm, or localized neurologic impairments.

10.3.1. Conservative Treatment of EDH

Epidural hematomas measuring < 30 cm^3 and that are <15 mm thick and have a <5 mm shift in patients with a GCS > 8 and no focal deficits can be followed up carefully and subjected to serial imaging (with repeat scans every 6–8 h).

Controversy appears to surround the nonsurgical management of acute epidural hematomas that are asymptomatic or nearly so. Regardless of whether this conservative approach may be contemplated for supratentorial hematomas far from the perimesencephalic cistern, many surgeons strongly recommend that all acute epidural hematomas in the temporal fossa or the posterior fossa be immediately removed, even if they are small. This is because these patients may initially appear relatively asymptomatic while harboring a "small" clot. The latter, however, may rapidly enlarge, leading to dramatic, life-threatening changes.

10.3.2. Operative Technique

An extensive fronto-tempro-parietal craniotomy is commonly carried out by keeping the patient's head on a horseshoe head rest or a three pins' head fixator (Figure 15). A wide question-mark- or c-shaped incision is made on the front or back of the external acoustic meatus. Skin along with temporalis muscle are removed as a single flap. Bone fractures (if any), either linear or depressed, are evident following exposure. Multiple burr holes are made. Small blood clots commonly come out through the burr hole. A wide craniotomy is carried out using a high-speed craniotome. Extradural blood clots are removed via suction, irrigation, and a coup scoop or spatula. A common source of bleeding is the middle meningeal artery, in which the blood coagulates, and the vein along with it, in which the blood also coagulates. Sometimes the surgeons need to coagulate and ligate the origin of the MMA at the origin foramen spinosum level.

Following the cauterization and ligation of MMA, the waxing of the foramen spinosum must be conducted.

The application of dural tack-up sutures is always recommended, both centrally and peripherally. This maneuver prevents epidural hematoma re-accumulation, which is particularly relevant for patients with acute hypo-coagulopathic disorders or those in whom a bleeding source was missed intraoperatively because of temporary spontaneous hemostasis (Quiñones-Hinojosa 2012).

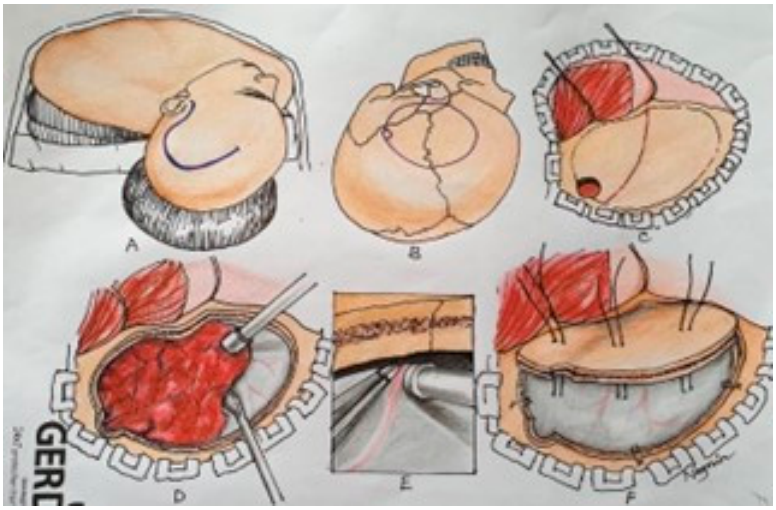


Figure 15. EDH surgical technique via the standard craniotomy. (**A**) The patient is put in the supine position, with a roll placed under their ipsilateral shoulder and their head turned to the opposite side. A large question-mark-shaped skin flap is marked out. If the patient suffers rapid neurologic deterioration due to mass effect, rapid decompression through a large temporal bur hole is initially carried out. (**B**) A large frontotemporal craniotomy is performed, with further exposure of the middle fossa being achieved by removing parts of the lateral sphenoid wing. (**C**) When the bone flap is turned, the hematoma is rapidly removed with suction, irrigation, and cup forceps. (**D**) In most cases, the principal bleeding source is the middle meningeal artery or the bone of its main branches lying in the dura mater, and this blood easily coagulates. (**E**) In other cases, the bleeding may originate either from the foramen spinosum or from a fracture running across the temporal bone, and the hemorrhage can be arrested by plugging it with bone wax. (**F**) After meticulous hemostasis in the epidural space is achieved with bipolar diathermy and hemostatic agents, "tack-up" sutures are placed at the periphery and in the middle of the bone flap, which is then reapplied. Source: Figure by authors.

10.3.3. Causes of Death from EDH

- Respiratory failure;
- Cerebral edema;
- Secondary pontine hemorrhage;
- Herniation of the uncas.

11. Acute Subdural Hematoma (ASDH)

ASDHs is more common than EDH. It occurs due to the rupture of surface or bridging vessels via the acceleration or deceleration types of violent head trauma. Here, blood accumulates between the dura mater and pale brain surface. Another cause of ASDH is associated with a burst temporal or frontal lobe. A rare cause is a rupture of the small cortical artery due to an overlying fracture of the bone.

In descending order of frequency, ASDHs usually occur over the cerebral hemisphere convexities, along the tentorium cerebelli, and along the falx cerebri. ASDHs look crescent-shaped on imaging and do not cross the midline (Figures 16 and 17) (Kim and Gean 2011).

Here, the lucid interval is not typical like that for EDH. In total, 10–40% of individuals with ASDH may experience a lucid interval. Patients commonly deteriorate rapidly.

The poor outcome of ASDHs largely results from simultaneous cerebral cortical injuries such as contusion, brain swellings, and DAI. Overall, 60–80% of patients are in a state of coma during diagnosis. The accumulation of 100–150 mL of blood may be fatal in acute ASDH.

CT scanning is the ideal tool for investigation. CT findings according to time frame are summarized in Table 4.

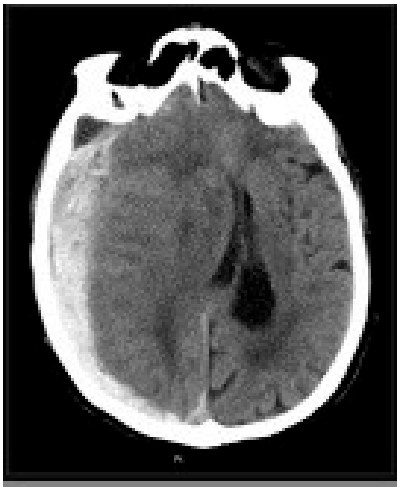


Figure 16. CT scan showing the typical crescentic shape of acute SDH in temporal convexity area with midline shift and biventricular effacement. Source: Figure by authors.

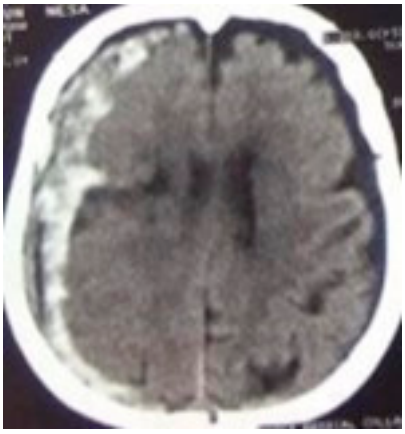


Figure 17. CT scan of the crescentic shapes of an acute subdural hematoma with no midline shift. Source: Figure by authors.

Table 4. ASDH density changes visible on CT scans over time (Figures 16 and 17).

Category	Time Frame	Density on CT
Acute	1–3 days	Hyperdense
Subacute	4 days–2/3 weeks	Isodense
Chronic	Usually, more than 3 weeks and less than 3–4 months	Hypodense (trending toward CSF density)
	After nearly 30–60 days	May take lenticular shape (akin to EDH) with density greater than CSF and less than fresh blood

Source: Authors' compilation based on data from Greenberg (2010); Vella et al. (2017).

11.1. Etiologies

1. Rupture of connecting bridging or veins.
2. Tear of inferior cerebral vein entering the venous sinuses at the skull base.
3. Rift or tear in dural venous sinuses.
4. Damage to cortical veins.
5. Contusion and/or laceration of the brain and dura.
6. Damage to earlier adhesions between the dura and brain.
7. Secondary to pathology, e.g., cerebral tumor, aneurysm, or hematological disorder.
8. Drugs (such as dicoumarol, heparin, and warfarin).

11.2. Management

11.2.1. Indications of Surgery

1. ASDH with a thickness of more than 10 mm, regardless of GCS;
2. ASDH with a midline shift of more than 5 mm, regardless of GCS;
3. GCS < 8 and ICP > 20 mmHg—thickness or midline shift is of less concern;
4. GCS < 8 and asymmetric or fixed and dilated pupils;
5. GCS < 8 and decrease of 2 or more points on GCS after hospital admission;
6. Secondary injury to the brain that is frequently associated with ASDH (Bishokarma 2018).

11.2.2. Surgical Treatment

Treatment consist of a wide craniotomy with a durotomy and removal of the subdural hematoma (Figures 18 and 19).

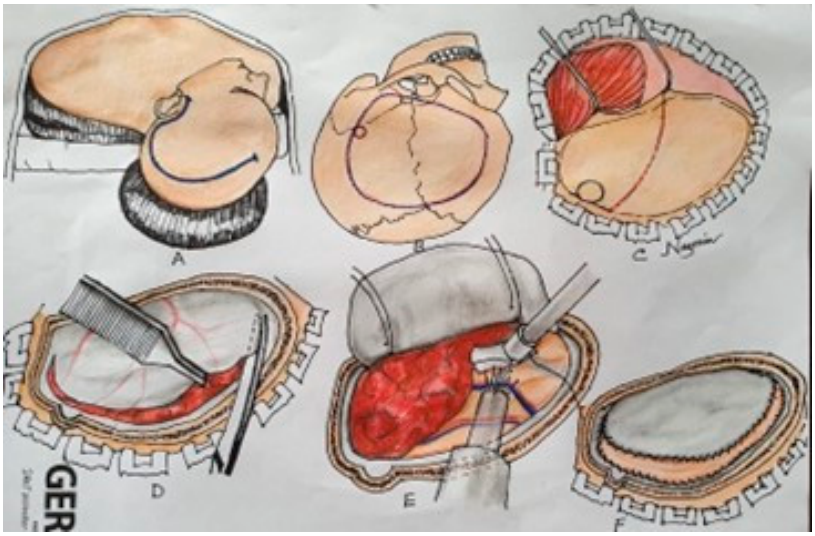


Figure 18. Schematic presentation of surgical evacuation of ASDH: (**A**) patient's position and incision; (**B**) marking indicating the site at which large fronto-parieto-temporal craniotomy will be performed; (**C**) subperiosteal dissection and removal of craniotomy bone flap; (**D**) durotomy; (**E**) evacuation of hematoma via gentle irrigation and suction; (**F**) expansile duroplasty through the pericranium. Source: Figure by authors.

