"MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"

By

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE
IN
RADIODIAGNOSIS

Under the
Guidance of

Dr. DEEPTI NAIK,
PROFESSOR
DEPT. OF
RADIODIAGNOSIS

DEPARTMENT OF RADIODIAGNOSIS, SRI DEVARAJ URS MEDICALCOLLEGE, TAMAKA, KOLAR-563101

2023

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. DEEPTI NAIK**, Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M. D. DEGREE IN RADIODIAGNOSIS**", the examination to be held in June 2023 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

Date:

Place: Kolar

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

Postgraduate in

Radiodiagnosis Sri

Devaraj Urs Medical

College

Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATIONAND RESEARCH, TAMAKA, KOLAR, KARNATAKA

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled "**MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"** is a bonafide research work done by **Dr. ALURU VENKATA SAI NIKHILENDRA REDDY,** under my direct guidance and supervision at Sri Devaraj Urs Medical College, Kolar**,** in partial fulfilment of the requirement for the degree of "**M.D. IN RADIODIAGNOSIS"**.

Date:

Place: Kolar

Dr. DEEPTI NAIK
Professor
Department of Radiodiagnosis
Sri Devaraj Urs Medical College
Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATIONAND RESEARCH, TAMAKA, KOLAR, KARNATAKA

CERTIFICATE BY THE HOD

This is to certify that the dissertation entitled **"MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"** is a bonafide research work done by **Dr. ALURU VENKATA SAI NIKHILENDRA REDDY,** under my supervision at Sri Devaraj Urs Medical College, Kolar**,** in partial fulfilment of the requirement for the degree of "**M.D. IN RADIODIAGNOSIS"**.

Date:

Place: Kolar

Dr. ANIL KUMAR SAKALECHA

Professor & HOD

Department Of Radiodiagnosis

Sri Devaraj Urs Medical College
Tamaka, Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATIONAND

RESEARCH, TAMAKA, KOLAR, KARNATAKA

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT AND PRINCIPAL

This is to certify that the dissertation entitled **"MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"** is a bonafide research work done by **Dr. ALURU VENKATA SAI NIKHILENDRA REDDY** under the direct guidance and supervision of **Dr. DEEPTI NAIK,** Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of university regulation for the award "**M.D. DEGREE IN RADIODIAGNOSIS**"**.**

Dr. ANIL KUMAR SAKALECHA

Professor & HOD

Department Of Radiodiagnosis,

Sri Devaraj Urs Medical College,

Kolar Tamaka, Kolar

Date:

Place: Kolar

DR. SREERAMULU P. N

Principal,

Sri Devaraj Urs Medical College,

Tamaka,

Date:

Place: Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION

AND RESEARCH TAMAKA, KOLAR, KARNATAKA

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethical committee of Sri Devaraj Urs Medical College,

Tamaka, and Kolar has unanimously approved

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

Post-Graduate student in the subject of

RADIODIAGNOSIS at Sri Devaraj Urs Medical College, Kolar

To take up the Dissertation work entitled

"MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP"

to be submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND

RESEARCH, TAMAKA, KOLAR, KARNATAKA,

Member Secretary

Sri Devaraj Urs Medical College,

Kolar–563101

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION

AND RESEARCH TAMAKA, KOLAR, KARNATAKA

COPY RIGHT

I hereby declare that Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

Date:

Place: Kola

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH

Tamaka, Kolar 563103

Certificate of Plagiarism Check

Title of the Thesis/Dissertation	MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW-UP.
Name of the Student	Dr. ALURU VENKATA SAI NIKHILENDRA REDDY
Registration Number	20RD1024
Name of the Supervisor / Guide	Dr. DEEPTI NAIK
Department	RADIO-DIAGNOSIS
Acceptable Maximum Limit (%) of Similarity (PG Dissertation /Ph.D. Thesis)	10%
Similarity	7%
Software used	Turnitin
Paper ID	1990663303
Submission Date	10/01/2023

Signature of Student

Signature of Guide/Supervisor

Professor
Dept. of Radio-Diagnosis
R.L.J. Hospital & Reaserch Centre
Tamaka, Kolar-563 101

HOD Signature

Professor & Head
Dept. of Radio-Diagnosis,
Sri Devaraj Urs Medical College
Tamaka, KOLAR-563 101

University Librarian

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

Coordinator UG and PG Program

Co-Ordinator,
UG&PG Program ,Faculty of Medicine,
Sri Devaraj Urs Medical College ,
Tamaka, Kolar- 563103

