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Original Research Paper

Radio-Diagnosis

ROLE OF NCCT BRAIN IN EMERGENCY CONDITIONS IN DAY-TO-DAY PRACTICE

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ABSTRACT Computed tomography (CT) scan of the brain is a good method of investigation to diagnose intracranial lesions as well as calvarial fractures in day-to-day emergency conditions. Paraparesis, paralysis, seizures & traumatic brain injury (TBI) is a major cause of morbidity and mortality worldwide. NCCT brain plays an important role in the evaluation, diagnosis, and triage of these patients. Here, we review role of NCCT brain including its indications, benefits, imaging protocols, and imaging findings for each these pathoanatomic entities. **Materials & Methods:** All patients with clinical presentation of seizures, paraparesis, paralysis, traumatic injury who underwent emergency brain CT scan are included in this study. Data on age, headache, vomiting, loss of consciousness or amnesis, post-traumatic seizure, physical evidence of trauma above the clavicles, alcohol intoxication & anticoagulant usgae were collected. All patients were scanned on 16 slice TOSHIBA scanners with same techniques at civil hospital, gandhinagar from the duration of 01/01/2022 to 30/06/2022. Brain CT scans were reviewed by the radiologists. The frequency of positive CT scans was determined fo each symptoms.

KEYWORDS : Computed Tomography (CT);Traumatic Brain Injury (TBI);Paraparess;Paralysis; Seizures.

INTRODUCTION:

Noncontrast computed tomography (NCCT) is the first-line imaging modality in the investigations of suspected minor ischaemic events & after traumatic brain injury. Readily available in most emergency departments, NCCT can rule out important stroke mimics (space-occupying lesion, subdural haemorrhage) and intracerebral hemorrhage. In a small proportion of patients with minor ischaemic cerebrovascular events (5%), the NCCT confirms acute ischaemia **(1),** which is an important finding for prognostication. Acute ischemia on NCCT has been shown to be associated with increased stroke recurrence and disability. **(1,2)** In addition, the presence of white matter disease & remote infarcts also adds prognostic value. A prospective study of > 2000 consecutive patients presenting to the emergency department with TIA or nondisabling stroks found that the presence of acute ischemia, age-indeterminate ischemia, and microangiopathy on NCCT was associated with a 23-fold higher risk of recurrent stroke at 90 days compared with patients without these factors.**(1)**

Minor head injury (MHI) is one of the most common injuries seen in emergency departments, **(3)** which has typically been defined as patients with a history of blunt trauma who present findings of a Glassglow Coma Scale (GCS) score of 13-15 on initial ED evaluation. **(4)** The brain computed tomography scan is a good investigation method to diagnose intracranial lesions. **(5)** Indications for CT scan might be different based on the main outcome measures, which could be the presence of any abnormal lesion in CT scan related to the trauma **(6-8)** or presence of lesions suggested by a surgical opinion. **(9)** But because of the cost, **(10)** time, and probable complications of radiation, **(11)** there has been significant disagreement about the indications for brain CT scan in the large number of MHI cases.**(12)**

Symptoms such as headache, vomiting, loss of consciousness or amnesia, post-traumatic seizures, signs such as physical evidence of trauma above the clavicels, skull fracture or contusion & raccoon sign, past history such as alcohol intoxication or coagulopathy & age more than 60 years have been discussed as the risk indicators for abnormalities in brain CT scan of patients with MHI. The damage incurred by MHI can be differentiated into primary and secondary mechanisms. Primary injury is typically defined as the direct mechanical damage caused by trauma. These injuries are usually apparent acutely and include fractures, intracranial hemorrhage, contusion and traumatic axonal injury. This type of injury is best detected with conventional CT and MR structural imaging techniques.

