CRANIOCEREBRAL TRAUMA IN NATIONAL HIGHWAY-CT EVALUATION AND CLINICAL CORRELATION

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M.D IN **RADIODIAGNOSIS**

By

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APRIL – 2013

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DEDICATED TO,

LATE MR. BASAVARAJ RAMPUR., MY BROTHER IN LAW WHO WAS VICTIM OF ROAD TRAFFIC ACCIDENT,

OUR BELOVED TEACHER LATE DR MOHD. BAHEER.,

ALL MY TEACHERS ...,

MY PARENTS, MY BROTHER..,

MY FIANCÉE, HER FAMILY AND MY ENTIRE FAMILY..,

MY COLLEAGUES..,

LIST OF ABBREVIATIONS USED

RTA -- Road traffic accident

TBI -- Traumatic brain injury

LOC -- Loss of consciousness

CT -- Computed tomography

MRI -- Magnetic resonance imaging

SDH -- Subdural hematoma

EDH -- Epidural hematoma

SAH -- Subarachnoid hemorrhage

AVF's -- Arteriovenous fistulas

DAI -- Diffuse axonal injury

IVH -- Intra ventricular hemorrhage

ACA -- Anterior cerebral artery

PCA -- Posterior cerebral artery

PICA-- Posterior inferior cerebellar artery

ICA -- Internal carotid artery

GCS -- Glasgow coma scale

TCDB -- Traumatic Coma Data Bank

NICE -- National Institute for health and Clinical Excellence

CHIP -- CT in Head Injury Patients

SIGN -- Scottish Intercollegiate Guidelines Network

ABSTRACT

CRANIOCEREBRAL TRAUMA IN NATIONAL HIGHWAY.

CT EVALUATION AND CLINICAL CORRELATION.

Objectives of the study:

- To evaluate and assess role of CT in localizing the injury to the particular intracranial compartment in patients with cranio-cerebral trauma.
- To correlate CT findings with Glasgow Coma Score and clinical findings.
- To study whether Glasgow Coma Score &/or clinical findings can be used as an indication for CT scan in order to avoid unnecessary CT scans.

Materials and Methods:

During the period of 12 months of our study, 300 cases with history of road traffic accident and suspected of cranio-cerebral trauma, who fulfilled inclusion criteria's were undertaken for study.

Relevant history of the patients was taken, which included age, sex, type of injury, principal presenting complaints such as headache, vomiting, loss of consciousness etc.

Relevant clinical findings were noted-such as external injuries over head, pupillary size and reaction etc.

Glasgow coma scale (GCS) was recorded as Normal :15, Mild head injury:13-14, Moderate head injury: 9-12 and Severe head injury: < 8.

Non contrast CT brain was performed with SIEMENS Esprit single slice Spiral CT unit.

CT findings were correlated with history, clinical findings and Glasgow coma scale.

Results:

Out of 300 patients, 249 were male patients (83%) and 51 were females patients (17%). Age distribution of patients with road traffic accidents showed highest in age group between 20-30 years (109 patients 36.33%).

Glasgow coma score was normal in 207 patients, mild in 13 patients, moderate in 51 patients and severe in 29 patients.

96 patients showed various types of intracranial bleeds. Subdural hematoma was commonest, seen in 59 patients (61.46%). Subarachnoid hemorrhage was seen in 37 patients, epidural bleed in 8 patients and intraparenchymal hematoma was seen in 47 patients. Skull fractures were seen in 105 patients. Mid line shift was seen in 16 patients and pneumochepalus in 16 patients. Some of the patients showed more than one abnormality.

When Glasgow coma scale alone is taken as test to identify intra cranial bleed, it showed sensitivity 88.5%, specificity 96.1%, positive predictive value 91.4%, and negative predictive value 94.7%. When Glasgow coma scale combined with relevant clinical findings is taken as test to predict intra cranial bleed, it showed sensitivity 97.8%, specificity 63.4%, positive predictive value 55.3%, and negative predictive value 99.2%.

Conclusion:

CT was able to localize accurately intra cerebral injuries to the particular intracranial compartment in patients with craniocerebral trauma.

Incidence of road traffic accidents was more in male population (83%). Peak age group was between 20 to 30 years in our study.

Subdural hematoma was most common form of intracranial injuries (61.46%).

Glasgow coma scale combined with relevant clinical findings was sensitive (Sensitivity 97.8%) method of predicting intracranial injuries which rarely mis-classified patient with intra cranial bleed as normal.(Negative predictive value 99.2%)

Glasgow Coma Score combined with relevant clinical findings can be used as an indication for CT scan in order to avoid unnecessary CT scans.

Key words: Cranio-cerebral trauma, Computed tomography, Glasgow coma scale.

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INTRODUCTION

Trauma is a major health problem and is a leading cause of death. Brain injury is the major contributor to death in multi trauma patients.[1] Motor vehicle accidents are the leading cause of head injuries followed by fall, assault, firearm wound and others.[2]

Computed tomography (CT) is one of the most comprehensive diagnostic modality for accurate localization of the site of injury in cranio-cerebral trauma. The ability of CT to rapidly demonstrate a surgically correctable lesion, fracture, and subarachnoid hemorrhage makes it modality of choice in evaluation of acute head injury.[3]

CT can accommodate life saving equipment, traction and patient monitoring devices. It is rapid, widely available, inexpensive and is easy to obtain.[4]

CT involves high doses of radiation, resulting in a marked increase in radiation exposure in the population. Although the risks for any one person are not large, the increasing exposure to radiation in the population may be a public health issue in the future.[5]

Magnetic resonance imaging (MRI) is not well suited to assess acutely injured patients. MRI requires more time to perform than Computed Tomography and is more susceptible to patient motion artifacts. The lack of signal from the bone and the relative inability to differentiate fresh hemorrhage from normal brain, impairs MRI's ability to detect fractures and acute hematomas, thereby limits its usefulness in acute head trauma. However MRI is more sensitive in detecting white matter injuries as well as in imaging brainstem and posterior fossa lesions.[6]

Correlation between CT findings with Glasgow scale and clinical outcome may give useful information, which may be helpful, in order to avoid unnecessary CT scans and exposure to radiation.

AIMS AND OBJECTIVES

1.To evaluate and assess role of CT in localizing the injury to the particular intracranial compartment in patients with cranio-cerebral trauma.

2.To correlate CT findings with Glasgow Coma Score and clinical findings.

3.To study whether Glasgow Coma Score &/or clinical findings can be used as an indication for CT scan in order to avoid unnecessary CT scans.

REVIEW OF LITERATURE

HISTORICAL PROSPECTIVE

Head injury is present in ancient myths that may date back before recorded history. Ancient Mesopotamians knew of head injury and some of its effects, including seizures, paralysis, and loss of sight, hearing or speech.[7] The Edwin Smith Papyrus, written around 1650–1550 BC, describes various head injuries and symptoms and classifies them based on their presentation and tractability. Ancient Greek physicians including Hippocrates understood the brain to be the center of thought, probably due to their experience with head trauma.

In the Middle Ages, physicians further described head injury symptoms and the term concussion became more widespread. Concussion symptoms were first described systematically in the 16th century by Berengario da Carpi.[8]

It was first suggested in the 18th century that intracranial pressure rather than skull damage was the cause of pathology after traumatic brain injury(TBI). This hypothesis was confirmed around the end of the 19th century, and opening the skull to relieve pressure was then proposed as a treatment.[7] The introduction of intracranial pressure monitoring in the 1950s has been credited with beginning the "modern era" of head injury.

Conventional radiographic examination of the human body dates back to the genesis of diagnostic radiology in 1895, when Wilhelm Roentgen produced the first x-ray film image of his wife's hand. Conventional radiography remains fundamental to the practice of diagnostic imaging. It was initial modality of choice for head injury assessment. Plain films virtually never demonstrate significant findings in the low-risk group and are inadequate to characterize or exclude intracranial injury in the high-risk group. Further, the absence of skull fractures on plain films clearly does not exclude significant intracranial injury. Other technique used was cerebral angiography where shift of major vessels was seen in extracranial hematomas. However small contusions and hematomas which did not displace arteries could not be identified.[9] The 20th century saw the advancement of technologies that improved treatment and diagnosis such as the development of imaging tools including CT and MRI.[10]

The first Computed Tomography (CT) machine used for clinical purposes, developed by the late Sir Godfrey Hounsfield, was installed at the Atkinson Morley Hospital, London, in 1973.[11] Each axial image of the head took several minutes to acquire and days to reconstruct at the Electromagnetic interference (EMI) laboratories. Over the next decade, CT machines became faster as the processing power of computers improved. In the mid-1980s, the development of slip-ring technology enabled continuous revolution of the X-ray unit, which reduced the acquisition time and allowed helical data to be acquired.[12] Since the late 1990s, the focus of development has been on reducing the size of the detectors in order that they could be arranged in multiple rows along the z-axis. This permits the acquisition of multiple ‘slices’ simultaneously, thus reducing acquisition time yet further. At the time of writing, ‘state of the art’ CT systems have up to 64 rows of detectors, allowing up to 4 cm

to be imaged per revolution, a revolution time around 0.4s, and with a resolution of approximately 0.4 mm.

The invention of Computed Tomography by G. N. Hounsfield in 1973, revolutionized the management of patients with acute cranio-cerebral trauma.[11]

For the first time non invasive technique slowly started replacing invasive techniques. CT scanning rapidly became an integral part of emergency room evaluation of patients with traumatic brain injury. Its ability to delineate skull fractures and related intracranial hematomas or contusions made the use of plain skull roentgenograms or cerebral angiography obsolete for this patient population.

NORMAL CT ANATOMY

Accurate knowledge of normal sectional anatomy is required in order to interpret CT images. Various Structures which can be visualized in axial sections of brain at various levels are:

Below the level of fourth ventricle

Anteriorly the section includes both the orbits and medial and lateral orbital walls, etmoidal sinuses, bilateral zygomatic bones and pterygopalatine fossa. In the middle, structures seen are basilar part of occipital bone, temporo mandibular fossa, mastoid process of temporal bone with mastoid air cells. Posteriorly medulla oblongata, caudal portion of both cerebellar hemispheres and cerebellar tonsils can be visualized. Laterally sigmoid sinus area is seen.

Most inferior portion of the fourth ventricle may be visualized in 50% of normal cases. Also included is the Cisterna Magna, triangular in shape with its base directed posteriorly towards the occipital bone.

The detailed anatomy of the ethmoid and sphenoid sinuses, foramen magnum, and other foramina at the base of the skull requires thinner sections.

At the level of fourth ventricle

Anteriorly the section includes the orbital roof, crista galli, sphenoid and frontal sinuses.

The middle cranial fossa is bounded anteriorly by the sphenoid bone, posteriorly by the petrous bone and medially by the lateral margin of supra sellar cistern and it contains the inferior and middle gyri of the temporal cortex. The anterior clinoids are visualized as paired high density structures. The dorsum sella is located in the midline. Internal auditory meatus, dorsum sella, and posterior clinoid processes can be visualized.

The lower pons is seen in front of the fourth ventricle, connecting to the cerebellar hemispheres by the middle cerebellar peduncles. The pons is outlined by CSF with the anterior and lateral pontine cisterns. Posteriorly, the fourth ventricle is outlined by the cerebellar nodulus in the midline and by the cerebellar hemispheres laterally.

When section is taken little higher up, in the center, the midbrain appears outlined by the suprasellar cistern, ambient cistern, and quadrigeminal cistern. The suprasellar cistern contains the internal carotid artery, the anterior and middle cerebral arteries, the optic chiasma, the infundibulum, the mammillary bodies, and the top of the basilar artery. Lateral to the midbrain are the temporal lobes. The temporal horn can be

seen in the middle of the temporal lobe at this level. The frontal lobes are separated from the temporal lobes by the sylvian fissure. Most inferior portions of the frontal lobe can be seen at this level. In the posterior fossa, the aqueduct of Sylvius is present behind the midbrain. The superior cerebellar surface is seen with separation of the two hemispheres by the superior vermis.

Third ventricular level

The frontal lobes can be seen separated by the inter hemispheric fissure between them. Superficially, the sylvian fissures extend medially to separate the frontal lobe from the temporal lobe. Medial to the medial surface of the sylvian fissure, the insular cortex, external capsule, putamen, and globus pallidus can be demonstrated. The third ventricle in the midline is seen as a slit like cavity, surrounded by the thalamus laterally. Behind the third ventricle, cerebellar vermis can be seen. Sometimes, the pineal gland behind the third ventricle is seen at this level. Occasionally, some calcification of the habenula occurs anterior to the pineal gland calcification. Within the lateral ventricle, the most commonly calcified choroid plexus can be identified. Posteriorly, the occipital lobes are visualized.

Low lateral ventricular levels

The most superior aspects of the frontal lobes are visualized. The superior portion of the frontal horns appears outlined by the head of the caudate nuclei laterally. Anteriorly, the frontal horns are shaped by indentation of the genu of the corpus callosum. The corpus callosum is seen separated from the cingulate gyrus by the cingulate sulcus. Posteriorly, the occipital horns are indented by the splenium of the corpus

callosum. Within the lateral ventricle, the choroid plexus can be visualized.

Mid lateral ventricular levels

The superior extension of the sylvian fissure and the superior temporal gyrus are seen. The central sulcus separates the frontal lobe from the parietal lobe. The most superior aspect of the frontal horns is indented laterally by the most superior aspect of the caudate nuclei. The posterior medial aspect of the occipital horns is seen bound by the white matter fibers of the splenium of the corpus callosum. The posterior portion of the cingulate sulcus, separating the cingulate gyrus from the occipital lobe, can be seen connecting to the posterior inter hemispheric fissure.

Above ventricular levels

The higher scan sequences include the most superior portion of the body of lateral ventricle. It is frequently possible to identify the longitudinal pre central, central and post central fissures because of relatively constant position. Above the ventricular system the centrum semi ovale will be visualized. The two hemispheres are separated by the falx, which is best visualized on post contrast scan. Posteriorly in the midline the triangularly shaped superior sagittal sinus is visualized. This is an important landmark.[13,14]

Mechanisms of head trauma

Projectile or penetrating wounds and non missile injury are the two basic mechanisms of traumatic brain damage.

Projectile or penetrating wounds

Penetrating trauma to the head can be sub classified as superficial (object remains trapped in extra cranial soft tissues), tangential (object traverses and exits extra cranial soft tissues), penetrating (object penetrates calvarium and remains in it), and perforating (object penetrates and exits calvarium). Severity of a bullet wound is strongly influenced by missile orientation during its path through tissue and whether the projectile fragments or deforms. Wounds are most severe when the missile is large and traveling at high velocities and if it fragments or yaws early in its path through tissue. Tissue crushing and stretching are the major mechanisms of injury in these cases. Elasticity and tissue density, as well as thickness of the affected body part, strongly affect the wound produced.[15,16]

Non missile injury

The majority of non projectile traumatic brain injury (TBI) is caused by shear-strain forces. These are mechanical stresses on brain tissue that are induced by sudden deceleration or angular acceleration and rotation of the head. [17] Shear-strain injuries may be extensive and severe, are often multiple and bilateral, and frequently occur when there is no direct blow to the head.

Rotationally induced shear-strain forces typically produce intra axial lesions in the following predictable locations :

1. Brain surface (cortical contusions)
2. Cerebral white matter (so called diffuse axonal injury)

3. Brainstem

4. Along penetrating arteries or veins

Direct impact is significantly less important than shear-strain forces in the genesis of most TBI. With direct blows there is localized skull distortion or fracture and the underlying blood vessels and brain are damaged in a much more focal fashion as the transferred energy dissipates quickly. The typical results are cortical contusions and superficial lacerations localized to the immediate vicinity of the calvarial lesion. Although some extra axial lesions such as epidural hematoma are frequently associated with skull fracture, significant extracerebral hemorrhage often occurs in the absence of direct blows and is due to shear-strain forces.[18]

Classification of traumatic brain injuries

Traumatic brain lesions can be clinically classified as

Primary brain damage: Primary traumatic craniocerebral lesions results from direct application of external force to the calvarium and intra cranial contents. When these forces strain the cerebral tissue beyond the structural tolerance, injury results. The injury pattern reflects the type of strain, which may be compressive, tensile or shear in nature.

Secondary brain damage: Secondary manifestations of craniocerebral trauma often develop and are frequently more devastating than the initial injury. These occur as a consequence of primary lesions, usually as a result of mass effect or vascular compromise. These secondary effects include herniation syndromes, ischemia, diffuse cerebral edema, and secondary infarctions and hemorrhages.[19]

Traumatic cranio-cerebral lesions

Primary lesions

Scalp hematoma/ lacerations

Skull fractures

Extra axial lesions

- Epidural hematoma
- Subdural hematoma
- Subarachnoid hemorrhage

Intra axial lesions

- Parenchymal hematomas
- Cortical contusions
- Diffuse axonal injury
- Deep cerebral grey matter injury
- Intra ventricular/ choroid plexus hemorrhage
- Brian stem injury

Secondary lesions

Cerebral herniations

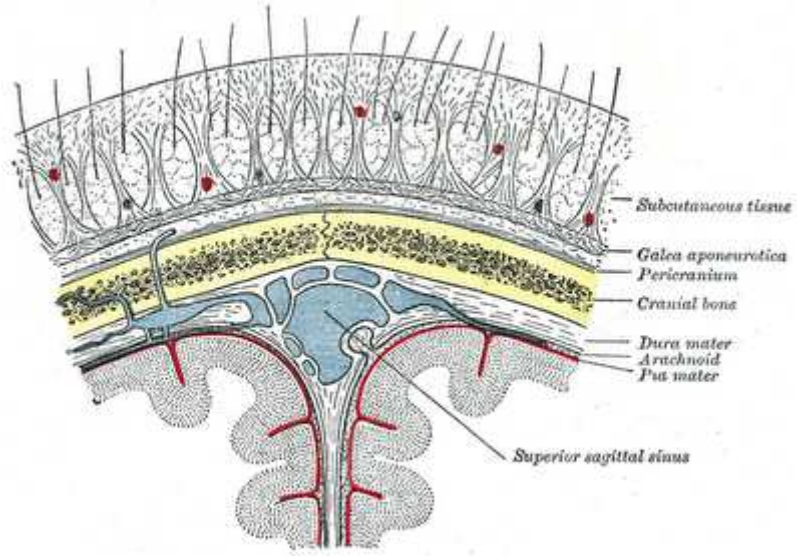
Traumatic ischemia, infarctions

Diffuse cerebral edema

Hypoxic injury

PRIMARY LESIONS

Scalp hematoma/ lacerations



The human scalp is prone to injury because of its exposed position. It helps to absorb the force of trauma to the skull, particularly glancing blows. The movement of the galea allows a tangential blow to slip off with no more damage than a laceration. When interpreting CT scans for head trauma it is useful to begin by examining the extra cranial structures for evidence of scalp injury or radio opaque foreign bodies. Scalp soft tissue swelling is often the only reliable evidence of the site of impact.[20]

Type of scalp wounds

- Uncomplicated small lacerations
- Perforating lacerations
- Contused lacerations with a varying degree of devitalisation of surrounding tissue
- Massive avulsions
- Sub–galeal hematoma

Sub Galeal Hematoma

At impact, the skull tends to move more rapidly than the scalp. Such movement may result in tearing of the connecting blood vessels between the scalp and the skull. This incongruity of movement may result in subcutaneous or subgaleal hematomas, even without any apparent bruising or laceration of the scalp.

On CT the subgaleal hematoma appears as a well defined soft tissue swelling of the scalp, located between the galea aponeurotica and the pericranium.[20]

Calvarial fractures

Calvarial fractures, as in fractures elsewhere in the body can be classified by several features, including location, size, comminution (fragments into multiple parts), diastasis (widening of sutural joints), and most importantly, the presence of any associated depressions.[21]

Types

Linear Fracture

Linear fractures that run parallel in the axial plane are easily missed on CT scans and are usually negligible if no associated brain injury is demonstrated. Most dramatic complication of a linear fracture is an epidural hematoma, resulting from tearing of the underlying meningeal vessels.

Depressed Fracture

A fragment is considered depressed when its outer table is displaced below the level of the inner table of the adjacent skull. Although skull films are frequently diagnostic, the depression can easily be identified with CT. Depressed fractures near the vertex or the skull base may require coronal reconstructions. Delineation of a depressed fracture is important because a depressed fracture may cause an underlying dural tear, extra cerebral hematoma, or brain contusion. Although compound depressed fractures frequently require surgical treatment, the majority of such injuries are managed conservatively. The goals of treatment are the prevention of infection and post traumatic seizures and the amelioration of neurologic deficits. Depressed fractures adjacent to venous sinuses are usually left in place because of the danger of uncontrollable hemorrhage when fragments are removed.[22]

Stellate Fracture

Occurs when there is a small area of impact which leads on to several fracture lines radiating from a central point giving the appearance of a star.[23]

Basilar Skull Fracture

If a basilar skull fracture is clinically suspected, high-spatial-resolution CT with a thickness of 3 mm or less is required to delineate a fracture. Furthermore, CT may suggest a basilar skull fracture before it becomes clinically evident. An air-fluid level in the paranasal sinuses or the mastoid air cells should be viewed as secondary to a basilar skull fracture until proven otherwise.

Basilar fractures are important because of associated injuries to cranial nerves and vessels adjacent to skull base.[24]

The basilar fractures include

- Fractures involving the paranasal sinuses and if associated with tear of the dura cause meningitis and pneumocephalus.
- Fractures of the sella turcica, which later may cause endocrinal abnormalities.
- Petrous bone fractures
- Longitudinal fractures

Longitudinal fractures are more common and are usually associated with CSF Otorrhoea (through torn tympanic membrane) and CSF rhinorrhoea (through eustachean tube). This can result in ipsilateral facial nerve palsy and sensorineural hearing loss.