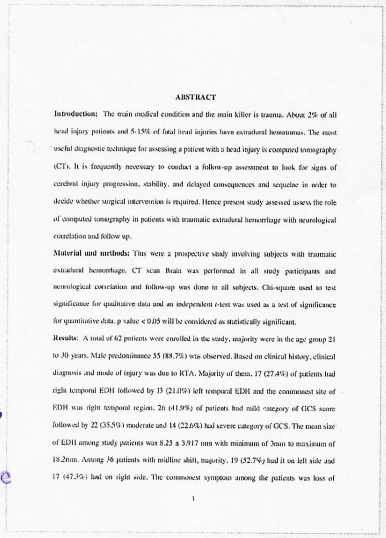
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	Dr. Nikhilendra Reddy
Assignment title:	PG dissertation
Submission title:	Multidetector computed tomography evaluation in traumati...
File name:	Dr._Nikhilendra_reddy_-_thesis_after_plagiarism_reduction.d...
File size:	3.89M
Page count:	90
Word count:	16,310
Character count:	87,482
Submission date:	10-Jan-2023 03:23PM (UTC+0530)
Submission ID:	1990663303



ABSTRACT

Introduction: The main medical condition and the main killer is trauma. About 2% of all head injury patients and 5-15% of fatal head injuries have extradural hematomas. The most useful diagnostic technique for assessing a patient with a head injury is computed tomography (CT). It is frequently necessary to conduct a follow-up assessment to look for signs of cerebral injury progression, stability, and delayed consequences and sequelae in order to decide whether surgical intervention is required. Hence present study assessed assess the role of computed tomography in patients with traumatic extradural hemorrhage with neurological correlation and follow up.

Material and methods: This were a prospective study involving subjects with traumatic extradural hemorrhage. CT scan Brain was performed in all study participants and neurological correlation and follow-up was done in all subjects. Chi-square used to test significance for qualitative data and an independent *t*-test was used as a test of significance for quantitative data. p value < 0.05 will be considered as statistically significant.

Results: A total of 62 patients were enrolled in the study, majority were in the age group 21 to 30 years. Male predominance 55 (88.7%) was observed. Based on clinical history, clinical diagnosis and mode of injury was due to RTA. Majority of them, 17 (27.4%) of patients had right temporal EDH followed by 13 (21.0%) left temporal EDH and the commonest site of EDH was right temporal region. 26 (41.9%) of patients had mild category of GCS score followed by 22 (35.5%) moderate and 14 (22.6%) had severe category of GCS. The mean size of EDH among study patients was 8.23 ± 3.917 mm with minimum of 3mm to maximum of 18.2mm. Among 36 patients with midline shift, majority, 19 (52.7%) had it on left side and 17 (47.3%) had on right side. The commonest symptom among the patients was loss of

1

10/1/23

10/1/23

Document Viewer

Turnitin Originality Report

Processed on: 10-Jan-2023 15:24 IST
ID: 1990663303
Word Count: 16310
Submitted: 1

Multidetector computed tomography evaluation ... By Dr. Nikhilendra Reddy

Similarity Index	Similarity by Source	
7%	Internet Sources:	6%
	Publications:	4%
	Student Papers:	2%

include quoted | include bibliography | excluding matches < 14 words | mode:
quickview (classic) report | print | refresh | download

<1% match (Internet from 11-Dec-2020)
https://radiopaedia.org/articles/extradural-haemorrhage?lang=us

<1% match (Internet from 24-Nov-2020)
https://www.ncbi.nlm.nih.gov/books/NBK518982/

<1% match (student papers from 15-Jan-2017)
Submitted to Mansoura University on 2017-01-15

<1% match (student papers from 06-Aug-2022)
Submitted to Mansoura University on 2022-08-06

<1% match (Internet from 04-Oct-2022)
https://pjmhsonline.com/2018/july_sep/pdf/925.pdf

<1% match (student papers from 23-May-2021)
Submitted to University of Suffolk on 2021-05-23

<1% match ()
Febin Ross Joseph Jeyamony,. "Role of Magnetic Resonance Imaging Diffusion Weighted and Apparent Diffusion Coefficient Evaluation in Brain Infarction", 2020

<1% match (Internet from 20-Aug-2021)
https://msjonline.org/index.php/ijrms/article/download/2122/2698

<1% match ()
Tariq, Muhammad, Mian, Mushtaq ahmad, Ali, Muhammad, Ayub, Shahid, Dad Khan, Waseem, Daud Khan, Sohail. "Outcome of the Traumatic Extradural Hematoma on the Basis of Size of Extradural Hematoma", 'Pakistan Society of Neurosurgeons', 2021