Secondary injury mechanisms are varied, and relate to disruption of the blood brain barrier, production of reactive oxygen species and resultant oxidative stress, metabolic dysfunction, inflammation and excitotoxicity. These processes are mediated at the cellular level which is currently below the resolution of conventional imaging; however, they are believed to greatly contribute to the long term morbidity and disability associated with MHI. When severe, macroscopic manifestations of secondary injuries may become apparent as diffuse cerebral hyperemia, cytotoxic and/or vasogenic edema, and tissue ischemia.

Roles For Ct Imaging

(1) Acute, Moderate and Severe TBI—

Rapid imaging helps differentiate patients who require urgent/emergent neurosurgical intervention from those who can be safely monitored or sent home. Non contrast multi-detector CT (MDCT) has become the consensus choice for the initial imaging study after acute, moderate to severe TBI ,because it is fast, ubiquitous, very sensitive to calvarial injury and radio-opaque foreign bodies (e.g., gunshot fragments), and it is highly accurate for detecting injuries requiring emergent neurosurgical attention--namely hemorrhage, herniation and hydrocephalus. MDCT has also been shown to be useful for predicting clinical outcomes, and the NCCT findings have been incorporated into a number of outcome prediction rules.

(2) Acute Mild TBI—

Mild TBI, as defined by a GCS ≥13, is a misnomer in many cases as patients in this category often experience long-term debilitating symptoms that may interfere with normal daily activities. Nor does this definition for mTBI imply an absence of structural abnormalities on imaging. In a recent prospective

study evaluating imaging features in mTBI patients, When imaging is clinically indicated, non contrast CT is the primary initial modality of choice for evaluation of acute mTBI **(13, 14)**. After clinical screening, the majority of patients with mTBI for which imaging is indicated will have normal noncontrast CTs (i.e. "Uncomplicated" mTBI).

(3) Short-term Follow-up Imaging—

While MDCT is recommended in patients with neurological deterioration following TBI, studies have shown little benefit for routine follow up imaging**(15).** Among patients with initial MDCT positive for intracranial traumatic injury, only certain attributes including subfrontal/temporal hemorrhagic contusion, use of anticoagulation, age over 65 years, and volume of ICH >10 cc have shown high risk for progression**(16)**. At many institutions it is commonplace to obtain routine follow up MDCT in patients on anticoagulation, even when the initial MDCT is negative for acute intracranial pathology, though the clinical utility of this practice is not well established **(17)**. One recent prospective study evaluating patients with mild head trauma who were on anticoagulation found that repeat head CT imaging revealed hemorrhagic changes in only 1.4% of such patients after negative initial scan.**(18)**

(4) Imaging Subacute/Chronic TBI—

Subacute and chronic TBI are best evaluated with MRI **(19)**, which outperforms CT in its ability to identify parenchymal atrophy, white matter injury, and microhemorrhage. Imaging is indicated in patients who experience new, persistent, or worsening symptoms. NCCT should be performed to evaluate subacute/chronic TBI if MRI is contraindicated or unavailable.

(5) Vascular Imaging—

Intravenous contrast administration is not necessary or useful in the evaluation of TBI unless arterial or venous injury is suspected. NCCT findings can identify patients at increased risk for traumatic vascular injuries.**(20)** Patients with skull base fractures, particularly through the carotid canal, have much higher incidence of arterial injuries. Other findings that should raise suspicion for arterial injury include epistaxis, LeFort II and III facial fractures, high cervical spine fractures, GCS ≤8, or TAI36.

(6) Pediatric Imaging—

Head trauma is a common imaging indication in children and recommended imaging studies largely mirror those recommended for adults for given indications **(20)**. Children are more susceptible to harmful effects of ionizing radiation than adults and every effort should be made to avoid unnecessary examinations, particularly CT. In balance, however, diagnostic head CT should not be avoided when clinically indicated because of an overemphasized concern for the relatively small radiation dose associated with modern scanners. When CT is performed, dedicated pediatric CT protocols should be used to keep the dose as low as possible (see http://www.imagegently.org for more information). MRI does not expose patients to ionizing radiation; however, it does present its own challenges in pediatric patients. Children may require general anesthesia to tolerate MR examinations, which are much longer and more sensitive to motion than CT.