• Fractures of orbital roofs results in bilateral periorbital hematomas, black eye (raccoon eyes).[25]

Pneumocephalus

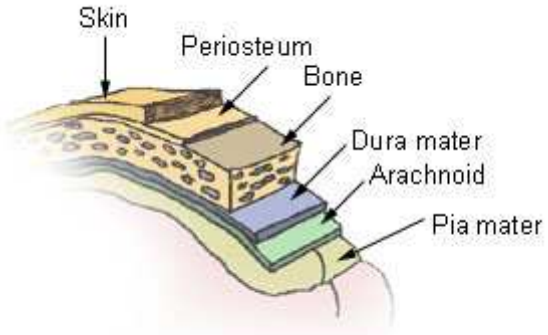
Air loculi in the extracerebral spaces typically indicate traumatic air entry resulting from fracture of a paranasal sinus or mastoid air cells abutting the dura. When associated with a dural tear, they may be complicated by CSF leakage, empyema, meningitis, or brain abscess. Most posttraumatic CSF leaks cease spontaneously, and the responsible fractures may never be visualized.

Growing Fracture

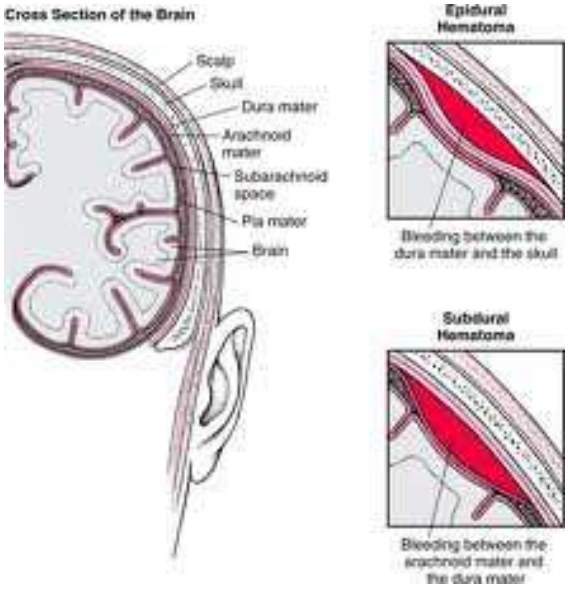
Enlarging skull fractures have been described in 0.75% of skull fractures in children. Most are associated with a history of significant trauma, that causes the skull fracture, dural tear, and contusion of the underlying brain. Growing fractures commonly occur in the growing skull, although occasionally such lesions have been reported in adults. The ongoing normal growth of the child's brain is considered an aggravating factor. Children with growing fractures present weeks to years after injury with a pulsatile mass or less commonly with a depressed calvarial defect. The lesions seem to occur when a skull fracture in childhood is accompanied by a dural tear or rent that allows focal protrusion of the arachnoid membrane into the fracture gap. The unabating CSF pulsations transmitted to the entrapped arachnoid result in bone remodeling at the fracture site and enlargement of the fracture line. Frequently an area of underlying porencephaly or encephalomalacia results from an associated contusion that occurs during the initial trauma, rather than being caused by the fracture enlargement.[26]

Extra axial hemorrhages

Anatomy of Fluid Spaces.



Meninges of the CNS



The imaging appearance of post traumatic fluid collections is best understood in relation to the meningeal layers covering the brain. The pia is the deepest layer, covering the brain surface and lining the cortical gyri. It also lines the perforating vessels, forming a perivascular space, the Virchow-Robin space, which communicates with the subarachnoid space. The subarachnoid space is the space between the arachnoid and pia. In normal patients, this is a thin space containing a small amount of

cerebrospinal fluid (CSF). The arachnoid is attached to the pia and to the brain surface by fine arachnoid trabeculations. The arachnoid follows the general contour of the brain, but does not extend into the sulci.

The subdural space lies between the inner layer of the dura and the underlying arachnoid. The subdural space normally contains a small amount of fluid, which differs only slightly from CSF. Bridging cortical veins traverse the subdural space. Because the dura is firmly attached to the skull and the arachnoid is attached to the cerebrum, most brain motion occurs across the subdural space, placing the bridging cortical veins at risk for tear.

The dura is a fibrous, 2-layer membrane that is peripheral to the arachnoid. The inner meningeal layer and outer periosteal layer are tightly bound to each other and to the skull. The 2 leaves of dura separate to enclose the venous sinuses. The falx and the tentorium are reflections of the dura. The meningeal layer has a rich capillary network that is responsible for the organization and resolution of SDH. The potential space between the periosteal layer of the dura and the inner table of the skull is the epidural space. Running within the epidural space are branches of the middle meningeal artery. The periosteal layer is tightly adherent to the margins of the cranial sutures; therefore, most epidural fluid collections do not cross the suture margins.[27]

Epidural hematomas

Epidural hematomas lie in the space between the inner table of the skull and periosteal layer of dura. These layers are normally fully attached, but may be forcibly separated by direct traumatic impact, especially if there is an associated fracture. Most commonly, EDHs occur in the temporal and parietal regions where they arise from injuries to branches of the middle meningeal artery. This artery lies in a groove in the inner table of the skull and is thus vulnerable to injury, especially when a fracture is present. EDHs generally result from severe head trauma and are found most commonly in young adults. The compliance of the skull in children, the firm attachment of the dura to the skull in the elderly, and the increased incidence of severe head trauma in young adults are factors contributing to this distribution of injury.

A venous EDH can occur when there has been disruption of a major venous sinus. Venous EDHs often involve a basilar fracture extending to the torcular Herophili or the transverse sinus. These hematomas are uncommon (15%) compared with the arterial EDH (85%).[28]

Epidural hematomas are found in only 1% to 4% of patients imaged for craniocerebral trauma, although-EDHs represented 10% of fatal injury in the Glasgow autopsy series. A classic "lucid interval" between the traumatic episode and onset of coma or neurologic deterioration is seen in only half the patients with EDH. Delayed development or enlargement is seen in 10% to 30% of EDHs and usually occur within the first 24 or 48 hours. Late hematomas develop in 20% of moderate to severely head injured patients who do not have signs of cerebral contusions on initial post trauma CT studies.[29]

EDHs are characteristically biconvex or lentiform, Uncommonly, EDHs may be bilenticular, crescentic, or irregular in shape. The shape is determined by the dura, which is firmly adherent to the inner table of the skull. The biconvex shape results from the firm attachment of the dura to the inner table of the skull. The margins of the hematoma are limited by the bony sutures. Unlike SDHs, EDHs are not limited by the falx and tentorium. Careful examination of the CT at bone windows allows identification of a skull fracture in 90% or more of adults with an arterial EDH.[30]

The most common location for an EDH is over the temporal lobe, followed in frequency by the parietal, frontal, and occipital lobes, with the posterior fossa the least common location.[31]

EDHs may be classified as acute, subacute, and chronic. An acute EDH is heterogeneous in attenuation, containing areas of hyperdense blood and isodense serum. The subacute EDH is homogeneously hyperdense in attenuation, consisting of solid blood clot. Heterogeneous or decreased attenuation, as well as an enhancing membrane, are characteristics of a chronic EDH as a result of the degradation of blood products. Peripheral enhancement representing dura and membrane formation between the EDH and adjacent brain parenchyma may be seen in chronic EDHs with the use of intravenous contrast material. Delayed EDH is most commonly secondary to slow venous bleeding from rupture of a dural sinus. Other causes of delayed EDH are pseudoaneurysm rupture of an epidural vessel and arteriovenous fistula.[32] In some instances, when minimal neurologic signs and symptoms are present, an EDH may undergo spontaneous resolution, which is visualized on serial CT scans. Some EDHs expand 1 or 2 weeks after injury and then gradually resolve. This expansion correlates with an increase in or persistence of symptoms.

The imaging criteria in determining whether an EDH may be treated conservatively are

(1) a diameter of less than 1.5 cm and

(2) a minimal midline shift of less than 2 mm. The patient must be neurologically intact without focal deficit.[33]

Parenchymal abnormalities are not present with EDHs as frequently as they are with acute SDHs. Prompt surgical evacuation of the EDH often results in marked clinical improvement. Little or no residual EDH is evident on postoperative scans.

Posterior fossa EDHs are rare; however, delay in detection may result in fatal brain stem injury. Many posterior fossa EDHs are not readily visualized on CT because of beam-hardening artifact. The use of contrast material facilitates the detection by demonstrating displacement of the dural sinuses, the torcular herophili, or both, as well as displacement of the dura away from the calvarium.[34]

Subdural hematoma:

Traumatic acute subdural hematoma is among the most lethal of all head injuries. Mortality rates range from 50% to 85% in some reported series. Subdural hematomas (SDH) are seen in 10% to 20% of all cranio-cerebral trauma cases and occur in up to 30% of fatal injuries.[35]

Hemorrhage into the potential space between the pia arachnoid and the dura is termed a subdural hematoma. Acute subdural hematomas result from significant head injury and are caused by the shearing of bridging veins. This occurs because of rotational movement of the brain with respect to fixation of these veins at the adjacent venous sinus. The subdural portion of the vein is not ensheathed with arachnoid trabeculae as is the subarachnoid portion and therefore is the weakest segment of the

vein. Acute subdural hematomas also occur in the setting of the burst lobe. Intra cerebral clot is in direct continuity with the subdural hematoma.
Penetrating injury can also result in acute subdural hematoma.[36]

The elderly and infants are particularly susceptible to this injury because their bridging veins are stretched. The elderly are likely to suffer from age-related volume loss, whereas, children less than 2 years of age may have benign enlargement of the subarachnoid spaces. Low-pressure venous bleeding separates the arachnoid from the dura, stretching the remaining intact veins, which may result in further venous rupture and enlargement of the hematoma. A SDH may be located at the point of direct impact, (ie, a coup injury) or opposite the point of direct impact, (ie, a contrecoup injury). Hemorrhage in these locations can be understood by considering the motion of the brain with respect to the skull and the subsequent rupture of the cortical veins. After initial impact, the brain moves back and forth with respect to the skull. This motion alternately stretches and compresses the cortical veins, leading to bleeding in coup and/or contrecoup locations.

Because of the loose connection between the arachnoid and the dura, a SDH spreads over a considerable area and usually has a crescent configuration overlying the convexity of the brain. SDH can also form along dural reflections such as the falx and the tentorium.[37] Occasionally an SDH may be biconcave, simulating the appearance of EDH. This biconcavity can be seen particularly when the SDH is large. Common sites for SDH are over the frontoparietal convexities and in the middle cranial fossa. Isolated interhemispheric and parafalcial SDHs are common in cases of non accidental trauma. Bilateral SDHs are also more frequent in child abuse.[38]

Types and CT appearances

Acute subdural hematoma

The classic CT appearance of an acute SDH is a crescent-shaped homogeneously hyperdense extra axial collection that spreads diffusely over the affected hemisphere However, up to 40% of acute SDHs have mixed hyper/hypodense areas that reflect unclotted blood, serum extruded during clot retraction, or CSF within the subdural hematoma due to arachnoid laceration. [37]

The classic homogeneous, hyperdense appearance of acute SDH is not always present. The SDH may appear mixed rather than homogeneous in attenuation. It may have one of three patterns: marginal hypodensity, central irregular areas of hypodensity, or laminar areas of hypodensity. The low density may be secondary to unclotted blood or possibly CSF resulting from traumatic arachnoid tears. The mixed density SDH is usually larger and has more mass effect than the classic hyperdense SDH.[37]

Rarely, acute SDHs may be nearly isodense with the adjacent cerebral cortex. This occurs with coagulopathies or severe anemia when the hemoglobin concentration reaches 8 to 10 g/d1. The majority of acute SDHs are hyperdense because of the attenuating properties of the hemoglobin molecule.[39]

Sub acute subdural hematomas

With time, subdural hematomas undergo clot lysis, organization, and neomembrane formation. The evolution of an untreated, uncomplicated SDH follows a predictable pattern. Sub acute SDHs become nearly isodense with the underlying cerebral cortex within a few days to a few weeks after trauma. Effacement of the cortical sulci over the cerebral convexity and mass effect on the ventricular system or midline shift indicate the presence of an isodense SDH. Mass effect on the ventricular system may be negligible or absent when bilateral collections are present. Delayed contrast-enhanced CT performed 4 to 6 hours after injection of contrast material reveals enhancement of the subdural collection. Contrast-enhanced CT may also demonstrate enhancement of cortical veins that are displaced medially from the inner table of the calvarium; however, this enhancement is not seen in all cases. The use of rapid high-dose contrast enhancement allows the more accurate detection of an isodense SDH. The majority of patients with an isodense SDH may have three or four of the following signs: posterior displacement of the ipsilateral anterior horn of the lateral ventricle, compression of the ipsilateral posterior and temporal horns, anterior displacement of the ipsilateral posterior and temporal horns, anterior displacement of the ipsilateral glomus calcification and widening of the contralateral ventricle. In a patient with a history of head trauma, the presence of small compressed ventricles with the absence of cortical sulci should raise the possibility of bilateral isodense SDHs.[40]

Chronic subdural hematoma

Chronic SDHs are most commonly hypodense on CT. Chronic SDHs are typically crescentic; however, they may be biconcave or lentiform as a result of fluid absorption into the hematoma. Another possible cause is the formation of adhesions. A capsule composed of a

capillary-rich membrane develops and surrounds the SDH. This capillary-rich membrane is responsible for repeated episodes of re-bleeding and subsequent increase in size of the SDH. As the SDH enlarges, the patient becomes symptomatic, showing signs of increased intracranial pressure, hemiparesis, or intellectual and personality change. Repeated episodes of re-bleeding may result in a mixed-density collection containing areas of hypodense, isodense, and increased density. A chronic SDH may appear hyperdense from acute hemorrhage, simulating an acute process. Fluid-fluid levels may be seen as blood products settle in the dependent aspect of the subdural collection, which becomes hyperdense relative to the superior aspect. Enhancement of the SDH may be seen with delayed CT obtained 3 and 6 hours after intravenous administration of contrast material.[41]

Unusual Locations of SDH

Posterior fossa extra axial hematoma are very rare. They may occur due to rupture of dural sinuses, bridging veins, secondary to trauma. These are also seen in difficult deliveries, where it is secondary to leak in tentorium produced by severe moulding. It is usually crescentic in shape. A central/ eccentric, uni/bilateral lesion may be present.[42]

Inter hemispheric Subdural Hematoma

These are usually unilateral and are produced by bleeding from bridging veins between the superior sagittal sinus and the parieto-occipital cortex. The inter hemispherical lesion has a straight medial border and a convex lateral border as it displaces the brain parenchyma away from the midline. The two inter hemispheric subdural spaces do not communicate across the midline.

The inter hemispheric fissure is the most common site for subdural hematoma in children. Inferior extension of the hematoma along the tentorium is not uncommon.

Sub temporal Subdural Hematoma

Occasionally subdural hematoma occurs below the temporal and occipital lobes and thus lies in the middle cranial fossa floor anteriorly and the superior tentorial surface posteriorly. This may be obscured on axial scanning due to volume averaging with the bony floor of the middle cranial fossa. The temporal horn is elevated and displaced anteromedially. Alternatively the sub temporal hematoma may extend posteriorly along the superior aspect of the tentorium.

Peritentorial hematoma appears as a sheet like hyperdensity along the tentorium with a hazy lateral margin and a sharp medial margin.

Prognosis

Acute subdural hematoma with neurologic dysfunction indicates poor prognosis. Mortality is more in patients who are comatose at the time of initial examination.

Calcified Subdural Hematoma

Most patients with calcified SDH are in the 2nd – 4th decades of life and may have a history of trauma 10-20 years before discovery of the lesion. Calcification is crescentric located next to the inner table of skull vault. The calcification develops in the thickened membranes of the hematoma, which undergoes gradual degeneration and transformation of its connective tissue.

Subdural Hygroma

These are subdural collections of CSF or chronic subdural hematomas in which liquefaction has occurred. These may occur following a tear in the arachnoid membrane causing CSF flow into the subdural space. This is a valve mechanism and therefore it is unidirectional. It is more common in infants and younger children than in aged patients.

CT findings

Commonest site is fronto–temporal and parietal region. It is crescent shaped with density values same as that of the CSF. Ventricles may upper compressed or enlarged.[43]

Subarachnoid hemorrhage

Overall, trauma is the most common cause of subarachnoid hemorrhage (SAH). Others include aneurysm rupture and dural AVFs. The incidence of SAH associated with head trauma has been reported to range from 33% to 60%.[44]

Sources of SAH in trauma include tearing of pial vessels, extra axial extension of a hemorrhagic contusion, and redistribution of intraventricular hemorrhage caused by damage to subependymal veins. Often the highest concentration of SAH occurs contralateral to the side of direct impact. Traumatic SAH most often occurs in superficial cerebral sulci near calvarial fractures and/or cerebral contusions. In particularly severe traumas, injury to one or more of the arteries in the vertebro basilar circulation can result in massive SAH at the base of the brain.[45]

CT findings

SAH has a characteristic appearance. In the acute and sub acute phases, CT shows hyper attenuation in the sulci and basal cisterns and occasionally also in portions of the ventricular system. SAH is commonly seen in the interpenduncular and ambient cisterns, Sylvian fissures, and occipital horns of the lateral ventricles. Occasionally, hemorrhage in the region of the ambient cistern represents the most pronounced area of hemorrhage. It has been suggested that this may occur as a consequence of impaction of a vein along the edge of the tentorial incisura.[39] Differentiation of SAH from subdural hemorrhage (SDH) can be difficult in certain locations, particularly along the tentorium cerebelli. A useful hint for recognizing SAH is extension of the blood into the cerebral sulci.[46]

SAH can result in communicating hydrocepalus, presumably as a result of interference with reabsorption of CSF at the level of the arachnoid villi. Less frequently, obstructive hydrocephalus can result from impairment of CSF flow at the level of the cerebral aqueduct.

Another sequela of SAH is arterial vasospasm, although this complication is more common in the setting of aneurysmal rather than traumatic SAH. If severe enough, vasospasm can result in cerebral infarction corresponding to the vascular territories of the affected arteries. As with infarction as a result of other causes, CT shows hypo attenuation and loss of gray-white differentiation. Vasospasm may be suspected if there is a change in the patient's clinical examination or if increased arterial velocities are detected on transcranial Doppler ultrasonography.[45]

Intra axial lesions

Contusions

Cortical contusions are the second most common primary traumatic neuronal injury. Cortical contusions represent 45% of primary intra axial traumatic lesions. Compared to DAI, cortical contusions are less frequently associated with initial loss of consciousness unless they are extensive or occur with other abnormalities such as shearing injury or secondary brain stem trauma.

Contusion is bruising of the brain caused by a direct contact and is most often seen at the point of impact. Capillary injury and edema are the result. When the brain moves relative to the skull, the inferior frontal lobes are at increased risk for cortical contusion as they slide over the irregular floor of the anterior cranial fossa. The injury may be limited to the portions of the gyri adjacent to the skull with relative sparing of the underlying white matter. In patients with more severe injury, the volume and depth of tissue injury are greater and the small hemorrhages may coalesce into an intracerebral hematoma. The decision whether to evacuate the hematoma is complex, based in part on the amount of mass effect seen on imaging, the patient's intracranial pressure, and overall condition.

Surrounding the hematoma is an area of edema characterized by markedly decreased cerebral blood flow. Ultrastructural abnormalities described include vascular endothelial swelling and capillary compression. The edema may increase for several days following injury and may contribute to an increase of the intracranial pressure. Healing begins during the first week following injury and, in the next weeks to months, the lesion shrinks to a gliotic scar.[47,48]

Because contusions occur when brain contacts a dural ridge or bony protuberance, they occur in very characteristic locations. Nearly half of all cases involve the temporal lobes, most frequently the temporal tip, inferior surface, and cortex around the sylvian fissure. One third occur in the frontal lobes, particularly along the inferior surface and around the frontal poles. Twenty-five per cent are parasagittal or "gliding" contusions (so called because the convexities of each hemisphere are anchored to the dura by arachnoidal granulations. When the brain abruptly shifts at the time of impact, the sub cortical tissue "glides" more than the cortex. The inferior surfaces of the cerebellar hemispheres are less common sites of cortical contusion.[49]

CT appearance

Contusions appear as areas of heterogeneous increased density mixed with or surrounded by areas of decreased or normal density. Mass effect may or may not be present, depending on the size of the lesion. Contrecoup injuries typically involve the inferior surface of the frontal and temporal lobes. In the posterior fossa, contusions may be difficult to detect because of beam-hardening artifact from adjacent bony structure. In some instances, no lesion can be identified, and the presence of indirect signs such as effacement of the pontine, cerebellopontine angle, and perimesencephalic cisterns must be used to suggest the presence of brain stem injury.

Hemorrhagic contusions have a typical pattern of evolution that occurs over several months. The initial area of heterogeneous increased density progresses to an area of decreased density within 1 week. At 2 weeks, the contusion is not visualized because it is isodense with brain parenchyma. Enhancement occurs during the acute and sub acute stages.