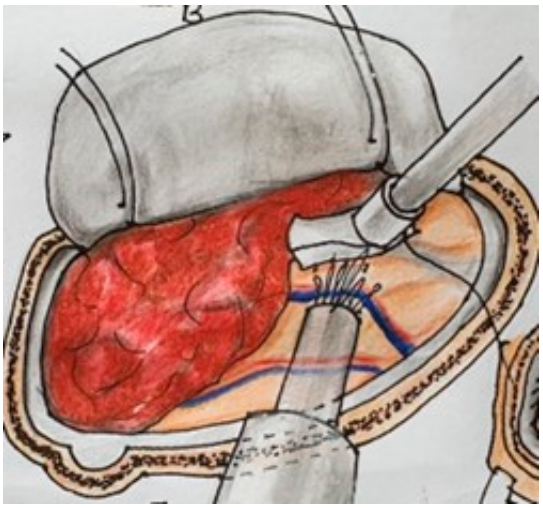


Figure 19. Diagrammatic perioperative picture of acute SDH showing clotted blood being sucked out using a suction device and gentle irrigation. Source: Figure by authors.

In some cases, a duroplasty is needed.

Some cases require a decompressive hemicraniectomy and a duroplasty depending upon the state of brain swelling.

11.2.3. SDH Removal Technique

A standard fronto-temporo parietal trauma craniotomy is performed. Once the bone flap is taken away, inverse U-shaped or Y-shaped or X-shaped (cruciform) dural openings can be adopted depending on the neurosurgeon's experience. A wide cruciate dural incision is carried out. Starting from the area of maximum clot thickness, the clot is evacuated with copious irrigation and suction. A bleeding point on the surface of the brain could be a connecting vein or an artery that can be coagulated via bipolar diathermry, or a hemorrhage could be caused by ragged dural borders or decaying brain tissue. The connecting vein is occasionally visibly avulsed, and it can be effortlessly coagulated with diathermy. Areas of a cortical contusion larger than 1 to 2 cm in diameter, with irreversibly injured brain that appears mottled, should be aspirated gently.

If a tear formed across a venous sinus, the neurosurgeon should be ready to deal with profuse sinus bleeding, and the sinus tear needs to be completely observable. The edge of the craniotomy may have to be expanded toward the sinus; at the same time, profuse bleeding may make this a difficult maneuver. In addition, it may be important to repair the sinus rip properly without interfering with sinus blood flow. In some circumstances, massive red cell transfusions may be required. Sinus ligation may be performed, but neurosutgeons choose to ligate solely the anterior quarter of the superior sagittal sinus while avoiding ligation involving more posterior segments of superior sagittal sinus.

After homeostasis has been ensured, the dura is sealed with a water-tight closure. Depending on the status of the brain, the bone flap is either replaced or preserved in situations where significant brain edema/brain swelling is observed. The reciprocal of intraoperative brain swelling in acute subdural hematomas is found when the hematoma is essentially non-pulsatile. Under these circumstances, visual confirmation of a poorly perfused brain indicates rapid attempts at tissue reperfusion. Because the status of cerebral pressure autoregulation is not usually assessable intraoperatively, caution should be exercised when iatrogenically raising the patient's blood pressure with dopamine, noradrenaline, or both in an attempt to induce re-expansion of the retracted brain.

If rebound brain swelling develops, fast intravenous administration of 25% mannitol (0.75–1 g/kg) should follow along with rapid intravenous fluid replacement. This therapeutic combination is justified because mannitol simultaneously increases CBF and decreases brain volume. Acute subdural hematomas are more frequently associated with contusions than EDH. Brain swelling is also more frequent in acute subdural than epidural hematomas. Therefore, decompressive surgery may be necessary under the following circumstances:

(1) The clot itself is not very thick, but the hemorrhagic contusion is sizable, leading to a mass effect (frequently compressing basilar cisterns);
(2) The clot itself is not very thick, but hemispheric swelling may be pronounced in proportion to the size of the hematoma;
(3) The clot is large and associated with contusions, and acute "rebound" brain swelling is found after clot removal (Quiñones-Hinojosa 2012).

ASDHs do not provide the rewarding outcome that is usually seen for EDH patients after surgical intervention. SDHs are the worst form of traumatic injury affecting the brain.

This is because of associated parenchymal brain injury and the subsequent secondary brain injury that is commonly present with ASDH. Mortality rates range from 40–60%.

11.3. Risk Factors for Worse Outcome

1. Age: younger patients generally have more favorable outcomes than patients older than 65;
2. The period of loss of consciousness and poor GCS;
3. Signs of brain herniation and brain stem injury;
4. Surgery within 2 h of injury has a more favorable outcome and lower mortality rate compared to that for patients who undergo surgery after 2 h.

11.4. Comparison Between EDH and ASDH

Comparison between EDH and ASDH is shown in Table 5.

Table 5. Epidural vs. subdural hematomas.

Hematoma Type	Extradural	Subdural
Site	Between the skull bone and the dura	Between the arachnoid matter and the dura
Involved vessel	Tempero-parietal (most likely vessel)—Middle meningeal artery Frontal—anterior ethmoidal artery Occipital—sigmoid or transverse sinuses Vertex—superior sagittal sinus	Bridging or connecting veins
Symptoms	Lucid interval after period of initial unconsciousness	Slowly incrementing headache and confusion
CT appearance	Biconvex lenticular shape—restricted by suture lines	Crescent-shaped—transcends suture lines

Source: Authors' compilation based on data from Habeeb (2017).

12. Chronic Subdural Hematoma (CSDH)

A CSDH is an encapsulated accumulation of old blood frequently liquefied and situated in between the dura mater and the pial brain surface. The cortical vein may be ruptured with minimum head trauma, particularly for elder individuals with cortical atrophy of the brain.

12.1. Sign and Symptoms of CSDH

The onset of the symptoms of CSDHs is generally delayed by 4–7 weeks.
Clinical features:

- Dementia
- Unconsciousness
- Seizures
- Numbness
- Dizziness
- Amnesia
- Nausea and vomiting
- Changes in personality
- Ataxia and/difficulty walking
- Altered respiratory patterns
- Gaze palsy or abnormal eye movement
- Fluctuating LOC
- Irritability
- Pain
- Headache (either constant or fluctuating)
- loss of orientation
- Weakness
- Anorexia
- Aphasia or slurred speech
- Lake of muscle control
- Deafness or tinnitus
- Blurring of vision

12.2. Neuroimaging

12.2.1. CT Findings of CSDH

CSDHs are crescent-shaped when viewed in a CT scan, having a concave surface away from the skull. A fresh subdural hemorrhage is hyperdense at first, but as cellular materials dissolve, it becomes hypodense. The hemorrhage becomes isodense with brain tissue after 3–14 days and may thus be ignored (Figure 20). It will eventually become more hypodense than brain parenchyma (Figure 21).

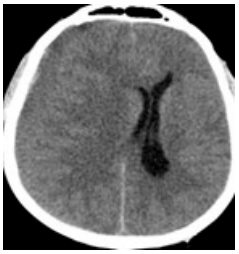


Figure 20. CT scan showing isointense right-sided mass with a midline shift—suggestive of chronic SDH. Source: Figure by authors.

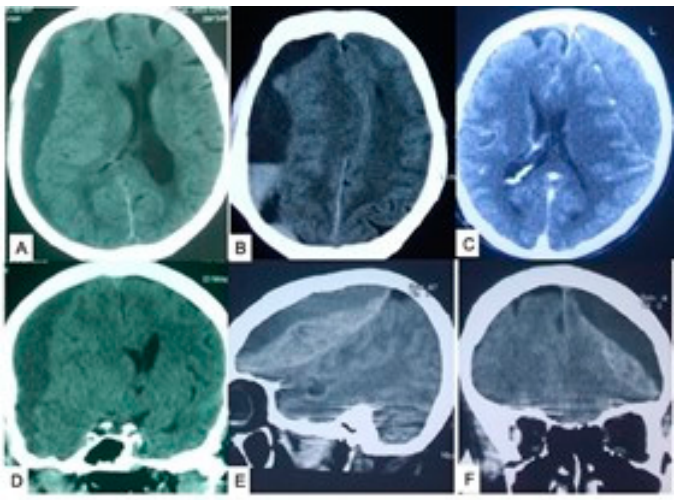


Figure 21. CT head shows fluid levels in chronic SDH (**A**–**F**). Source: Figure by authors.

12.2.2. MRI Findings

The diagnosis of a CSDH and numerous loculations, intra-hematoma membranes, fresh bleeding, hemolysis, and capsule size is more sensitive when using magnetic resonance imaging (MRI). Primary or metastatic dural illnesses can be detected using contrast-enhanced MRI.

T1: If the hemorrhage is stable, it looks isointense with respect to CSF; nevertheless, if there is a further hemorrhage or infection, it can seem hyperintense with respect to CSF.

T2: The hematoma looks isointense with respect to CSF if it is stable. If the hematoma bleeds again, it will seem hypointense.

12.3. Etiology

Although trauma is the commonest cause of a CSDH, intracranial hypotension and coagulation problems may also be to blame.

12.3.1. Post-Traumatic

In the vast majority of instances, a definitive trauma history can be acquired. Most of these cases involve mild brain damage, while in some situations, moderate to severe injury may be the cause. It is possible for this injury to be minor and go unreported. Some cases may arise as a result of neurosurgical procedures. Bridging/connecting veins in the subdural portion are weaker than those in the subarachnoid portion due to their thin walls, circumferential distribution of collagen fibers, and absence of outside support by arachnoid trabeculae.

Acute SDH or subdural effusion can lead to a CSDH (SDE) (Simmons and Luks 2013). It is suspected that matrix metalloproteinase is involved in the formation of CSDH. Approximately 50% of asymptomatic post-traumatic SDEs develop into CSDHs. Bridging vein rupture, hemorrhage from the wall of hygroma owing to neocapillaries, increased vascular permeability, accelerated fibrinolysis as well as increased protein component in the hygroma are some of the ideas for the etiology of traumatic SDE progressing into CSDH. Inflammatory cytokines are greater in SDE as well as CSDH than in peripheral venous blood. SDE and CSDH are hypothesized to be different stages of the same inflammatory reaction with different signs and symptoms (Kim and Gean 2011; Feng et al. 2008).

12.3.2. Intracranial Hypotension

CSF leaking could result in intracranial hypotension, which could contribute to the development of a CSDH.

12.3.3. Spontaneous Intracranial Hypotension

Without prior trauma or hematological abnormalities, spontaneous intracranial hypotension can be the etiology of CSDH, particularly in young and middle-aged people. Intracranial hypotension can be diagnosed via spinal MRI as well as radionuclide cisternography. Even among elderly patients taking anticoagulants, the existence of a spontaneous spinal CSF fistula should be explored in relation to CSDH (Kim and Gean 2011).

12.4. Pathology

An exterior membrane, a hematoma cavity, and an internal membrane make up a CSDH. Hematoma fluid is usually a non-clotting liquid. Hematomas are usually liquid, but mixed lesions with solid components sometimes occur. The growth of CSDHs has been linked to recurrent hemorrhaging, more exudates from the outer membrane, and CSF entrapment.