(7) Stroke-

Noncontrast head CT (NCCT) is the first-line diagnostic test for emergency evaluation of acute stroke due to its speed of imaging, widespread availability, and low cost. The window width and center level settings—measured in Hounsfield units: HUs—used for computed tomographic (CT) scan review are known to influence both lesion conspicuity and diagnostic accuracy. Numerous studies suggest that detection of early ischemic change (EIC) on NCCT can predict both functional outcome and the risk of intracranial hemorrhage (ICH)]. Specific features relevant to stroke assessment include hyperdense middle cerebral artery (MCA)/basilar signs, focal parenchymal hypoattenuation (notably of the insular ribbon or lenticular nuclei for MCA infarcts), and cerebral swelling manifested by sulcal or ventricular effacement or loss of cortical grey-white differentiation. Decreases in CT attenuation accompanying early stroke are small; therefore, their conspicuity may be increased by using narrow window settings centered at approximately the mean attenuation in HUs of gray and white matter.

Routine Ct Protocol

CT imaging is typically acquired helically in the axial plane on a MDCT scanner. An axial slice thickness of 2.5 mm is commonly employed for evaluating the brain parenchyma, though, thinner reformats (0.625 mm) using a bone kernel is recommended for the detection of subtle fractures. Helical acquisition allows the generation of reformatted images in different planes (typically coronal and sagital), as well as volume rendered 3D reformats, which can aid in the detection of subtle intracranial hemorrhage and fractures **(22, 23)**. Modern CT scanners also typically employ dose reduction software, which reduces patient radiation exposure.

Imaging Findings Of Traumatic Brain Injuries

Intracranial pathology can be subdivided to anatomic location, the most basic distinction being whether it localizes to the brain parenchyma (intra-axial) or outside the brain tissue (extra-axial). Vascular injuries, while typically extra-axial, are addressed separately in this discussion to highlight differences in the imaging workup.

(A) Extra-axial Lesions

Three intracranial, extra-axial spaces—epidural, subdural, and subarachnoid spaces—are potential sites for posttraumatic pathology, most often hemorrhage. . NCCT is excellent at detecting acute hemorrhage, which appears hyperdense to the surrounding brain parenchyma typically measuring between 50 and 70 Hounsfield units. In general, the density of a hematoma decreases as it ages, which can create challenges for identifying subacute and chronic hemorrhages that may appear isodense to the surrounding brain parenchyma.

(B) Epidural Hematoma—

Epidural hematomas (EDH) form as blood collects in the potential spaces between the inner table of the skull and the dura. EDHs can be arterial or venous in origin, and may relate to direct bleeding from fractured bone into the epidural space. Arterial epidural hematomas most commonly arise from laceration of meningeal arteries--typically the middle meningeal artery in the temporal or temporoparietal region**(25-29)**. In adults, an overlying skull fracture is present in the vast majority of cases. Occasionally (9%), EDHs can occur from stretching and tearing of meningeal arteries in the absence of fracture. The latter occurs commonly in children due to transient deformation and depression of the calvarial vault **(25).**

EDHs classically appear as a lenticular-shaped hematoma, which can cross dural attachment sites, but cannot cross cranial sutures. On CT, acute EDHs generally appear hyperdense. Some hematomas can appear heterogeneous with intrinsic irregular areas of low density, termed the "swirl sign," an important imaging finding that corresponds to extravasation of hyperacute unclotted blood**(30)**. EDHs with this sign tend to rapidly expand and warrant urgent surgical consultation. Rarely, it may be difficult to differentiate a small EDH that does not have the classic shape from a subdural hematoma (SDH) on CT.