Contusions may be isodense if they consist of equal amounts of hemorrhage and edema. The use of intravenous contrast material allows the detection of these isodense lesions as contrast material accumulates in areas of blood–brain barrier breakdown. Focal encephalomalacia becomes evident by 1 month after injury.[50]

Diffuse axonal injury

DAI is sustained as a result of shearing of axons related to the differential acceleration of tissues of different densities, most often in the setting of trauma involving, rapid, high-magnitude acceleration or deceleration. It can occur with or without a direct blow to the head. Estimates suggest that DAI occurs in more than 50% of all severe head trauma and in more than 85% of the subset related to motor vehicle collisions. These injuries tend to be diffuse, bilateral, and occur in very predictable locations. The characteristic shearing injuries are microscopic axonal bulbs or "retraction balls". Disruption of penetrating blood vessels at the corticomedullary junction, corpus callosum and internal capsule, deep gray matter and upper brainstem produce numerous small hemorrhagic foci that may be the only gross pathologic markers of DAI.[51]

The most common locations for this type of injury are at the gray-white matter junctions, in the corpus callosum and in the brainstem. Most DAI lesions occur at the gray-white matter junction, and they are most common in the frontal and temporal lobes. Lesions can occur elsewhere in the cerebral hemispheres and in the cerebellum. Callosal injuries, the second most common location, are most often sustained in the region of the splenium, presumably because this portion of the corpus callosum is relatively mobile in relation to the falx cerebri.

DAI is graded by its anatomic location. Grade I injuries are isolated to the gray-white matter junction mainly in parasagittal white matter of the cerebral hemispheres, plus presence of lesions in the corpus callosum is grade II and brainstem increase the grade to III, respectively. Overall, the presence of DAI worsens a patient's prognosis, and the higher the grade, the more dire the prognosis. DAI is believed to be a contributing factor explaining persistent neurologic problems, including memory and attention deficits and headaches, which occasionally plague even patients who suffered mild head trauma.[52]

CT findings

CT findings in DAI include diffuse cerebral swelling, corpus callosal hemorrhage, and SAH. Hemorrhage may also be present in the area of the third ventricle and hemispheric white matter. Although CT scans do not reveal the actual axonal lesion demonstrated with histology, they do show the associated edema and hemorrhage.[53]

CT underestimates the extent of DAI because the punctate hemorrhages resolve quickly and because only a minority (approximately 19%) of diffuse axonal injuries are macroscopically hemorrhagic. For CT to reveal the shearing injuries acutely, the lesion must be hemorrhagic, fairly large, or both. When present, CT findings consist of small areas of hemorrhage, most commonly at the gray-white matter interface, and less commonly in the brainstem and corpus callosum. The sensitivity of the CT also decreases following the acute injury as the hemorrhagic products degrade and become isodense with brain. [54]

Sub cortical grey matter (deep cerebral) and Brain stem injuries

These are less common than DAI and contusions these account less than 5 to 10% of primary traumatic brain injuries. Most are induced by shearing forces that cause disruption of multiple small perforating blood vessels.

Brainstem injury can occur as a primary or secondary event in TBI. If the brainstem is displaced during trauma, the dorsal lateral aspect of the brainstem may contact the free edge of the tentorium at the tentorial incisura. A small area of either hemorrhagic or non hemorrhagic contusion may result. The location of the injury is characteristic. The extent of injury depends not only on the nature of the trauma but also on variations in the size and shape of the tentorial incisura.

Secondary brainstem hemorrhage that results from increased intracranial pressure and/or descending transtentorial herniation is referred to as Duret hemorrhage. Caudal displacement of the brainstem can produce distortion of blood vessels in the inter peduncular cistern. Injury to these vessels may result in single or multiple secondary hemorrhages currently called Duret hemorrhages. The Duret hemorrhages can be distinguished from direct traumatic brainstem injuries by their location. The Duret hemorrhages are typically in the central pons, whereas direct injuries are located in the dorsal lateral pons.

CT can be normal in these patients. Petechial hemorrhages can sometimes be seen in the dorsolateral brainstem, periaqueductal region and deep gray matter nuclei. MR depicts these brainstem lesions nicely.[55]

Intraventricular and choroid plexus hemorrhage

IVH is identified in 1% to 5% of all patients with closed head injury Traumatic IVH is thus relatively uncommon and usually reflects severe injury. Most cases of IVH are associated with other manifestations of primary intra axial brain trauma such as DAI, deep cerebral gray matter, and brainstem lesions. Prognosis is poor, although patients with isolated IVH typically have a somewhat better outcome. Disruption of subependymal veins, shearing injuries, and basal ganglionic hemorrhage with subsequent rupture into the adjacent ventricle are thought to cause most cases of traumatic IVH.

Imaging. CT manifestations of acute IVH are high density intraventricular blood with or without a fluid fluid level. Occasionally, focal choroid plexus hematomas can be identified in the absence of frank IVH. Most cases of traumatic ICH have hemorrhagic foci in the adjacent deep gray matter nuclei or white matter. Subarachnoid hemorrhage is also commonly associated with IVH.[56]

Secondary effects of craniocerebral trauma

Cerebral edema.

Brain swelling and edema occur commonly in patients with head trauma. Brain swelling is observed more commonly in children than in adults. Minor episodes of trauma may result in brain swelling. Patients may experience a lucid interval after the episode of trauma, followed by the sudden onset of headache, nausea, vomiting, and loss of consciousness. SAH and contusions are often seen in association with this type of abnormality.[57]

Severely increased intracranial pressure may produce ischemia as a secondary complication. The cerebral ischemia in turn leads to more edema, even greater increases in intracranial pressure, and results in decreased brain activity. Patients with persistent swelling have a short survival and high mortality.

On a cellular level, active transport mechanisms at the cell membrane may become disabled. The intracellular calcium is normally maintained at a lower concentration and extracellular calcium by active transport. Following injury, the active transport mechanism can become impaired, allowing influx of calcium. The influx of calcium can lead to secondary cellular injury as well as the release of various neurotransmitters into the extracellular space. The role of calcium channel blockers in the treatment of brain trauma is an area of active research. The blood-brain barrier, which normally restricts the movement of proteins and other solutes into the extracellular space, fails, increasing the vasogenic edema.

Cerebral swelling is seen on CT as unifocal or diffuse areas of low attenuation. There are signs of mass effect such as effacement of cortical sulci and effacement of cisterns. The ventricles or midline structures may be displaced and there may be loss of normal differentiation between gray and white matter.[58]

Midline shift

When the expanding mass lesion is predominantly unilateral, the midline structures are shifted to the opposite side. The interventricular septum, third ventricle and the pineal gland can define the degree of midline shift. As this shifting proceeds, the cingulate gyrus herniates under the free edge of the falx cerebri, pressing the corpus callosum

downward on the side of the mass lesion. The falx may also be shifted away from the mass. If the process is extensive and severe, sub falcine herniation may compromise one or both pericallosal arteries leading to infarction in their territory of distribution.

The medial portion of the temporal lobe on the side of the lesion (uncus and hippocampal gyrus) are pressed against the side of the midbrain. This is part of the process of obliteration of the cisterna ambiens. Nonetheless, in patients with head injury there is correlation between the occurrence of significant midline shift and the incidence of signs of severe neurologic dysfunction.[59]

Cerebral herniations

Cerebral herniations are caused by mechanical displacement of brain, cerebrospinal fluid, and blood vessels from one cranial compartment to another.

Subfalcine herniation.

Here, the cingulate gyrus is displaced across the midline under the inferior free margin of the falx cerebri. Initial displacements may be relatively minor. With larger herniations the ipsilateral lateral ventricle is compressed and the contralateral ventricle enlarges as the foramen of Monro becomes obstructed. Vascular displacements also occur with subfalcine herniation. The ipsilateral anterior cerebral artery (ACA) and deep subependymal veins are shifted across the midline. In severe cases the ACA and its branches may become compressed against the falx. Callosomarginal artery occlusion may result in secondary ischemia and infarction.[60]

Uncal Herniation

Uncal herniation is sub divided into anterior, posterior and complete types depending on the extent of involvement of medial temporal lobe. Anterior uncal herniation is herniation of the posterior hippocampal gyrus into the broader anterior region of the incisura of the tentorium cerebelli. Posterior uncal herniation occurs when the para hippocampal gyrus, lingual gyrus or isthmus of the gyrus forticatus are displaced inferiorly through the incisura. In complete uncal herniation anterior and posterior herniations become continuous and the brain stem is displaced to the opposite side. Uncal herniation can interfere with CSF circulation via effacement of the subarachnoid spaces at the level of the incisura. This causes narrowing of the cerebral aqueduct or direct compression of the third ventricle in the region of the pineal body. Earliest consistent clinical manifestation is a unilaterally fixed and dilated pupil due to mass effect on the oculomotor nerve. Later, bilateral motor signs evolve, followed by decerebrate rigidity. Bilateral motor signs evolve, followed by decerebrate posturing, coma, progressive loss of brain reflexes and respiratory arrest may ensue if not intervened.

Descending transtentorial herniation.

This is by far the most common type of transtentorial herniation. The uncus and parahippocampal gyrus of the temporal lobe are initially displaced medially and protrude over the free tentorial margin. At early stages of descending transtentorial herniation the ipsilateral side of the suprasellar cistern is effaced. As the brainstem is shifted away from the herniating temporal lobe, the ipsilateral cerebellopontine angle cistern is initially enlarged. Progressive obliteration of the suprasellar cistern occurs with increasing supratentorial mass effect until, with severe

bilateral descending herniation, the tentorial incisura is completely plugged from displacement of both temporal lobes and the lower diencephalon into the midline basal subarachnoid spaces. In cases with severe descending transtentorial herniation, CT scans show obliteration of all basal cisterns. The anterior choroidal, posterior communicating, and posterior cerebral arteries (PCA) are all displaced infero-medially in severe descending herniations. The PCA may become compressed against the tentorial incisura, resulting in occipital lobe ischemia or infarction. Inferior kinking and occlusion of perforating vessels that arise from the circle of Willis can result in basal ganglia and midbrain infarction Other manifestations of descending transtentorial herniation include periaqueductal necrosis, secondary midbrain, or "Duret," hemorrhage and "Kernohan's notch" as well as compressive cranial neuropathies.[61]

Ascending transtentorial herniation.

Infratentorial traumatic injuries are less common than their supratentorial counterparts. Trauma-induced upward herniation of the vermis and cerebellar hemispheres through the tentorial incisura is therefore much less common than descending temporal lobe herniation. With ascending herniations the central lobule, culmen, and superior surface of the cerebellum are displaced cephalad through the tentorial incisura. Imaging studies show the superior vermian cistern is effaced and the fourth ventricle is compressed and displaced anteriorly.

With increasing upward herniation the quadrigeminal cistern is deformed and the midbrain displaced anteriorly. Aqueductal compression may result in obstructive hydrocephalus.[62]

Tonsillar herniation

Also known as “Foraminal impaction” or “cerebellar cone”. When the cerebellar tissue herniates inferiorly, tongues of compressed tonsilar folia extend through the foramen magnum for a variable distance into the upper cervical spinal canal. In severe cases, the cortex of the tonsils becomes necrotic and subsequently sclerotic. The common causes include cerebellar hematoma, posterior fossa venous epidural hematoma and sometimes diffuse cerebral swelling can be the cause.

Clinically, the severe tonsilar herniation can result in Cushing’s triad-respiratory depression, elevated blood pressure and a decrease in the pulse rate due to medullary compression. If the posterior inferior cerebellar artery (PICA) is compromised by the herniating tonsil it results in “Wallengberg’s Syndrome”.

A small percentage of normal patients, in whom, the caudal loop of the PICA extends below the level of the foramen magnum, are more prone to vascular compromise. On CT, tonsillar herniation causes effacement of the cisterna magna. Associated causes like epidural hematoma or cerebellar hematoma are also found.[63]

Transalar (trans sphenoidal) herniation.

This is less common than subfalcine or transtentorial herniations. In descending transalar herniation the frontal lobe is forced posteriorly over the greater sphenoid ala, causing backward displacement of the sylvian fissure, the horizontal middle cerebral artery, and the temporal lobe. In ascending herniations the temporal lobe, sylvian fissure, and middle cerebral artery are displaced up and over the sphenoid ridge.[61]

DIRECT VASCULAR INJURY

Trauma to the extra cranial and intracranial blood vessels and dural sinuses that leads to thrombosis and occlusion may occur in head injury. Damage to the vessels of the scalp, dura and brain sometimes results in aneurysms or arterio-venous fistulas which represent specific symptoms. A delay in treatment frequently results in irreversible cerebral damage, blindness or even death.

Injury to the internal carotid artery in the neck

Traumatic occlusion of the ICA in the neck due to direct contusion, stretching or shearing is rare and is usually caused by traffic accidents, violence or falls. Most of the traumatic occlusions are at or near the bifurcation of the common carotid artery. Other mechanisms of occlusion include intimal tear, sub intimal hemorrhage, subsequent vascular spasm and thrombosis. Signs and symptoms are typical of carotid artery insufficiency and include diminished level of consciousness, hemiparesis or hemiplegia, aphasia and seizures. Carotid angiography confirms the diagnosis.[64]

Aneurysms

Trauma is the commonest cause of the false aneurysms of the cervical internal carotid artery. In traumatic aneurysms, blood escapes through the torn wall of the carotid, but the escaping blood is confined by the intact adventitia and adjacent tissue layer, particularly deep cervical fascia. When large enough, these aneurysms may bulge into the pharynx and interfere with swallowing. Angiography is essential to confirm diagnosis.

Carotid Injury at the Base of the skull Thrombosis

Two primary sites for the onset of traumatic thrombosis at its entrance into the cranial cavity are:

1) In the petrous pyramid
2) Fracture middle cranial fossa involving the body of sphenoid.

Injury to Vertebral and Basilar Arteries

The anatomic position of the vertebral-basilar system makes it particularly susceptible to trauma because of its numerous relationships to bones of the cervical spine and skull.[65] The different sites of involvement are as follows,

- Cerebral herniation through foramen magnum resulting in compression of one or both vertebral arteries against the bony walls.
- Direct compression or contusion against the clivus.
- In cervical hyper extension injury, condylar compression of the vertebral artery as it traverses the first cervical vertebra.
- Forward bending and compression by the odontoid process in atlanto-axial dislocation.
- Violent trauma and rotation of the neck.

Patients with vertebral artery occlusion may present either with abrupt onset of coma, often leading to death or with transient symptoms of brainstem ischemia. Vertebral angiography is the investigation of choice.

Vertebral Arterio–Venous Fistula

The vertebral artery is susceptible to A-V fistulas by missile wounds or blunt trauma, due to very close proximity of the artery and adjacent veins within a confined space. This can occur at any age. Symptoms include dizziness, a continuous murmur with systolic accentuation, cardiac enlargement and possible heart failure.[66]

Carotid Cavernous Fistula (CCF)

Carotid cavernous fistula is an abnormal connection between the ICA and cavernous sinus. Majority of the cases are traumatic in origin. Bilateral post traumatic carotid cavernous sinus fistula may occur in association with a fracture of the sella turcica. CCF usually presents with bruit, proptosis, redness or swelling of the conjunctiva, double vision, blurred vision, orbital pain and frequent complaint is pulsation of the involved eye. CCF can be demonstrated by substraction angiography as well as CT.[65,66]

Injury to the Meningeal Vessels

Aneurysm

Linear fracture of the skull, primarily of the temporal bone, may cause injury to the adjacent vessels, particularly the middle meningeal artery. The result may be a rent in the vessel wall leading to a false aneurysm and slight EDH.[66]

A-V Fistula

The fistula develops between the intracranial portion of the middle meningeal artery or its anterior branches and the accompanying veins, in the greater petrosal or spheno parietal sinus or a diploic vein. Carotid angiography shows these two lesions clearly.[67]

Injury to the Dural Venous Sinuses

Most frequently involved sinuses are superior sagittal sinus and transverse sinuses. The sinus can be injured by a penetrating or non penetrating injuries. Depression of a fragment at the fracture site can also cause this. Damage to the dural wall of the sinus or extension of thrombosis inward from abrasions of the scalp or damaged emissary veins probably constitute the most important causes of sinus thrombosis.[65,66]

SEQUELAE OF TRAUMA

Encephalomalacia

Pathologic residua of closed head injury vary from microscopic changes associated with DAI (such axon retraction balls, microglial clusters, and foci demyelination) to more extensive confluent areas of gross parenchymal loss and deep cerebral or generalized cortical atrophy. Encephalomalacic foci appear as low-density non enhancing areas on CT scans.

Blood degradation products may complicate the imaging appearance of encephalomalacia. Secondary changes of volume loss such as ventricular and sulcal enlargement are often present.[68]

HYDROCEPHALUS

Hydrocephalus can occur after sub arachnoid hemorrhage or intraventricular hemorrhage as a result of either impaired CSF reabsorption at the level of the arachnoid granulations or obstruction at the level of the aqueduct or fourth ventricular outflow foramina. Mass effect from cerebral swelling or an adjacent hematoma can cause hydrocephalus by compression of the aqueduct or outflow foramina of the fourth ventricle. Asymmetric lateral ventricular dilatation can be produced by compression of the foramen of Monro.[69]

Pneumocephalus

Skull base fracture with dural tear and direct communication with an air-containing paranasal sinus may lead to acute and chronic pneumocephalus. Intracranial air can occur in virtually any compartment: extracerebral (epidural, subdural, subarachnoid spaces) or intracerebral (brain parenchyma, cerebral ventricles). Air collections can be diffuse or focal. When they are focal they are often referred to as "pneumatoceles".

Intracranial air is easily identified as very low attenuation foci on CT scans. Epidural air tends to remain localized and does not change with alteration in head position. Subdural air often forms an air-fluid level within the subdural space, is confluent, and changes with head position. Subarachnoid air typically is multifocal, non confluent, and droplet-shaped, often located within the cerebral sulci. Intraventricular air, like intraventricular hemorrhage, is typically seen only with severe head trauma. Intraventricular pneumocephalus rarely occurs in isolation and is usually seen with skull base or mastoid fractures that also lacerate the dura. Intravascular air is uncommon and typically only seen with mortal injury.[68]

Ischemia/ infarction

Post traumatic ischemia or infarction can result from raised intracranial pressure, embolization from a vascular dissection or direct mass effect on cerebral vasculature from brain herniation or an overlying extra axial collection. In addition, patients may suffer diffuse ischemic damage from acute reduction in cerebral blood flow or from hypoxemia secondary to respiratory arrest or status epilepticus.

Patterns of infraction from focal mass effect include – Anterior cerebral artery infarction from subfalcine herniation; Posterior cerebral artery infarction from uncal herniation; and posterior inferior cerebral artery infarction from tonsillar herniation.[70]

Cephaloceles and Leptomeningeal Cysts

Herniation of brain, meninges, CSF, or a combination of all three may occur at the site of a dural laceration and dehiscent skull defect. These acquired cephaloceles can occur at any location but are-common in the basifrontal area. Occasionally, acutely increased intracranial pressure combined with surgically or traumatically induced dural and calvarial defects results in extrusion of cerebral tissue and accompanying vessels through the dura into the epidural-and subgaleal spaces.[65]

CSF Leaks and Fistulae

Approximately 80% of CSF fistulas result from skull base fractures. These fistulae are generally basifrontal in location, with drainage into the ethmoid or sphenoid sinuses. Recurrent meningitis complicates 20% of such cases. Although a CSF fistula can develop many years after trauma, 70% occur within 1 week.[65]

Radionuclide cisternography is highly sensitive for the presence of CSF extravasation, however CT scanning with intrathecal contrast is required for detailed anatomic localization of the defect.[72]

HEAD INJURY ASSESSMENT

History

Some patients may describe the events leading to head injury, but more often it is the relative or persons at the scene of the accident who can give a more accurate account. The factors to be noted are,

1) **Alteration of level of consciousness:** This relates to the severity of diffuse brain damage and may range from few seconds to several weeks. It also determines whether patient has improved or deteriorated since the time of accident. If there is a progressive worsening in the level of consciousness of the patient since the time of accident it indicates that there is a progressive intracranial pathology.

2) **Period of post-traumatic amnesia:** This is a period of amnesia occurring after the head injury. It reflects the severity of damage and in severe injuries may last for several weeks.

3) **Cause and circumstance of injury:** Detailed account of how the event took place is important because the patient may collapse or crash his vehicle as a result of some preceding intracranial event. For e.g. subarachnoid hemorrhage, vertigo, epileptic seizures, History of Ethanol Intake etc. More the force of impact, the greater the risk of associated extracranial injuries.

4) **Presence of headache and vomiting:** These are common symptoms after an injury and if they persist a possibility of intracranial hemorrhage and increased intracranial pressure must be considered.

5) **History of seizures:** This is important because, with seizures occurs a rise in intracranial pressure which may further deteriorate the patients condition.

6) **ENT bleed:** These points to the presence of basal skull fractures.

Examination

The points to be kept in mind examining a patient of head injury are,

- Level of consciousness
- Pupillary response
- Limb weakness
- Eye movements
- Evidence of external injury

Level of consciousness

To asses a person's conscious state following brain injury, widely used system is the Glasgow Coma Scale (GCS). The Glasgow Coma scale evaluates a person's level of consciousness and degree of brain injury. The scale standardizes consciousness assessment and helps doctors predict patient prognosis. The Glasgow Coma Scale was first published in 1974 by Graham Teasdale and Bryan J. Jennett, two Glasgow University neurology professors.[73] The Glasgow Coma Scale evaluates visual, motor, and verbal responses to stimuli from three categories. Each response - or lack there of - by the patient is scored. The score from each category is added and assessed. The total Glasgow Coma Scale score helps doctors identify a person's conscious state and extent of brain injury. A lower score indicates more severe brain injury and poorer prognosis.

Eye opening

Spontaneous - 4

To verbal command - 3

To pain - 2

None - 1

Verbal response

Oriented - 5

Confused speech - 4

Inappropriate words - 3

Incomprehensible words - 2

None - 1

Motor response

Obeys command - 6

Localizes pain - 5

Withdrawal from the pain - 4

Abnormal flexion to pain - 3

Abnormal extension to pain - 2

None - 1

If during a short interval several different responses occur, Teasdale and Jannet recommended that the best response in each category be used in noting patients reaction.[73] To follow trends in individual progress, however it is better to report both the best and the worst responses. Right and left motor responses should be recorded separately. In patients who obey commands motor response to single stage

commands can be divisible into brisk and sluggish responses. This distinction separates the patient who is quite lethargic but follows commands after much stimulation from the patient who is nearly alert and requires little prompting to respond to verbal commands.