Older CSDHs (after 40 days of trauma) generally show numerous capillaries and thin-walled sinusoids along with patent, larger blood vessels. Vessels are commonly occluded by a thrombus in the fibrotic external membrane of a sixty-or-more-day-old hematoma. The external capsule may ossify or calcify in some cases.

Recurrent bleeding, more exudates from the external membrane, osmotic mechanisms, and fast enlargement due to CSF entrapment are probable causes of the expansion of CSDH (Kim and Gean 2011; Quiñones-Hinojosa 2012; Gooch et al. 2009).

12.5. Management

Symptomatic and mass effects generating a CSDH need surgical evacuation.

12.5.1. Burr-Hole Craniostomy

A burr-hole craniostomy is a common procedure for treating a primary, uncomplicated CSDH with a low recurrence risk and low morbidity.

Technique

The patient is put in supine position on a horseshoe headrest in the operation room. To allow for safe tilting of the operating table, the patient is fastened to the table. Based on the patient's comorbidities and the neurosurgeon's preference, general or local anesthesia may be used. At the time of anesthetic induction, a single dosage of prophylactic broad-spectrum antibiotics is administered.

Over the hematoma's maximal width, a pair of 14 mm burr holes are bored around 7 cm apart (commonly one frontal as well as one parietal, with the proper location calculated based on a CT scan). A cruciate incision is used to open the dura mater, which is then coagulated by bipolar diathermy. Using a 50 mL syringe, the subdural collection is flushed away with warmed Ringer solution until the subdural collection becomes clear. A soft catheter can be utilized to irrigate the hematoma cavity across long distances. Through the burr hole, a soft silicon drain with three smooth side holes along with a blunt tip is placed into the subdural space (Figure 22). Gel foam is applied in the burr hole. The skin is closed in multilayers.

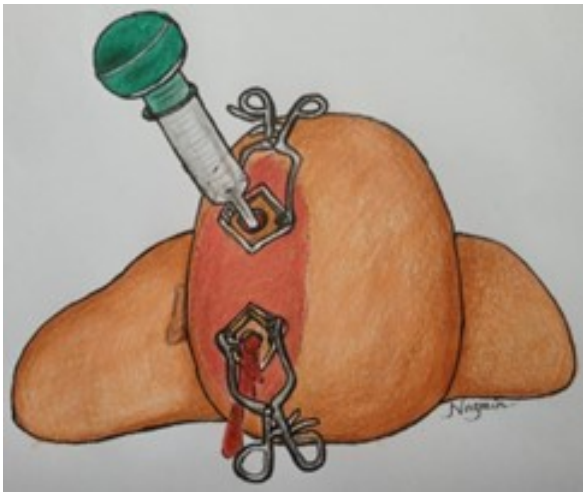


Figure 22. A diagrammatic picture of burr holes and the irrigation of a CSDH. Source: Figure by aurhors.

12.5.2. Mini Craniotomy and Partial Membranectomy

Mini craniotomy and partial membranectomy with irrigation, followed by closed-system drainage, are regarded as a potential management method for CSDHs when there are multiple septations present.

12.5.3. Large Craniotomy with Extended Membranectomy

In non-liquefied and solid hematomas, multilayer intrahematomal loculations, and calcified or organized CSDHs, a larger craniotomy using the 'extended membranectomy' technique may be required, as opposed to a micro craniotomy via the partial membranectomy technique (Kim and Gean 2011; Gooch et al. 2009).

13. Subdural Hygroma

When the arachnoid mater is disrupted, CSF may enter the subdural space, and heavy accumulation of CSF leads to compression of the cerebral parenchyma. A subduro-peritoneal shunt may be required in some cases to treat this hygroma.

14. Traumatic Intracerebral Hematoma (ICH)

This is an acute intracerebral hematoma defined as follows:

- Small ICH—when the size of the hematoma is between 1 and 3 cm;
- Average ICH—when the size of the hematoma size is between 3 and 5 cm;
- Large ICH—when the hematoma is over 5 cm in diameter.
- ICH is conservatively managed in a variety of circumstances:
  - (1) In the event of an intracapsular location;
  - (2) When there is little or no mass effect (except, perhaps, for the contribution of peri-hematoma edema);
  - (3) When there is severe acute hypo-coagulopathy.

Repeat CT 6–8 h after admission or the development of new neurologic localizing signs indicates that it is necessary to re-evaluate these initially small hematomas.

If the intraparenchymal hematoma exhibits a relevant pressure effect upon conducting the first CT scan and is extracapsular, one should consider emergency removal via standard craniotomy and corticectomy while attempting to preserve a layer of clotted blood against the walls of the hematoma cavity. The latter maneuver is conducted to minimize the risk of clot re-accumulation. Proper hemostasias must be ensured before closure of the dura to avoid hemorrhage.

14.1. Surgical Management of Traumatic ICH

14.1.1. Surgical Indication of Evacuation

Yamaki et al. thought that only ICHs greater than 3 cm in diameter were of clinical importance. Surgery should be advised for every patient with an intracerebellar hematoma larger than 3 cm (Yamaki et al. 1990).

McLaurin and McBride treated surgically an ICH that was between 10 and 75 mL in volume. For an ICH in the temporoparietal region (McLaurin and McBride 1956), Andrews et al. suggested that such a procedure should be considered for lesions greater than 30 mL in volume (Andrews et al. 2012).

15. Decompressive Craniotomy (DC) in Head Injury

This term refers to surgical decompressive measures other than cerebral decompression due to hematoma removal alone. In the management of acute traumatic intracranial hypertension associated with brain swelling, decompressive surgery is required for approximately 20% of patients. The main factor governing the acquisition of successful results in decompressive surgery appears to be its timing. Little or no benefit can be found when decompressive procedures are carried out after irreversible bilateral pupillary dilatation has developed. Bifronto-temperoparietal decompressive craniectomy in cases with diffuse traumatic head injury and resistant ICH results in decreased ICP and short ICU stay, according to the DECRA (decompressive craniectomy in diffuse traumatic brain injury) randomized clinical trial. However, when compared to individuals receiving normal care, DC may be linked to poor long-term neurological prognosis and equivalent mortality at 6 months.

15.1. DC Techniques

Large fronto-temperoparietal DC (at least either 12 $\times$ 15 cm or 15 cm in diameter) is advised over a mini fronto-temporoparietal DC to allow decreased mortality and better neurological results in cases with severe head injuries (Figure 23).

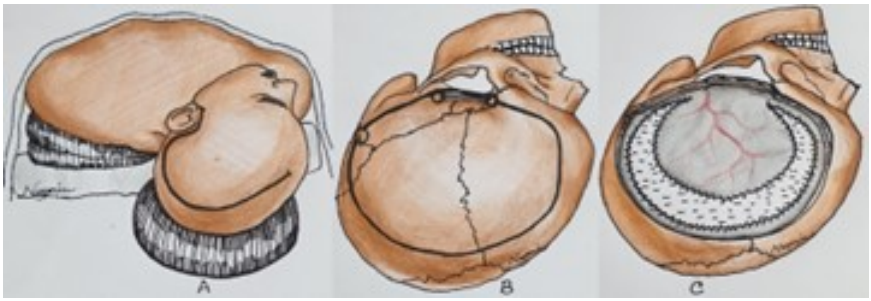


Figure 23. Unilateral decompressive craniectomy. (**A**) The dotted line represents the usual skin incision made during a unilateral decompressive craniectomy. To preserve adequate vascular supply. The length of the incision (distance B) should not exceed its width (distance A). (**B**) A myo-cutaneous flap is depicted. The dotted line represents the usual extent of the craniectomy. (**C**) The dotted line on the dura mater represents our preferred method for opening the dura. Source: Figure by authors.

The dura opens in a C shape, its base running along the sphenoidal ridge. To reduce the risk of harming the protruding brain, the dural incision is made 5–10 mm away from the craniectomy margin. A hemi-craniectomy can be helpful when there is a midline shift and (possible) swelling (e.g., for a SDH with tissue damage). To achieve a sufficient decrease in ICP and reduce the risk of trans-calvarial herniation, the latter of which is linked to brain tissue injuries and cortical venous occlusion at the bone margin, adequate hemi-craniectomies should be performed, and the bone flap should be big enough, with a diameter of at least 11–12 cm antero-posteriorly.

For diffuse (bi-hemispheric) cerebral injuries with intractable intracranial hypertension, bifrontal DC (Figures 24 and 25) is a therapeutic option. A bifrontal DC runs from the anterior cranial fossa floor, posteriorly to the coronal suture and bilaterally to the temporal floor. To permit the brain to expand sufficiently, the dura mater must be opened considerably. The dura is left open with a layer of hemostatic agent, pericranium, or temporalis fascia or closed with dural grafts as well as superior sagittal sinus sectioning or sparing, using various procedures (Bohman and Schuster 2013). The DC must be extended to the floor of the middle cranial fossa in individuals with a temporal lobe contusion or edema producing midbrain compression.

The superior sagittal sinus is divided anteriorly along its attachment to the skull base, and the dura is cut in a C-shaped pattern on each side of the midline (Gooch et al. 2009; Hall 2014; Carney and Ghajar 2007).

Many problems can develop after a decompressive craniotomy, including infection, CSF leaking, and sinking skin flap syndrome (Figure 26).

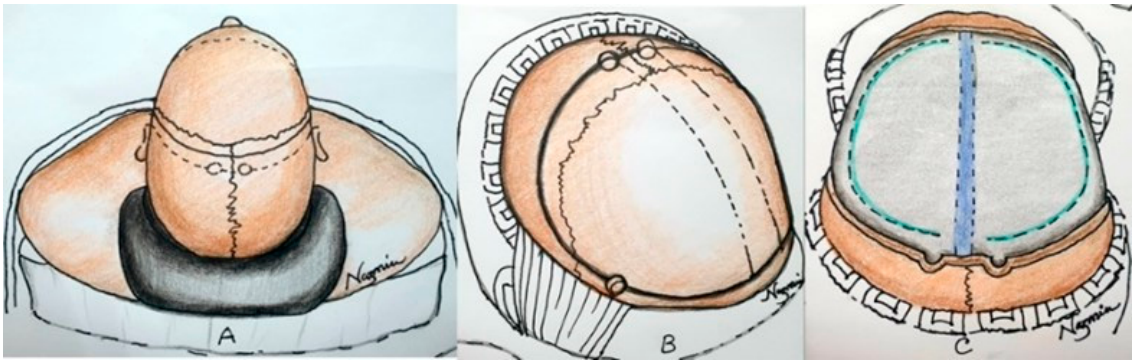


Figure 24. Bifrontal decompressive craniectomy. (**A**) The dotted line represents the usual skin incision made for bifrontal decompressive craniectomy, which should be kept behind the hairline. (**B**) Bi-coronal myo-cutaneous flap is depicted anteriorly. The dotted line on the skull represents the usual extent of a craniectomy. Subtemporal decompression can be seen. (**C**) The bone flap has been removed. The dotted line on the dura mater represents our preferred method for opening the dura. Source: Figure by authors.