<1% match (Internet from 14-Jul-2019)

ACKNOWLEDGEMENT

*I owe debt and gratitude to my parents **Sri. A. RAMIREDDY** and **Smt. A. SARASWATHI**, along with my sister **Dr. PRANITHA REDDY** and my brother-in-law **Dr. SIVAKARTHIK REDDY** for their moral support and constant encouragement during the study.*

*With humble gratitude and great respect, I would like to thank my teacher, mentor and guide, **Dr. DEEPTI NAIK,** Professor, Department of Radio-diagnosis, Sri Devaraj Urs Medical College, Kolar, for their able guidance, constant encouragement, immense help and valuable advices which went a long way in moulding and enabling me to complete this work successfully. Without their initiative and constant encouragement this study would not have been possible. Their vast experience, knowledge, able supervision and valuable advices have served as a constant source of inspiration during the entire course of my study. I would like to express my sincere thanks to **Dr. ANIL KUMAR SAKALECHA,** Professor and Head Department of Radio-diagnosis, Sri Devaraj Urs Medical College for, valuable support, guidance and encouragement throughout the study. I would also like to thank **Dr. RAJESWARI,** Assoc. Professor Department of Radio-diagnosis, **Dr. HARINI BOPAIAH,** Assoc. prof., Department of Radio-diagnosis, Sri Devaraj Urs Medical College for their wholehearted support and guidance.*

*I would like to thank **Dr. BUKKE RAVINDRA NAIK, Dr. DARSHAN A.V., Dr. RAHUL DEEP G., Dr. ANEES, Dr. DIVYA TEJA PATIL, Dr. YASHAS ULLAS L., Dr. AASHISH, Dr. CHAITHANYA, Dr. MONISHA, Dr. VARSHITHA** and all my teachers of Department of Radio diagnosis, Sri Devaraj Urs Medical College and Research Institute, Kolar, for their constant guidance and encouragement during the study period.*

I am extremely grateful to the patients who volunteered to this study, without them this study would just be a dream.

*I am thankful to my **postgraduates, especially Dr. REVANTH R.B., Dr. ARUN RAJKUMAR, Dr. MADAN KUMAR, Dr. SANDEEP REDDY, Dr. PRAVEEN , Dr. KALATHURU UHA SAI, Dr. LYNN JOY, Dr. MAHIMA KALE, Dr. SURYAKANTH ANNE, Dr. SIVA SIDDANTH and Dr. RISHI PRAJWAL** for having rendered all their co- operation and help to me during my study.*

*My sincere thanks to **Mr. SUNIL, Mrs. NASEEBA** along with rest of the computer operators.*

*I am also thankful to **Mr. RAVI, Mr. ALEEM** and **Mr. SUBRAMANI** with other **technicians** of Department of Radiodiagnosis, R.L Jalappa Hospital & Research Centre, Tamaka, Kolar for their help.*

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
MDCT	Multidetector Computed Tomography
EDH	Extra dural hematoma
CNS	Central nervous system
RTA	Road traffic accident
GCS	Glasgow coma scale
CT	Computed tomography
MRI	Magnetic Resonance Imaging
TBI	Traumatic Brain Injury
CBF	Cerebral Blood Flow
SDH	Subdural hematoma
CSF	Cerebrospinal fluid
TCDB	Traumatic Coma Data Bank

ABSTRACT

Introduction: Trauma is the major health problem and is a leading cause of death. Extradural hematomas occur in approximately 2% of all patients of head injuries and 5-15% of fatal head injuries. CT is the single most informative diagnostic modality in the evaluation of a patient with a head injury. Follow-up assessment is frequently necessary to detect progression and stability and evidence of delayed complications and sequalae of cerebral injury which can determine whether surgical intervention is necessary. Hence the present study assess the role of computed tomography in patients with traumatic extradural hemorrhage with neurological correlation and follow up.

Material and methods: This was a prospective study involving subjects with traumatic extradural hemorrhage. CT scan Brain was performed in all study participants and neurological correlation and follow-up was done in all subjects. Chi-square used to test significance for qualitative data and an independent *t*-test was used as a test of significance for quantitative data. p value < 0.05 will be considered as statistically significant.

