

## Review article

**Roles of diagnostic imaging techniques in traumatic brain injury**

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**ABSTRACT**

Traumatic brain injury (TBI) remains a serious medical issue worldwide, because of its high prevalence, temporary disability and mortality rate. TBI is more common in young and middle-aged, i.e. the most active in the labor market, social relations of part of the population. TBI is damage to the brain from an external mechanical force, leading to temporary or permanent impairments of cognitive, physical, and psychosocial functions, with an altered state of consciousness. The diagnosis of TBI is a clinical decision, however, imaging, particularly Computed Tomography (CT) scan is a key in diagnosis, classification, prognosis, and follow-up. In this review, we have included the status of diagnosing TBI in both basic and clinical research. The objective of this study is to provide a brief overview of the various subtypes of traumatic injuries, their complications, along with both the non-invasive and invasive monitoring modalities necessary instances, with a brief analysis and literature review. For a detailed understanding and study of the topic, we included the CT scan data from our own research conducted at the GE LightSpeed VCT.

**Keywords:** Traumatic brain injury; cerebral hematoma; skull fractures; diagnostic strategies; CT scan.

**INTRODUCTION**

Traumatic brain injury (TBI) is one of the most common types of injury and accounts for about 40% of total injuries. Every year, 69 million individuals worldwide are estimated to undergo TBI (1,2). The highest rates of TBI reported in a very young age group (0-4y) as well as in adolescents and young adults (15-24y), There is even a high incidence in the elderly (>65y) (2, 3). The two common causes of TBI were falls and motor vehicle accidents. Even though there is an increased prevalence of TBI cases, the death rate is lower, but the increased rate of patients with TBI having disabilities (4).

**Worldwide incidence and volume of TBI**

According to Peden (5), about five million people die every year because of TBI. Thus, the death rate from TBI is 83 per one million of the population, which is 9% of all causes of death. The long-term loss of working capacity due to TBI accounts for 12% of all causes of disability (1-5). The incidence of TBI was highest in the USA and Canada (1299/100,000 people, 95% CI 650–1947), Europe (1012/100,000 people, 95% CI 911–1113), and lowest in the Africa (801/100,000 people, 95% CI 732–871). In regional populations, the higher prevalence of TBI annually

was seen in the populations of South-East Asian Region (18.3 million) and Western Pacific Region (17.3 million; 6).

The frequency of TBI in Russia ranges from 1.6-7.2/1,000 people/year, i.e., more than 600,000 people/year. In Kyrgyzstan, the frequency is 4.0/1,000 people and a mortality rate of up to 11/100,000 people (7).

As per pathogenesis, TBI is divided into primary and secondary brain damage. The primary injuries are caused by the effects of traumatic force on the bones of the skull, the casing and the brain tissue, the cerebral vessels, and the lymph system (8).

These disorders arise from different types of primary injury and result in a variety of cell damage, resulting in a wide range of damage processes. A head injury causes primary damage to the nerve cell membrane, white matter structure, the vascular course, and the triggering of secondary damage mechanisms: metabolic stress and ion disorders. As a result, a whole complex of biochemical and molecular changes causes the death of neurons (4,5).

The objective of this study is to provide a brief overview of the various subtypes of traumatic injuries, their complications, along with both the non-invasive and invasive monitoring modalities

necessary instances, with a brief analysis and literature review. For a detailed understanding and study of the topic, we included the Computed Tomography (CT) scan data from our own research conducted at the GE LightSpeed VCT.

### Role of imaging modalities

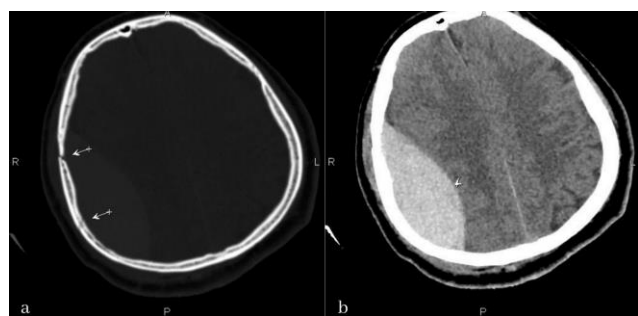
In many cases, if a patient suffering from a head injury and his CT scan of the brain gives us a conclusion about the presence of hemorrhage in the brain, it has been categorized into intra-axial hematoma (hemorrhage inside the brain) or extra-axial hematoma (hemorrhage outside the brain; 2).

