Computed Tomography of Head Trauma

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Abstract

"Prompt recognition of treatable injuries is critical to reduce mortality and CT of the head is the cornerstone for rapid diagnosis[2]. Follow up assessment using CT is frequently necessary to detect progression and stability of lesions and evidence of delayed complications and sequelae of cerebral injury, which can determine whether surgical intervention is necessary".

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 haemorrhage from normal brain, impairs MRI's ability to detect fractures and acute haematomas, 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 fossalesions"[4]

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Introduction

"Immediate and instantaneous death following cranial trauma occurs due to unpreventable primary brain injuries. However, death occurring within 24hrs of craniocerebral trauma can be averted by timely institution of diagnostic and therapeutic measures that could prevent secondary brain insults."

"Sir Godfrey Housfield described CT in 1973 and thereby spawned an imaging revolution"[1]

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Aim and Objectives

"To describe various spectrums of haemorrhages those occur in head trauma

with aid to CT."

Review of Literature

Based on a principle first described in 1917 and proved by an Austrian mathematician J. **Radon** that "a three dimensional object could be reproduced from an infinite set of all its projections, **G.N.Hounsfield** developed a technique with the help of a computer and called it Computerized Axial Tomography or CAT scanning. The first clinical prototype EMI head scanner (Mark I) was installed in early 1972 at Atkinson Morley's Hospital, London".

Jennet and Bond in 1975 proposed a practical scale to assess the outcome of severe brain damage and divided the outcome of these patients into death, vegetative stage, severe disability, moderate disability and normal.

The CT findings were divided into three groups - Minor, Moderate and Major and attempted to correlate them with severity of trauma based on the clinical status.

Minor:

- Local areas of edema

Moderate:

- Edema up to 1/3 of cerebral hemisphere and mild shift of midline structures.
- Localized contusion.

Major:

- Severe edema more than ½ of cerebral hemisphere.
- Hemorrhage anywhere.
- Pronounced midline shift.

They also identified the entity known as delayed traumatic cerebral hematoma.

Scotti et al in the same year stated that, overlap in the three groups of "acute, sub acute and chronic SDH occurred due to difference of speed of resorption of extravasated blood for each patient, difficulty in obtaining precise information on initial trauma and rebleed in an old hypodense SDH"[16]. In initial era of CT, usually all SDH were confirmed angiographically but with the later scanner with improved resolution, CT became the mainstay of SDH diagnosis.

Dublin, French et al[17](1977) did a retrospective analysis and concluded that:

a) CT has an accuracy approaching 100% in diagnosis of intra and extra cerebral collections ofblood.
b) Angiography is no longer needed but for isodense subdural hematoma.
c) The temporal course and CT density of subdural hematoma does not always correlate because of re-bleed into chronic subdural haemorrhage. Hence, instead of describing subdural hematoma as acute, subacute and chronic, they should be classified as hyper, iso andhypodense.
d) Early detection of intraventricular hemorrhage is possible and prompt drainage can improvesurvival.
e) Improvement of intracerebral hemorrhage should be diagnosed on changes in mass effect.

Barathamand **Dennyson**in 1972 reported the development of delayed intracerebral haematoma in complicated cases of head injury. The age distribution of these patients resembled that of SDH and frequently both conditions coexisted. This pointed to the possibility of similar etiological factors operating in the production[25].

Lipper et al put forth that both extra and intra axial delayed traumatic intracerebral haemorrhage (DTICH) were commoner than previously thought and were due to failure of regulation of blood flow[26]. Thedelayed extradural collections are due to diffuse brain injury, relief of tamponade effect (post surgical) and reaccumulation of fluid at site of original hematoma. These delayed collections, according to them were associated with poor outcome.

Studies indicating predictive usefulness of CT in traumatic brain injuries were published, few of which are illustrated below. **Levi L Guiburd JN et al** in the same article in 1990 pointed out the poor prognosis associated with deeper lesions of DAI[28]. They using the GCS and CT findings classified brain injury into severe (GCS <9), moderate (GCS 9-12) and minor (GCS 13-15) types.

Kido DK, Cox C et al in 1992 showed clear-cut correlation of Glasgow coma score and Glasgow coma outcome score after head injury, with CTlesionsize regard less whether lesion was intra or extra axial. Lesion that was more than 4100 cumm had two fold higher risk and poor outcome than those with abnormal scans[29].

Suzanne Laughlin, Walter Montanerain their article in 1998 mentioned that "CT is the primary procedure for evaluating intracranial complications of acute head injury. Access is relatively easy in spite of the ventilators, monitoring equipment, and traction devices that these patients often need. Scan times are relatively short, and the images are very sensitive for acute hemorrhage and cerebral edema. Excellent bony detail makes CT the best modality for assessing fractures of the skull base, calvarium, and facial bones"[35].

Physics of Computed Tomography

"A thin cross section of the head, a tomographic slice, is examined from multiple angles with a thin pencil beam of X-ray. The transmitted radiation was counted by scintillation detector, which was fed into the computer for analysis by a mathematical algorithm and reconstructed as a Tomographic Image. The physics can be discussed under the followingheadings"[38].