The physician, however should not limit the examination to parameters of consciousness in Glasgow Coma Scale which are eye opening, verbal response and motor response. Of equal importance in the initial examination of the patient with impaired consciousness are vital sings, pupils, eye movements and motor power. When these features are coupled with GC Scale a dependable and rapid overall neurological status is obtained. The GC Scale provides a simple grading of arousal and functional capability of the cerebral cortex. Because of the widespread nature of the brain injury in addition to cortical function, brain stem function must also be assessed. This is best done by observation of the pupil and the ocular motility.

Pupillary Response

Light reflex tests optic (IInd) and oculomotor (IIIrd) nerve function. Testing the IInd nerve function after head injury is important. Injury to it may result in permanent visual dysfunction. It is the IIIrd nerve function which is more useful, indicator of an expanding intracranial lesion. Herniation of the medial temporal lobe through the tentorial hiatus directly damages the IIIrd nerve resulting in pupil dilatation with impaired or absent reaction to light. The pupil dilates on the side of expanding intracranial lesion and is an important localizing sign. With further increase in intracranial pressure, bilateral IIIrd nerve palsies may occur. Pupillary dilatation when being recorded should be

represented in terms of millimeters. Also the pupillary symmetry should be noted i.e., equal or unequal.

Limb Movement

One should determine the limb movements by comparing the response in each limb to painful stimuli. Hemiparesis and hemiplegia usually occur in limbs contralateral to the side of the lesion because of pressure of the expanding mass on ipsilateral cerebral peduncle. But this may also occur in the ipsilateral limb. This is due to the indentation on the contralateral cerebral peduncle by the edge of the tentorium cerebelli (Kernohans notch).

Eye Movement

Presence or absence of eye movement is of limited value in the immediate management, but provides an useful prognostic guide. Eye movements may occur spontaneously or can be elicited reflexly by head rotation (Oculocephalic reflex) or by caloric stimulation (Oculo vestibular reflex). The latter is usually done by instillation of cold saline into the ear and looking for the eye response on that side. If there is any suspicion that the ear has been damaged, then air instead of saline can be used to avoid infection.

Abnormal eye movements may result from brain stem dysfunction, damage to the nerves supplying the extra ocular muscles or damage to the vestibular apparatus. Absent eye movements relates to the low levels of responsiveness and indicates a gloomy prognosis.

Evidence of External Injury

The presence of these features confirm the occurrence of head injury, but traumatic intracranial lesions can occur without evidence of external injury. With laceration one should explore the wound with a gloved finger for evidence of depressed fracture. Also a scalp hematoma may be over the depressed fracture which may then be missed. The importance of this lies in that, it may be the cause of meningitis later.

CLASSIFICATION OF HEAD INJURY

The severity of head injury is defined by the Traumatic Coma Data Bank (TCDB) on the basis of Glasgow Coma Scale (GCS) scores. Severe head injury includes those patients with a GCS score of '8' or less following neurosurgical resuscitation and those who deteriorate to that level within 48 hours of admission. This accounts for nearly 10% of the cases of head injury admitted to the hospital.

Moderate head injury includes those patients with a GCS score of 9-12. Many studies also include patients with higher GCS scores who undergo surgical intervention for removal of intracranial hematoma.

Mild head injury includes all patients with GCS score between 13-14. A GCS score of 15 is taken as normal.

Various guide lines for indication of CT in head injury patients.

Some of the important guidelines for indication of CT in head injury patients includes Canadian CT head rules, CHIP Prediction Rules for CT in Head Injury Patients , developed in the Netherlands, The National Institute for Health and Clinical Excellence (NICE) and the Scottish Intercollegiate Guidelines Network (SIGN) etc.

Canadian CT head rules includes [74]

High Risk (for neurological intervention)

- GCS < 15 at two hours after injury.
- Suspected open or depressed skull fracture. This is a clinical rather than X-ray diagnosis.
- Any sign of basal skull fracture (hemotympanum, "raccoon" eyes, cerebrospinal fluid otorrhea /rhinorrhea, Battle's sign).
- Vomiting 2 or more episodes.
- Age > 65.

Medium Risk (for brain injury on CT)

- Amnesia before impact > 30 minutes
- Dangerous Mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 3 feet or 5 stairs).

CT in Head Injury Patients (CHIP Prediction Rules) rule developed in the Netherlands includes [75]

A CT is indicated in the presence of 1 major criterion

- Pedestrian or cyclist versus vehicle
- Ejected from vehicle
- Vomiting
- Posttraumatic amnesia 4 hour or more
- Clinical signs of skull fracture
- GCS score < 15
- GCS deterioration 2 points or more (1 hour after presentation)
- Use of anticoagulant therapy
- Posttraumatic seizure
- Age 60 years or more

A CT is indicated in the presence of at least 2 minor criteria

- Fall from any elevation
- Persistent anterograde amnesia
- Post traumatic amnesia of 2 to 4 hours
- Contusion of the skull
- Neurologic deficit
- Loss of consciousness
- GCS deterioration of 1 point (1 hour after presentation)
- Age 40 to 60 years

National Institute for Health and Clinical Excellence (NICE) includes[76,77]

1. **Selection of adults for CT Scan**

Urgent scan if any of the following (results within 1 hour):

- Glasgow Coma Scale (GCS) < 13 when first assessed or GCS < 15 two hours after injury
- Suspected open or depressed skull fracture
- Signs of base of skull fracture
- Post-traumatic seizure
- Focal neurological deficit
- > 1 episode of vomiting (SIGN guidance suggests 2 distinct episodes of vomiting)
- Coagulopathy + any amnesia or loss of consciousness since injury

A CT scan is also recommended (within 8 hours of injury) if there is either:

- More than 30 minutes of amnesia of events before impact
- Or any amnesia or loss of consciousness since injury if:
- Aged ≥ 65 years
- Coagulopathy or on warfarin
- Dangerous mechanism of injury
- Road traffic accident (RTA) as a pedestrian
- RTA - ejected from car
- Fall > 1 m or > 5 stairs

Selection of children (under 16 years) for CT Scan

- Urgent scan if any of the following:
- Witnessed loss of consciousness > 5 minutes
- Amnesia (antegrade or retrograde) > 5 minutes
- Abnormal drowsiness
- ≥ 3 Discrete episodes of vomiting
- Clinical suspicion of non accidental injury
- Post-traumatic seizure (no PMH of epilepsy)
- GCS < 14 in emergency room
  (Paediatric GCS < 15 if aged < 1)
- Suspected open or depressed skull fracture or tense fontanelle
- Signs of base of skull fracture
- Focal neurological deficit
- Aged < 1 - bruise, swelling or laceration on head > 5 cm
- Dangerous mechanism of injury (high-speed RTA, fall from > 3 m, high-speed projectile)

Computed tomography-source of radiation exposure.[5]

The increase in CT use and in the CT-derived radiation dose in the population is occurring just as our understanding of the carcinogenic potential of low doses of x-ray radiation has improved substantially, particularly for children.

In biologic material exposed to x-rays, the most common scenario is the creation of hydroxyl radicals from x-ray interactions with water molecules; these radicals in turn interact with nearby DNA to cause strand breaks or base damage. X-rays can also ionize DNA directly. Most radiation-induced damage is rapidly repaired by various systems within the cell, but DNA double-strand breaks are less easily repaired, and occasional mis-repair can lead to induction of point mutations, chromosomal translocations, and gene fusions, all of which are linked to the induction of cancer and genetic abnormality.

Although the individual risk estimates shown are small, the concern about the risks from CT is related to the rapid increase in its use. small individual risks applied to an increasingly large population may create a public health issue some years in the future. On the basis of such risk estimates and data on CT use from 1991 through 1996, it has been estimated that about 0.4% of all cancers in the United States may be attributable to the radiation from CT studies. By adjusting this estimate for current CT use this estimate might now be in the range of 1.5 to 2.0%.

There are three ways to reduce the overall radiation dose from CT in the population.

The first is to reduce the CT-related dose in individual patients. The automatic exposure-control option on the latest generation of scanners is helping to address this concern.

The second is to replace CT use, when practical, with other options, such as ultrasonography and magnetic resonance imaging (MRI).

The third and most effective way to reduce the population dose from CT is simply to decrease the number of CT studies that are prescribed.

From an individual standpoint, when a CT scan is justified by medical need, the associated risk is small relative to the diagnostic information obtained. However, if it is true that about one third of all CT scans are not justified by medical need, and it appears to be likely, perhaps 20 million adults and, crucially, more than 1 million children per year in the United States are being irradiated unnecessarily.

MATERIALS AND METHODS

Source of data:

Cases for the above study were collected from R.L. Jalappa Hospital and Research Center which is attached to Sri Devaraj Urs Medical College, Kolar.

Research methodology:

1. The study includes evaluation of 300 cases over a period of one year, that is, from DEC 2010 to NOV 2011.
2. All cases with history of road traffic accident and suspected of cranio-cerebral trauma, referred to department of Radiodiagnosis for CT scan within 24 hours of injury were included in the study.
3. Relevant history of the patients was taken, which includes, age, sex, type of injury, principal presenting complaints such as headache, vomiting, loss of consciousness.
4. Relevant clinical findings were noted-such as external injuries over head, pupillary size and reaction etc.
5. Glasgow coma scale (GCS) recorded as follows :

Grades Scores

Normal: 15

Mild head injury: 13-14

Moderate head injury: 9-12

Severe head injury: < 8

5. Non contrast CT brain was performed with SIEMENS Esprit single slice Spiral CT unit, on all the patients.

6. CT findings were correlated with history, clinical findings and Glasgow coma scale.

Inclusion criteria

- All cases with history of road traffic accident and suspected of cranio-cerebral trauma, referred for C.T scan, within 24 hours of injury.

Exclusion criteria:

- Cases referred for CT scan after more than 24 hours of injury.
- History of assault, fall, firearm injury etc.
- Patients in whom Glasgow scale could not be evaluated such as tracheal intubation, severe eye swelling, drug over dose or intoxication, metabolic disturbances, spinal cord injury etc.
- Children under 5 years of age.

CT performed using Seimens SOMATOM ESPRIT single slice spiral CT.

NECT of brain was performed with the patient in supine position.

The exposure settings used were 130kVp and 80-100mAs.

Orbitomeatal line was used as reference and scans were taken with 15 degree gantry tilt. Axial CT brain sequential sections were obtained from orbitomeatal line up to vertex using 3mm axial sections in infratentorial region and 5 mm axial sections in supratentorial region.

The Computed Tomography findings in patients with cranio-cerebral trauma were noted in wide range of window level and window widths to note bleed, fractures and other relevant findings. (window level/ window width for brain 35/70, bone 600/3000, subdural hematomas and collections 70/300).

Various CT findings noted are

- Fractures
- Contusions
- Cerebral edema
- Epidural hematoma
- Subdural hematoma
- Subarachnoid hemorrhage
- Intra parenchymal hematoma
- Intraventricular hemorrhage
- Herniations
- Pneumocephalus
- Hydrocephalus
- Infarcts

RESULTS

During the period of 12 months of the study 300 patients with history of road traffic accidents were studied.

Clinical findings

Out of 300 patients, 249 were male patients (83%) and 51 were females patients (17%)**. (Table 1, Chart1)**

Age distribution of patients with road traffic accidents showed highest in age group between 20-30 years (109 patients 36.33%) followed by 30- 40 years (65 patients). **(Table 2, Chart 2)**

Out of 300 patients-

207 patients showed normal Glasgow coma score (GCS) at the time of examination.

93 patients showed abnormal GCS at the time of examination.

Out of 207 patients with normal GCS -

130 patients had no obvious clinical findings.

77 patients had one or more clinical findings.

Glasgow coma score was normal in 207 patients.

Glasgow coma score was abnormal in 93 patients.

Severity of head injury by traumatic coma data bank which is based on GCS score was

Mild in 13 patients

Moderate in 51 patients

Severe in 29 patients (**Table 4, Chart 4**)

History of loss of consciousness was most common presenting symptoms in head injury patients (147 patients) followed by history of vomiting in 101 patients and abnormal papillary reaction in 29 patients. **(Table 5)**

CT findings

96 patients showed various types of intracranial bleeds.

Subdural hematoma was commonest seen in 59 patients (61.46%). Subarachnoid hemorrhage was seen in 37 patients, epidural bleed in 8 patients and intraparenchymal hematoma was seen in 47 patients. **(Table 6, Chart 5)**

Skull fractures seen in 105 patients. Mid line shift was seen in 16 patients and pneumochepalus in 16 patients. **(Table 7)**

Some of the patients showed more than one abnormality.

Correlation between Glasgow coma scale, clinical and CT findings

Out of 207 patients with normal Glasgow coma score 11 patients had intracranial bleeds.

Out of 13 patients who had mild GCS score 6 patients had intracranial bleeds.

Out of 51 patients who had moderate GCS score all had intracranial bleeds.

Out of 29 patients who had severe GCS score 28 had intracranial bleeds. The one patient who did not have intracranial injuries with severe GCS had facial bones fractures and blunt intra-abdominal injury. (**Table 4, Chart 4**)

16 cases who had mid line shift were classified under severe head injury (13 patients) and moderate head injury (3 patients) by GCS scale. (**Table 8, Chart 6**)

Sensitivity, specificity, positive predictive valve and negative predictive values of Glasgow coma scale to predict intracranial bleed was calculated with following data.

- Patients who had normal GCS and no intracranial bleed (True Negative)- 196 patients.
- Patients who had abnormal GCS and showed intracranial bleed on CT (True Positive)- 85 patients.
- Patients who had normal GCS and showed intracranial bleed on CT (False Negative)- 11 patients.

- Patients who had abnormal GCS and no intracranial bleed (False Positive)- 8 patients.

When Glasgow coma scale alone is taken as test to identify intra cranial bleed, it showed Sensitivity 88.5%, specificity 96.1%, positive predictive value 91.4%, and negative predictive value 94.7%. (**Table 9**)

Sensitivity, specificity, positive predictive valve and negative predictive values of Glasgow coma scale combined with relevant clinical findings to predict intracranial bleed was calculated with following data.

- Patients who had normal GCS and clinical findings and no intracranial bleed (True Negative)- 130 patients.
- Patients who had abnormal GCS and/or clinical findings and showed intracranial bleed on CT (True Positive)- 93 patients.
- Patients who had normal GCS and clinical findings and showed intracranial bleed on CT (False Negative)- 2 patients.
- Patients who had abnormal GCS and/or clinical findings and no intracranial bleed (False Positive)- 75 patients.

When Glasgow coma scale combined with relevant clinical findings is taken as test to predict intra cranial bleed showed Sensitivity 97.8%, specificity 63.4%, positive predictive value 55.3%, and negative predictive value 99.2%. (**Table 9**)

Two out of 132 patients with normal GCS and clinical findings had minor intracranial bleed on CT— one patient had a small contusion in left frontal lobe and the other had left tentorial bleed. These two patients had good clinical outcome on follow up.

Total number of cases who had normal CT findings were 159, 53% of our total study population. (**Table 10, Chart 7**)

Out of the 159 patients who had normal CT findings, 94 (31.33% of total study population), **(Table 12**) had normal GCS and clinical findings. This figure accounts for the more than half of (59.12%) of patients with normal CT. (**Table 11, Chart 8**)

OBSERVATIONS

Table 1: Gender distribution among study population.

Gender	No. of patients	Percentage(%)
Male	249	83
Female	51	17
Total	300	100

Chart 1: Gender distribution among study population.

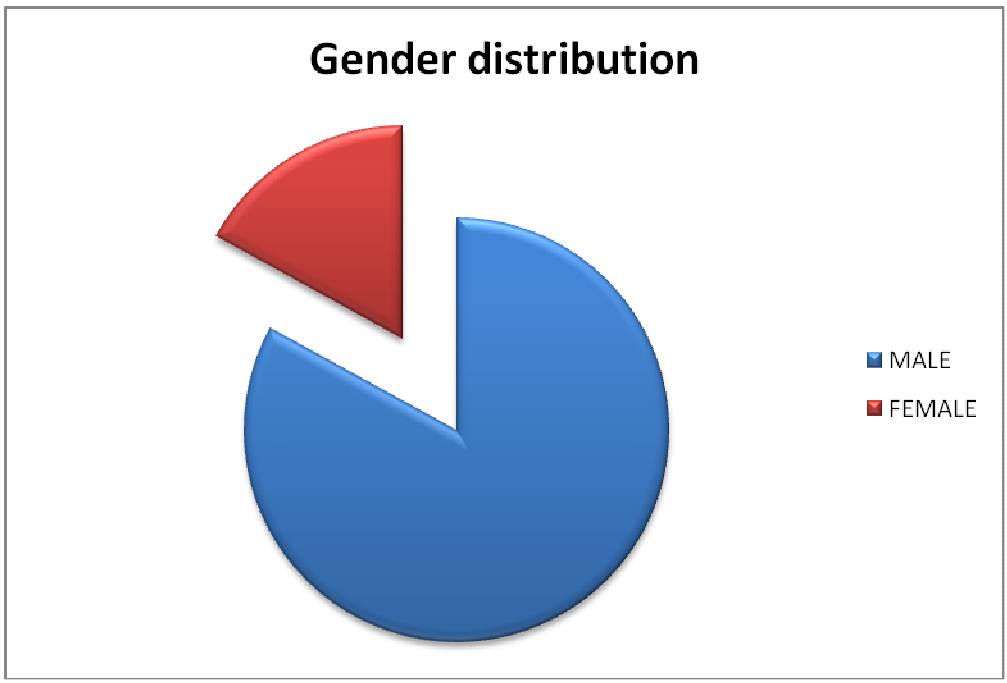


Table 2: Age distribution among study population.

Age (yrs.)	No. of patient	Percentage(%)
Below 10	11	3.67
10-20	20	6.67
20-30	109	36.33
30-40	65	21.67
40-50	51	17
50-60	31	10.33
60-70	8	2.67
80-90	0	0
Total	300	100

Chart 2: Age distribution among study population

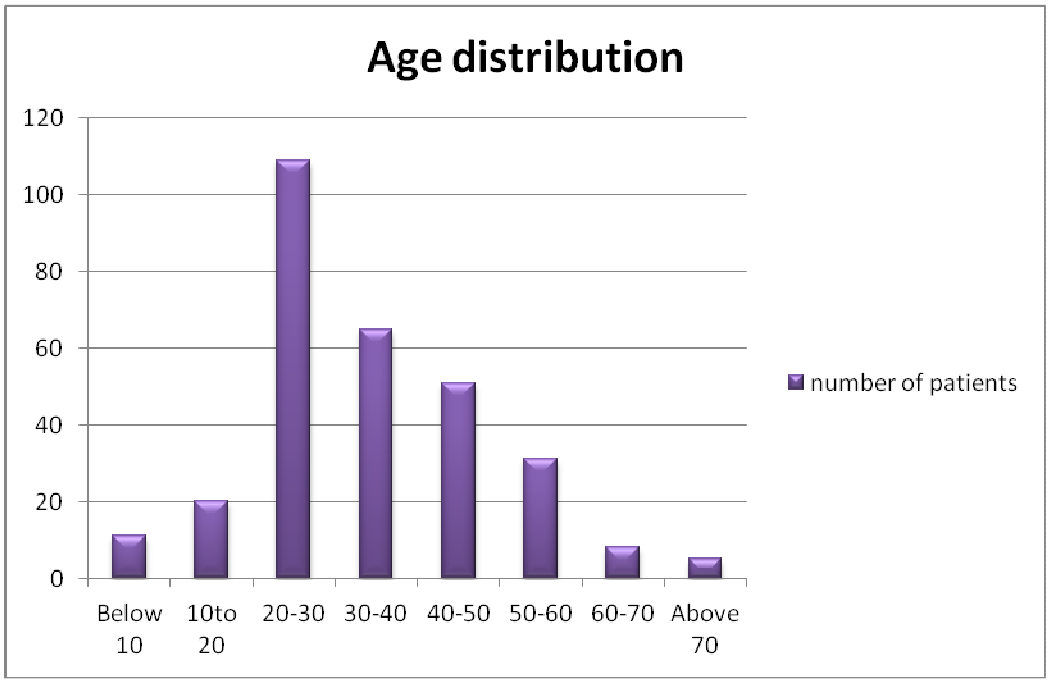


Table 3: Gender and age distribution among study population.

Age	Male		Female		Total
In years	**Cases**	**Percentage (%)**	**cases**	**Percentage (%)**	
< 10	5	2.01	6	11.76	11
10-20	16	6.43	4	7.84	20
20-30	98	39.36	11	21.59	109
30-40	54	21.69	11	21.59	65
40-50	42	16.87	9	17.65	51
50-60	23	9.24	8	15.69	31
60-70	7	2.81	1	1.96	8
> 70	4	1.60	1	1.96	5
Total	249		51		300

Chart 3: Gender and age wise distribution among study population.