The different imaging techniques used in imaging of traumatic injuries of the brain such as X-rays used for screening, in older days but it can miss skull fractures. CT has been the preferred modality in the acute setting and best for detecting acute traumatic hemorrhages and skull fractures, which saves times compared to Magnetic Resonance Imaging (MRI; 9,10). MRI of the brain is not preferred in the acute setting as it is time-consuming and not readily available. Mostly used in special cases, if neurological deterioration/suspected diffuse axonal injury (DAI), which detects complications developing over time, failure to improve with treatment.

### Clinical presentation

#### Extradural hemorrhage (EDH)

EDH originates from the medial meningeal artery (MMA) and is associated with a skull fracture-the bone fractures that cause damage to an MMA branch which results in EDH. It is bled in the extradural space between the outer layer of the dura (endosteal layer) and the inner table of the skull vault. Clinically associated with lucid interval and most common site is a temporoparietal junction. The shape is typically biconvex (lenticiform) and EDH does not cross the cranial sutures. It is not limited to venous sinuses, but it can cross and elevate/displace venous sinuses (Table 1) (9).



**Fig. 1.** Axial non-contrast CT scan of the brain:  
Extra-axial biconvex hyperdense collection  
associated with a fracture consistent with  
extradural hematoma

### CT scan

In most cases, EDHs are seen on CT scans of the brain. They are typically biconvex (lenticiform) in shape, and most frequently beneath the squamous part of the temporal bone. EDHs are hyperdense, somewhat heterogeneous, and sharply demarcated. Depending on their size, secondary features of mass effect (midline shift, subfalcine, and uncal herniation) may be present. When acute bleeding is occurring at the time of CT, the non-clotted fresh blood is typically less hyperdense, and a swirl sign (internal hypodensity within the bleed may suggest continuous bleed, which requires urgent management) may be evident. Post-contrast extravasation may be seen rarely in the case of acute EDH and peripheral enhancement due to granulation and neovascularization can be seen in chronic EDH (Fig. 1).

### MRI

MRI demonstrates the displaced dura that appears as a hypointense line on T1 and T2 sequences which are helpful in distinguishing it from a subdural hematoma. Acute EDH appears isointense on T1 and shows variable intensities from hypo- to hyperintense on a T2 sequence. Early subacute EDH appears hypointense on T2 while late subacute and chronic EDH is hyperintense on both T1 and T2 sequences. Intravenous contrast displaced or occluded venous sinus in case of the venous origin of EDH (2).

Angiography is rarely done. Tram-track sign may be seen due to contrast extravasation from the MMA and draining into the paired, parallel middle meningeal veins (2).

**Subdural hemorrhage (SDH)** is originated from bridging veins. **Bleeding in the subdural space:** large potential space that can accommodate a large amount of blood before causing mass effect on the underlying brain. **Spontaneous SDH in the elderly:** may be asymptomatic or present with mild headache or transient but repetitive neurological deficits-termed pseudodementia. **Concavo-Convex shape:** the inner margin is concave; the outer margin is convex. **Pseudo empty Delta sign:** the acute SDH lining the walls of the superior sagittal sinus may make the walls look hyperdense while the contents look relatively hypodense resembling the Empty Delta sign of sinus thrombosis (Fig. 2, Fig. 7). **Comma-shaped SDH:** SDH along the falx cerebri spreading along the tentorium cerebelli. Not limited to cranial sutures but limited to Dural folds like falx cerebri, falx cerebella, and tentorium cerebelli. **Subacute SDH:** Is so-dense to the brain and may be difficult to detect, where MRI is more useful. **Chronic SDH/Subdural hygromas:** bilateral hypodense (Cerebrospinal fluid (CSF) density) crescentic collections (Table 1). CSF levels may be

seen if a fresh hemorrhage occurs. SDH in neonates and infants could raise suspicion of non-accidental injury/Battered baby syndrome (11).