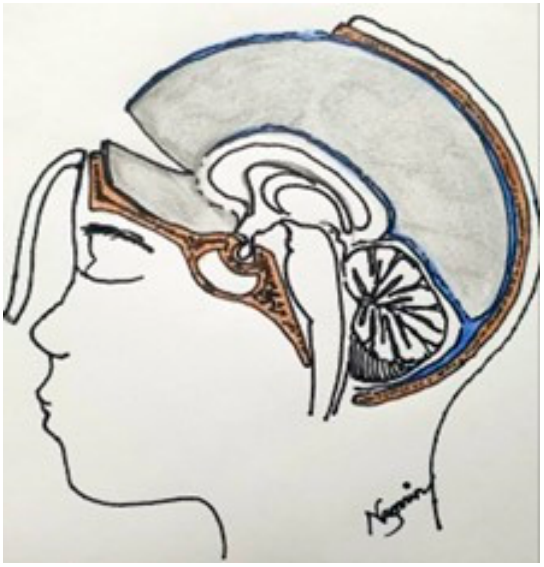


Figure 25. Diagrammatic figure depicting bifrontal decompressive surgery and cutting of falx. Source: Figure by authors.

15.2. Cranioplasty

After a previous DC for TBI or ischemic or hemorrhagic disease or even the excision of cranial tumors, cranialoplasty is a surgical treatment used to reconstruct a cranial vault defect.

Timing of surgery: 1–6 months after decompressive surgery.

Materials used in Cranioplasty:

- Autologous bone graft;
- Titanium (mesh or plate);
- Synthetic substitute of bone (in liquid form);
- Solid biomaterial (customized prefabricated implant matching the appropriate contours as well as shape of the skull) (Gooch et al. 2009; Carney and Ghajar 2007).

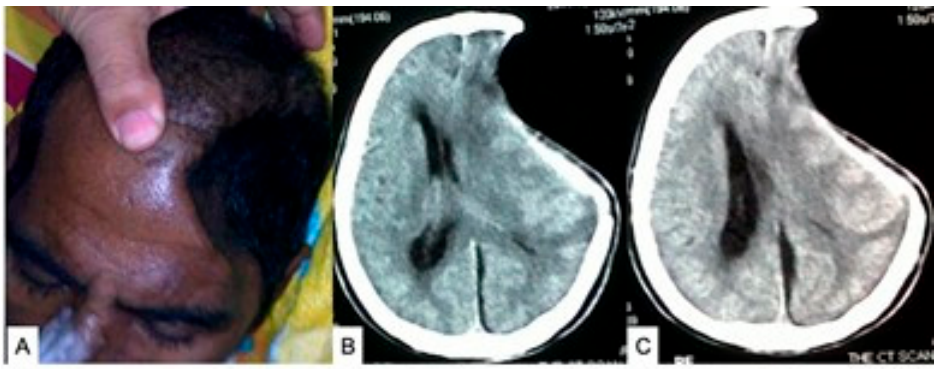


Figure 26. Post-DC sinking skin flap syndrome (SSFS): (**A**) the head of a patient with SSFS; (**B**,**C**) CT scan of the head (axial views) of the patient. Source: Figure by authors.

15.2.1. Procedure

Autologous bone harvested from the abdomen or from a bone bank is laid on the pericranium and fixed using a miniplate and screws or via a custom plate made of titanium or porous plastic (Figures 27 and 28).

First, a CT scan is conducted to measure the defect, and a virtual composite model is created. According to the model, a custom implant that identically matches the shape of the defect of the patient is created. This custom-made implant inserted over the defect through the previous incision or a new incision according to the situation. Implants are secured with the bone adjusted via miniplates or screws. The wound is closed in either single or multiple layers after setting up a subgaleal drain.

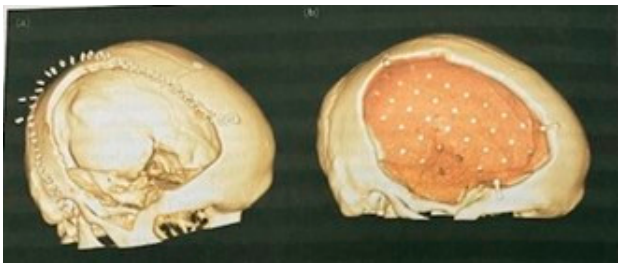


Figure 27. 3-D CT before and after cranioplasty. Source: Figure by authors.

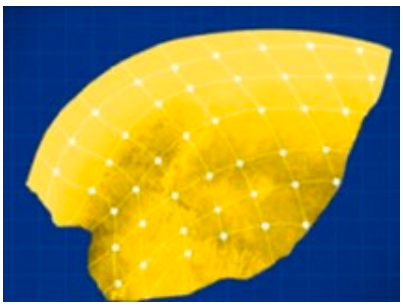


Figure 28. Computer-generated virtual composite. Source: Figure by authors.

15.2.2. Complications of Cranioplasty

- Infection (which may be treated with appropriate antibiotics);
- Postoperative clots needing drainage;
- Wound dehiscence;
- Stroke;
- Seizure;
- DVT and thromboembolism;
- Pneumonia;
- Heart attack;
- Urinary infection.

16. Cerebellar Injury

Cerebellar damage causes sluggish and disorganized motions. When walking, people with cerebellar abnormalities tend to stagger and sway.

Injury to the cerebellum may cause the following symptoms: (1) lack of motor coordination (asynergia), (2) an inability to asses distance and when to stop after indicating a given point to travel towards (dysmetria), (3) an inability to execute fast alternating movements (adiadochokinesia), (4) intention tremors, (5) an ataxic gait, (6) a tendency to fall, (7) hypotonia, and (8) scanning speech and nystagmus.

The fastest recovery happens in the first three months after a moderate TBI, and most people return normal by six months. If an affected individual still has symptoms after 6 months, these symptoms will most likely go away or improve significantly within a year of the incident.

17. Brain Stem Injury

An injury to the brain stem can be catastrophic and deadly.

A brain stem injury can result in dizziness or motor weakness, and more severe forms can cause paralysis, coma, or death.

A brain stem injury can be caused by the following:

1. Stretching of the peduncles;
2. Deceleration against the basi-sphenoid as well as dorsum sellae;
3. Lateral movement of the peduncles against the tentorial margin;
4. Force avulsion of the cranial nerves;
5. Stretching of the brain stem's vascular pedicles.

Brain stem damage can be difficult to recover from, and therapy differs based on the degree of the injury. The initial phase of recuperation is usually started in a hospital or a specialized facility. Physiotherapy can be used to assist a person.

18. Death

According to the Uniform Determination of Death Act proposed in 1981, death is defined as follows:

1. A condition in which circulatory and pulmonary functions are irreversibly lost.
2. Brain death is the irreversible loss of all functioning of the brain. In 1968, the Harvard Medical School Ad Hoc Committee published a definition of brain death or permanent coma as "loss of brain functioning."

18.1. Causes of Death via Head Injury

Head injuries can cause death in the following ways: injury to vital brain centers, such as the posterior hypothalamus, mesencephalon, or medulla oblongata; respiratory failure/paralysis via an uncal or other form of brain herniation caused by a traumatic expanding mass lesion or brain swelling; and other causes, including infection, hypostatic pneumonia, pulmonary embolism, or renal failure.

18.2. Brain Death

The diagnosis of brain death requires three conditions to be met: a persistent coma, no brainstem reflexes, the inability to breath independently.

When a painful stimulus is applied, coma is confirmed. A patient is confirmed to have a coma when he or she does not open their eyes, respond verbally, or move their limbs in reaction to a painful stimulation. During the twentieth century, mechanical breathing and life support technologies allowed patients with serious brain damage to be kept alive in intensive care units (ICUs) for longer periods of time.

It is critical to distinguish brain death from various types of serious brain damage that might result in a vegetative state in which few brain functions are preserved and recovery may take time.

There is no way to reverse the brain stem's permanent loss of function, and even if a ventilator is employed, the heart will eventually stop beating.

18.2.1. Clinical Significance

Before brain death can be determined, certain conditions must be met:

1. There should be evidence of a coma etiology. Confounding disorders, such as severe metabolic, endocrine, and acid–base imbalance, should be ruled out. If a drug overdose is suspected, 5 half-lives of drug clearance should be allowed to pass while adjusting renal and hepatic functioning.
2. The patient's core body temperature should be at least 36 °C.
3. A systolic blood pressure (SBP) of >100 mm of mercury (mmHg) should be observed. Using vasopressors like noradrenaline or dopamine, as well as vasopressin, is a common way to achieve this.

Brain death can be evaluated via clinical tests, the apnea test, and ancillary tests.

I. Physical examination: Physical examination in this case covers the examination of brain stem reflexes and a patient's response to pain.

The administration of painful stimuli to particular locations such as the supraorbital notch, the sternum, and the anterior axillary fold causes a lack of responsiveness to central pain. In brain death, neither eye response nor motor reflexes are detected. It is vital to note that patients with brain death may still have some spinal reflexes.

Reflexes, especially gag reflexes, are lost when the brain dies.

- CN II: Loss of light reflex: pupils should be mid-dilated (4–9 mm) as well as light-insensitive.
- CN III, IV, and VI: Eye motion in response to head movement is lost (doll's eyes).
- CN V and VII: Corneal reflex loss.
- CN VIII: Absence of oculovestibular reflex (Caloric test): The eyes will not migrate toward the irrigated ear when each are irrigated with 60 mL of ice water.
- CN IX: Absence of the gag reflex.
- CN X: Cough reflex loss.

II. Apnea test: This test is positive in case of brain death. When respiratory effort ceases and artificial ventilatory support is temporarily turned off, then PaCO2 levels will rise. The PaCO2 level will be >60 mmHg.

III. Ancillary tests: Ancillary diagnostics for detecting cerebral blood flow stoppage include angiography of the brain; the gold standard for the evaluation of CBF is four-vessel angiography. When there is no blood flow to the brain, this can indicate brain death.

18.2.2. Considerations for and Consequences of Brain Death

Once brain death is diagnosed, the brain is no longer working.

An examination that includes an apnea test is required to diagnose brain death in adults. Recent guidelines, however, advocate two distinct brain death investigations as the minimum norm in youngsters.

If the patient is a probable candidate for organ donation or is pregnant and the decision is made to prolong assistance for the unborn, organ support, with artificial ventilation and drugs to keep normal blood pressure, may be initiated following an announcement of brain dead (Carney and Ghajar 2007).

18.2.3. Vegetative State

In a state of wakefulness, a patient will open their eyes but not react to the external environment. A vegetative patient with a functioning brain stem has the following capacities:

1. Some state of consciousness (awake but not aware);
2. Unassisted normal respiration.

19. Burst Lobe Syndrome

This condition is an intracranial hemorrhage affecting the temporal or frontal lobe, commonly associated with subdural and subarachnoid hemorrhages and contusion (Figure 29). It occurs due to high-energy trauma and results in a high mortality rate.