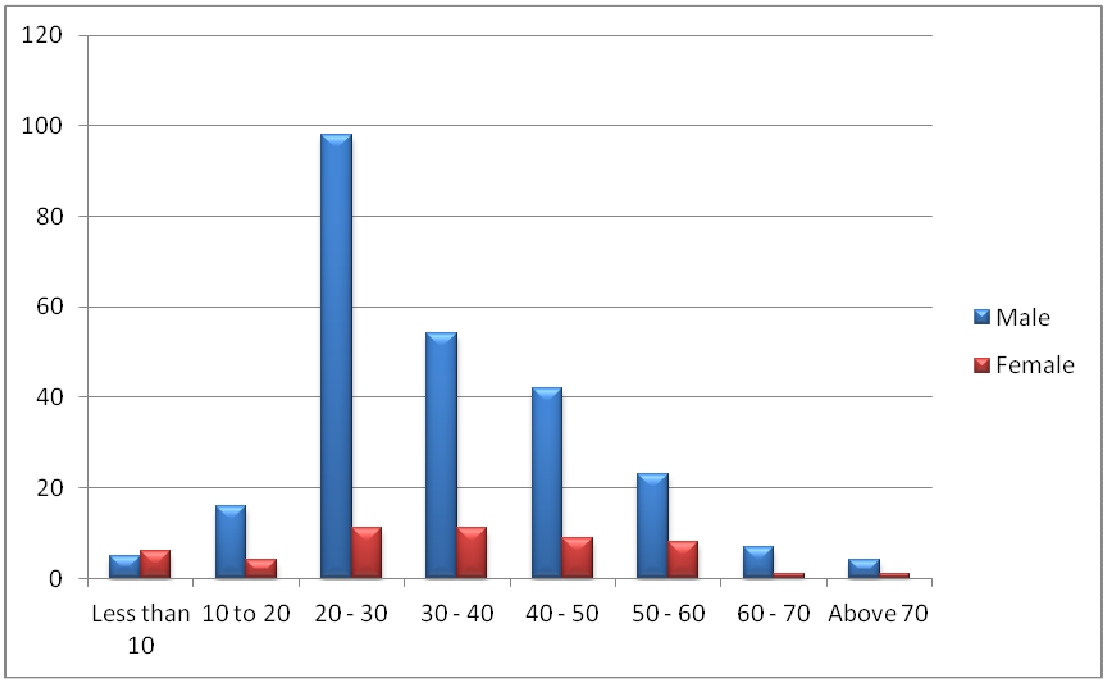


Table 4: Glasgow coma score among study population and showing intracranial bleeds.

Glasgow coma scale classification	Number of patients	Number of patients with intracranial bleeds
Normal (15)	207	11
Mild (13-14)	13	6
Moderate (9-12)	51	51
Severe (less than 8)	29	28*

***Note: One patient classified under severe GCS score had blunt intra abdominal injury, facial bone fractures but showed no intracranial bleed.**

Chart 4: Glasgow coma score among study population.

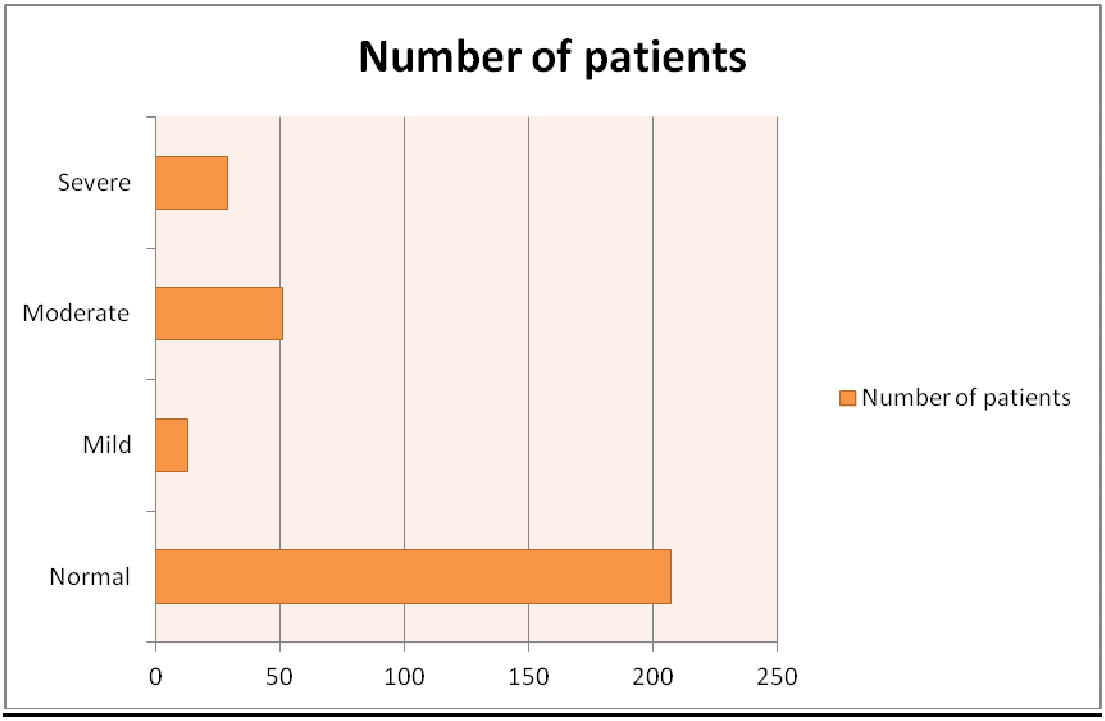


Table 5. Relevant clinical findings among study population and their frequency among all the patients (168) who had abnormal clinical findings

S.No	Clinical findings	Number of patients	Percentage(%)
1	History of loss of consciousness	147	87.5
2	History of vomiting	101	60.12
3	Abnormal papillary reaction	29	17.26

Note: A patient may show one or more of the above findings.

Table 6: Percentage of various bleeds in all patients with intracranial bleeds. (96 patients had various types of intracranial bleeds)

Type of bleed	Number of patients	Percentage(%)
Subdural	59	61.46
Subarachnoid	37	38.54
Epidural	8	8.3
Intraparenchymal	47	48.95

Note : A patient may show one or more of the above findings.

Chart 5: Various bleeds among study population.

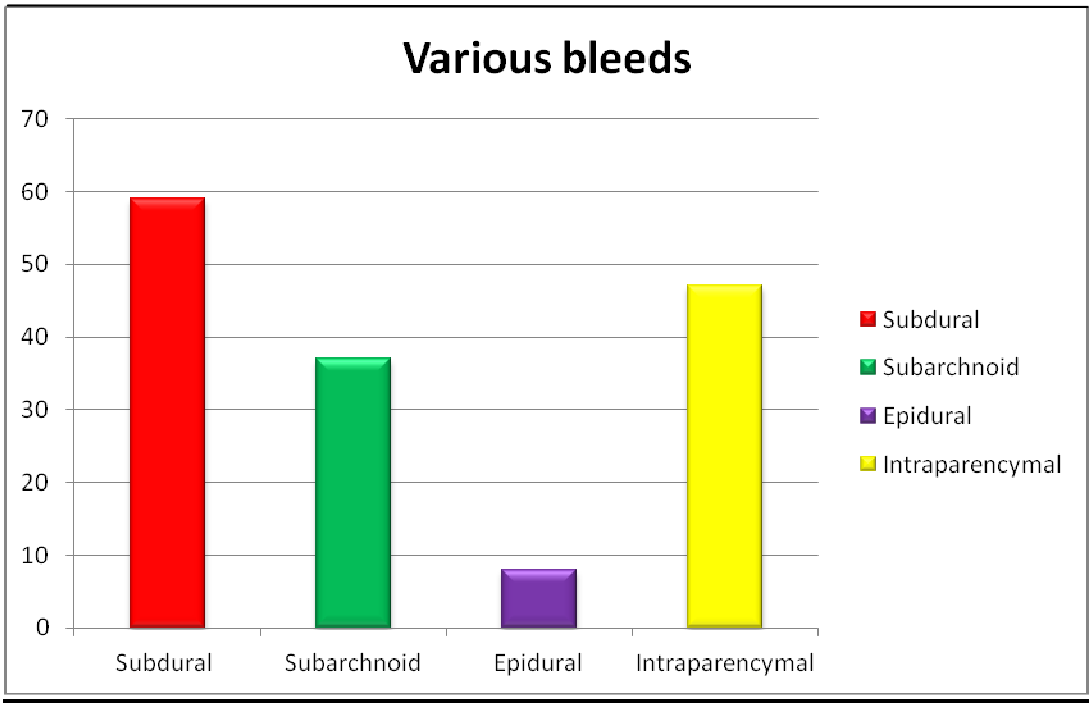


Table 7. C T findings among all patients (141) who had one or more abnormal CT findings.

S. No	C T Findings	Number of patients	Percentage (%)
1.	Subdural hematoma	59	41.84
2.	Subarachnoid hemorrhage	37	26.24
3.	Epidural hematoma	8	5.67
4.	Contusions	47	33.33
5.	Skull fractures	105	74.47
6.	Midline shift	16	11.35
7.	Pneumochephalus	16	11.35
8.	Diffuse Cerebral edema	19	13.48

Table 8. Patients with Mid line shift showing severity of head injury in GCS

Glasgow coma scale	Patients with mid line shift	Percentage (%)
Mild	0	0
Moderate	3	18.75
Severe	13	81.25
Total	16	100

Chart 6. Patients with Mid line shift showing severity of head injury in GCS

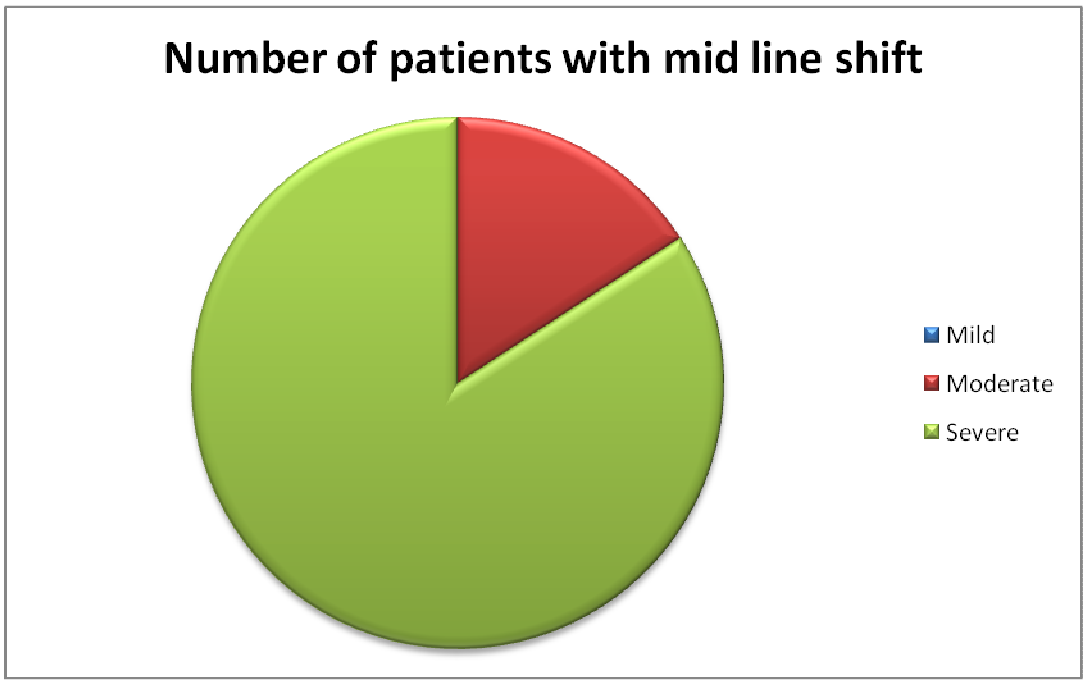


Table 9. Sensitivity and specificity of GCS and GCS combined with clinical findings in prediction of intracranial bleeds.

Test	**GCS**	**GCS combined with clinical findings**
Sensitivity	88.5	97.8
Specificity	96.1	63.4
Positive predictive value	91.4	55.3
Negative predictive valve	94.7	99.2

Table 10: Frequency of normal and abnormal CT findings in patients who underwent CT scan in our study.

CT findings	Number of patients	Percentage (%)
Normal	159	53%
Abnormal	141	47%
Total	300	

Chart 7 : Frequency of normal and abnormal CT findings in patients who underwent CT scan in our study.

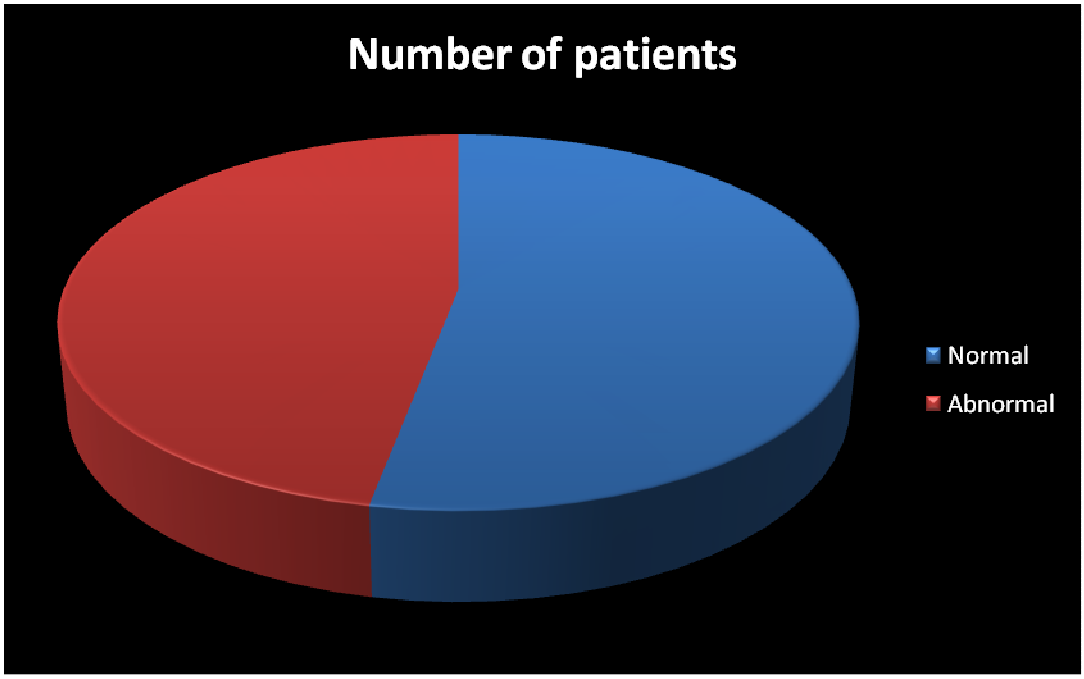


Table 11 :Frequency of patients with normal CT findings who had normal and abnormal GCS and/ or clinical findings scan in our study.(Total number of patients with normal CT findings 159)

GCS and/ or clinical findings	Number of patients	Percentage (%)
Normal	94	59.12%
Abnormal	65	40.88%
Total	159	

Chart 8 : Frequency of patients with normal CT findings who had normal and abnormal GCS and/ or clinical findings scan in our study.(Total number of patients with normal CT findings 159)

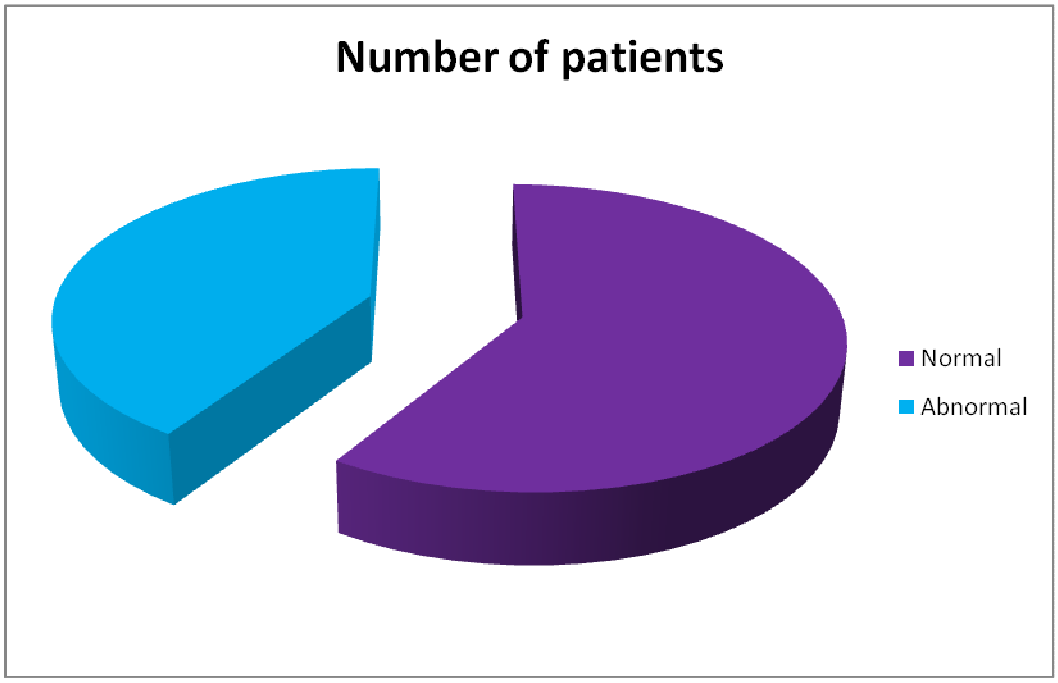


Table 12: Number of patients with normal CT who had normal GCS and clinical findings.

	Number of patients	Percentage (%)
Normal CT findings	159	53%
Normal CT, GCS and clinical findings	94	31.33%

FIGURES & LEGENDS

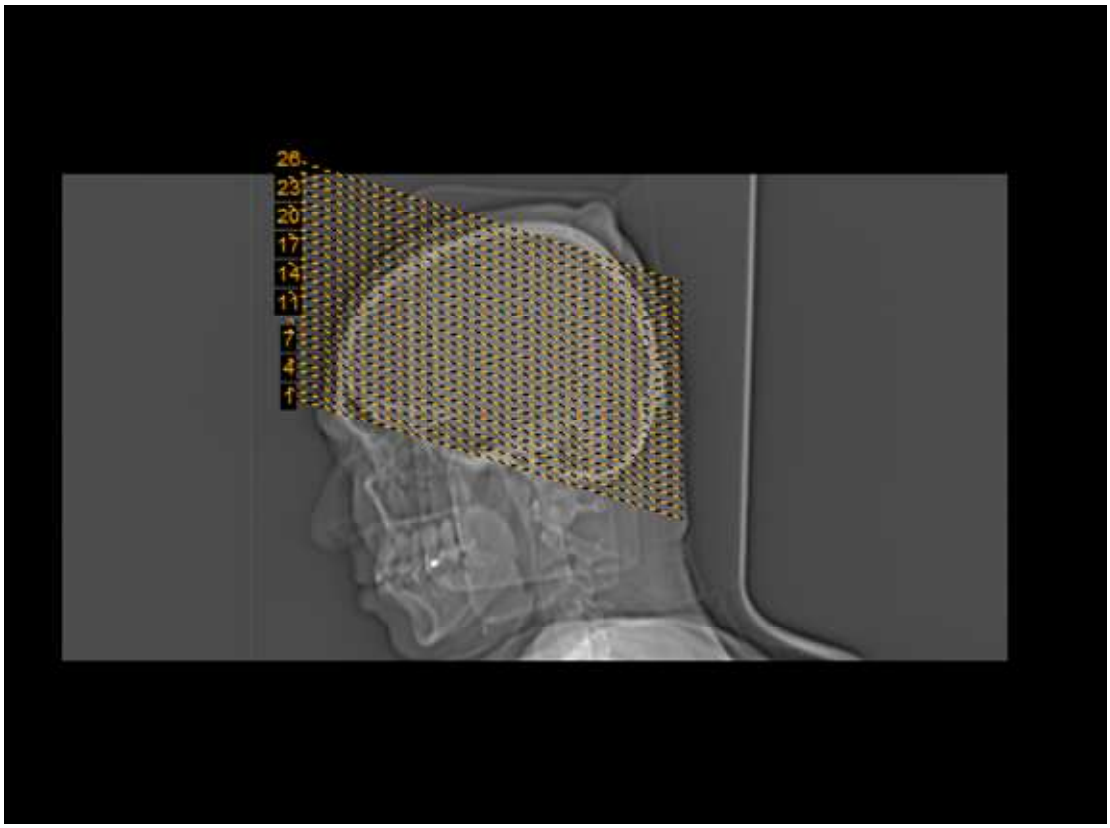


Figure 1: CT scan of brain. Topogram and Planning.

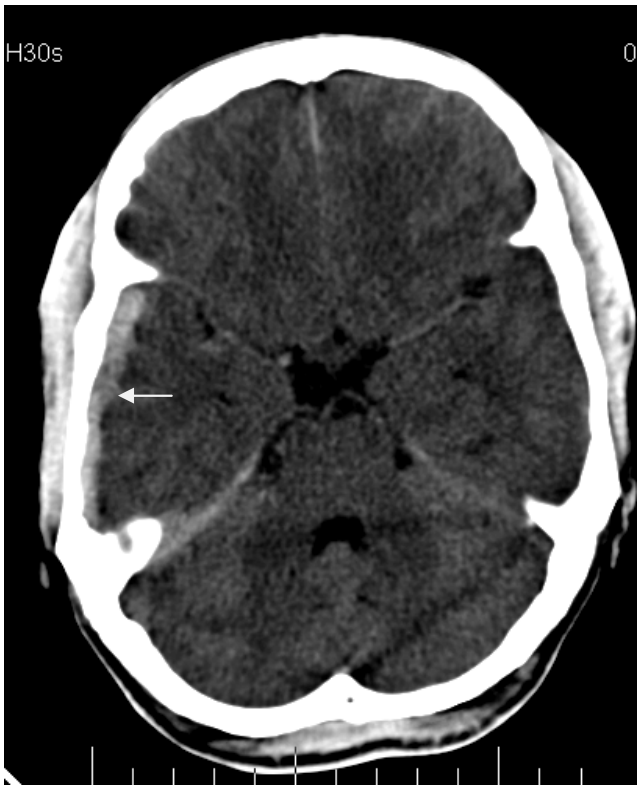


Figure 2: Subdural hematoma in right temporal convexity (arrow).