Venous EDHs are less common than those of arterial origin. **(25-29)** They typically are caused by laceration of a dural

sinus in conjunction with fracture of the overlying skull **(31)**. The posterior fossa (transverse or sigmoid sinus injury), middle cranial fossa (sphenoparietal sinus) and parasagittal region (superior sagittal sinus) are the most common locations for venous EHD **(31)**. Venous EDHs, unlike SDHs, are not bound by dural attachments and will often extend both above and below the tentorium. Posterior fossa EDHs occur less frequently (2-29%), are more likely to be of venous origin (85%), and are associated with poorer outcomes than supratentorial lesions.**(32, 33)** Venous EDH may exhibit a more variable shape than those of arterial origin. Invariably, venous EDH are found adjacent to a fracture transgressing a dural venous sinus. These injuries have a high rate of associated dural sinus thrombosis or occlusion. When evaluating for venous sinus injury, it is important to distinguish external sinus compression by epidural blood from traumatic venous sinus thrombosis. In the latter, the dural venous sinus is usually irregular with a central sinus filling defect correlating to the thrombus.

(C) Subdural Hematoma—

Within the skull, the brain is relatively mobile and can move relative to the fixed dural sinuses. Injury to the bridging veins that traverse the subdural space connecting the brain to the dural sinuses results in the SDH.**(34-36)** While SDHs are most often found along the supratentorial convexities, they can also occur in the posterior fossa, along the falx cerebri, and adjacent to the tentorium **(37,38)**. Interhemispheric and tentorial leaf SDH are common in children, including cases of abuse resulting from violent shaking (shaken-baby syndrome) **(39,40,41)**. Although these hematomas are not specific for child abuse, their presence should lead to close consideration of the possibility.

Most SDHs manifest as a crescentic collection between the brain and / or inner table of the skull; interhemispheric and tentorial SDHs have a more linear morphology. All SDHs follow the typical appearance of evolving blood on CT and MR. While large acute hyperdense SDHs are readily apparent on CT, small subacute SDHs are often isodense and can be difficult to identify.

(D) Subarachnoid And Intraventricular Hemorrhage—

Subarachnoid hemorrhage (SAH) is a common finding in TBI. One large European series found evidence of SAH in 40% of patients with moderate-severe head injury**(42,43)**. Traumatic SAH is also the most common isolated finding in cases of mTBI and has been associated with poor outcome scores at 3 months after injury22. Acute traumatic SAH results from injury to small subarachnoid vessels or extension of intraparenchymal hemorrhage beyond the pial limiting membrane and into the subarachnoid space. Traumatic pseudoaneurysms typically do not occur in the acute setting. Acute SAH appears as curvilinear hyperdensity within the cortical sulci, sylvian fissures and basal cisterns on NCCT.

Intraventricular hemorrhage (IVH), considered a type of intra-axial injury by some authors, is addressed here because its pathophysiology and imaging findings largely overlap with SAH. IVH is also common in patients with head injury, occurring in 3% to 35% of cases, depending on the severity of trauma. Primary IVH may be caused by a variety of traumatic lesions including TAI, intracerebral hematoma (ICH), and contusions; however, the most prevalent etiology is thought to be the tearing of subependymal veins by rotational strain. Similar rotational forces are thought to cause callosal TAI and, indeed, one published series identified IVH in 60% of patients with TAI of the corpus callosum, but in only 12% of patients without callosal injury. Interestingly, another study found that the presence of IVH on admission NCCT was the only CT imaging finding predictive of grade II or III TAI on subsequent MRI.**(44)** IVH appears similar to SAH with the blood products appearing hyperdense to CSF on CT and hyperintense to CSF on FLAIR and T1WI with MRI28. A CSF-blood fluid level is often seen layering within the posterior aspect of the occipital horns (remember that the images are acquired with the patient in the supine position). Because the subarachnoid and intraventricular CSF spaces communicate, delayed imaging may demonstrate IVH due to recirculation of SAH and vice versa.