**Fig. 2:** Axial non-contrast CT scan of the brain: Hyper dense crescentic hematoma, extending across suture lines, i.e., acute subdural hemorrhage

**Subarachnoid hemorrhage (SAH)** a common cause is head injury and bleeding seen within the basal cisterns/sulcal spaces. SAH can result from disruption of small pial vessels, an extension of blood into the subarachnoid space from a contusion or hematoma, diffusion of intraventricular hemorrhage (12).

**CT:** Appears as linear or serpentine areas/finger-like extensions of high attenuation that conforms to the morphology of the cerebral sulci and cisterns - commonly seen in the Sylvian fissures and interpeduncular cistern (Fig. 3, Fig. 6).

**MRI:** Can't detect in acute stage than on CT. FLAIR images may show sulcal hyperintensity. Sub-acute and chronic SAH is better picked up on MRI (13).

**Table 1:** Characteristic differences between EDH and SDH for better consolidation (EDH vs SDH) (2).

	EDH	SDH
Origin of bleed	Middle meningeal artery	Bridging cortical veins
Side	Unilateral	Unilateral or Bilateral
Symptoms	Lucid interval followed by unconsciousness	Gradually increasing headache, confusion leading to altered sensorium
Associated fractures	Commonly associated with fractures	Not associated with fractures
Limited to	Cranial sutures (Not limited to dural folds/venous sinuses)	Dural folds/venous sinuses (Not limited to cranial sutures)
Shape	Biconvex	Concavo-Convex (crescentic/banana shaped)
Surgical drainage	Almost required	Assessed based on neurological status, mass effect and midline shift

EDH: Extradural Hemorrhage; SDH: Subdural Hemorrhage.



**Fig. 3:** Axial non-contrast CT scan of the brain: subarachnoid hemorrhage on the convective surface of the right frontal lobe.



**Fig. 4:** Brain contusion in the right hemisphere of the cerebellum without hemorrhagic component.



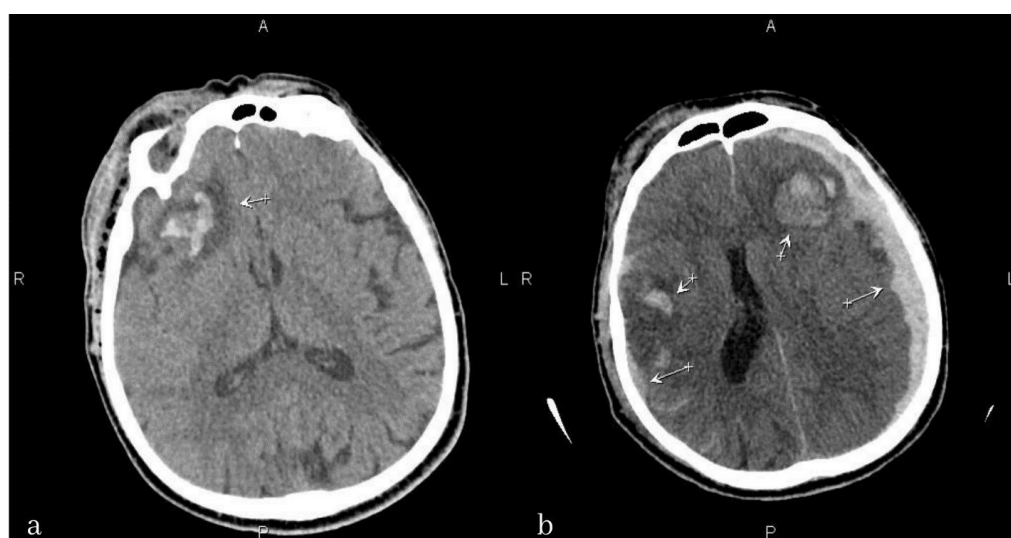
**Fig. 5:** Brain contusion without hemorrhagic component (arrow), subdural hematoma (arrowhead).

Parenchymal Contusions may be hemorrhagic/non-hemorrhagic. Seen in areas of the brain close to rough bony surfaces, hence commonly seen in orbitofrontal and temporal regions of the brain. On CT seen as ill-defined hyper densities (blood) with large surrounding hypo densities (marked peri bleed edema). On MRI, contusions can be observed as ill-defined areas of variable signal intensity on both T1-weighted and T2-weighted images, depending on the age of the lesions (Figs. 4, 5) (14).