Data Acquisition[38]

Data gathering techniques have developed in stages. These stages have been called "Generations".

1. 1st-generation: "Translate - Rotate, one detector"
2. 2ndgeneration: Translate –"Rotate, multiple detectors"
3. 3rdgeneration: Rotate –"Rotate"
4. 4thgeneration: Rotate –"Fixed"
5. "Other various geometries"

First Generation (Original EMI Scanner)

It employed a pencil beam of X-ray and a single detector. "The X-ray tube detector movements were both linear and rotatory (usually termed translate-rotate motion)."
A five view study of the head took about 25-30 minutes.

Second Generation (Translate-Rotate)

- Pencil beam of X-ray was replaced by a Fan-shapedbeam.
- Numberofdetectors increasedfrom1-30.
- Because of multiple detectors,the total linear movements werereduced.

Hence the time required for the CT Slice ranged from 10-90 seconds.

Third Generation (Rotate-Rotate)

- Here only rotation motion is required, the linear motion being completely eliminated.
- Scan time is 4-9seconds.
- Multiple detectors, up to 700 "are aligned along the arc of a circle, whose centeris the X-ray tube focal spot."
- Both Xenon ionization chamber and scintillation crystal detectors are used.
- Number of scan slices in each projection are equal to the no. of scan lines.

Fourth Generation (Rotate fixed)

Here "the detectors form a ring that completely surrounds the patient. The detectors do not move and the X-ray tube rotates in a circle inside the detector ring, the X-ray beam is collimated to form a fan - beam."

- Number of detectors may be more than2000.
- When the X-ray tube is on, the exposed detectors are read.
- One principal disadvantage of fan-beam is that it produces scattered radiation.

This can be controlled by the collimators of individual detectors, which absorb scatter radiation.

Image Reconstruction

"In Computed Tomography, a cross sectional layer of the body is divided into several tiny blocks and then each block is assigned a number proportional to the degree that the block attenuated the X-ray beam." The individual blocks are called "Pixels'" in a single plane and "Voxels" in its entire volume.

To assign a number to each pixel the amount of X-ray attenuation (x) has to be calculated. As the numbers of pixels are in terms of thousands, the calculations become complex and have to be solved with the help of algorithms in the computer (An algorithm is a mathematical method for solving a problem). "These algorithms attempt to solve the equations as rapidly as possible without compromising the accuracy."

The following are the three mathematical methods of image reconstruction,

1. "Back projection"
2. "Iterative methods"
3. "Analytical methods."

Pathophysiology and Mechanisms of Trauma

"The knowledge of pathophysiology and mechanisms involved in trauma provides an insight to the response of body to injury and this knowledge in turn forms a framework for logical therapy. Head injury asatermisusedtoencompass all injuries to the scalp calvarium and brain. Understanding the mechanisms underlying brain trauma, their basic pathology, and their imaging manifestations is therefore essential for the practicingradiologist"[47].

"The magnitude and distributionofatraumatic brain lesiondependsontheshape of the object causing trauma, the force of impact and whether the head is in motion at the time of injury. Finally the total injury produced by impact depends not only on the mechanical damage but also on the complex interaction of path physiological events that follow."

PATHOPHYSIOLOGY:

"The manifestations of head trauma can be divided into primary and secondary lesions."

"Primary lesions are those that occur at the time of trauma as a direct result of traumatic force.Secondary lesions occur as a consequence of primary lesions, usually as a result of mass effect or vascular compromise. This division is clinically important because secondary lesions are often preventable, whereas primary injuries, by definition, have already occurred by the time the patient arrives in the emergency room."

Primary injuries can be classified as *focal* or *diffuse.*

"Focal brain injuries include cerebral contusions, intracranial haemorrhages and epidural and subdural haematomas".

"Diffuse injury is typically called diffuse axonal injury (DAI). DAI occurs deeper in the brain than focal injuries and has been shown to be caused by shear forces[48,49]. DAI is more prevalent than previously believed and is associated with neurological deficit[50]."

Secondary insults are "physiological changes that occur following injury that furthers the progression of damage. Neural cells are vulnerable to ischemic events following primary injury[51]. Evidence shows that global and regional circulation in the brain is also impaired after primary injury[52,53]. Therefore, physiological insults such as hypoxia and hypotension lead to secondary injury and cell death. These two variables are independently associated with rise in mortality andmorbidity"[54].

1. Linear fracture:

Linear or fissure fractures of the skull vault imply a considerable degree of force deforming the cranium but spread over a wider surface area. Linear fractures constitute approximately 80% of all fractures of skull, with 50% occurring in the mid portion. The development of serious infective complications depends mainly on associated tearing of the dura and the overlyinglaceration of the scalp. Most dramatic complication of a linear fracture is an epidural hematoma, resulting from tearing of the underlying meningeal vessels.