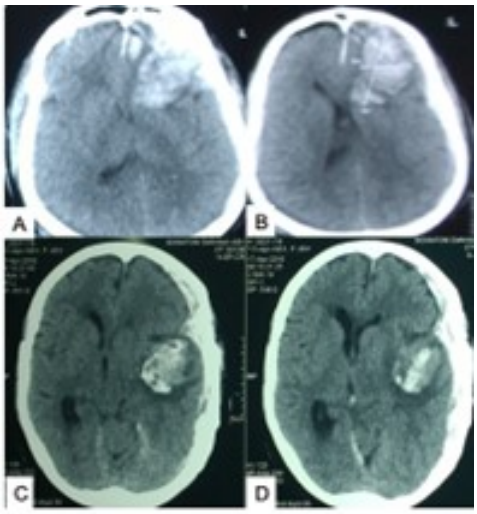


Figure 29. CT scans showing (**A**,**B**) burst frontal lobe; (**C**,**D**) the burst temporal lobe on left side. Source: Figure by authors.

19.1. Kluver–Bucy Syndrome

Kluver–Bucy syndrome is an uncommon behavioral disorder characterized by a bilateral anterior temporal lobe injury. It drives people to put things in their oral cavities and participate in sexual activities that are not appropriate. Visual agnosia (an inability to distinguish objects visually), a lack of typical fear and rage, memory impairment, distractibility, seizures, and dementia are all possible symptoms. Herpes encephalitis may also be linked to this condition (Das and Siddiqui 2021).

19.2. Amnesia Following Head Injury

Post-traumatic amnesia is commonly associated with concussion. Some patients may experience permanent retrograde amnesia. Such patients may make false accusations (Cantu 2001).

20. Penetrating Injury of the Brain

The dura mater is pierced in a penetrating head injury, also known as an open head injury (Mckee and Daneshvar 2015). Penetrating trauma can be produced by high-velocity projectiles (Figures 30 and 31) or low-velocity (Figure 32) items like rods, knives, or bone pieces from a vault bone fracture that penetrates the brain. Penetrating trauma to the head is a medical emergency that can result in permanent impairment or death.

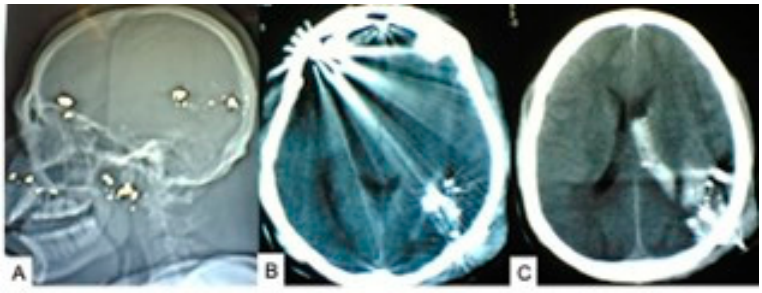


Figure 30. (**A**) X-ray of the skull (lateral view) showing multiple retained bullet fragments in different sites of the head. (**B**,**C**) CT scan (axial views) showing multiple retained bullet fragments with artifacts and parenchymal and lateral ventricular hemorrhages, respectively. Source: Figure by authors.

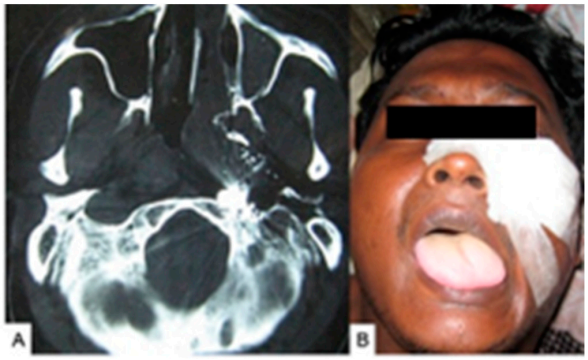


Figure 31. (**A**) CT scan of the head (axial view) (bony window) showing bullet fragments near the left hypoglossal canal that entered through the face. (**B**) Picture of a patient with left hypoglossal palsy. Source: Figure by authors.

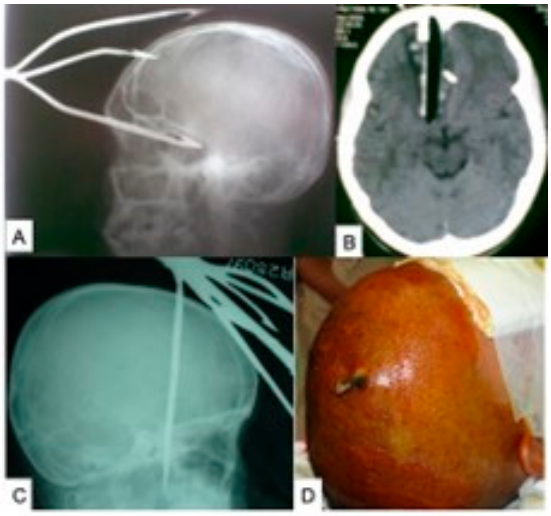


Figure 32. (**A**) X-ray of the head (lateral view) showing penetrating orbitocranial injury caused by a polyspike animal-hunting weapon locally called a 'Teta'. (**B**) CT scan (axial view) of the head showing a penetrating injury caused by a long fragment of wood file. (**C**) Head X-ray (lateral view) showing a penetrating head injury caused by a 'Teta'. (**D**) Painted head of a patient during operation showing a metallic peg nailed into the head by a political rival. Source: Figure by authors.

A penetrating head injury is defined as follows: "A wound where an object passes through the cranium but does not come out." A perforating head injury, on the other hand, occurs when an object moves through the head and comes out through an exit wound (Vinas and Pilitsis 2006). A penetrating brain injury (PBI) is a type of TBI that is a leading cause of death among young people. PBI is the most serious traumatic brain injury and usually counts as all TBIs excluding blunt head injuries (Kim and Gean 2011; van Rein et al. 2019; Kommaraju et al. 2019; Vlček et al. 2018; Cengiz et al. 2019). Penetrating trauma takes place when a foreign instrument pierces the skin, affecting the underlying tissues and leading to an open wound. Such injuries are most commonly caused by gunshots, explosive devices, and stab wounds.

Penetrating injuries can be classified into two categories depending on penetration speed:

- Bullet or shell fragment injuries, direct injury, or shockwave injury to adjacent brain tissue owing to a stretching brain injury are all examples of high-velocity penetration (Figures 30 and 31).
- Low-velocity penetrating injury: Injuries caused by sharp instruments like knives, with direct harm to brain parenchyma, are examples (Figure 32) (Das et al. 2015).
- The following factors influence the outcomes of a penetrating head injury.
- Path and location of intracranial fluid: Injuries wherein intracranial path crosses the midline, moves through the ventricles, or lands in the posterior cranial fossa have a significant mortality rate.
- Energy and velocity of entry: These factors are determined by a weapon or missile's characteristics. This type of injury occurs when energy from an instrument or object is transferred to the skull and the brain parenchyma beneath it. High-velocity projectiles are connected with a high death rate. The square of the velocity determines the kinetic energy involved. Three types of injury mechanisms have been identified.
- Primary injuries happen right away. Secondary brain trauma occurs after an initial injury. The breadth and severity of secondary brain injury have an impact on the final neurologic outcome. As a result, the primary priority in the emergency room is to deter or ameliorate disorders like hypotension, hypoxia, anemia, and hyperpyrexia, which can all exacerbate outcomes.

Certain elements play a role in making vital decisions and have prognostic significance. The following are some examples:

- Wounds at the entry and exit points;
- Intracranial fragments;
- The link between a missile's trajectory and cerebral vessel and air-filled skull base structures;
- The presence of air inside the cranial cavity.

20.1. Management of Penetrating Injuries

Medical and surgical treatment are required for patients who have suffered a penetrating head injury (Maragkos et al. 2018; McGrew et al. 2018; Milton et al. 2017; Elias et al. 2007).

20.1.1. Emergency Medical Management

In cases of penetrating head trauma, neurosurgical consultation is warranted, as many patients with a significant head injury will almost certainly need surgery.

Patient management should be conducted in accordance with ATLS guidelines. In the emergency department, one should not remove any piercing weapons from the skull until a trauma and neurosurgery examination has been conducted. In order to avoid further harm, the external part of the weapon should be stabilized and rendered immobile throughout the patient's transportation.

Conditions where endotracheal intubation is necessary:

- Inability to maintain proper ventilation;
- An insecure airway due to a low level of consciousness;
- Injury to the neck or pharynx.

20.1.2. Surgical Management

The presence of a hematoma is a common reason for conducting a neurosurgical procedure. Large hematomas should be evacuated without delay. Early decompression and cautious brain debridement may be required. In most circumstances, removing a deeply lodged bullet is not necessary. However, there are certain indications that it should be removed. These are the following:

- When there is an injury to the pterion, orbit, or posterior fossa that penetrates the skin;

- When an intracranial hematoma is present;
- When a pseudoaneurysm is present during the initial examination.

For low-velocity missile injuries in which the weapon is still inside the skull in situ, a craniotomy is required. For those who survive the initial damage, some crucial elements can influence the result; these patients rely on rapid as well as early neurosurgical operation, including the capacity to provide a standard level of care in the neurocritical care unit.

20.2. Gunshot Injury to the Head

Due to an increase in gang violence and general homicide rates, in many urban locations across the United States, gunshot injuries to the head are a prominent cause of TBI. Suicide and unintended accidents are two other examples. Suicide-related gunshot injuries to the head have a high death rate and are associated with substantial morbidity among survivors. Individuals who suffer TBIs as a result of self-inflicted gunshot injuries have a higher risk of death as well as a poorer prognosis than those who suffer TBIs as a result of accidental or intentional gunshot wounds (Vlček et al. 2018; UCLA Health 2021; Chotai and Than 2021).

A penetrating wound is defined as one in which the bullet penetrates but does not exit the cranium. A perforating wound occurs when a bullet enters and exits the cranium at the same time. When a projectile passes through the brain, it causes harm due to both direct penetration and the spread of a pressure wave from a high-velocity bullet going through brain tissue (greater than 2000 feet per second) (Chotai and Than 2021). Brain swelling is caused by both bleeding and damage caused by this pressure wave, and it can lead to death.

The severity of damage dealt by a gunshot wound depends on certain factors, such as the gun's caliber, the bullet's size and speed, and the trajectory and location of the injury. As it passes through the brain tissue or vascular structures, a bullet from a gunshot wound to the right frontal pole, located well above the skull base, seems to result in only modest clinical damage. A bullet traveling in a caudal direction from the left frontal pole into the temporal lobe and brainstem, on the other hand, would be fatal because it would pierce the eloquent cerebral parenchyma and injure key vascular structures within the cranial cavity. A bullet directed into vital intracranial vessels can cause a quickly developing blood clot in the brain, compressing crucial brain tissue and leading to immediate death. If the sufferer survives the initial phase, the main confounding situation is increased intracranial pressure (Chotai and Than 2021).