Results: A total of 62 patients were enrolled in the study, majority were in the age group 21 to 30 years. Male predominance 55 (88.7%) was observed. Based on clinical history, clinical diagnosis and mode of injury was due to RTA. Majority of them, 17 (27.4%) of patients had right temporal EDH followed by l3 (21.0%) left temporal EDH and the commonest site of EDH was right temporal region. 26 (41.9%) of patients had mild category of GCS score followed by 22 (35.5%) moderate and 14 (22.6%) had severe category of GCS. The mean size of EDH among study patients was 8.25 ± 3.917 mm with minimum of 3mm to maximum of 18.2mm. Among 36 patients with midline shift, majority, 19 (52.7%) had it on left side and 17 (47.3%) had on right side. The commonest symptom among the patients was loss of consciousness 38 (61.3%). Majority 57 (92.0%) of patients had good recovery followed by 5 (8.0%) had moderate recovery. The size of EDH was significantly larger among patients with midline shift and patients with moderate recovery had significantly larger EDH compared to those with good

recovery.

Conclusion: Multidetector Computed Tomography (MDCT) was found to be a useful and adequate diagnostic tool in the evaluation of patients presenting with head injury.

Keywords: Road Traffic Accidents, Head Trauma, Epidural Hemorrhage

INTRODUCTION

INTRODUCTION:

In a rapidly developing country like India steady increase in urbanization and industrialization has resulted in an exponential growth of road transportation and subsequently there is a steady increase in road traffic accidents (RTA) and has been referred to as "silent epidemic RTA's" has become a daily occurrence taking an increased toll on human lives and limbs. Most of these patients are in their prime (2nd and 3rd decade of life) and therefore have a direct social and economic effect besides the emotional burden of suffering a lifelong debilitating loss of function. Neurotrauma in the current scenario is not only identified, but evaluated & quantified. Previously, the mainstay of diagnosis of intracranial traumatic lesions was at best clinical evaluation, plain roentgenograms of skull and cerebral angiography. [1]

The incidence of traumatic brain injuries is common among the 21 - 40 years of age group followed by 41 - 60 years age group and below 20 years age group. The incidence among the above 61 years age group is comparatively less.[1]

Skin, subcutaneous fibro-fatty tissue, galea aponeurotica, loose areolar connective tissue, and periosteum are the five layers that make up the scalp. The skin is attached to the nearby layer of thick fibro fatty tissue and has sebaceous and sweat glands. Within this second layer, hair follicles and neurovascular systems are securely locked in place. A paradigm shift over the past ten years is discernible, and a sense of urgency for prompt diagnosis and neuroimaging assessment of trauma is visible. [2,3]

The advent of CT & the recent influx of newer generations of MDCT have revolutionized the understanding of traumatic brain injury.[4] Head CT has eased diagnosis and paved way for assessment of classification based on etiology, pattern of injury in correlation with pathoanatomical distribution and CT scoring systems viz, Marshall CT score and Rotterdam CT scores have aided in prognosticating outcomes

in neurotrauma.[5] Preserving the patient's life and remaining neurological function is the primary goal in treating patients with craniocerebral trauma. Neuroimaging plays a vital role in optimal management of these patients which depends on early and correct diagnosis.[6]

In head injury, Computed Tomography is the single most informative diagnostic modality in the evaluation of a patient. Besides facilitating rapid implementation, it can demonstrate significant primary traumatic injuries including extradural, subdural, intracerebral hematomas, subarachnoid and intraventricular hemorrhages, skull fractures, cerebral edema, contusions and cerebral herniations. Contribution of CT is crucial to complete injury assessment and forms the basis of patient management.[1] Prompt recognition of treatable injuries is critical to reduce mortality and CT of the head is the cornerstone for rapid diagnosis.[7] Not only is exact pictorial depiction of the effect of head trauma possible, but CT has also furthered the understanding of pathophysiology of head trauma. Technically superior 3rd and 4th generation scanners have decreased the scan time significantly and simultaneously increased the accuracy with which small lesions of minimally differing attenuation can be imaged. CT is currently the procedure of choice over MRI because it is faster and more readily available and it more easily accommodates emergency equipment and can easily enable, the detection of blood during the acute phase.[8]

Inability to use life supporting ferromagnetic equipment, inability to acquire bone details and cost factors further makes MRI inferior to CT in the evaluation of craniocerebral trauma. CT is a quick, cost effective, non-invasive method to assess time and the extent of cerebral injury and is an essential aid to triage patients to observation, medical or surgical management.[9,10] Sequential CT in a subset of patients with traumatic brain injury has made way for preemptive therapeutic interventions, often