Object has to be sufficiently large (greater than 2 square inches) so that skull penetration does not occur and sufficiently small so that contact phenomenaare not distributed widely over the surface of head.

2. Depressed fracture:

When one part of the skull has been driven inward, so that its outer table is at or below the level of the surrounding inner table, it is called a depressed fracture. More focused and intense contact forces are present on a smaller impact surface. This exceeds the elasticity of skull thus allowing skull perforation. The calvarium fails to rebound and bony fragments are displaced into the brain. The inner tableisusuallydisplacedmorethantheoutertable.

Such fractures may be either simple or compound. The simple fracture, in which the covering scalp remains intact, is uncommon and usually occurs in children. Compound fracture is one in which skull shatters into several fragments which are driven into the cranial cavity. This is usually associated with dural tear, which is a portal of entry for infection. Half of the depressed fractures occur in frontal area.

3. Basilarfracture:

"These are due to direct impact to the area of skull base (occiput, mastoid, supraorbital), or to energy transmitted to skull base from facial or mandibular impact, ortoremote effects of skull impact. Inthelastcase, stress waves that propagate fromthe impact point or changes in skull shape due to impact areusual."

The basilar fractures include

- Fractures involving the paranasal sinuses and if associated with tear of the dura cause meningitis andpneumocephalus.
- Fractures of the sellaturcica, which later may cause endocrinal abnormalities.
- Petrous bonefractures
  - Ongitudinalfractures
  - ransverse 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 (raccooneyes).

4. DiastaticFractures:

Traumatic separation of the cranial sutures results in a so-called diastatic fracture. Diastatic skull fractures are relatively common, especially in children. The width of normal sutures varies between different individuals. Typically, a width of 2 mm is

agreed to be the upper limit of normal, and this width is most frequently encountered in the lambdoid region. The key radiologic finding of a diastatic fracture is asymmetry between the two sides or between two different sutures. Although asymmetry can be seen with fracture of the coronal and lambdoidal sutures, the comparison is lacking when the sagittal suture is involved. Sutural diastasis can occur with or without an associated fracture. It often represents a continuation of a fracture line originating remote from the suture.

5. ComminutedFractures:
The majority of depressed skull fractures are associated with some degree of communication (i.e. multiple bone fragments). Comminute fractures are usually the result of severe blunt or penetrating trauma in which a large amount of energy is dissipated over a short period of time and within a small area. A child's skull is blessed with increased calvarial plasticity, so communited fractures (and fractures in general) are uncommon in children. Comminuted fractures are oftencompound.

Configuration:
The typical subdural haematoma has a crescentic configuration that conforms to the unyielding calvarium laterally and the cerebral cortex medially. Once extravagated the blood extends through the continuous subdural space of the convexity and there is little focal expansion into the underlying cortex. A sharp medial "beak" is noted at the portion where haematoma extends into the anterolateral portion of the sylvian fissure between the frontal andtemporal opercular regions. The haematoma is diffuse, covering most of the cerebral hemisphere. Therefore it, produces a holo hemispheric, circumferential vector of force on the underlying cortex nearby ventricles, which is rarely duplicated by other more focal space occupying masses.

Atypical Configuration:
These are encountered when haemorrhage occurs in a patient with abnormal calvarium, subdural space or cerebral parenchyma. Thus, an acute SDH may be localized when extensive parenchymal damage compresses the subarachnoid space, preventing free flow of blood over convexity. When the collection is more localized, it may bulge into adjacent cortex, producing a lentiform configuration.

Lentiform configuration is also seen when the subdural space is loculated by fibrotic bands secondary to previous, trauma or inflammation. In these cases a small localized unilocular subdural collection may develop or when haemorrhage is more extensive a multilocular lesion can be seen. It is important to identify multilocular collections as direct surgical drainage of each portion of haematoma may be necessary.

Occasionally a chronic subdural haematoma is encountered which does not have classic crescentic configuration. These lesions are often massive, occupying more than half of hemicranium. They occur in elderly patients with severe atrophy. The medial border of the lesion is straight and areas of mixed attenuation are seen within the subdural haematoma, secondary to episodes ofhaemorrhage.

Density Pattern:
i) Temporal Changes:
For approximately 5-10 days after trauma, the majority of subdural haematomas are hyperdense relative to adjacent brain. The haematomas may increase in density over the first one to two days, owing to retraction of the clot and a resultant decrease in water content. Over the next several days to weeks the haematomas will decrease in density, passing through an isodense phase between 7^{th} and 21^{st} days. After this time haematoma is hypodense. This temporal sequence is however subject to many variables making

accurate aging of SDH problematic. Thus an acute SDH may be isodense or hypodense in patients with low packed cell volumes (Blood density is dependent on the protein fraction of haemoglobin molecule and therefore the hematocrit) or when there is admixture of blood and CSF in subdural collection. The aging of SDH is however of limited clinical importance as therapeutic decisions are made on the basis of patients clinical status and findings on CT (e.g. size of lesion and mass effect) and not theoretical age of thehaematoma.

ii) Layering Phenomenon:

The density in a subdural haematoma is often non-homogenous. Commonly a haematoma is seen with a hypodense anti-dependent portion that is sharply demarcated from a hyperdense dependent portion. This phenomenon also known as the "hematocrit effect", occurs after liquefaction of the clotted blood, usually in the sub-acute or chronic phase. The denser elements within the hematoma settle outinthe more dependent portion of the collection. The sharpness ofthis demarcationisdependent on the degree of movement of the patients head, prior to scanning. A well-demarcated layering phenomenon is seen most often in patients who have been in a superposition for some time. In more active or agitated patients the change from hypodensity to hyperdensity is moregradual.

ii) Rebleed in SDH:

With multiple episodes of haemorrhage, successive organization of each new hematoma leads to the formation of multiple membranes in the collection. On CT a large cresentic area of varying ages, are seen. In general, the more chronic (hypodense) collections are present at the medial and lateral margins of the haematomas and the denser, more acute haemorrhage occurs more centrally. Clinically recognition of this pattern is important since a simple burr hole evacuation may not allow for complete removal of haematoma. A more extensive surgical procedure involving stripping of multiple membranes is often necessary for adequate removal.

C) Unusual Location ofSDH:

i) Posterior Fossa SDH: These are very rare.

Etiology*:* Due to rupture of,

- Dural sinuses
- Bridging veins
- Secondary to trauma.

CT finding in posterior fossa SDH are variable. A central or eccentric, single lesion or bilateral lateral lesions may be present. Its configuration is usually crescentic, but occasionally lentiform collection, indistinguishable from an EDH, is seen. When the lesion extends superiorly it has a sharp lateral margin where it abuts the tentorium. In the other hand supratentorial infratemporal lesions have sharp medial margins.

ii) Interhemispheric SubduralHematoma

"These are usually unilateral and are produced by bleeding from bridging veins between thesuperiorsagittal 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[64].

CT **Findings***: "*About 20% of lesions contain sufficient hemorrhage to be visible on CT. Most common finding is the small, petechial hemorrhages at the gray-white junction of the

cerebral hemispheres or corpus callosum. Ill defined areas of decreased attenuation on CT may occasionally be seen with non-hemorrhagic areas."

Cortical contusions:

"Contusions are typically superficial foci of punctuate or linear haemorrhages thatoccuralonggyralcrests.Theyareinducedbyb rainstrikingonanosseousridgeless often a dural fold and occur when differential acceleration / deceleration forces are applied to the head. Focal contusions may also be associated with a depressed skull fracture. Petechial cortical contusions tend to coalesce into larger hemorrhagic foci and often become more evident 24 to 48 hours after the initial trauma."

Location:

"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 percent 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."

Cerebral Herniations:

"The cranial cavity is functionally divided into compartments by combinations of bony ridges and dural folds. Cerebral herniations are caused by mechanical displacement of brain, cerebrospinal fluid and blood vessels from one cranial compartment to another. Brain herniation are the most common secondary effect of expanding intracranial masses. Various types of brain herniations are encountered which are as below"[67].

Table 1: Types of Cerebral Herniation

Subfalcine
Transtentoria
"Descending"
"Ascending"
"Transalar (trans-sphenoidal)"
"Descending"
"Ascending"
"Tonsillar"
"Miscellaneous e.g, transdural/ transcranial"

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 parahippocampal 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 posturing, coma, progressive loss of brain reflexes and respiratory arrest may ensue if not intervened.

Diffuse Cerebral Edema:

"Massive cerebral edema with intracranial hypertension is among the most life-threatening of all secondary traumatic lesions."

Although gross enlargement of one or both hemispheres may occur within hours after the traumatic insult, severe cerebral edema usually takes 24 to 48 hours to develop. Diffuse posttraumatic brain swelling is due to increased intravascular blood volume, increased brain water content, or both. Mortality rate in these cases approaches 50%.

"Cerebral edema may be cytotoxic, interstitial or vasogenic in origin. In the immediate post-traumatic interval, cerebral edema is vasogenic, reflecting breakdown of blood-brain barrier with leakage of intravascular contents into and around areas of damaged tissue."

"The pathogenesis is poorly understood, but appeared to be due to loss of normal cerebral autoregulation. The cerebellum and the brain stem are usually spared and may appear hyperdense relative to the cerebral hemispheres. Often the Falx and cerebral vessels appear dense mimicking acute sub-arachnoidhemorrhage."

CT Findings:

- "Generalised mass effect with effacement of sulci, suprasellar and quadrigeminal plate cisterns and compression of theventricular system."
- "Decrease attenuation of the brain parenchyma with loss of gray-white differentiation."
- "Effacement of the brain stem cisterns indicates severe mass effect and may herald impending trans-tentorialherniation."

The 'reversal' sign is similar. It is caused by diffuse hypodensity of the cortex and deep white matter compared to the normal density of the thalamus, brainstem, and cerebellum, all of which are perfused by the posterior circulation.

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 extracranialinjuries.