20.2.1. Surgical Treatment

Patients with a gunshot injury to the head should promptly and vigorously be resuscitated when they arrive at the hospital. An immediate CT scan should be conducted if blood pressure and oxygenation are satisfactory. The following considerations guide decisions regarding the surgical treatment of a gunshot wound:

- The spectrum of brainstem function;
- The CT scan findings of head;
- The LOC: According to the Glasgow Coma Scale (GCS), scored from 1 to 15, a patient with a score < 7 or 8 is deemed to be in coma (Chotai and Than 2021).

A fatal end-result is almost inevitable if individuals are in a severe coma with minimal indications of brainstem function as well as no signs of an intracranial hemorrhage. If a CT scan confirms a hematoma, an emergency craniotomy may be performed to remove the clot, debris, and devitalized tissue. Because pressure inside the skull is widespread, a decompressive craniectomy is often performed (Chotai and Than 2021).

20.2.2. Outcomes

Patients with long bullet tracks crossing the deep midline tissues of the cerebrum or brain stem have a poor prognosis. A gunshot that hits the brain's right cerebrum may produce motor and sensory deficiencies on the left side, and vice versa. The control of several tasks such as cognitive functions, speech, memory, and vision are carried out by both cerebral hemispheres. As a result, depending on which lobes of the brain are affected, a person's ability to execute specific activities may be limited (Chotai and Than 2021).

Because each hemisphere is separated into four lobes, a more superficial injury restricted to one hemisphere and one lobe is the "best-case scenario", minimizing the functional losses caused by the trauma. The acute and critical-care stages correspond to the first 1–14 days. The amount of tissue damage, the degree of edema, the pressure inside the brain during the early stage, and the functional extension of the lesion all influence the extent

and speed of recovery after an injury. To assist a survivor's functional restoration or adaptation to persistent disabilities, intensive neurorehabilitation may be employed. Recovery from a neurological condition might take anywhere from months to years (Chotai and Than 2021) (Box 1).

Box 1. Factors influencing initial gunshot injuries and outcomes (Chotai and Than 2021; Aarabi et al. 2014).

- The location of the entry and/or exit wound
- The site of the brain injury
- The bullet's fragmentation level
- Bullet caliber and weapon type—high velocity or low velocity
- The shooting range (distance between the gun and the victim)
- The time gap between the infliction of the gunshot wound and beginning of treatment
- The age and overall health status of the patient
- The initial GCS score
- Pupil status (dilation and reactivity)
- The state of the brainstem function and reflexes
- High blood pressure
- The state of oxygenation immediately after the trauma.

21. Pneumocephalus and Tension Pneumocephalus

21.1. Pneumocephalus

Pneumocephalus (Figure 33) is most commonly related to skull disruption, such as incidents following facial and head injury, skull base tumors, otorhinolaryngological surgery, or neurosurgery and, on rare occasions, incidents occurring spontaneously. It can also occur while scuba diving, though this is quite uncommon. Pneumocephalus has also been linked to neurosurgery techniques like deep-brain stimulation, which, while ostensibly safe for the patient, might result in brain displacement and consequent stereotactic inaccuracy (Elias et al. 2007; Sharim et al. 2015). Neurosurgeons strive to limit pneumocephalus volume and, as a result, brain shift during an operation.

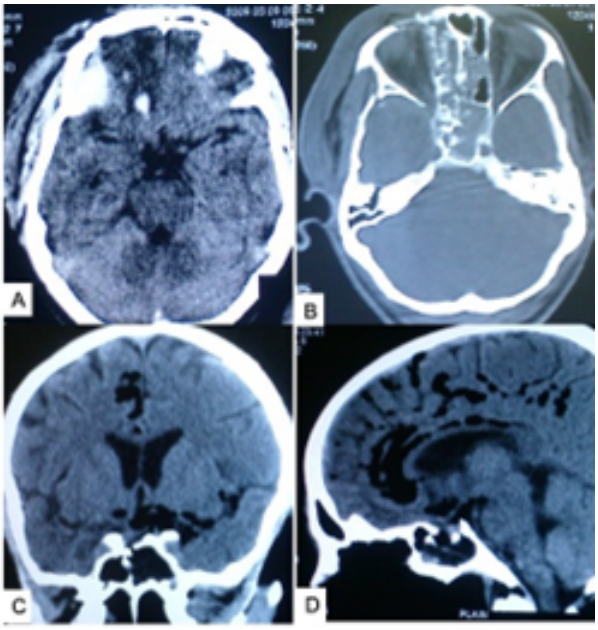


Figure 33. (**A**,**B**) CT scan of the head (axial views) following RTA showing right-side skull base fractures with right frontal contusion. (**C**,**D**) CT scans on the 24th day following RTA (at which point the patient developed CSF rhinorrhea), with coronal and sagittal views, respectively, showing sellar bony gap with pneumocephalus. Source: Figure by authors.

21.2. Tension Pneumocephalus (TP)

Tension pneumocephalus (Figure 34) is a neurosurgical emergency that happens when subdural air creates a pressure effect on the surrounding brain parenchyma, usually as a result of a ball valve system failure that permits air to enter the subdural region only one way. During the early postoperative phase, clinical

imaging of neurosurgical patients frequently reveals postsurgical pneumocephalus. The distinction between uncomplicated and tension pneumocephalus is critical in clinical practice since the latter is a neurological emergency. To confidently arrive at an accurate diagnosis, an understanding of the imaging findings regarding tension pneumocephalus and a strong index of suspicion are essential. It is a rare but life-threatening neurosurgical emergency that can happen after head trauma (especially if there are skull base or air sinus fractures), extradural injections, or complicated neurosurgical, spinal, air sinus, or craniofacial surgeries (Simmons and Luks 2013; Sweni et al. 2013; Ishiwata et al. 1988; Monas and Peak 2010). Because the symptoms and signs of TP are not specific, its detection should be carried out by recognizing the characteristic imaging signals of TP as soon as possible, allowing for life-saving emergency decompression (Simmons and Luks 2013; Monas and Peak 2010).

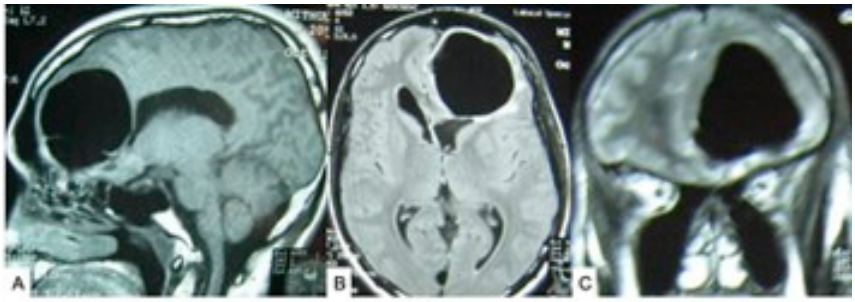


Figure 34. MRI of the brain (**A**–**C**) (sagittal, axial, and coronal views, respectively) showing tension pneumocephalus with mass effect that requires surgical decompression (through burr hole). Source: Figure by authors.

21.2.1. Clinical Presentation of TP

Tension pneumocephalus manifests itself in a variety of ways:

- Agitation;
- Deterioration of consciousness;
- Specific neurological impairments;
- Cardiac arrest.

There are several etiologies of this condition:

- Recent neurosurgery;
- Trauma with cerebrospinal fluid leaks;
- Infections of the paranasal air sinuses;
- Tumors in the paranasal air sinuses;
- The use of NO (nitrous oxide) as a local anesthetic.

21.2.2. Investigations

A plain X-ray can identify pneumocephalus, but a CT scan or MRI of the brain can identify pneumocephalus along with possible causes and sites of air entry (Figures 33 and 34).

21.2.3. Treatment

Post-traumatic or post-surgery pneumocephalus resolves spontaneously. Tension pneumocephalus requires urgent surgical decompression with treatment of the cause (Figure 34). Pneumocephalus with a spontaneous CSF fistula requires urgent fistula closure.

22. Vascular Injury to the Cranium

Blunt as well as penetrating wounds to the base of the skull might cause vascular damage. In 8.5% of instances, blunt base-of-skull fractures were shown to be linked to neurovascular damage (Feiz-Erfan et al. 2007). A clival fracture and fracture of 'the sellae turcica-sphenoid air sinus complex', in particular, have been linked to vascular damage.

The following are the most common vascular injuries related to head trauma:

- Aneurysms caused by trauma;
- Carotid-cavernous fistulas (CCF) (Figure 35);

- Post-traumatic (arteriovenous) AV fistulas, which are rarely arterial and generally relate to the venous sinuses such as the SSS (the superior sagittal sinus) or lateral sinuses.

Neurovascular injuries can be caused by blunt trauma to the neck, chest, or head that can injure the vessels perfusing the brain; these injuries can be caused by the following:

- Pressure or forceful compression from a seat belt during an MVA;
- Sudden violent movements (such as flexion and extension of the neck) that may occur during an MVA when a vehicle decelerates/accelerates (whiplash injury).

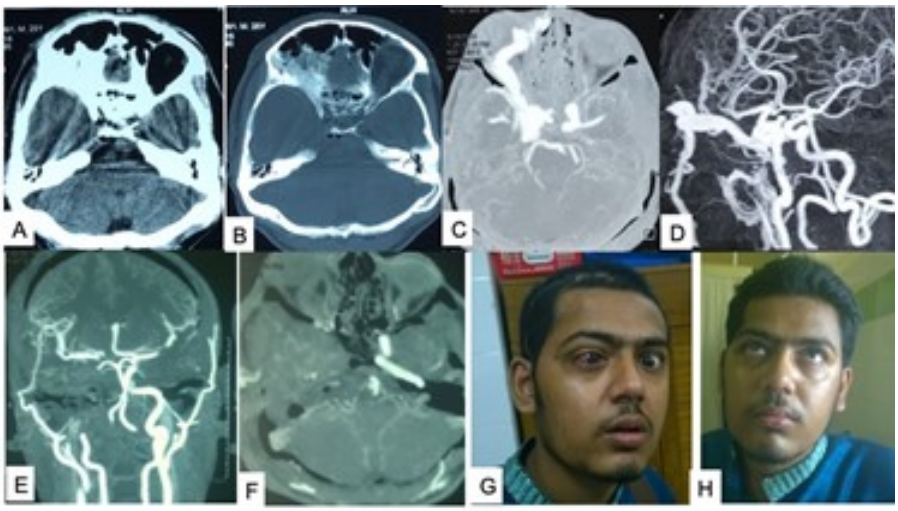


Figure 35. (**A**,**B**) CT scan of head (axial views) showing skull base fractures in sellar and parasellar zones after RTA. (**C**,**D**) CTA of head 3 months after RTA showing rightside high flow CCF. (**E**,**F**) Early postoperative CTA following right STA-MCA bypass occlusion of right ICA in Glasscock triangle (where CCF and right ICA are not seen). (**G**) Photograph of the patient three months after RTA showing ocular clinical features of right CCF. (**H**) Patient three months after microsurgical treatment of CCF. Source: Figure by authors, used with patient's consent.