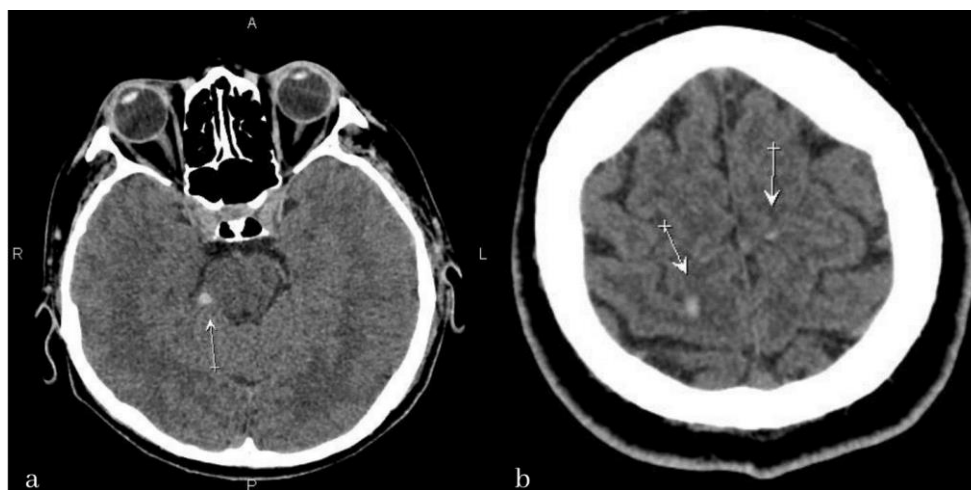
**DAI:** It is a “History Clincher” and follows the following sequence in most of the cases - non-improving altered sensorium in a head injury patient, with normal initial CT scan. DAI is shearing stress/force exerted on the brain, because of rotational acceleration and deceleration forces occur during a road traffic accident. When such shearing force is applied to a neuron, the neuron simply breaks off its point and the junction between the cell body and neurofilament. This is a permanent break and irreversible. So this Axonal level injury occurring in a diffuse manner is called DAI (Fig. 7) (15).

**CT/MRI Findings:** Small petechial bleeds are seen mainly at three sites - 1) Gray-White matter junction is the most common site that corresponds to the site of neuronal break/discontinuity, 2) Corpus callosum and 3) Dorsolateral brainstem.

Non-hemorrhagic foci of DAI are seen as focal hyperintensities in the brain on T2W/FLAIR images. MRI is an Investigation of choice in DAI (13).



**Fig. 6:** Axial non-contrast CT scan of the brain: Brain contusions with intracranial hemorrhages (short arrows) and Subdural hematomas (long arrows).



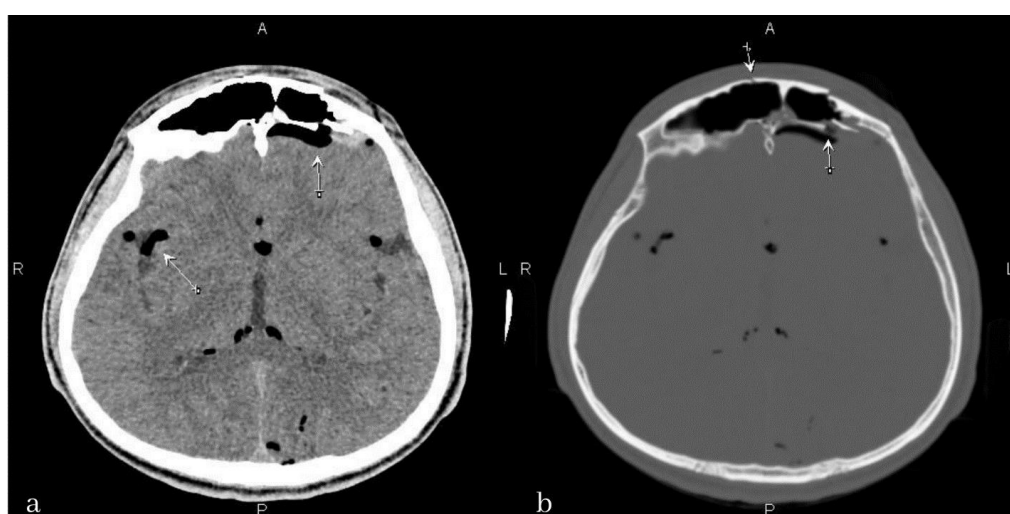
**Fig. 7:** Axial non-contrast CT scan of the brain: focuses on diffuse axonal damage.