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 beconsidered.
5) *History of seizures:* This is important because, with seizures occurs a rise in intracranial pressure which may further deteriorate the patients' condition.
6) *ENTbleed:* These points to the presence of basal skull fractures.

Table 2: TCDB Diagnostic Categories of Abnormalities Visualised on CT Scanning

Diffuse Injury Grade	CT Appearance
Diffuse injury - Grade I	"No visible intracranial pathology seen on CT"
Diffuse injury - Grade II	"Cisterns are present with midline shift of < 5 mm and / or lesion densities are present. Tissue tear in brain parenchyma."
	"No high mixed density region greater than 15 cc. May include bone fragments and foreign bodies"
Diffuse injury - Grade III	"Diffuse brain swelling, cisterns compressed or absent midline shift < 5 mm No high / mixed density lesion> 15 cc."
Diffuse Injury - Grade IV	"Diffusebrainswelling,cisterns arecompressed / absent, shift of midline structures > 5 mm, High / mix density lesion of more than 15 cc."

Methodology

Source of Data

"The present study was carried out in patients with head trauma, referred to Krishna hospital, Krishna institute of medical sciences, Karad, in the Department of Radio-diagnosis during a period from November 2017 To April 2019."

Sample Size

"The study comprised a total of one hundred patients with head injury referred to Krishna hospital,Karad."

Inclusion Criteria

1) "Patients of all age groups with head trauma."
2) "Head trauma that has occurred within 24 hours."
3) "Patients with head trauma treated as in-patients."

Exclusion Criteria

1) "Cranial trauma during childbirth."
2) "Patients with non traumatic intracranial bleed."

Plan of Study

A complete clinical history of the patients was noted on proforma, which included, age sex, type of injury. The type of trauma was further classified into Road traffic accidents, Falls, Assaults, industrial accidents and miscellaneous. Follow up of Patients during their hospital stay was performed.

After initial resuscitation, severity of the craniocerebral injury was graded with the help of "Glasgow Coma Scale" (GCS).

Eye opening

Spontaneous	4
To sound	**3**
To pain	2
Never	1

Verbal Response:

Oriented	5
Confused Speech	4
Inappropriate Words	3
Incomprehensible sounds	2
Never	1
Motor response:	
Obeys commands	6
Localises pain	5
Normal flexion (withdrawal)	4
Abnormal flexion	3
Extension	2
Nil	1

Rating:

Mild head injury ..... GCS of 13-15

Moderate head injury..... GCS of 9-12

Severe head injury ..... GCS of 8

EQUIPMENTS

The patients were scanned using **Somatom emotion 16 slice.**

- Matrixsize-512
- Slice thickness - 4.8mm, 2.4 mm
- K.V-80tol30.
- MAS -50 to270

CT PROTOCOL

"Patients were examined with CT scanner in the supine position.Proper immobilization and positioning of head was achieved in all patients.Unco-operative patients were sedated by giving I.V Diazepam (5-20 mg). The Gantry tilt was given in the range of ± 0-20 degrees, so as to parallel the scan plane to the orbito-meatalline.Bone algorithms & wide window settings were studied to visualise the various craniocerebral changes."

Statistical Methods

"Rates, ratios and percentages of different diagnosis and outcome made by Computed tomography will be computed and compiled with the help of SPSS (version 20) software. Chi square test will be used for comparison of CT findings of different variables and P value will be calculated."

Results

"A total of hundred patients of sustained head trauma with positive findings on CT scan were included in the present study. Out of 100, 70 patients were male and 30 were female."

Table 3: Sex wise Distribution in head trauma

Sex	No. of Cases	Percentage
Male	70	70
Females	30	30
Total	**100**	**100.00**

Table 4: Sex wise and age wise Distribution in head trauma

Age group (years)	Male	Female	Total
0 to 10	9.00	4.00	13.00
11 to 20	11.00	4.00	15.00
21 to 30	21.00	8.00	29.00
31 to 40	17.00	4.00	21.00
41 to 50	5.00	2.00	7.00
51 to 60	3.00	5.00	8.00
More than 61	4.00	3.00	7.00