22.1. Diagnosis

A CT scan and CTA are generally utilized for diagnosing traumatic vascular damage. Catheter angiography is conducted if a vascular injury is suspected but not conclusively seen via CTA. Cerebral DSA is carried out in an angiography suite in a hospital. An arterial catheter is introduced into the artery through the groin, and contrast is injected into the arteries directly, allowing the abnormality to be seen more clearly in the images taken. An MRI of the brain or spine of a patient who has suffered a stroke may be performed in some conditions to assess the patient's spine or brain, and this may also reveal blood vessel injuries.

High-energy injuries have a higher risk of vascular damage, and high-energy blunt trauma is associated with a higher chance of amputation. Damage to nearby tissues and structures may necessitate a one-time intervention or, in the case of severe polytrauma, a multidisciplinary treatment incorporating advanced life support techniques. Irreversible alterations of the neurological and musculoskeletal systems are generally observed after 6 h of limb ischemia, albeit the exact start time must be determined. If the ischemia process is iatrogenic, the time can be calculated from the time when blood flow was interrupted (e.g., pressure dressing, tourniquet).

22.2. Medical Examination

Due to a significant mortality risk in the case of a misdiagnosis, the decision to perform early surgery, particularly when a substantial hemorrhage is obvious, is critical within the first minutes of assessment. With a sensitivity of over 90%, most patients with "hard signals" of vascular injury need prompt surgery; on the contrary, if no "hard sign" is observed, the risk of vascular injury is low (Cheaito et al. 2016).

An algorithm for managing patients with vascular injury to the cranium (like a vascular injury of any other part of the body) is shown below (Figure 36).

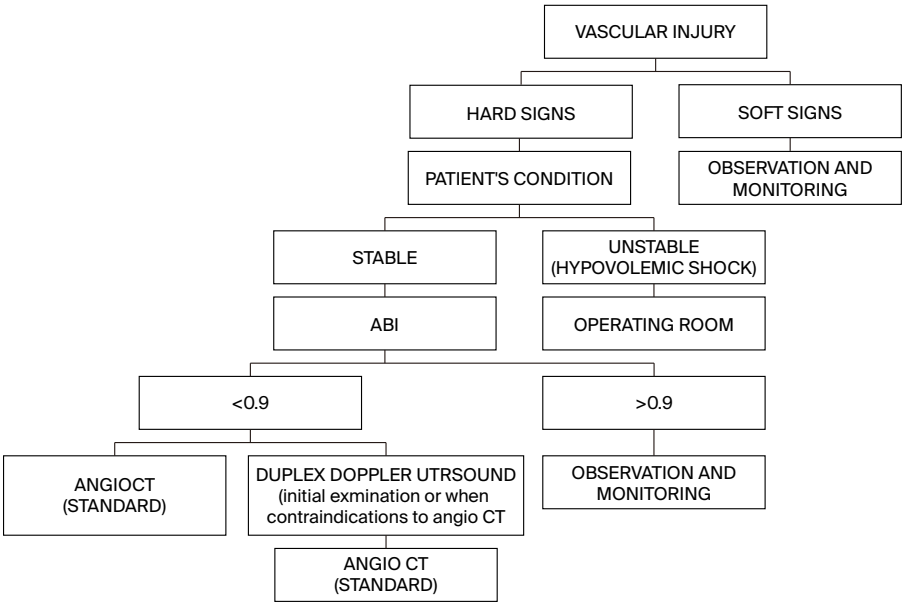


Figure 36. An algorithm for managing patients with vascular injury to the cranium. ABI—ankle–brachial pressure index. Source: Authors' compilation based on data from Krzysztof et al. (2019).

Vascular injuries are uncommon, yet they are among the most serious and complex situations for medical experts to correctly evaluate and treat. The fast development of minimally invasive procedures in numerous neurology departments has increased the extent of iatrogenic vascular damage that can be accounted for. Iatrogenic injuries are common, despite their rarity, as a result of the larger number of minimally invasive operations that require the assistance of a vascular surgeon.

22.3. Treatment

The type of treatment to be applied is determined by the degree of damage. Minor injuries are carefully monitored, and a patient may be prescribed blood thinners to prevent a thrombus from developing as well as detaching and spreading to the cerebrum. A stent can be put endovascularly into an injured artery in more severe cases to keep it open and prevent it from becoming obstructed. The artery may need to be repaired surgically in some cases.

22.4. Dissection of Arteries and Pseudoaneurysm

Dissection of the carotid as well as vertebral arteries is a major cause of stroke in young people, causing up to 25% of strokes in this population (Schievink 2001). Intracranial and extracranial dissections are both possible, with the latter being the more prevalent. These can happen on their own, be linked to certain medical illnesses such as connective tissue diseases (Ehlers–Danlos syndrome, fibromuscular dysplasia, and Marfan syndromes), or happen as a result of a traumatic incident. Carotid and vertebral artery dissections can cause aneurysmal expansions at the dissection site, particularly in the event of subadventitial dissections (Boström and Liliequist 1967). Dissecting pseudoaneurysms are the most common type of pseudoaneurysm. The percentage of dissections that result in pseudoaneurysm development has been observed to range between 5% and 40% (Guillon et al. 1999). They may be fusiform or saccular, and their appearance varies depending on where they are found (Touzé et al. 2001). They can be seen as a sudden occurrence or as a result of a follow-up angiography (MRA/CTA/DSA) (Levy et al. 1994; Nguyen Bui et al. 1993; Provenzale 1995). Because pseudoaneurysms have the potential to develop and become symptomatic.

Management techniques for dissections with associated pseudoaneurysms are not well defined, with some authors claiming that these aneurysmal structures are benign and thus only require medical management with antiplatelet or anticoagulation agents, while others advocate using endovascular or surgical interventions to avoid aneurysm rupture or thrombus formation (Provenzale 1995 Dennis et al. 2012; Holle et al. 2009). Stenting is the most frequently utilized endovascular intervention, with data in the literature progressively supporting its safety and efficacy.

The majority of dissecting pseudoaneurysms will not develop clinical features or exhibit expansion in a follow-up. For dissecting pseudoaneurysms, medical/conservative treatment may be adequate as an initial management strategy. Stenting or other endovascular techniques can safely treat the a few cases of pseudoaneurysms that grow and produce symptoms. Aneurysm site and size, smoking history, and hyperlipidemia are all important influencers of the progression as well as treatment of these conditions.

22.5. Carotid-Cavernous Fistula (CCF) and AV Fistula

A CCF is aberrant communication between the carotid artery and/or its branches as well as the cavernous sinus, a major vein. The cavernous sinus (CS) receives blood from the brain, orbit, and pituitary gland and is located behind the eye. A CCF can be either direct (high-flow) (Figure 35) or spontaneous (indirect/low-flow). A CCF can occur due to trauma or spontaneously. A traumatic CCF can arise when the intracavernous carotid artery is ruptured due to a head injury. Injuries ranging from minor falls to severe piercing wounds can cause head trauma. Endovascular treatment can also cause traumatic CCFs. A ruptured cavernous carotid aneurysm is the commonest etiology of spontaneous CCFs; these fistulas, on the other hand, can be congenital arteriovenous connections that occur spontaneously in the presence of collagen vascular disease, atherosclerosis, or hypertension.

22.5.1. Clinical Features

In the days and weeks after a close head trauma, the development of a direct CCF is common. Chemosis, pulsatile proptosis, and ocular bruit are the traditional trifecta that patients exhibit (including blood flow sounds coming from the eye). These fistulas can cause proptosis, diplopia, and vision loss (Figure 35).

The onset of an indirect CCF is usually gradual, with a milder appearance. They do not always exhibit the usual triad of symptoms. Because of the convoluted arterialization of the conjunctiva, patients with a CCF frequently have chronic red eyes. Often, an ocular bruit goes unnoticed.

22.5.2. Treatment

Microvascular neurosurgery or endovascular methods can be used to treat CCFs. Because of the decreased morbidity and mortality, an endovascular method is preferable. However, all CCFs are not susceptible to both types of management.

Endovascular Treatment

Traditional treatment for direct CCFs has involved occlusion of the fistula with deployment of detachable balloons transarterially while preserving the ICA (internal carotid artery). Due to the lack of disposable balloons, further treatment options include a covered stent and transarterial fistula coiling with stent support to keep the ICA open. A transvenous method utilizing platinum coils may be indicated if a transarterial route is impractical or ineffective. This can be carried out either surgically through the superior ophthalmic vein or through the femoral route through the inferior petrosal sinus.

Indirect CCFs can sometimes spontaneously resolve. In low-risk CCFs, manual carotid compression may be undertaken, as it can cure about 30% of fistulas. Compression is not recommended for patients with retrograde cortical venous system filling because of the danger of a cerebral hemorrhage. Either a transarterial or transvenous technique should be used to treat these patients.

Surgical Treatment

CCFs are surgically treated using a craniotomy and surgical clips occluding the ICA distal as well as proximal to the CCF site. The venous outflow is subsequently stopped by packing the cavernous sinus with acrylate glue, fascia, or Surgicel. To prevent a stroke, based up on the cerebral blood flow, a branch of the external carotid artery (ECA) may need to be connected to the MCA (middle cerebral artery), i.e., EC-IC bypass (Figure 35).

22.6. Traumatic Scalp AV Fistula

AVFs of the scalp are quite uncommon and mainly occur due to severe injuries to the superficial scalp veins. Car accidents, penetrating trauma from sharp weapon attacks, diving accidents, and iatrogenic causes such as punch-graft hair transplantation and temporomandibular joint arthroscopic surgery are all examples of insults

that might cause this. Single case reports (Davis and Nelson 1997; Dogan et al. 2008; Fukuta et al. 1994; Lanzieri et al. 1985; Mathis et al. 1994) constitute the majority of the literature on scalp AVF after hair transplantation. Surgical ligation and excision, selective angiography, and embolization and direct-puncture embolization have all been effective in treating these lesions, with full recuperation in 100% of cases and no documented complications as of yet. Polyvinyl alcohol microparticles, absolute alcohol, coils, acrylic glue, and Onyx material all have been used to successfully embolize scalp AVF (Mathis et al. 1994). Traumatic scalp AVF (Figure 37) is a rare vascular disease in which the high-flow arterial as well as low-flow venous systems communicate (Badejo and Rockwood 1987; Li et al. 2007). There is no capillary bed between the scalp's arterial feeding vessels and the draining veins; therefore, there is a direct connection (Badejo and Rockwood 1987). It manifests as a disfiguring pulsatile swelling with a wide range of clinical symptoms, including local discomfort, headaches, bruits, tinnitus, hemorrhage, epilepsy, and scalp necrosis (Mohanty and Rao 1976).