**Pneumocephalus:** Most common cause trauma suggests basal skull fracture with air coming from the paranasal sinuses. Tension Pneumocephalus occurs when subdural air causes a mass-effect over the underlying brain parenchyma, often from a ball-valve mechanism causing one-way entry of air into the subdural space. In the Mount Fuji sign, air may separate and compress the frontal lobes, creating a widened interhemispheric space that mimics a picture of a volcano just like Mount Fuji in Japan (Fig. 8). Treatment is emergent surgical decompression. CT scan is an Investigation of choice.

**Skull fractures:** Skull fractures are best imaged with CT of the brain. CT is not only sensitive in the detection of fractures but is also able to exquisitely characterize their extent and allow for surgical planning. Furthermore, it is obtained at the same time as the brain is imaged. If the force of the impact is

excessive, the bone fractures at or near the site of the impact and cause damage to the underlying structures within the skull such as the membranes, blood vessels, and brain. A simple fracture is defined as having one bone fragment; a compound fracture exists when there are 2 or more bone fragments. Below we list the most common types of skull fractures.

**Linear fractures:** The most common, and usually require no intervention for the fracture itself. They have the form of thin lines that do not cause bone fragments to shift and rarely require emergency measures. These types of fractures are the least dangerous. Typically, they do not cause the loss of consciousness. It can cause damage to the membranous arteries and the formation of EDH (Figs. 9, 10, 11, 12).



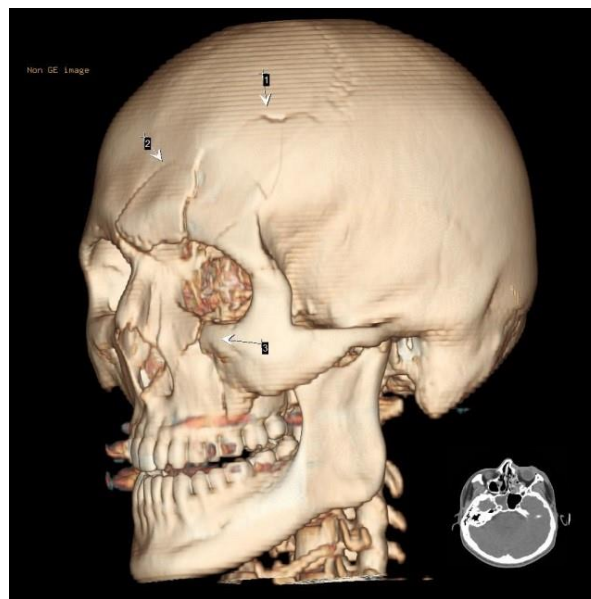
**Fig. 8:** Cranial CT scan showing Pneumocephalus. a) Axial non-contrast CT scan of the brain: gas bubbles (pneumocephaly) b) Fractures of the external and internal walls of the frontal sinus.