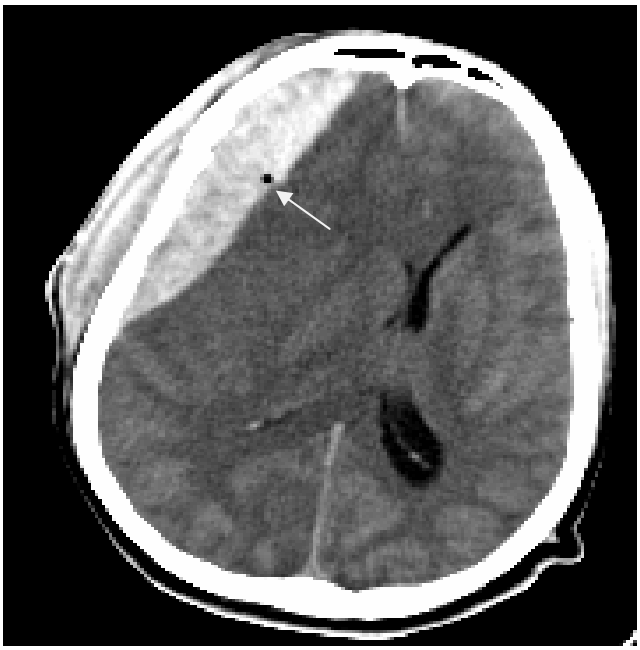


Figure 3: Subdural hematoma is seen in right frontotemporal region causing mid line shift towards left. Fracture of overlying frontal bone along with small pneumocephalus noted (arrow).

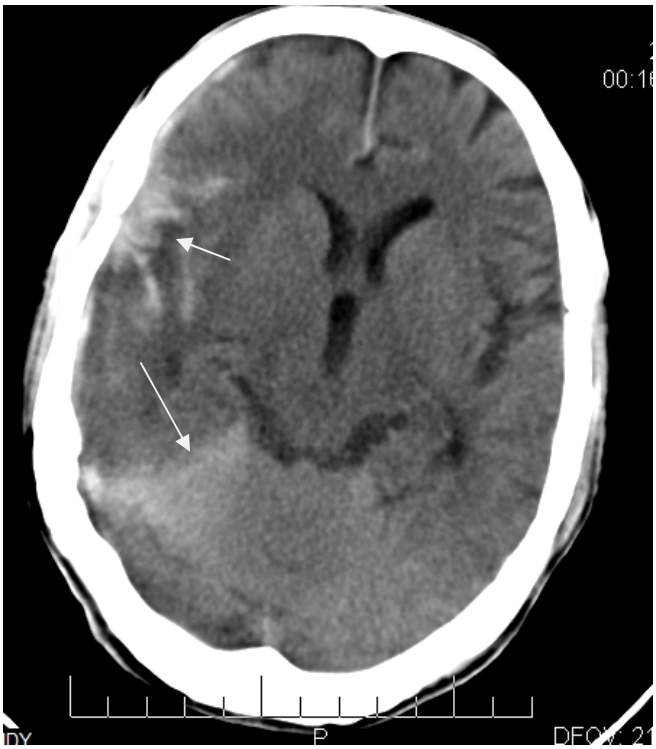


Figure 4: Subdural and subarachnoid bleed densities along right frontotemporal region (short arrow). Subarachnoid hemorrhage is seen extending into right sylvian fissure. Subdural hematoma also seen along right tentorium cerebelli (long arrow).

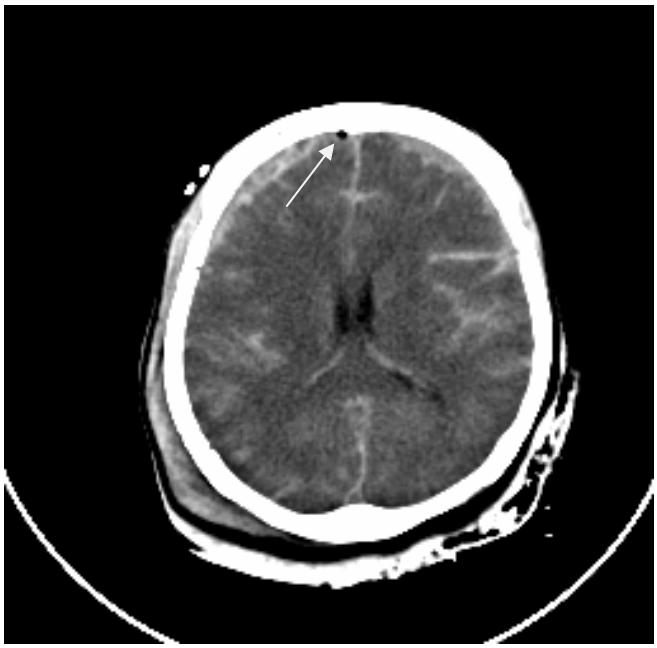


Figure 5: Subdural hematomas seen in bilateral frontal region with small pneumocephalus (arrow) and subarachnoid hemorrhage is noted diffusely involving interhemishperic fissures and bilateral cortical sulci.

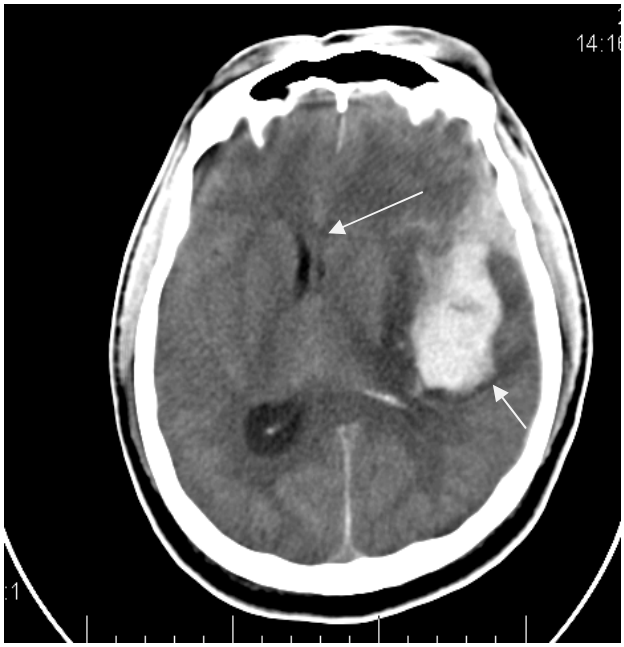


Figure 6: Large intraparencymal hematoma is seen in left temporal lobe with surrounding edema (short arrow) and subdural hematoma is seen in left fronto-temporal region. There is also mid line shift towards right (long arrow).

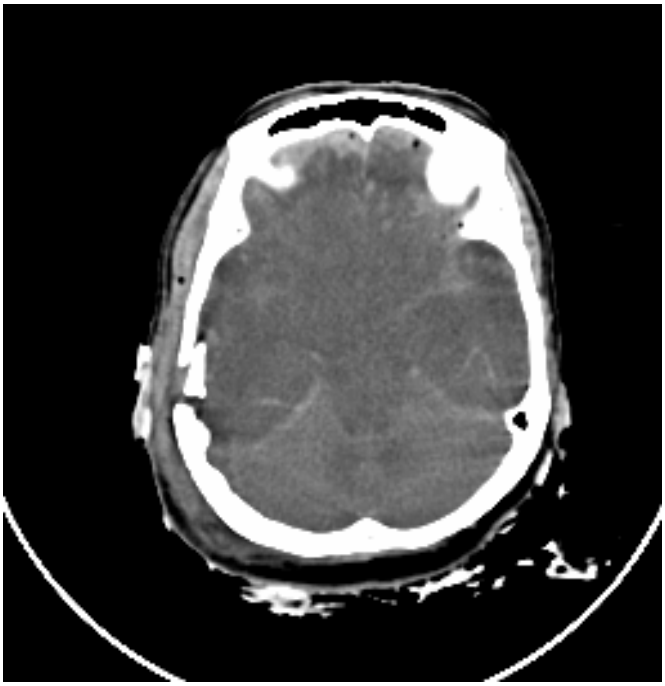


Figure 7: Diffuse cerebral edema seen obliterating bilateral cortical sulci and basal cisterns with diffuse subarachnoid bleed. Displaced fracture of right temporal bone seen. Subdural hematoma is seen in bilateral frontal lobes.

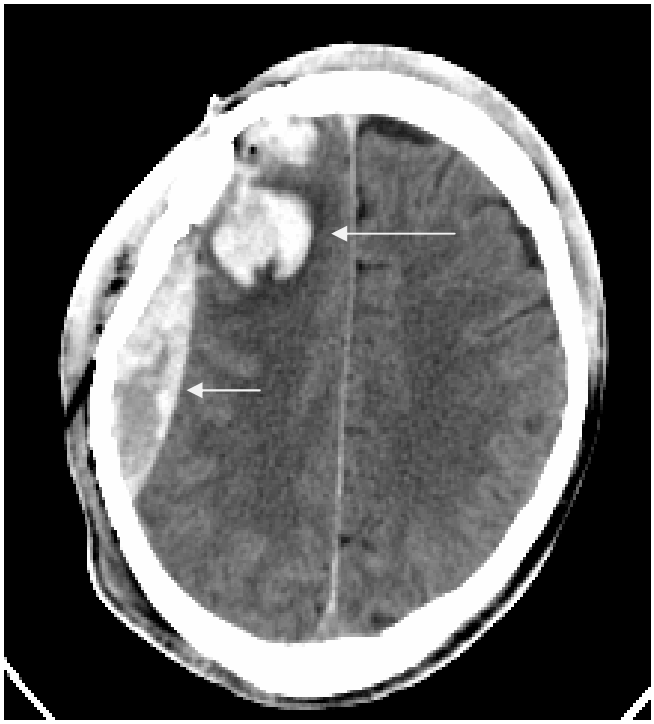


Figure 8: Epidural hematoma is seen in right fronto parietal convexity (short arrow). There are intraparencymal hematomas with minimal surrounding edema in right frontal lobe with fracture of frontal bone (long arrow).

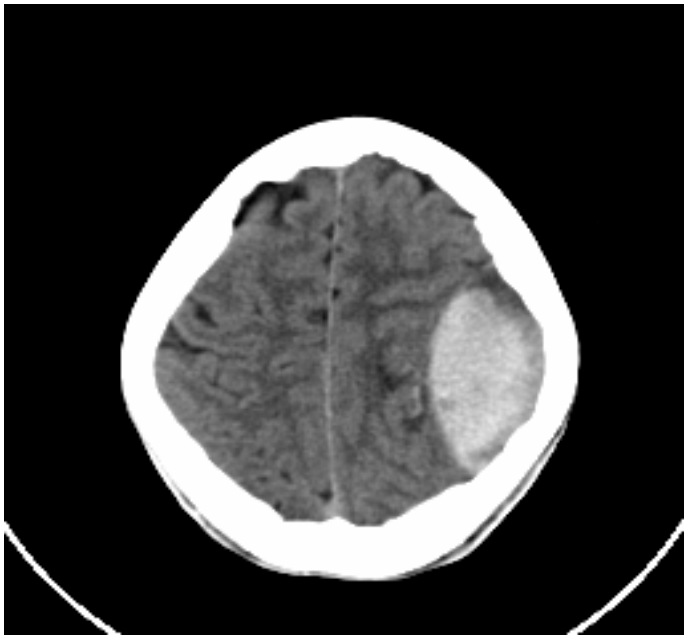


Figure 9: Epidural hematoma is seen in left high parietal region.

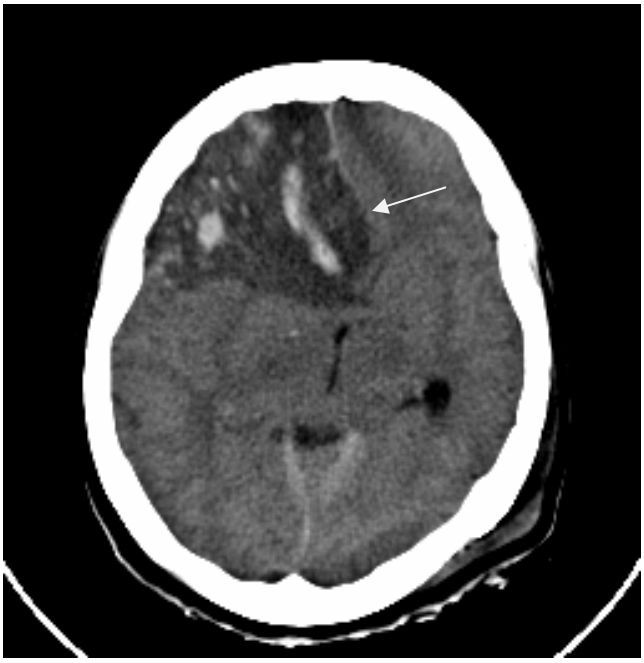


Figure 10: Multiple hemorrhagic contusions in right frontal lobe with significant surrounding edema and causing mass effect and midline shift towards left (arrow).

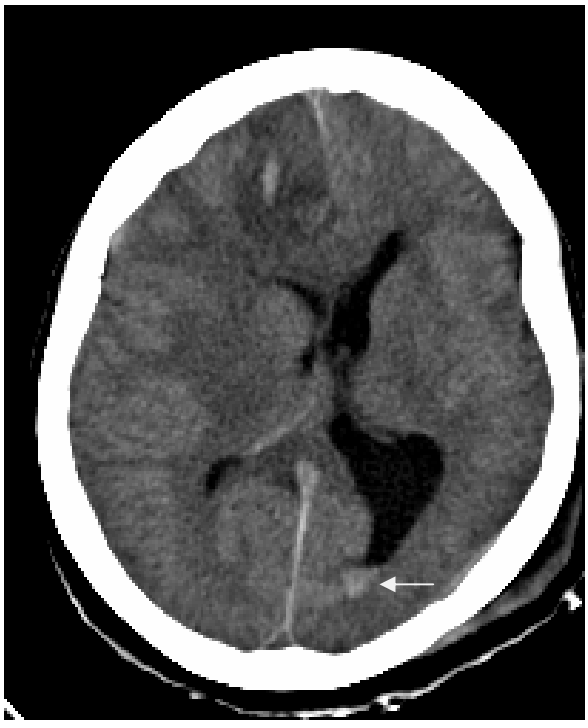


Figure 11 Same patient in higher section shows mass effect causing compression of ipsilateral lateral ventricle and dilatation of contra-lateral ventricle with intraventricular hemorrhage within (arrow).

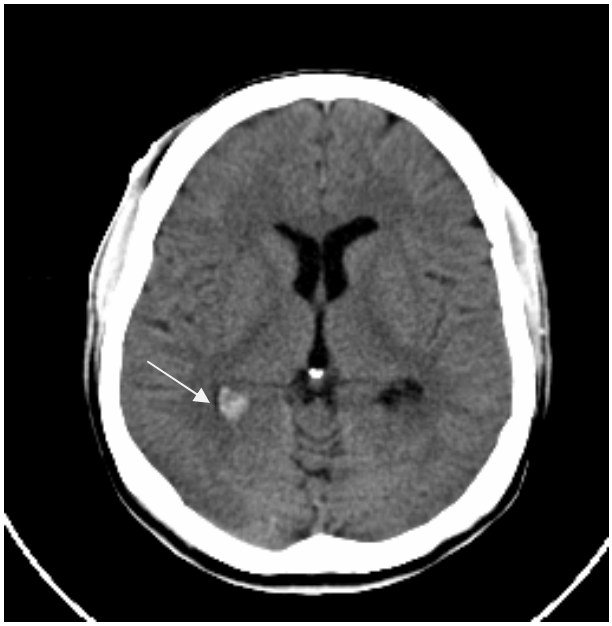


Figure 12. Patient with isolated intra ventricular hemorrhage in right lateral ventricle (arrow).

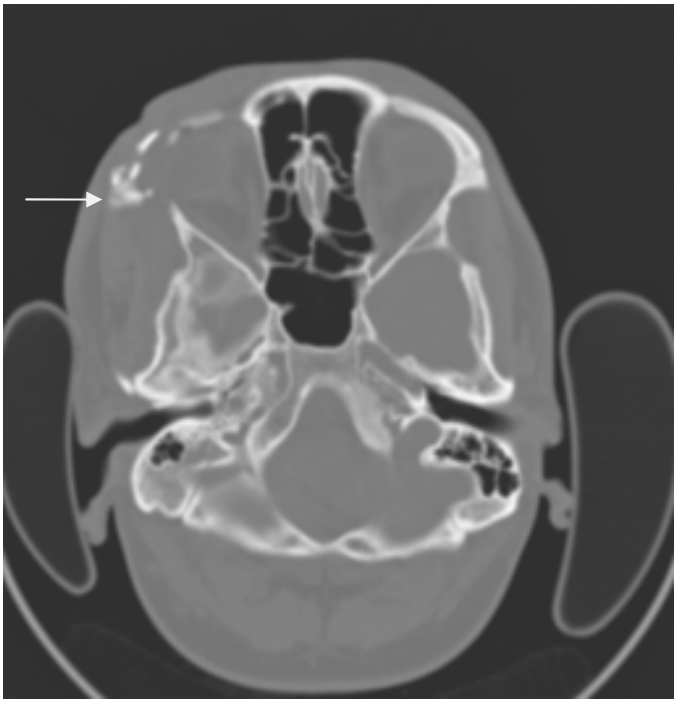


Figure 13.Patient with comminuted and displaced fracture of lateral wall of right orbit and supra orbital margin of frontal bone (arrow).

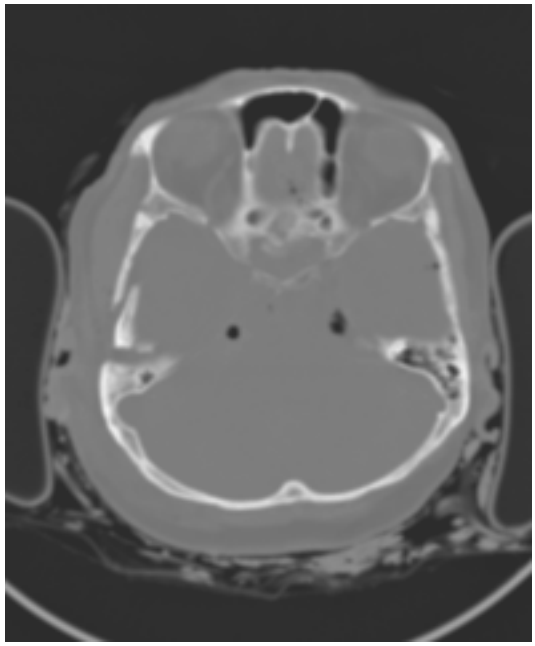


Figure 14. Patient with pneumocephalus and displaced fracture of right temporal bone in bone window.

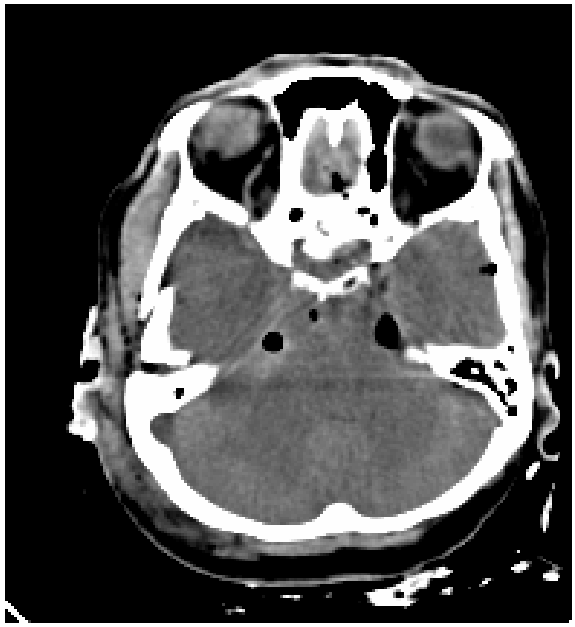


Figure 15.Same patient with pneumocephalus and displaced fracture of right temporal bone shown in cerebral window.

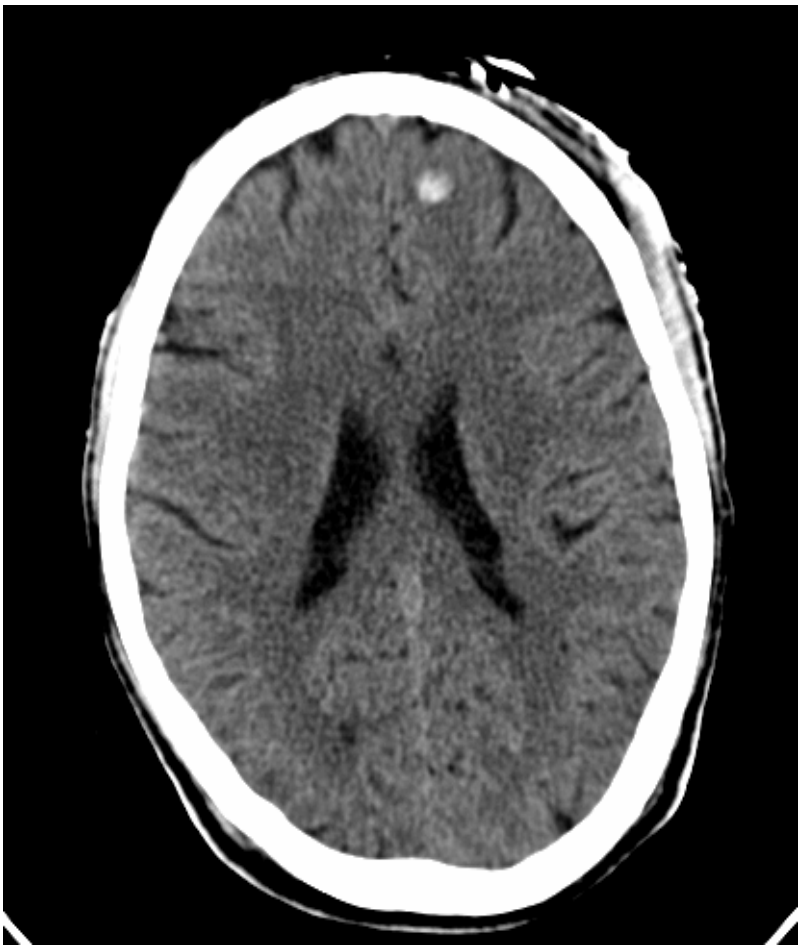


Figure 16: There is small intra-parenchymal hematoma in left frontal lobe in one patient who had normal GCS and clinical findings at the time of presentation. Patient had good clinical out come on follow up.