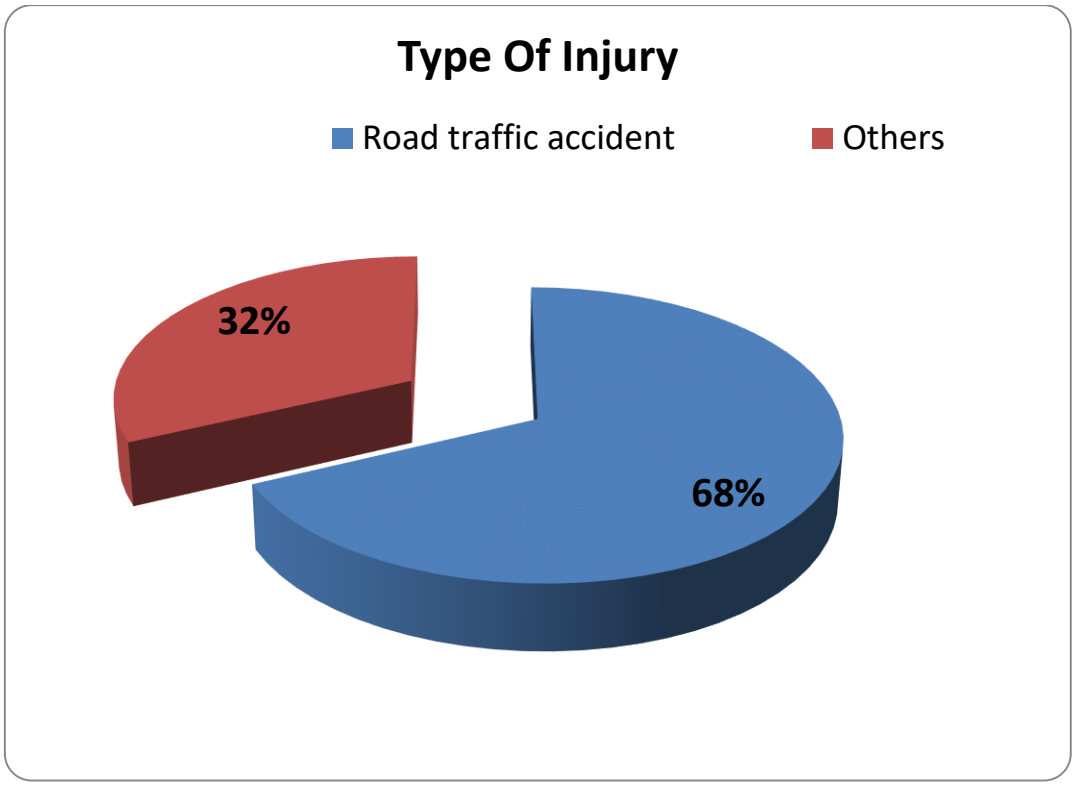

Figure 3: Incidence of Different Modes of Injury

Table 5: Grading of Head Injury Based on GCS Score

Type of Head Injury	No. of Cases / Percentage
Mild (13 –14)	30
Moderate (9-12)	26
Severe (< 8)	44
Total	100.00

According to the study, "cases with severe head injury with GCS score of <8 were the commonest accounting for 44% of all cases followed by cases with mild head injury with GCS score of 13-14 accounting for 30% cases and with moderate head injury were least common accounting for 26%."

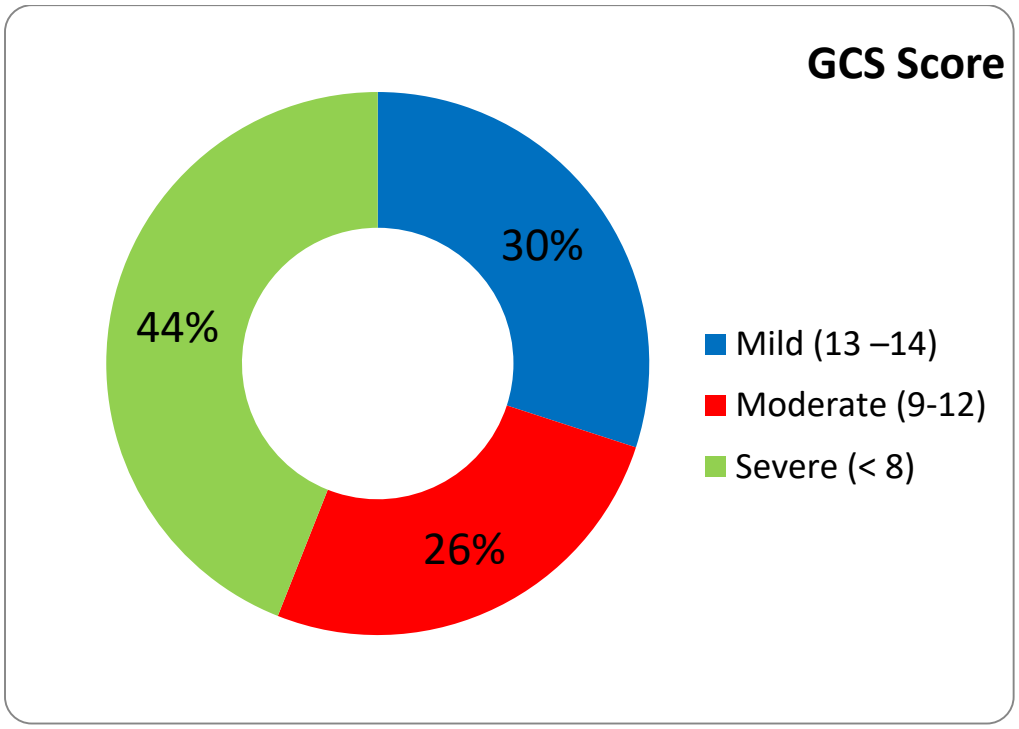

Figure 4: Grading of Head Injury Based on GCS Score

Table6:Incidence of various haemorrhages in patients who expired
In our study, 24 patients expired.