Open surgical excision (Figure 37), obstruction of the feeding arteries, trans-arterial or trans-venous embolization, and intralesional sclerosant injection all have been used to treat these patients (Badejo and Rockwood 1987).

Hair transplantation treatments, while generally low-risk, can occasionally result in arteriovenous fistulas in the scalp. In the ultimate treatment of these lesions, both open surgical as well as endovascular treatments are often safe and effective. When deciding on the best treatment technique, pay close attention to the anatomy of the fistula. Its appearance after the intervention should also be taken into account.

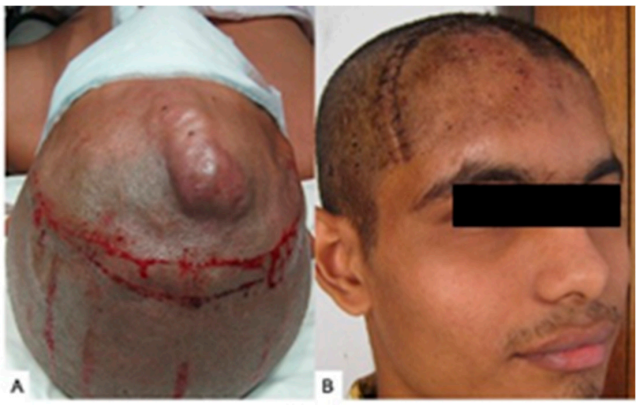


Figure 37. (**A**) Perioperative picture of patient with frontal scalp AV fistula that developed after blunt scalp injury. (**B**) The patient without an AV fistula following an operation. Source: Photos by authors.

22.6.1. Prognosis and Treatment

If left untreated, patients may develop cosmetic flaws. Endovascular occlusion, surgical resection, or direct injection of sclerosing agents are all alternative treatments.

23. Cranial Nerves Injury

Injuries to the cranial nerve (CN) can range from minor annoyances to life-threatening complications. Practitioners in a variety of specialties, including neurosurgery, otolaryngological surgery, head–neck surgery, ophthalmological surgery, oral and maxillofacial surgery, neurology, and general surgery, may encounter patients who may be at risk for these injuries. As a result, anatomic principles, proper history taking, clinical examination, and diagnostic evaluation are all significant components of their professions.

23.1. Facial Nerve Injury

Facial nerve damage has a wide range of consequences that have a substantial influence on one's quality of life. The facial nerve is one of the most frequently injured CNs, and a damaged nerve has a critical impact on various physiologic functions such as tear secretion, saliva secretion, and shutting of the eyelids. Furthermore, the spectacular disfigurement resulting from damage may have social and psychological consequences, making the prevention of iatrogenic nerve injury critical. Therapeutic improvements in recent years have boosted both medicinal and surgical therapeutic options for treating disfigurements, though the surgical option may play an essentially increasing role in the management of iatrogenic nerve injuries.

23.1.1. Traumatic Facial Nerve Injuries

Clinically, the major etiology of seventh-nerve damage is traumatic temporal bone fractures. There are numerous approaches for arranging temporal bone fractures when looking at relevant clinical circumstances. The classification of temporal bone fractures as transverse or longitudinal is one useful paradigm for determining the possibility of seventh-nerve damage. This classification is based on whether the temporal fracture is parallel to the petrous pyramid's long axis. Longitudinal fractures are significantly more common, accounting for 75–95% of all fractures. Only about one-fourth of these injuries result in seventh-nerve impairments. When the nerve is affected, the cause is usually local inflammation, edema, and compression. Transverse fractures, on the other hand, though being significantly less common, have a greater frequency of related seventh-nerve injury, which occurs in almost 50% of cases (Hasso and Ledington 1988). These types of facial nerve damage seem to result in nerve transection and frequently involve occipital damage. Aside from temporal bone fractures, additional common etiologies of traumatic seventh-nerve injury include direct intratemporal fossa injury from penetrating/gunshot wounds as well as extratemporal wounds from a variety of sources.

23.1.2. Surgical Facial Nerve Injury

The occurrence of iatrogenic seventh-nerve injury, especially during tympanic cavity surgery, is quite rare. When it comes to extratemporal injury, parotidectomy is the most common cause. If negative margins cannot be established, malignant tumors, especially those encasing the facial nerve, necessitate the deliberate sacrifice of the seventh nerve. Nerve transection discovered during surgery, whether deliberate or unintentional, should be corrected immediately. Local edema, trauma, nerve manipulation, or still-active local anesthetics are the most common causes of postprocedural facial palsy, even when a surgeon is confident that the nerve is safe.

23.2. Olfactory Nerve Injury

The olfactory nerve is a special sensory nerve that is responsible for olfaction. People who have olfactory nerve dysfunction live a life with lower quality (Svider et al. 2014). Dysgeusia is caused by a distorted or nonexistent sense of olfaction, which makes it difficult to enjoy a meal. The diminished ability to perceive toxic smoke or an adjacent fire is a potentially life-threatening matter for persons with olfactory impairment. In such circumstances, a lack of early diagnosis may result in injuries or fatalities that may have been otherwise avoided.

23.3. Other Cranial Nerves Injuries

Though rare, any cranial nerve can be affected by a traumatic brain injury, especially penetrating injuries (Figure 31). But postsurgical paralysis of cranial (any) nerves is quite common and is conducted according to the approach, the site, and the nature of the pathology.

23.4. Medicolegal Aspects of Cranial Nerve Injury

Iatrogenic cranial nerve injuries may be a possible issue for malpractice since the aforementioned ailments have severe consequences for basic activities connected with sustaining a positive and good quality of life. Malpractice litigation has increased exponentially in the last 30 years, resulting in higher malpractice insurance costs and the exercise of defensive medicine (Brenner and Smith 2004). These developments, in combination with other variables, have attributed to increased healthcare expenses in the US. As a result, practicing surgeons may find it beneficial to learn the fundamental principles and rules of CN injury litigation, particularly for application in preoperative counselling as well as the informed consent process.

24. Traumatic CSF Rhinorrhea and Otorrhea

CSF rhinorrhea or otorrhea indicates that the subarachnoid space is open to the nasal cavity or tympanomastoid cavities. Because such a conection poses a serious risk of intracranial infection, the disease must be appropriately recognized, and the link must be closed. The identification and localization of a CSF leak can be difficult at times. There are also some widespread misconceptions about the utility and precise role of positive contrast computed tomographic cisternography. When there is direct discussion about the exact task being asked of the diagnostic procedure, the interdisciplinary team performs well. The best therapy options for patients will come from effective communication and focused imaging.

CSF leaks can be classified as either acquired or congenital. Trauma, frequently with fractures that result in a dural defect; surgical leaks; infections; and benign or malignant neoplasms are all acquired causes. The most common causes of congenital CSF leaks include developmental bone communication channels and accompanying dural deficits, as well as more serious developmental anomalies such as meningoencephaloceles and inner-ear dysplasia. CSF rhinorrhea is caused by the breach of the partitions that separate the subarachnoid space from the nasal cavity and/or paranasal sinuses. The most prevalent cause of CSF rhinorrhea from the anterior cranial fossa is head trauma, followed by endoscopic sinus surgery (ESS) or more extensive sinonasal operational procedures. A CSF fistula after ESS is thought to occur in less than 1% of cases. Conditions that raise ventricular pressure may have a role in the development of CSF fistulas. Congenital conditions such as developmental meningoencephaloceles, arachnoid granulations, or simply developmental areas of larger-than-normal bone abnormalities can cause CSF fistula. CSF rhinorrhea is a term used to describe an unusually fluid, unilateral nasal discharge caused by CSF leaks. It may also be detected during a search for a cause of meningitis, particularly recurring meningitis caused by bacteria with a nasal origin.

CSF leaks are caused by an osteodural defect that creates an improper connection between the CSF space and the neighboring paranasal air sinus or tympano-mastoid cavity, causing CSF rhinorrhea or otorrhea (Lloyd et al. 2008). The extension of infection from the sinonasal cavity puts affected individuals at risk of meningitis, the prevalence of which can be as high as 19–50% in individuals with persistent leakage (Daudia et al. 2007; Aarabi and Leibrock 1992). In spite of advances in drug treatment, the increasing risk of life-threatening consequences underlines the importance of early diagnosis, the correct dentification of the leak's location, and the execution of quick action to prevent morbidity. Endonasal endoscopic surgery for CSF rhinorrheas has become the standard of therapy due to its higher success rate as well as lower morbidity analogous to transcranial procedures (Zweig et al. 2002). Neuroimaging is important in the preoperative workup because it helps to pinpoint the location of the leak and determine the exact size of the osteodural gap. It also assists in endoscopic repair by promoting real-time anatomical neuro-navigation utilizing multiplanar imaging as well as endoscopic views, allowing the operating neurosurgeon to avoid critical structures (Kacker et al. 2005). CSF leaks are easier to diagnose when imaging is used.

25. Post-Head-Injury Syndromes

Post-traumatic brain injury syndrome (PHIS) is a symptom complex that consists of dizziness, headache, cognitive impairment, and neuropsychiatric symptoms and is a common complication of TBI. Mild TBI can happen when the head is struck, when the head hits an item, or when the brain accelerates or decelerates without any external trauma to the head.

It is divisive, particularly in its extended form. The clinical features are hazy, subjective, and widespread among different people. The patient population is diverse, with varying degrees of head as well as brain trauma.

The features of individual patients may influence how the injury manifests. The pathology that underpins this condition is unknown. Test abnormalities may or may not be present, and when they are, they are not consistent with a clear pattern.

- A loss of consciousness lasting <30 min is considered a mild head injury/concussion.
- PTA (post-traumatic amnesia) is a type of amnesia that lasts less than 24 h after a distressing event has taken place (this is a period wherein people are confused, act strangely, and are unable to remember what has just happened)

It is important that only around 10% of reported minor head injuries/concussions result in loss of consciousness, so do not rely on this as a sole indicator (Dean et al. 2012).

25.1. Symptoms of Concussion

In people who have suffered a moderate head injury, the symptoms of concussion include headache, irritability, difficulty concentrating, dizziness, confusion, nausea, a difficulty processing or memorizing information, light sensitivity, and vision distortion.

Following a minor head injury, there is a small possibility for the development of problems that may demand emergency care in the early stages.

Concussion symptoms usually go away after a few days or a few weeks, but some people may suffer from these symptoms for a much longer period. A symptom group known as "post-concussion syndrome" persists after a mild head injury or concussion (Dean et al. 2012).

25.2. Management of Concussion

While there is no one-size-fits-all treatment for concussion, most people recover effectively with good medical care, plenty of rest, and professional assistance, as needed.

Family members and employers must be notified of the potential implications of a mild head injury/concussion, and suitable measures must be established. These could include not rushing returning to work, limiting short-term stress, and avoiding alcohol (Dean et al. 2012).

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