(E) CSF Leak

CSF leak is a complication in approximately 1-3% of TBI cases **(45)** and is typically associated with a basilar skull fracture. These cases usually present with CSF rhinorrhea (~80%) or otorrhea (~20%) within the first 48 hours after traumatic injury, though cases can present months to years after the initial insult with meningitis or orthostatic headaches **(45, 46)**. CSF rhinorrhea or otorrhea should prompt the search for a skull base fracture on thin section maxillofacial or temporal bone CT11. Common fractures involve of anterior cranial fossa (especially the frontal sinus or cribriform plates), sella and sphenoid sinus, and temporal bone.

CT cisternography is rarely used for initial evaluation, but can be helpful for problem solving in patients with multiple skull defects, negative initial CT imaging, or to confirm a CSF fistula when the diagnosis is unclear. In this setting, intrathecal contrast media is injected to opacify the CSF spaces. A positive study shows abnormal increased attenuation within a paranasal sinus, nasal cavity or middle ear secondary to contrast passage through the skull base defect.

(F) Intra-axial Injury

Intra-axial injuries refer to lesions within the brain parenchyma. Primary traumatic intra-axial lesions include the cortical contusion, intracerebral hematoma, TAI and brain stem injury. There are also secondary intra-axial injuries that can occur as a result of brain swelling and ischemia.

(G) Parenchymal Contusion

Parenchymal contusions occur when the brain forcibly impacts the irregular surface of the overlying skull, which typically occurs at (coup injury) or opposite (contrecoup) the site of blunt trauma. Contusions frequently are multifocal and bilateral, usually involving the superficial grey matter. They typically occur in the temporal (46%) and frontal (31%) lobes. These injuries tend to occur immediately adjacent to the petrous bone and posterior to the greater sphenoid wing in the temporal lobe and just superior to the cribriform plate, orbit roof, planum sphenoidale, and lesser sphenoid wing in the frontal lobe. Contusions less often involve the parietal and occipital lobes (13%) and cerebellum (~10%) Contusions frequently contain hemorrhagic foci ranging in size from punctate cortical surface petechiae to much larger confluent regions of hemorrhage occupying an entire lobe.

(H) Intracerebral Hematoma—

Intracerebral hematomas (ICH) result from injury to intraparenchymal arteries or veins secondary to rotational strain or penetrating trauma**(47-49)** and are usually located in the fronto-temporal white matter or basal ganglia. Differentiation from hemorrhagic contusions or TAI can be challenging; ICHs collect between relatively intact parenchyma in contrast to hemorrhagic contusions wherein hemorrhage is within a larger area of injured edematous brain. Prognosis of isolated ICH is generally good, but worsens when the lesion coexists with marked mass effect, TAI, or multiple basal ganglia haemorrhages**(47, 48)**. Temporal lobe hematomas are especially unpredictable, because even a relatively small lesion can lead to uncal herniation.

(I)traumatic Axonal Injury

Traumatic axonal injury (TAI) is one of the most common and important types of primary injury found in patients with all grades of head trauma. TAI typically results from rotational strain, and manifests as scattered, often bilateral, small white matter lesions with or without hemorrhagic components. When there are greater than 3 foci of radiographically evident TAI involving at least two separate lobes of the brain and the corpus callosum, the term diffuse axonal injury (DAI) is used.

TAI tends to be multifocal. The severity of injury worsens as involvement of the deep anatomic structures become affected**(50-52)**. In the Adams Classification for TAI, Grade 1 injuries involve the lobar white matter, particularly the grey-white junction frontal and temporal lobes. Grade 2 injuries extend to involve the corpus callosum, particularly the splenium. Grade 3 injuries involve the dorsolateral aspect of the upper brainstem. **(50-52)**

TAI lesions are usually ovoid with their long axis oriented in the direction of the involved axonal tracts. They range in size from 2 to 15 mm--peripheral lesions tending to be smaller than more central ones. Overall, CT has poor sensitivity for TAI. Only about 10% of TAI display the classic CT findings of punctate hemorrhage in the characteristic white matter loci91.