Hematomas	Percentage
EDH	8.33
SDH	16.66
ICH	33.33
SDH with secondary cerebral Oedema	**41.66**

According to the present study, **subdural haematoma with associated cerebral oedema-no of cases:10 (41.66%)** was the most common lesion noted in patients who expired followed by intracerebral hemorrhage (no of cases:8)(33.33%). **Only large sized ICH (Vol~>40cc) were associated with mortality.**

10 out of 14 cases with SDH related deaths had cerebral oedema likely responsible for higher mortality than ICH. Extradural haemorrhage (no of cases:2) (8.33%) was the least common lesion noted in these patients.

Table 7: Mortality on basis of GCS

Glasgow Coma Score	No. of cases	Death	Percent
More than 8	44.00	18.00	40.90
9 to 12	26.00	5.00	19.23
13 to 14	30.00	1.00	3.33

According to the study, "poor outcome was noted with a GCS score of <8.Patients with GCS score of <8 had a mortality of 40.90% followed by 19.23% in patients with GCS of 9-12 and 3.33% in patients

with 13-14 GCS score. **Only 1 case out of 30 with GCS 13-14 category expired likely due to patient suffered other body parts injuries.**Outcome is therefore poor with low GCS score."

"X^2=25.49 and p<0.001, which shows that the relationship is significant".

IMAGES

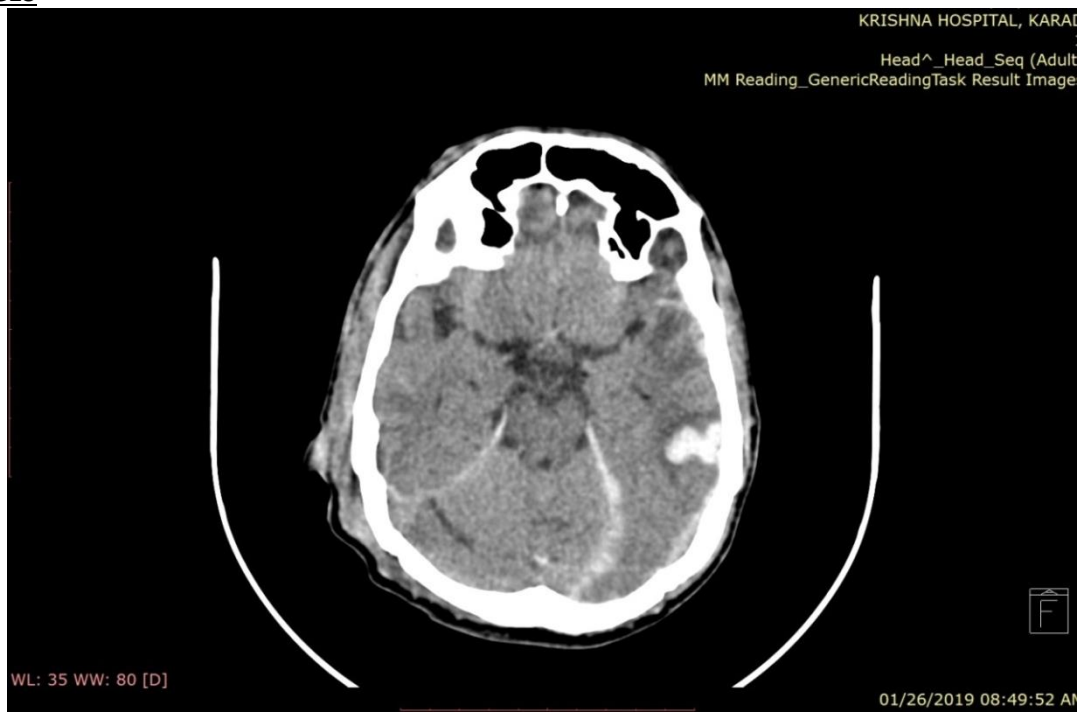

Image 1: "Axial CT Section in 26 year old man with history of RTA showing Hemorrhagic contusion in left temporal lobe"