**Fig. 9:** Linear fracture of the occipital bone.



**Fig. 10:** Major bone linear fracture.



**Fig. 11:** Linear fracture of the frontal bone, maxilla (arrowed).



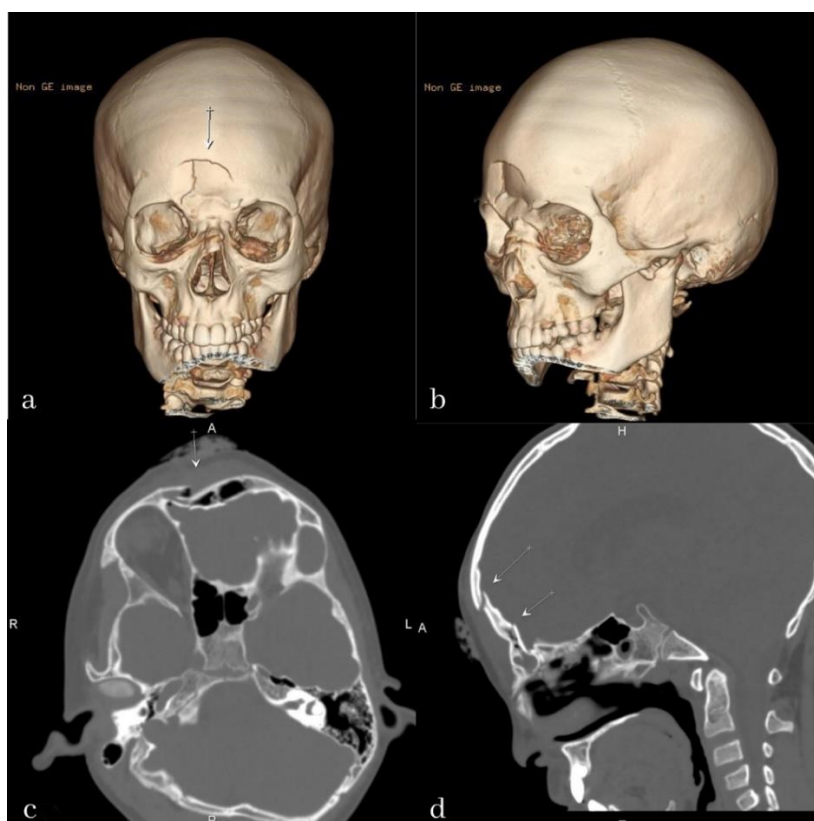
**Fig. 12:** Linear fracture of the base of the left anterior cranial fossa (arrowed).



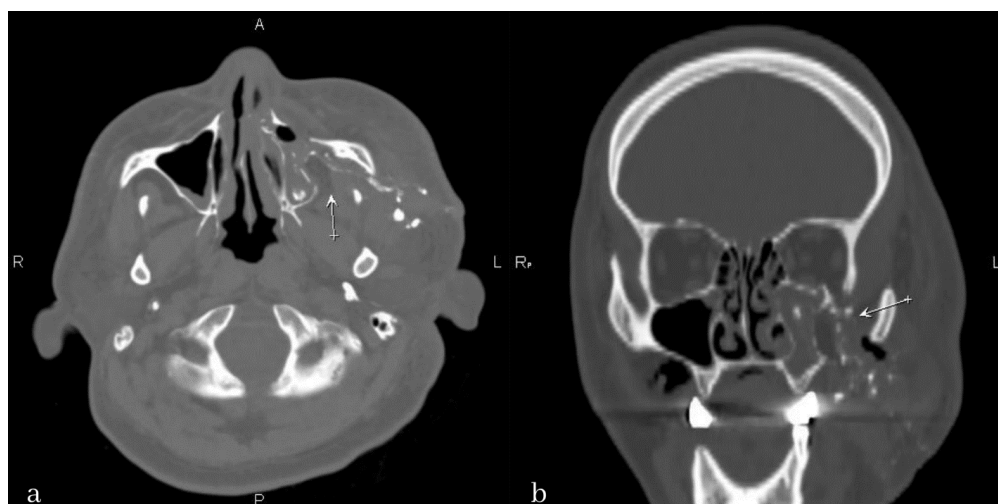
**Depressed fractures:** Usually comminuted, with broken portions of bone displaced inward and may require surgical intervention to repair underlying tissue damage. Depressed fracture or “signature fracture” caused by a heavy weapon with a small striking surface (hammer). The fractured bone is driven inward, and its shape may have indicated the type of weapon with which it is produced, therefore it is known as a signature fracture. A depressed fracture of the frontal or parietal bone can occur before or during labor because of compression of the fetal head by the maternal pelvis; or it can occur during delivery, usually because of a forceps operation (Fig. 13).

**Comminuted fracture:** Mostly caused by vehicular accidents “falls from height”, and blow from a weapon with a large striking surface (A heavy iron bar). The bone is broken into two or more pieces (Fig. 14).