(J) Brain Stem Injury—

Brainstem injury (BSI) can be divided into primary injuries that result directly from the initial impact and secondary injuries which develop subsequently **(53-55)**. Primary brainstem injury can be divided into 4 categories. The first type occurs when severe posterior displacement of the brain forces the dorsolateral upper brainstem to directly impact the tentorium producing laceration or contusion**(56)**. This mechanism is thought to be uncommon and, unlike brainstem TAI, it is not necessarily associated with more diffuse white matter injury.

The other types of BSI are indirect, the most common of which is brainstem TAI **(57).** Brainstem TAI is invariably seen in the context of similar axonal lesions in the supratentorial white matter. A third type of primary BSI is characterized by multifocal scattered petechial hemorrhages concentrated in the deep central white matter, hypothalamus, thalamus, and periaqueductal rostral brainstem **(58)**. These lesions usually carry a grim prognosis. While both entities share a shear-strain etiology, unlike brainstem TAI, this entity is not associated with lobar white matter, corpus callosum, or superior cerebellar peduncle lesions. It is worth noting that distribution of these petechial hemorrhages differs from the secondary (Duret) hemorrhages described below. The final type of primary BSI is pontomedullary separation or rent, which is caused by a hyperextension-induced tear of the ventral brainstem at the pontomedullary junction **(59, 60)**. This injury may range from an incomplete tear to complete brainstem avulsion and is typically, but not invariably fatal.

(J) Secondary Injuries

Much of the morbidity and mortality associated with TBI relates to secondary injuries. Mass effect from hematoma or cerebral swelling in a fixed intracranial volume causes increased intracranial pressure and can lead to herniation. Herniation refers to the displacement of brain parenchyma into a different compartment. Subfalcine herniation occurs when the cingulate gyrus is displaced beneath the free edge of the falx cerebri. It can lead to further complications including distal anterior cerebral artery (ACA) territory infarcts(the callosomarginal branch runs along the cingulate sulcus) and contralateral hydrocephalus secondary to obstruction of the foramen of Monro. Uncal herniation occurs when downward pressure forces the medial temporal lobes to descend below the tentorial incisura into the ambient cistern where they compress the brainstem or posterior cerebral artery (PCA). Mass lesions or edema in the posterior fossa can result in upward herniation of the superior cerebellum through the incisura and compress the superior cerebellar arteries (SCA). Increased pressure from above can force the cerebellar tonsils inferiorly into the foramen magnum causing mass effect on the medulla and compression of the posterior inferior cerebellar arteries (PICA).

Downward transtentorial herniation can lead to BSI secondary to mechanical compression. Duret haemorrhages are typically centrally located collections of blood in the tegmentum of the rostral pons and midbrain associated with a grim. These secondary brainstem hemorrhages are thought to result from stretching/tearing of upper brainstem penetrating arteries during sudden downward transtentorial herniation. Focal brainstem infarcts may also occur via the same mechanism.

Both CT and MR are excellent at evaluating for herniation and hydrocephalus, though MR performs better in the posterior fossa where CT is limited by beam hardening artifact. Signs of herniation include midline shift, effacement of the basilar cisterns and cerebral sulci, and (often contralateral) ventricular entrapment. Hypoxic-ischemic brain injury typically results from a period of catastrophic cardiac or vascular compromise.

On imaging it is associated with diffuse cerebral swelling and edema. Classic CT signs include the grey-white reversal sign(i.e., the white matter appears denser than the grey matter) and pseudo-subarachnoid hemorrhage (i.e., the perimesencephalic and sylvian cisterns appear hyperdense to brain; the falx and tentorium also appear abnormally hyperdense).

(K) Stroke:

Specific features relevant to stroke assessment include hyperdense middle cerebral artery (MCA)/basilar signs, focal parenchymal hypoattenuation (notably of the insular ribbon or lenticular nuclei for MCA infarcts), and cerebral swelling manifested by sulcal or ventricular effacement or loss of cortical grey-white differentiation . Decreases in CT attenuation accompanying early stroke are small; therefore, their conspicuity may be increased by using narrow window settings centered at approximately the mean attenuation in HUs of gray and white matter.