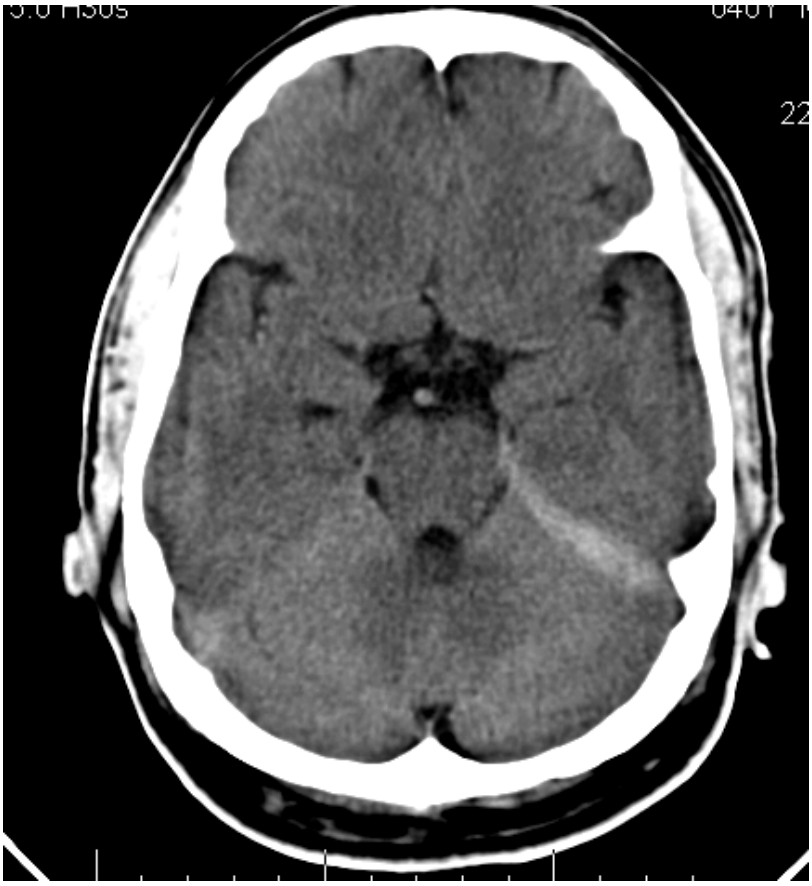


Figure 17: There is subdural hematoma in left tentorium cerebelli in another patient who had normal GCS and clinical findings at the time of presentation. Patient had good clinical outcome on follow up.

DISCUSSION

CT is one of the most comprehensive diagnostic modality for accurate localization of the site of injury in cranio-cerebral trauma. CT helps in early and timely diagnosis of the lesion precisely. CT has substantial impact over instituting appropriate treatment and timely surgical intervention.

Various clinical parameters used for planning management and predicting the prognosis of a patient with cranio-cerebral trauma are based on neurological dysfunction which is due to structural damage to the brain. This structural damage can be efficiently detected by CT scan. It is worthwhile to utilize this modality as the primary modality of choice in acute cranio-cerebral trauma.

In our study by incidence, male population was predominantly involved (83%) in road traffic accidents. Age wise most involved age group is between 20 to 30 years (36.33%) followed by 30 to 40 years (21.67%).

An enumerative population study by National Head and Spinal Cord Injury Study, (NHSCIS) showed the incidence rate for head injury range with highest incidence being 15–24 years and the male incidence being more than twice that for females.[78]

Another study by Kraus JF, Black MA et al., showed that males having a 2.2 times higher rate than females in head injury and rates were highest for males aged 15-24 years.[79]

Our study also showed similar findings. Universally male patients and working young population are more prone to craniocerebral trauma.

Subdural hematoma is the most common form of intracranial bleed (61.46%) followed by intraparenchymal hematoma (48.95%) among 96 patients who showed intracranial bleeds on CT in our study.

Maas AI, Stocchetti N et al., study shown that SDH was the most common type, present in 30% of the patients. EDH, IPH and SAH were present in 22% each.[80]

Our study correlates with above study.

Gentry LR et al, study showed cortical contusions represent 45% of primary intra axial traumatic lesions.[81]

Our study showed 48.95% of intracranial bleed was by contusions.

Patients with midline shift, and an abnormal third ventricle had significantly lower GCS scores in study by J M Wardlaw et al.[82]

Our study also showed significant number of cases (81.25%) with midline shift were classified under severe head injury by GCS.

Study by P. A. Gómez, R. D. Lobato, et al., shown multivariate analysis showed that advanced age, a lower GCS (13-14) and the presence of skull fracture, and focal signs, significantly increased the incidence of abnormal computed tomography (CT) findings.

Patients with 13-14 GCS had a significantly higher incidence of initial loss of consciousness, of skull fracture, abnormal CT findings.[83]

In a study by Miller EC, Derlet RW et al, patient with Glasgow Coma Scale score of 15 underwent CT of the head after loss of consciousness (LOC) or amnesia to event. Traumatic intracranial abnormality was identified on CT of the head in 84 (6.1%). Nausea and vomiting and signs of head trauma were significantly more common in the group with abnormal CT findings, only 0.2% had findings on cranial CT that indicated a need for surgery.[84]

By using four risk factors (headache, nausea, vomiting, and a depressed skull fracture), the investigators obtained 65% sensitivity and 63% specificity in detecting abnormal CT results when one or more of these risk factors was present.[85]

Similarly, in another series, the small percentage of patients who had a normal GCS score after minor head trauma and who had a positive finding on head CT also had one or more of seven findings, including headache, vomiting, drug or alcohol intoxication, seizure, or physical evidence of trauma above the clavicles.[76]

Various guide lines have been given for indication of CT in head injury patients.

Some of the important guidelines includes Canadian CT head rules, CT in Head Injury Patients (CHIP Prediction Rules) rule developed in the Netherlands, The National Institute for Health and Clinical Excellence (NICE) and the Scottish Intercollegiate Guidelines Network (SIGN).(Mentioned in pages 52 to 55)

We have found following results.

Glasgow coma scale combined with relevant presenting complaints is more sensitive (97.8%) in predicting intracranial bleed, rather than GCS alone (sensitivity 88.5%).

GCS combined with relevant presenting complaints showed negative predictive value of 99.2%. This means that it rarely mis-classifies patient with intra cranial bleed as normal.

Two patients who had normal GCS and clinical findings but showed small intracranial bleeds, had good clinical out come without any neurological deficit on follow up.

In our study 159 patients (53%) of patients had normal CT findings.

By using GCS combined relevant presenting complaints as test to predict intracranial injury, “unnecessary” CT scan could have been avoided in 94 patients out of total study population of 300 (31.33%).

CONCLUSION

Incidence of road traffic accidents was more in male population.

Peak age group involved in road traffic accident was between 20 to 30 years followed by 30 to 40 years in our study.

A variety of abnormalities were detected in CT scan.

Subdural hemorrhage was most common form of intracranial bleed.

Most of the cases with midline shift had moderate or severe head injury by GCS. None of the cases with normal or mild GCS had midline shift.

Glasgow coma scale combined with relevant clinical findings is sensitive method of predicting intracranial injuries.

GCS combined with relevant clinical findings rarely mis-classifies patient with intra cranial bleed as normal.

Glasgow Coma Score combined with relevant clinical findings can be used as an indication for CT scan in order to avoid unnecessary CT scans.

SUMMARY

Craniocerebral trauma is leading cause of death and trauma related fatalities in motor vehicle accidents.

CT is one of the most comprehensive diagnostic modality for accurate localization of the site of injury in cranio-cerebral trauma. CT helps in early and timely diagnosis of the lesion precisely. CT has substantial impact over instituting appropriate treatment and timely surgical intervention.

The study included 300 patients who presented with history of road traffic accidents. Relevant history, clinical findings and Glasgow coma scale was taken before subjecting to CT brain.

Intracranial injuries were correlated with history, clinical findings and GCS.

Study showed that younger and male population was predominantly involved in road traffic accidents.

Subdural hematoma was the most common type of intracranial bleed.

Most patients with mid line shift were classified under moderate or severe head injury by GCS scale.

When Glasgow coma scale combined with relevant clinical findings is used as test to predict intracranial bleed, it proved to be more sensitive than GCS alone.

High negative predictive value of GCS combined with relevant clinical findings (99.2%), rarely mis-classifies patient with intra cranial bleed as normal.

CT involves high doses of radiation, resulting in a marked increase in radiation exposure in the population. Although the risks for any one person are not large, the increasing exposure to radiation in the population may be a public health issue in the future.

By using GCS combined with relevant clinical findings as test to predict intracranial injury, CT scan could have been avoided in 94 patients (31.33%) in our study.

Glasgow Coma Score combined with relevant clinical findings can be used as an indication for CT scan in order to avoid unnecessary CT scans. Radiation exposure to population can be hence reduced.

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Annexure :I

PROFORMA

Name:

Date:

Hospital no.:

Age:

Sex:

CT no.:

Type of injury:

Presenting complaints:

1. Loss of consciousness-
2. Lucid interval-
3. Vomiting-
4. Seizures-
5. Headache-
6. Bleeding from any orifices-
7. Any other complaints-

Family history:

Past history:

Personal history:

1.Alcohol intake-

2. Others-

General examination:

1.Build-

2.Mental status-

3.Pupillary reaction-

4.Associated injuries-

Vital examination:

1.Blood pressure-

2.Pulse rate-

3.Respiration rate-

4.Temperatue-

5.Other systems-

Glasgow coma scale:

1.Eye opening-

2.Motor response-

3.Verbal response-

Computed tomography findings:

1.Fractures-

2.Contusions-

3.Subdural hematoma-

4.Subarachnoid hemorrhage-

5.Epidural hematoma-

6.Cerebral edema-

7.Intraventricular bleed-

8.Mass effect (midline shift)-

9.Pneumocephalus-

10.Hydrocephalus-

11.Infarcts-

12.Any other findings-

Annexure :II

ABBREVIATIONS USED IN MASTER CHART

Sl.No- Serial number

HOSP.No.- Hospital number

CT No- CT number

M-Male

F-Female

Y-Years

T.INJ- Type of injury

RTA- Road traffic accident

L.O.C- Loss of consciousness

LCD IN- Lucid interval

VOM- Vomiting

H.ACHE- Headache

SZS- Seizures

ALC-Alcohol intake

PUP-Pupillary reaction

GCS-Glasgow coma score

E-Eye opening

V-Verbal response

M-Motor response

SYN-Other systemic injury

FRCT-Fracture

CON-Contusions

C.ED-Cerebral edema

EDH-Epidural hematoma

SDH-Subdural hematoma

SAH-Subarachnoid hemorrhage

IVB-Intraventricular hemorrhage

MDS-Midline shift

PNEU-Pneumocephalus

HYD-Hydrocephalus

INF-Infarcts

Y-Yes (Present)

N-No (Absent)