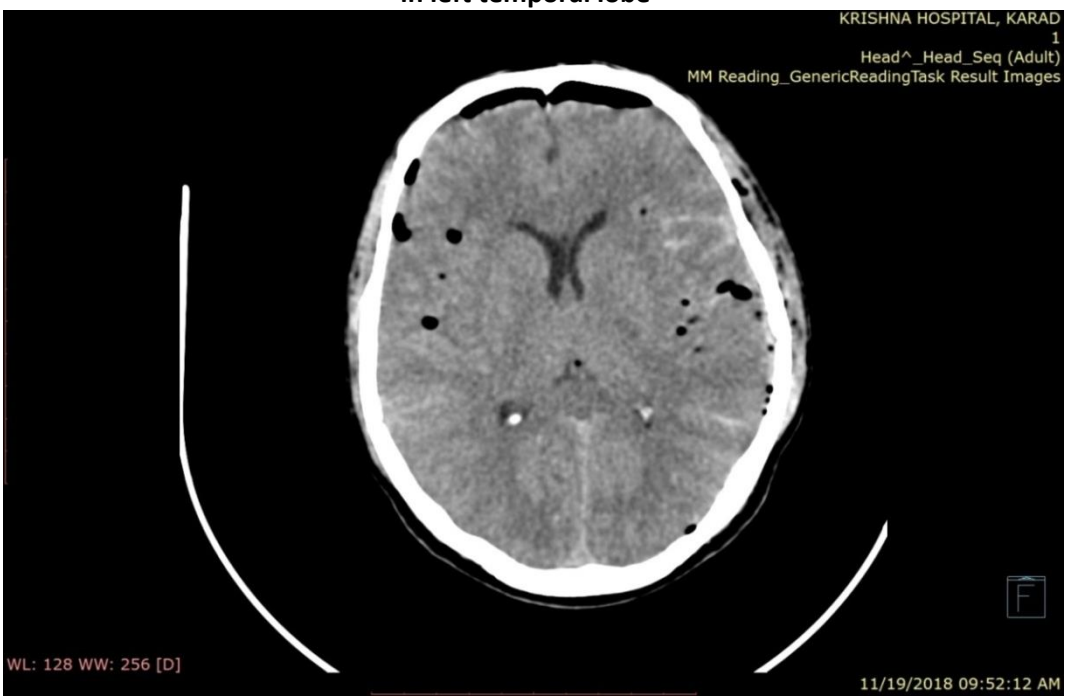

Image2: "Axial CT Section in 40 year old man with history of RTA showing Pneumocephalus"

Discussion

In the present study patients in the age group of 21-30 years formed the bulk of the study. Study by Ogunseyinde AO et al[77] also stated that "head injury was common in patients younger than 35 yrs. Fary Khan et al[78] (2003) in their study mentioned that peak incidence of traumatic brain injuries were between 15-35 years age group and MasihSaboori et al[75] (2007) reported a mean age of 29yrs for patients of head injury. By the studies it is noted that head injury is seen commonly in socially and economically productive age group of the population and hence has an impact on the financial aspect of the family."

"Contusion and cerebral oedema was found to be the commonest intracranial lesion detected on CT accounting for 43% and 45%, respectively in the present study. Dublin[17] also reported similar observation (40%)."

Table 8: Comparative studies of Incidence of Subdural Hematoma

Author	SDH (%)
MasihSaboori et al	34.70
Igun GO	60.00
Ogunseyinde AO	28.70
Present study	39

"Subdural hematoma was found to the commonest type of hemorrhage noted accounting for 39% in the present study. Incidence reported in other studies were MasihSaboori et al[75] (34.7%), Igun GO (60%), Ogunseyinde AO et al[77](28.7%)."

"Intracerebral bleed accounted for 21% of lesions in the present study, whereas a slightly higher incidence of 26.3% was noted in the study conducted by Ogunseyinde AO et al[77]."

"Intraventricular hemorrhage was the least common lesion noted with an incidence of 3 % in the present study. Le Roux PD etal[82](1992) and Lee J.P et al[83] (1991) in their studies had stated that IVH is noted in 1% to 5% of all patients with head injury. Traumatic IVH is thus relatively uncommon and usually reflects severeinjury."

"Extradural hematoma was found to be associated with an overlying fracture in 93.75% of cases in the present study. Igun GO[79] reported 100% association of EDH with an overlying fracture. A blow to the calvarium resulting in fracture of the adjacent bone causes displacement of dura away from the inner table of skull resulting in damage to underlying vessel thus causing extradural hematoma."

"The commonest hemorrhage found in patients who expired was subdural hemorrhage with an incidence of 58.33 %. This can be attributed to the more severe impact of trauma to cause the hemorrhage. SDH is associated with worse outcome because it generally is caused by high velocity injuries resulting in more primary brain injury.Cooper et al[84] in his study stated that mortality due to subdural hematoma was between 35% to 50%".

"Intracerebral hematoma was seen in 33.33 % of patients who expired. It is also another major cause of deaths after subdural hemorrhage."

"EDH was seen in only 8.33% of patients who expired. Bricolo A.P et al[85] and Smith HK et al[86] in their studies stated that mortality with EDH is approximately 5%. Since EDH is usually associated with low velocity injury, it results

in little primary injury to brain and causes poor outcome only if the expanding hematoma is allowed to compress the brain."

Conclusion

"Head injury causes more deaths and disability than any other neurologic condition before age 40 and is most commonly caused by road traffic accidents. Males are worst affected due to head injuries."

"Management of brain injury can be done by neuroimaging techniques which can provide some of the most important diagnostic, prognostic, and path physiological information. Imaging modalities can help assess intracranial hemorrhage, fractures, and other structural lesions. Beside the correct diagnosis itself the time to establish a diagnosis above all has a crucial impact on successful management and good outcome of these patients.Computed tomography is a simple, inexpensive, highly effective and safe imaging modality and provides the ability to rapidly evaluate patients with acute head injuries."

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