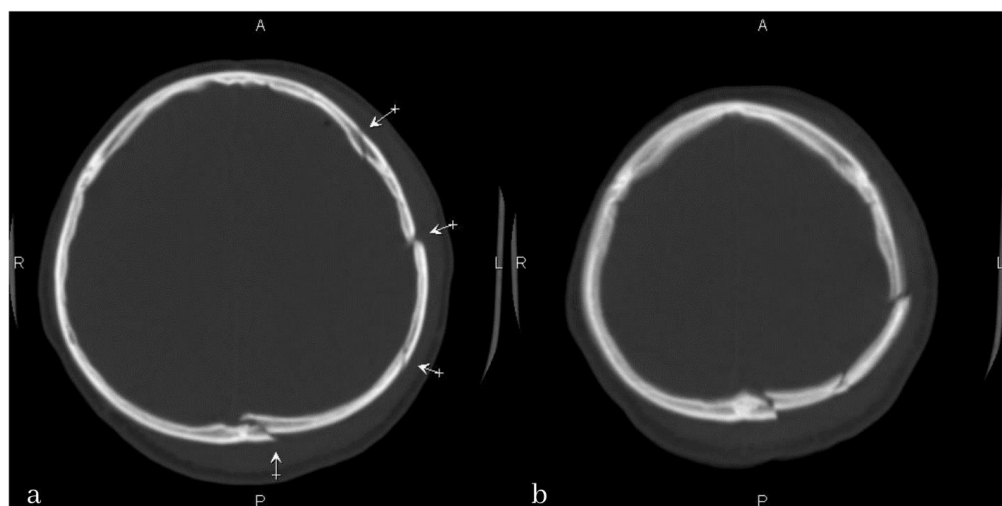
A fracture in conjunction with an overlying laceration that tears the epidermis and the meninges or runs through the paranasal sinuses and the middle ear structures, bringing the outside environment into contact with the cranial cavity is called a compound fracture (Fig. 15).



**Fig. 13:** Frontal bone fracture



**Fig. 14:** Comminuted fracture. a) Comminuted fracture of the external wall of the left paranasal sinus b) Multiple small foreign bodies in the subcutaneous soft tissue (arrowed).



**Fig.15:** Compound bone fracture of the cranial vault on the left, with linear fractures of the frontal and left parietal bones.

## Complications

Long-term sequelae of head trauma include: Encephalomalacia/gliosis, chronic SDHs/CSF hygromas, chronic traumatic encephalopathy, depression, anxiety, and alcohol. Increased risk of schizophrenia, bipolar disorder, and organic mental disorders. Severe mass effect can result in midline shift associated with worse prognosis, cerebral herniation requires urgent treatment and hydrocephalus can also be a chronic non-mass effect related complication.

## Diagnostic imaging studies for TBI

As we discussed earlier, CT remains a vital tool in the assessment of patients with TBI in the acute setting, as it rapidly gives critical information that directly impacts the management of acute TBI patients (7, 2). CT reveals a hematoma, midline shift, compression of ventricles, hydrocephalus, and depressed fractures that necessitate surgical intervention. CT allows us to triage patients who need surgical intervention versus conservative care. Imaging findings of CT are often compared with MRI as a gold standard or correlated with six months' clinical outcome.

MRI is superior to CT in terms of sensitivity to detect subtle abnormalities and address injury in the posterior fossa and brainstem. MRI is indicated for patients with neurological symptoms not explained by CT abnormality or mild TBI patients with persistent symptoms. The course of hemorrhage and traumatic intracranial hematoma shows that increased traumatic intracranial hematoma occurs earlier in the post-traumatic course (within the first 24 h). Therefore, when the initial CT scan shows

abnormality (hematomas), follow-up with head CT is recommended to assure stability of the abnormality.

Sensitivity and specificity of CT is lower than MRI with Gradient Echo Sequences (GES) or Susceptibility weighted imaging (SWI). When head CT is normal, yet there is a discrepancy between imaging findings and clinical symptoms, one should consider obtaining a brain MRI for accurate assessment of TBI. Typical MRI protocols for TBI include T1-weighted sagittal images, coronal fluid-attenuated inversion recovery (FLAIR) images, T2-weighted axial images, coronal GRE, or SWI images, and diffusion-weighted imaging (DWI). No intravenous contrast is necessary for the assessment of TBI on MRI (16).