A- Abnormal papillary reaction

P-Present

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
1	Ramalakhmamma	680056	60y	F	484	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	Y	N	N
2	Srihari	730164	30y	M	405	RTA	Y	N	Y	N	N	N	P	N	3	3	5	Y	Y	N	N	N	N	N	N	Y	N	N
3	Fajil khan	709930	26y	M	226	RTA	Y	N	Y	N	N	N	P	N	3	4	5	Y	Y	N	N	Y	N	N	N	N	N	N
4	Revanna	666375	35Y	M	165	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
5	Srinivas	666696	37y	M	193	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
6	Balakrishna	717312	27Y	M	258	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
7	Lakminarayanamma	667225	35y	F	221	RTA	Y	N	N	N	N	Y	P	N	4	5	6	N	N	N	N	Y	N	N	N	N	N	N
8	Manjunath	668084	30y	M	265	RTA	N	N	N	N	N	N	P	Y	4	5	5	N	N	N	N	N	N	N	N	N	N	N
9	Rathnamma	668319	36y	F	290	RTA	Y	N	N	N	N	N	P	N	3	4	5	N	N	N	N	Y	Y	N	N	N	N	N
10	Venkataram reddy	680414	35y	M	509	RTA	Y	N	Y	N	N	N	P	N	2	3	3	Y	N	N	N	Y	N	N	Y	Y	N	N
11	Shankar	712169	22y	M	389	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
12	Rajendra	677891	30y	M	313	RTA	Y	N	Y	N	N	N	P	N	3	3	4	N	N	N	N	Y	N	N	N	Y	N	N
13	Gnanaprakash	728293	45y	M	311	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
14	Ganesh	668945	30y	M	343	RTA	Y	N	N	N	N	Y	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
15	Karhik	669208	9y	M	357	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
16	Natarajchari	669287	40y	M	359	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
17	Subarmani	669512	40y	M	362	RTA	Y	N	N	N	Y	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
18	Ramaiah M	669535	52y	M	369	RTA	Y	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
19	Narayanappa	668902	60y	M	373	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
20	Ravikumar	669616	35y	M	376	RTA	Y	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
21	Muda chari	669931	40y	M	398	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
22	Venkatesh	730166	23y	M	406	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
23	Prakash	669944	30y	M	402	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
24	Bayamma	670276	36y	F	419	RTA	N	N	N	N	N	N	A	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
25	Muniraju	709934	35y	M	227	RTA	N	N	N	N	N	N	P	Y	4	5	5	Y	N	N	N	N	N	N	N	N	N	N
26	Shrinivasamurthy	670584	45y	M	440	RTA	N	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
27	Myelagouda	712689	35y	M	421	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
28	krishnappa	67822	38y	M	449	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
29	Lakmidevamma	670821	22y	F	450	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
30	Murali	729288	22y	M	361	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
31	Buche gouda	6822984	40y	M	148	RTA	Y	N	Y	N	N	Y	P	N	2	4	4	Y	Y	N	N	N	N	N	N	N	N	N
32	Dinkar	670840	45y	M	453	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
33	Arif ullah	670856	30y	M	456	RTA	N	N	N	N	N	Y	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
34	venkatarama	683102	30y	M	169	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
35	Ramesh	670887	28y	M	458	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
36	Byregouda	670895	30y	M	459	RTA	Y	N	Y	N	N	N	P	N	2	1	1	N	N	N	N	Y	N	N	Y	N	N	N
37	Nandini	670894	25y	F	460	RTA	Y	N	N	Y	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
38	Veena	670898	8y	F	461	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
39	Parvathamma	670897	24y	F	462	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
40	Saroja R Dannieal	671259	58y	F	491	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
41	Kannappa	790581	24y	M	474	RTA	Y	N	Y	N	N	Y	P	N	3	4	4	Y	Y	Y	N	Y	Y	N	N	Y	N	N
42	Venkatachalapathi	671277	35y	M	494	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
43	Manjunath	671492	23y	M	513	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
44	Krishnappa	733470	50y	M	157	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
45	Padmavathi	671551	35y	F	516	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
46	Devraj	680146	40y	M	489	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	Y	N	N	N	N	N	N
47	Muniyappa	671569	45y	M	520	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
48	Jayanna	671572	55y	M	521	RTA	N	N	N	N	N	N	P	N	4	5	6	N	Y	N	N	N	N	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
49	Shiraj pasha	671788	48y	M	532	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
50	Devamma	671787	55y	F	533	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
51	Nagarajathnamma	671786	45y	F	534	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
52	Saraswathi	672159	73y	F	547	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
53	Nagaraj singh	676286	23y	M	226	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
54	Jayaram	672169	36y	M	549	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
55	Suleman	672440	24y	M	566	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
56	Rajappa	682307	45y	M	112	RTA	Y	N	Y	N	N	Y	P	N	1	1	1	Y	N	Y	N	Y	Y	N	N	N	N	N
57	Nagesh	667043	55y	M	212	RTA	Y	N	Y	N	N	Y	A	N	1	1	1	Y	Y	Y	N	Y	Y	N	Y	N	N	N
58	Bhavana	672789	8y	F	593	RTA	Y	N	N	N	N	N	P	N	4	5	6	N	N	N	N	Y	N	N	N	N	N	N
59	Naresh	673066	18y	M	603	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
60	Shankar	719825	40y	M	434	RTA	Y	N	Y	N	N	N	A	N	1	1	1	Y	Y	Y	N	Y	Y	N	N	N	N	N
61	Satish	670837	18y	M	452	RTA	N	N	Y	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
62	Hemaraj	673141	22y	F	5	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
63	Mustafa	708326	40y	M	137	RTA	Y	N	N	N	N	N	P	Y	3	5	6	Y	N	N	N	N	N	N	N	N	N	N
64	Manjunath	673413	5y	M	17	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
65	venkataramappa	673428	63y	M	19	RTA	N	N	Y	Y	N	N	P	N	3	4	4	N	Y	N	N	N	Y	N	N	N	N	N
66	Varshika	673730	10y	F	42	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
67	Pavan	673737	27y	M	43	RTA	Y	N	Y	N	N	N	P	N	2	4	3	N	N	N	N	N	Y	N	N	N	N	N
68	Swarna	673732	35y	F	44	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
69	Vijaiya	673736	45y	F	45	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
70	Muniyappa	674282	45y	M	84	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
71	Narayanamma	674480	45y	F	98	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
72	Ponmari	674336	53y	F	95	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
73	Srinivas	674616	30y	M	114	RTA	N	N	Y	N	N	Y	P	Y	3	5	6	N	N	N	N	N	N	N	N	N	N	N
74	Paramesh	713714	45y	M	501	RTA	Y	N	Y	N	N	N	P	N	2	3	4	Y	N	N	Y	N	N	N	Y	N	N	N
75	Venkatamma	675206	65y	F	151	RTA	Y	N	Y	N	N	N	A	N	4	5	6	Y	N	N	Y	Y	N	N	N	Y	N	N
76	Venkateshappa	670560	55y	M	437	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
77	RAGHAVENDRA	669916	18Y	M	399	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
78	Shankarappa	674900	38y	M	132	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
79	Gohar	674986	40y	M	136	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
80	Sadananda reddy	670816	45y	M	448	RTA	Y	N	Y	N	N	Y	A	Y	4	4	5	Y	N	N	N	N	N	N	N	N	N	N
81	Muniramappa	674990	70y	M	139	RTA	Y	N	Y	N	N	N	P	N	2	4	4	N	Y	N	N	Y	Y	N	N	N	N	N
82	Baskhar	672168	45y	M	548	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
83	Enayath	674988	30y	M	138	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
84	Mahesh	675341	22y	M	161	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
85	Vinay Kumar	674146	24y	M	203	RTA	Y	N	Y	N	N	N	P	N	4	5	6	N	Y	N	N	N	N	N	N	N	N	N
86	Ali	734164	35y	M	208	RTA	Y	N	N	N	N	N	P	N	3	4	5	Y	Y	N	N	N	N	N	N	N	N	N
87	Venkatesh	676508	25y	M	241	RTA	Y	N	Y	N	N	N	A	N	2	4	4	N	N	Y	N	N	Y	N	N	N	N	N
88	Rajendra	676550	30y	M	243	RTA	N	N	N	N	N	N	P	Y	3	5	6	N	N	N	N	N	N	N	N	N	N	N
89	Ganesh	679498	34y	M	447	RTA	Y	N	N	N	N	N	P	N	4	4	5	Y	N	N	N	Y	N	N	N	N	N	N
90	Ashok kumar	678098	22y	M	347	RTA	Y	N	Y	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
91	Papanna	676702	75y	M	260	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
92	Gowri	686885	40y	F	270	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
93	Raja lakshmi	687445	45y	F	271	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
94	Nagraj	676892	28y	M	272	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
95	Venkateshappa	674605	55y	M	111	RTA	Y	N	Y	N	N	Y	A	N	2	4	1	Y	N	Y	N	Y	Y	N	Y	Y	N	N
96	Parnja	677140	14y	M	285	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
97	Manjunath	681323	24y	M	48	RTA	Y	N	N	N	N	Y	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
98	Nanjegouda	674596	50y	M	109	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
99	Shafulla Khan	677234	50y	M	297	RTA	Y	N	Y	N	N	N	P	N	4	5	6	N	Y	N	N	N	N	N	N	N	N	N
100	Satish	670827	21y	M	451	RTA	Y	N	Y	N	N	N	P	N	1	1	1	Y	N	Y	N	N	Y	N	Y	Y	N	N
101	Ravi kumar	677739	22y	M	318	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
102	Aravind kumar	578045	20y	M	343	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
103	Rangaswamy	678076	35y	M	345	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
104	Naveen	682229	26y	M	95	RTA	N	N	N	N	N	Y	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
105	Ramaswamy	678427	60y	M	361	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
106	Shakti prasad	678425	13y	M	363	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
107	Srinivaspur	678436	45y	M	366	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
108	Amarnath	678438	27y	M	365	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
109	Ramesh	678484	25y	M	370	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
110	Shrinivas	680481	32y	M	524	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
111	Byanna	677115	55y	M	286	RTA	Y	N	N	N	N	N	A	N	1	3	2	Y	N	N	N	Y	N	N	Y	N	N	N
112	Munegouda	679218	20y	M	431	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
113	Mohammed zuberulla	673087	33y	M	607	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
114	Suresh	679772	22y	M	462	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
115	Narasimha murthi	680057	40y	M	483	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
116	Prameelamma	676558	30y	F	245	RTA	Y	N	Y	N	N	N	P	N	3	4	4	Y	N	N	N	Y	N	N	N	N	N	N
117	Gopal	676579	40y	M	246	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
118	Sunil	674610	20y	M	113	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
119	Abdul rouf	674901	50y	M	131	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
120	Harish	680448	23y	M	521	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
121	Ramesh	680450	24y	M	522	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
122	Shabeer ahmed	672770	28y	M	589	RTA	Y	N	Y	N	N	N	P	Y	2	3	3	Y	Y	N	N	N	N	N	N	N	N	N
123	Shrinivas	680485	26y	M	526	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
124	Ramakrishna	682302	38y	M	109	RTA	Y	N	Y	N	N	N	P	N	2	3	3	Y	Y	N	N	N	N	N	N	N	N	N
125	Ramesh	681108	35y	M	21	RTA	Y	N	Y	N	N	Y	P	Y	4	5	5	N	N	N	N	N	N	N	N	N	N	N
126	Nagaraj	681120	24y	M	23	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
127	Upenra	681305	23y	M	44	RTA	N	N	N	N	N	Y	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
128	Laxminarayana	680396	51y	M	506	RTA	Y	N	Y	N	N	N	A	N	1	1	1	Y	Y	Y	N	Y	N	N	Y	N	N	N
129	Vimal	681328	32y	M	49	RTA	Y	N	N	N	N	Y	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
130	Gajendra	671547	35y	M	515	RTA	N	N	Y	N	N	N	P	N	3	4	4	Y	Y	N	N	N	N	N	N	N	N	N
131	Subramani	681621	25y	M	71	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
132	Venkatesh reddy	682227	60y	M	94	RTA	Y	N	N	N	N	Y	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
133	Velumuruga	682235	22y	M	97	RTA	Y	N	Y	N	N	Y	A	N	2	4	4	Y	Y	N	N	N	N	N	N	Y	N	N
134	Gowtham	668628	12y	M	311	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
135	Gopalappa	682278	50y	M	103	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
136	Jayappa	682289	62y	M	62	RTA	Y	N	N	N	N	N	P	N	3	4	5	N	Y	N	N	N	N	N	N	N	N	N
137	Sonu	682295	23y	M	107	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
138	Akram pasha	676300	26y	M	277	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
139	Srinivasaiah	670869	63y	M	455	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
140	Vinay Kumar	673199	25y	M	7	RTA	Y	N	Y	N	N	Y	P	N	3	4	4	Y	N	Y	N	Y	Y	N	N	Y	N	N
141	Sandeep	682560	24y	M	114	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
142	Bodamma	682639	75y	M	118	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
143	Manjamma	6822633	30y	F	119	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
144	Narayanaswamy	682665	43y	M	126	RTA	N	N	N	N	N	N	P	N	4	4	6	N	N	N	N	N	N	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
145	Ramesh	682673	22y	M	127	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
146	Venkataraj	680497	34y	M	530	RTA	Y	N	Y	N	N	N	P	N	1	1	2	Y	N	N	Y	Y	N	N	N	N	N	N
147	Anjanappa	682979	28y	M	147	RTA	Y	N	Y	N	N	Y	A	N	2	4	4	Y	Y	N	N	Y	N	N	N	Y	N	N
148	Santosh	677469	27y	M	307	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
149	Gopi	683390	27y	M	167	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
150	Shashi	683401	50y	M	168	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
151	Ranganath	672739	22Y	M	586	RTA	N	Y	Y	N	N	N	P	N	2	3	5	Y	Y	Y	N	N	N	N	N	N	N	N
152	Venkateshappa	671556	26y	M	518	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
153	Prabhavathi	706143	35y	F	30	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
154	Narayanappa	706702	60y	M	46	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
155	Krishnamurthy	706395	40y	M	55	RTA	Y	N	Y	N	N	Y	P	N	3	3	5	N	N	N	N	Y	N	N	N	N	N	N
156	Rakesh	706397	22y	M	59	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
157	Pradeep kumar	706944	25y	M	61	RTA	N	N	Y	N	N	Y	P	Y	4	5	5	N	N	N	N	N	N	N	N	N	N	N
158	Manjunath	707031	28y	M	70	RTA	Y	N	Y	N	N	N	P	N	4	4	4	N	Y	Y	N	Y	N	N	Y	N	N	N
159	Arjun	707351	6y	M	84	RTA	Y	N	N	N	Y	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
160	Yunus pasha	675315	30y	M	155	RTA	N	N	Y	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
161	Ram krishna reddy	708900	45y	M	165	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
162	Yellappa	709302	30y	M	194	RTA	Y	N	N	N	N	Y	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
163	Shankara	709309	21y	M	197	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
164	Subramani	709318	22y	M	200	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
165	Shantamani	709597	48y	M	210	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
166	Anil	731686	31y	M	47	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
167	Darshan raj	723275	8y	M	32	RTA	Y	N	Y	N	N	N	P	N	4	4	4	Y	N	N	N	Y	N	N	N	N	N	N
168	Mani	730906	22y	M	453	RTA	Y	N	Y	N	N	Y	P	N	2	4	4	Y	Y	N	N	N	N	N	N	N	N	N
169	Narula khan	709931	24y	M	218	RTA	Y	N	Y	N	N	N	P	N	4	5	5	N	N	N	N	Y	Y	N	N	N	N	N
170	Erappa	736791	45y	M	342	RTA	Y	N	Y	N	N	N	A	N	1	1	1	Y	Y	N	Y	N	Y	N	Y	N	N	N
171	Krishnappa	710127	32y	M	240	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
172	Ramachandrappa	710500	27y	M	262	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
173	Venugopal	724561	27y	M	104	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
174	Gopala krishna	710805	35y	M	279	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
175	Eyanth	710824	28y	M	281	RTA	Y	N	N	N	N	N	A	N	2	3	4	N	Y	N	N	N	N	N	N	N	N	N
176	Nagaraja	679163	45y	M	420	RTA	Y	N	Y	N	N	N	A	N	1	1	1	Y	N	N	N	Y	Y	N	N	N	N	N
177	Zabiulla	710908	20y	M	293	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
178	Geetha B L	711515	36y	F	336	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
179	Akkammal	711810	44y	F	352	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
180	Masappa	711806	35y	M	355	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
181	Muninarayanappa	682306	38y	M	111	RTA	Y	N	Y	N	N	Y	P	N	3	4	4	Y	N	N	N	Y	N	N	N	N	N	N
182	Kupendra	712097	27y	M	382	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
183	Kiran	706107	17y	M	27	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
184	Shankara	724634	47y	M	110	RTA	Y	N	Y	N	N	N	A	N	1	1	1	Y	Y	N	N	Y	Y	N	N	N	N	N
185	Ramesh	665996	35y	M	133	RTA	Y	N	Y	N	N	N	P	N	4	4	5	Y	N	N	N	Y	N	N	N	Y	N	N
186	Vijaya gouda	712687	30y	M	422	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
187	Mukesh N	710893	27y	M	292	RTA	Y	N	Y	N	N	N	P	N	3	4	4	Y	N	N	Y	N	Y	N	N	N	N	N
188	Sriram	721268	28y	M	521	RTA	Y	N	Y	N	N	N	A	N	3	4	5	Y	N	Y	Y	N	Y	N	N	N	N	N
189	venkataramappa	665567	50y	M	10	RTA	Y	N	Y	N	N	N	A	N	2	3	3	Y	Y	N	N	Y		N	N	N	N	N
190	Gowramma	715667	50y	F	31	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
191	Venkatesh	714478	45y	M	35	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
192	Subharayappa	715024	55y	M	55	RTA	Y	N	Y	N	N	N	P	N	2	6	3	N	Y	N	N	N	N	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
193	Thirumalappa	715259	45y	M	92	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
194	Rajappa	716004	30y	M	158	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
195	Abdul rashad	716228	55y	M	173	RTA	Y	N	Y	N	N	N	P	N	4	5	3	N	N	N	N	N	Y	N	N	N	N	N
196	Bhaskar	716314	30y	M	178	RTA	Y	N	Y	N	N	N	P	N	4	5	3	N	N	N	N	N	Y	N	N	N	N	N
197	Nagaraj	742085	48y	M	99	RTA	Y	N	Y	N	N	N	P	N	2	4	4	Y	Y	N	Y	N	N	N	N	N	N	N
198	Ramachandra reddy	716557	40y	M	190	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
199	Velu	716654	22y	M	207	RTA	Y	N	N	N	N	N	P	Y	4	5	6		N	N	N	N	N	N	N	N	N	N
200	Nayaz pasha	716669	22y	M	213	RTA	Y	N	N	N	N	N	P	N	3	5	6	N	N	N	N	N	N	N	N	N	N	N
201	Asaithabi	716685	59y	M	217	RTA	N	N	N	N	N	N	P	N	4	5	6	N	Y	N	N	N	N	N	N	N	N	N
202	Kanappa	665629	50y	M	107	RTA	Y	N	Y	N	N	Y	P	N	4	5	6	Y	N	N	N	Y	Y	N	N	N	N	N
203	Balaji	717308	24y	M	256	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
204	Venkateshappa	731663	50y	M	44	RTA	Y	N	Y	N	N	Y	P	N	3	4	5	Y	Y	N	N	N	N	N	N	N	N	N
205	Venkalachalapathi	718216	25y	M	318	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
206	Srinivas	718239	25y	M	319	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
207	Ravi kumar	718542	26y	M	344	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
208	Gunashegaran	712126	50y	M	384	RTA	Y	N	Y	N	N	N	P	N	3	4	4	Y	N	Y	N	Y	Y	N	N	N	N	N
209	Dodda subba reddy	718860	70y	M	369	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
210	Noorjan	719245	35y	F	399	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
211	Munivenkatappa	719579	35y	M	412	RTA	Y	N	Y	N	N	N	A	N	1	2	1	N	N	Y	N	Y	N	N	Y	N	N	N
212	Prabhakar	678869	53y	M	393	RTA	Y	N	Y	N	N	N	P	N	2	4	5	Y	Y	Y	N	Y	Y	N	N	N	N	N
213	Naveen T K	709935	30y	M	225	RTA	Y	N	Y	N	N	N	A	N	1	1	5	Y	Y	N	N	Y	N	N	N	N	N	N
214	Chandrappa	673122	35y	M	610	RTA	Y	N	Y	N	N	N	P	N	2	3	3	Y	N	Y	Y	N	N	N	N	Y	N	N
215	Manoharan	720547	20y	M	479	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
216	Lakhmamma	720655	32y	F	485	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
217	Ramappa	668634	55y	M	314	RTA	Y	N	Y	N	N	N	A	N	3	3	3	Y	N	N	N	Y	Y	Y	N	N	N	N
218	Madesh	721661	30y	M	546	RTA	Y	N	N	N	N	Y	P	Y	4	4	6	N	N	N	N	N	N	N	N	N	N	N
219	Nilakanth	731360	25y	M	21	RTA	Y	N	Y	N	Y	N	A	N	2	3	3	Y	Y	N	N	Y	Y	N	N	N	N	N
220	Bahadur	722240	45y	M	616	RTA	Y	N	Y	N	N	N	A	N	1	1	1	N	Y	Y	N	Y	Y	N	N	N	N	N
221	Shashidhar	722498	28y	M	631	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
222	Anil	722573	17y	M	637	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
223	Anjaneya gouda	722917	24y	M	12	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
224	Amaravathi	730257	42y	F	423	RTA	Y	N	Y	N	N	N	A	N	2	3	3	Y	N	N	N	Y	Y	N	Y	N	N	N
225	Venkatesh	717566	22y	M	273	RTA	Y	N	Y	N	N	N	P	N	3	4	4	Y	N	N	N	N	Y	N	N	N	N	N
226	Raju sharma	727312	30y	M	250	RTA	Y	N	Y	N	N	N	P	N	2	3	3	Y	N	N	N	Y	Y	N	Y	N	N	N
227	Suresh kumar	724635	35y	M	111	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
228	Gunaz begum	724852	59y	M	121	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
229	Gangulamma	725302	40y	M	141	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
230	Shivanna	726131	55y	F	181	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
231	Suresh	726139	20y	M	183	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
232	Gangappa	726346	60y	M	195	RTA	Y	N	Y	N	N	N	P	N	4	4	4	N	N	N	N	Y	Y	N	N	N	N	N
233	Mamata	726650	22y	F	212	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
234	Baghyamma	726673	51y	F	215	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
235	Asma	726919	20y	F	230	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
236	Munirama	728938	25y	M	340	RTA	Y	N	Y	N	N	N	P	N	3	4	4	Y	N	N	N	Y	N	N	N	Y	N	N
237	Chandra shekar	727313	28y	M	252	RTA	Y	N	Y	N	N	N	P	N	3	4	4	N	N	N	N	Y	Y	N	N	N	N	N
238	Syed amjad pasha	727343	28y	M	254	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
239	Shashidhar	741776	35y	M	81	RTA	Y	N	Y	N	N	N	P	N	3	4	5	Y	N	N	N	Y	N	N	N	Y	N	N
240	Narayanaswamy	727652	45y	M	275	RTA	Y	N	Y	N	N	N	P	N	3	4	5	N	N	N	N	Y	Y	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
241	Muniraju	677160	32y	M	287	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
242	Ramesh	728301	25y	M	314	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
243	Shadulla	709949	30y	M	233	RTA	Y	N	Y	N	N	N	A	N	4	3	3	Y	Y	N	N	N	N	N	N	N	N	N
244	Shantamma	732046	40y	F	74	RTA	Y	N	Y	N	N	N	P	N	3	4	5	Y	Y	N	N	Y	N	N	N	Y	N	N
245	Chand pasha	730150	35y	M	402	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
246	Sankappa	720539	70y	M	476	RTA	Y	N	Y	N	N	Y	A	N	1	1	1	Y	Y	Y	N	Y	Y	N	Y	N	N	N
247	Byrappa	718833	30y	M	366	RTA	Y	N	Y	N	Y	N	P	N	2	3	4	Y	Y	N	N	Y	Y	N	N	N	N	N
248	Narayanappa	730199	65y	M	410	RTA	Y	N	Y	N	N	N	P	N	2	3	4	N	N	N	N	Y	Y	N	N	N	N	N
249	Srikanth	730202	41y	M	413	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
250	Kumari	716548	48y	F	188	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
251	Venkatesh	730606	5y	M	439	RTA	Y	N	Y	N	Y	N	P	N	3	4	6	N	N	N	N	Y	N	N	N	N	N	N
252	Santamma	730602	60y	F	440	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
253	Kenchappa	730605	72y	M	441	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
254	Venkatesh	711842	48Y	M	359	RTA	N	N	N	N	N	N	P	N	3	5	6	Y	N	N	N	N	N	N	N	N	N	N
255	Narayanaswamy	731345	45y	M	17	RTA	N	N	N	N	N	Y	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
256	Renuka	731352	30y	F	20	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
257	Sarojamma	682969	40y	M	145	RTA	Y	N	Y	Y	N	N	A	N	4	5	6	Y	Y	N	N	Y	N	N	N	N	N	N
258	Sabrin taj	731368	23y	F	22	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
259	Narayanaswamy	731371	40y	M	24	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
260	Vijakanth	719592	27y	M	416	RTA	Y	N	Y	N	Y	N	P	N	3	2	3	Y	N	N	N	Y	N	N	N	N	N	N
261	Suguna	734882	18y	F	240	RTA	Y	N	Y	N	N	N	P	N	2	4	3	Y	Y	Y	N	Y	N	N	Y	N	N	N
262	Chetan kumar	731925	16y	M	56	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
263	Bharath	731911	38y	M	57	RTA	Y	N	Y	N	N	N	P	N	3	4	4	N	N	N	N	Y	N	N	N	N	N	N
264	praveen	731910	28y	M	58	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
265	Venkatesh reddy	727566	55y	M	263	RTA	Y	N	N	N	N	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
266	Harshita	732022	28y	F	71	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
267	Ashok	732042	22y	M	73	RTA	Y	N	N	N	N	Y	P	N	4	5	6	N	Y	N	N	N	N	N	N	N	N	N
268	Sajjad ahmed	721881	50y	M	553	RTA	Y	N	Y	N	N	N	A	N	2	3	3	Y	N	N	N	Y	Y	N	Y	N	N	N
269	Kumar reddy	732119	26y	M	82	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
270	Harish	732795	26y	M	117	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
271	Khadar sab	732817	53y	M	122	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
272	Srinivasa	712771	35y	M	432	RTA	Y	N	N	N	N	N	P	N	3	5	6	Y	Y	N	N	N	N	N	N	N	N	N
273	Chinnarayanappa	733518	60y	M	162	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
274	Bya reddy	733527	50y	M	164	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
275	Muninarayanappa	733706	60y	M	167	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
276	Mune gouda	734061	42y	M	192	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
277	Subadramma	734107	30y	M	198	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
278	Shameer	734165	35y	M	206	RTA	Y	N	Y	N	N	Y	P	N	3	3	4	N	Y	N	N	N	N	N	N	N	N	N
279	Reshma	713627	25y	F	496	RTA	Y	N	Y	N	N	N	P	N	3	3	4	Y	Y	N	N	N	N	N	N	N	N	N
280	Muneer	734163	35y	M	209	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
281	Ramaiah	734184	45y	M	211	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
282	Obala reddy	668803	75y	M	320	RTA	Y	N	Y	N	N	Y	A	N	2	3	3	Y	Y	N	N	Y	Y	N	N	N	N	N

MASTER CHART

SL NO	NAME	HOSP. NO	AGE	Sex	CT No.	BRIEF HISTORY						ALC	PUP	SYN	GCS			CT FINDINGS										
						T. INJ	L.O.C.	LCD IN	VOM	H.ACHE	SZS				E	V	M	FRCT	CON	C.ED.	EDH	SDH	SAH	IVB	MDS	PNEU	HYD	INF
283	Srinivas reddy	734865	35y	M	242	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
284	Venkaramappa	735644	42y	M	276	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
285	Vijay kumar	736004	25y	M	294	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
286	Nagendra	736379	32y	M	318	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
287	Varalaksmi	736387	5Y	F	319	RTA	N	N	Y	N	Y	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
288	Prem kumar	681613	18y	M	69	RTA	Y	N	N	N	Y	N	P	Y	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
289	Harish	741264	21y	M	53	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
290	Krishnamurthy	741534	54Y	M	54	RTA	Y	N	Y	N	N	N	P	N	2	4	4	N	N	N	N	Y	N	N	N	N	N	N
291	Rathnamma	741539	55y	F	55	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
292	Manjunath	731965	25y	M	64	RTA	N	N	N	N	N	N	P	N	4	5	6	Y	N	N	N	N	N	N	N	N	N	N
293	Srinivas gouda	741997	42y	M	87	RTA	N	N	Y	N	N	N	P	N	4	4	6	N	Y	N	N	N	N	N	N	N	N	N
294	Priyanka	742056	6Y	F	96	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
295	Shikhamani	710808	50y	F	265	RTA	Y	N	Y	N	N	N	P	N	4	4	4	Y	N	N	N	Y	N	N	N	N	N	N
296	Zulekha	742055	6y	F	108	RTA	Y	N	N	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N
297	Selvan	742391	38y	M	121	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
298	Sowmya	742775	13y	F	142	RTA	N	N	N	N	N	N	P	N	4	5	6	N	N	N	N	N	N	N	N	N	N	N
299	Vijay kumar	742863	43y	M	151	RTA	Y	N	Y	N	N	N	A	N	1	1	1	N	Y	N	N	N	N	N	N	N	N	N
300	Renuka	743416	19y	F	172	RTA	N	N	Y	N	N	N	P	Y	4	5	6	N	N	N	N	N	N	N	N	N	N	N

SL NO	NAME	HOSPITAL NO	AGE	SEX	IP/OP	CT SCAN NO	DATE	ADDRESS

				BRIEF HISTORY
TYPE OF INJURY	LOSS OF CONSCIOUSNESS	LUCID INTERVAL	VOMITTING	HEDEACHE

			PAST HISTORY
SEIZURES	BLEED OR DISCHARGE FROM ORIFICES	ANY OTHER	

PERSONAL HISTORY	OTHERS	FAMILY HISTORY		
ALCOHOL			BUILD	MENTAL STATUS

GENERAL EXAMINATION		VITAL EXAMINATION			
PUPILLARY REACTION	ASSOCIATED OTHER INJURIES	BP	PULSE	RESPI RATE	TEMP

OTHER SYSTEMS	GCS			
	EYE OPENING	MOTOR RESPONSE	VERBAL RESPONSE	FRACTURES

CONTUSIONS	CEREBRAL OEDEMA	EXTRA DURAL BLEED	SUB DURAL BLEED

CT FINDINGS		
SUB ARACHNOID BLEED	INTRA PARENCHYMAL BLEED	INTRA VENTRICULAR BLEED

MASS EFFECT	**PNEUMOCEPHALUS**	**HYDROCEPHALUS**	**INFARCTS**	**OTHER FINDINGS**