(L) Benign / Malignant Brain Tumours:

Although any suscpiciopus brain tumours are definitely diagnosed on contrast study or MRI study, NCCT brain is useful in identification of some brain tumours like menigngioma, pitutary tumours, cystic tumours etc. Meningioma feature on NCCT are calcified well-defined rounded intra-axial lesion. Whereas pitutary tumours features on NCCT are hypo / isodense lesion within the pitutary fossa. Cystic tumours features on NCCT are well-definded homogenously fluid-density intra-axial lesion.

(M) Infective / Inflammatory Sol:

Cerebral abscess on NCCT can be diagnosed by hypodense lesion with surrounding cerebral edema. Tubercuous / NCC can be diagnosed by ring shaped iso / hypodense intra-axial lesion. However, for the confirmation of these lesions, contrast study maybe reqiured.

Statistical Analysis Of Data

Qualitative variables such as the presence of various NCCT features were expressed as percentages. Total numbers of 300 patients were included in the study. Out of which, normal CT findings are seen in almost half of the patients. In rest of the patients, positive findings were seen and its percentile calculation done in below mentioned table.

NO	NCCT FINDINGS	NO OF PATIENTS	PERCENTAGES
1	EXTRA-DURAL HAEMORRHAGE	65	21.66%
2	SUB-DURAL HAEMORRHAGE	45	15%
3	SUB-ARACHNOIS HAEMORRHAGE	40	13.33%
4	INTRA-VENTRICULAR HAEMORRHAGE	18	6%
5	INTRA-PARENCHYMAL HAEMORRHAGE	24	8%
6	INFARCT	75	25%
7	BENIGN / MALIGNANT SPACE OCCUPYING LESION	3	1%
8	INFECTIVE / INFLAMMATORY SOL	1	0.33%

DISCUSSION

NCCT brain is essential for establishing diagnosis, understanding early prognosis, identifying the pathogenesis of the event, guiding pathogenesis- specific stroke prevention treatments. Multi-detector CT remains the preferred first line imaging study for moderate and severe traumatic brain injury as it can quickly identify patients who require urgent neurosurgical intervention. NCCT brain is also useful in identifying benign / malignant lesions like meningioma, pituitary tumours, cystic tumours, etc. NCCT brain is also useful in infective / inflammatory conditions like tuberculoma, neurocysticercosis, brain abscess, etc.

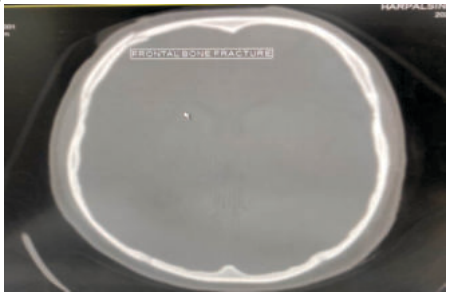

FIGURE 1: Frontal bone fracture on right side on non-contrast CT study.

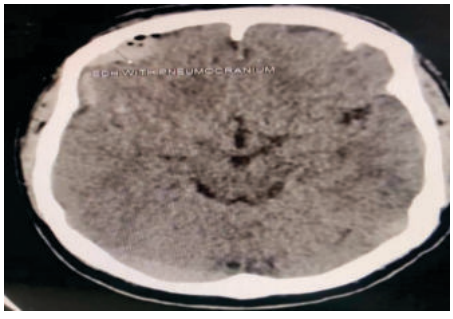

FIGURE 2: Frontal lobe EDH with few foci of pneumocranium on non-contrast CT study.

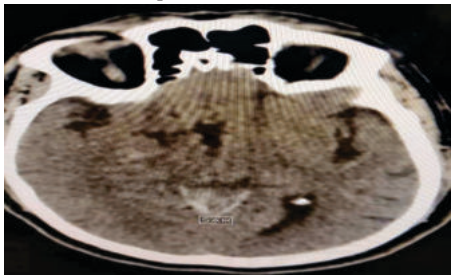

FIGURE 3: Thin strip of sub-arachnoid haemorrhage along basal cisterns on non-contrast CT study.