Functional imaging studies using single-photon emission computerized tomography (SPECT) or positron emission tomography (PET) scans have been used to evaluate patients with mild TBI where CT or MRI showed no detectable abnormalities.

## Role of imaging in the prediction of injury or outcome in TBI

As earlier discussed, imaging plays a major role for TBI patients, however, caution is required to clarify requirements in imaging among patients with head trauma, given ionizing radiation risk and overall medical costs involved. It is estimated that CT scans contribute approximately 45% of the US population's collective radiation dose from all medical x-ray examinations. This is particularly serious among pediatric trauma patients. Even though radiation exposure is a concern for both adults and children, children are more sensitive to radiation than adults



and have a longer life expectancy, leading to more chances for radiation-related illness. When the pediatric patient needs head CT after trauma, pediatric head CT protocol endorsed by many professional organizations such as the American College of Radiology, Society of Pediatric Radiology, American Academy of Pediatrics, and American Academy of Family Physicians was strongly encouraged.

Prediction rules for TBI can safely avoid head CT without adverse consequences. The most widely cited prediction rules include the New Orleans Criteria (NOC; 17), the Canadian Head CT Rule (CCHR), and the CT in Head Injury Patients (CHIP) rule (18). If a patient presents with a moderate-to-severe head injury, head CT is indicated to assess the extent of the injury and the need for surgical intervention. For patients with mild TBI (Glasgow Coma Scale (GCS) <13 or higher), it is critically important to define a group of patients with TBI without missing a patient

with significant TBI that necessitates surgical intervention. There are a few prediction rules for mild TBI.

Clinical symptoms and risk factors commonly used in the clinical prediction rules are: age, headache, vomiting, intoxication, amnesia, post-traumatic seizure, signs of skull fracture, GCS score, and GCS score deterioration after trauma. The primary outcome of interest is head CT abnormality, and the secondary outcome is neurosurgical intervention. The summary of prediction rules is shown in below Table 2. These prediction rules have different specificity; however, the sensitivity for detecting lesions requiring neurosurgical intervention is 100%. Specificity for NOC, CCHR, and CHIP are 24%, 32%, and 23%, respectively. Comparison between NOC and CCHR reports that both NOC and CCHR are highly sensitive for predicting neurosurgical intervention; however, CCHR is more specific than NOC.

**Table 2.** Comparison of three predictions rules: NOC, CCHR and the CHIP for use of CT in patients with minor head injuries.

	NOC	CCHR	CHIP
Headache	Major	-	-
Vomiting	Major	Major ( $\geq 2$ episodes)	Major
Post-traumatic seizure	Major	Excluded	Major
Intoxication	Major	-	-
Persistent anterograde amnesia	Major	-	Minor
Age	Major (>60 years)	Major ( $\geq 65$ years)	Major ( $\geq 60$ years) or minor (40-60 years)
Clinical signs of skull fracture	Major	Major	Major
Contusion of the skull	Major	-	Minor
Signs of facial fracture	Major	-	-
Contusion of the face	Major	-	-
GCS score deterioration	-	Major	Minor ( $\geq 2$ points) or minor (1 point)
Pedestrian versus vehicle	-	Minor	Major (also cyclist)
Ejected from vehicle	-	Minor	Major
Fall from height	-	Minor	Major
Prolonged post-traumatic amnesia	-	Minor (>30 min)	Major ( $\geq 4$ h) or minor (2 to <4 h)
GCS score <15 at presentation	Excluded	-	Major
Loss of consciousness	Inclusion	Inclusion	Minor
Neurologic deficit	Excluded	Excluded	Minor
Anticoagulation therapy	-	Excluded	Major

NOC: New Orleans Criteria; CCHR: Canadian Head CT Rule; CHIP: CT in Head Injury Patients; GCS: Glasgow Coma Scale.

## Availability of data and material

Data are available from the corresponding author upon reasonable request.

## CONFLICTS OF INTEREST

Authors declare that there is no conflict of interest.

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