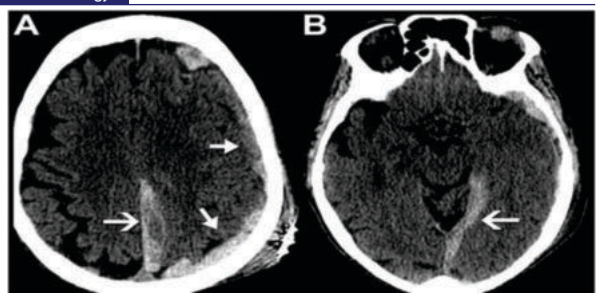

FIGURE 4: Left parietal scalp laceration reveals hyperdense blood along the left convexity (**A**, closed arrows), left posterior falx cerebri (**A**, open arrow), and tentorium cerebelli (**B**, open arrow) on non-contrast CT study.

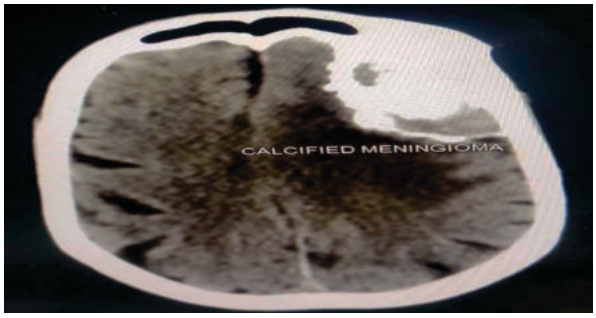

FIGURE 5: Left fronto-parietal lobe calcified meningioma on non-contrast CT study.

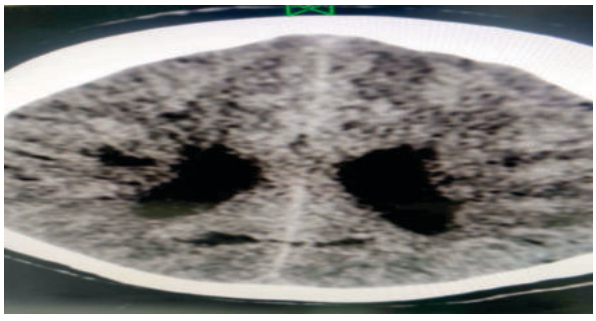

FIGURE 6: Right ganglio-capsular region lacunar infarct on non-contrast CT study.

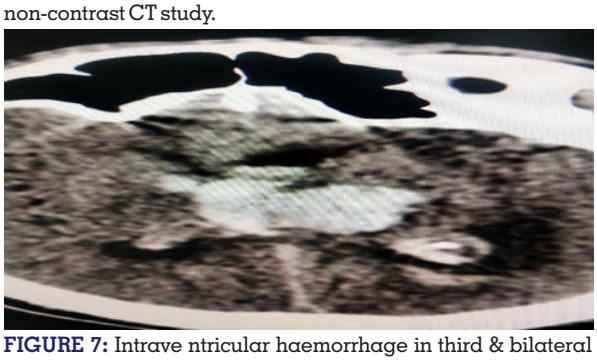

FIGURE 7: Intrave ntricular haemorrhage in third & bilateral lateral ventricles on non-contrast CT study.

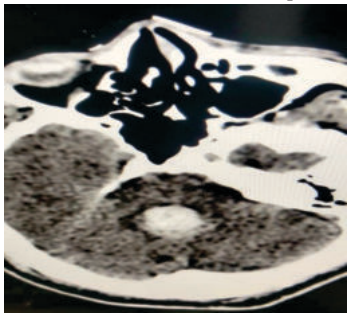

FIGURE 8: Intraventricular haemorrhage in fourth ventricle on non-contrast CT study.

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