ameliorating the effect of trauma and translating to decreased morbidity and mortality. However, varying school of thoughts pertaining to necessity of repeat CT scans in these patients exist, some studies affirm the need of serial Head CTs [5,6,11] in the absence of clinical deterioration while others are opposed to the same. [9,10,11] Till date, particularly in the Indian Scenario there is no general consensus on the standardized number and frequency of the CTs to be done is available.[9] Role of CT in Diagnosis and management of traumatic brain injury (TBI) is crucial to improve patient outcomes. Computed tomography (CT) scan is the optimum tool for quick and accurate detection of intracranial hemorrhage in the initial stage, the guidelines on use of repeat CT differ among institutions. Given that the value of repeat CT in TBI management is still unclear, definitive evidence is needed in order to guide clinical decisions on routine advice of repeat CT to patients with TBI.[12] It can have a progressively worsening course and an early diagnosis and timely management are critical to its treatment. CT is a quick, cost effective, non-invasive method to assess time and extent of cerebral injury and is an essential aid to triage patients to observation, medical or surgical management. [12]

Since very few structured studies have been conducted till date, we conducted this study to evaluate and assess the role of computed tomography in patients with traumatic extradural hemorrhage with neurological correlation and follow up.

NEED FOR STUDY:

Craniocerebral trauma results in a substantial number of deaths and permanent disabilities around the world. Head injuries account a significant cause of death in younger age groups. Death occurring within 24 hours of craniocerebral injury can be averted by timely institution of diagnostic and therapeutic measures that could prevent secondary brain insults.

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

CT is a quick, cost effective, non-invasive method to assess time and extent of cerebral injury and is an essential aid to triage patients to observation, medical or surgical management. It has also made the neuroradiologist an essential member of the trauma care unit.

The aim of the present study was to evaluate and assess the role of computed tomography in patients with craniocerebral injury and to study the various craniocerebral changes that occur in trauma to head with aid of CT.

AIMS & OBJECTIVES

OBJECTIVES OF STUDY:

1. To evaluate the imaging findings of extradural hemorrhage on multidetector computed tomography.
2. To correlate the size of extradural hemorrhage and midline shift with neurological features of patient.
3. To evaluate the prognosis of patients with neurological deficits on follow up.

REVIEW OF

LITERATURE

REVIEW OF LITERATURE:

Historical Review:

In the late 19th century, in the year 1895 **Wilhelm Conrad Roentgen**, Professor of Physics and Acting Rector of the University of Wurzburg discovered x-rays - the first imaging modality. He received the first Nobel Prize in Physics in 1901 for that discovery. [13]

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. [13]

Hounsfield along with Ambrose in 1973 presented the first paper on "Computerized Axial Tomography (CAT) - A new method utilizing X-rays" at the 32nd Annual Congress of the British Institute of Radiology. It was a great leap for medical science.[13]

In order to read the transmission of x-rays through the skull at different angles, crystal detectors were utilized in a revolving scanning device that was described. Using this information, absorption values of the material contained in the head were calculated and shown on a computer to exhibit a series of cranial slice images that illustrated the transverse anatomy of the brain. They claimed that their method was approximately 100 times more sensitive than a conventional x-ray equipment, allowing for the

detection of changes in soft tissues with nearly similar densities. The separation of tissues with barely different densities is made possible by the discovery of absolute values of the tissues' absorption coefficients. They afterwards received the name Hounsfield Units (H.U.).[13,14]

New, Scott et al, in 1974 reported the accuracy of CT scanning as proved by surgery and angiography. With experience they said that they were able to recognize extradural hematoma from intracerebral hematoma with relative ease. [14]

In a different publication, they discussed the CT findings of intracerebral hemorrhage and suggested that the partial volume effect brought on by large pixel sizes and low resolution makes it difficult to diagnose intracerebral hematomas.[14]

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. [15]

Baker in September 1975 stressed that CT would minimize hospitalization and more invasive and hazardous procedures by delineating normal brain. It helped clinicians at Mayo Clinic with diagnosis, prognosis and therapeutic regimen and thus affected the clinical practice of neurology and neuroradiology. [15,16]

Evans confirmed the Baker-presented facts in 1976. He reported that, since the introduction of this technique in their institute, air studies had diminished by 66%, cerebral angiography procedures by 34% and radionuclide brain scans by 29% in patients of various neurological diseases. [17]

A comprehensive account of retrospective study of hundred cases of head trauma by CT was published in April 1976 by **Merino- De Villasante J. and Taveras.**

[18] They concluded that

1. CT should be the first neuroradiological procedure to be performed in head injury and should be of best possible quality.

2. They opined that there is a direct relationship between severity of clinical presentation and CT demonstration of abnormality.

3. They divided their CT findings- 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 1/3rd of cerebral hemisphere and mild midline shift.
- 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.[18]

Ambrose and Gooding et al in 1976 advocated sequential scans in patients who fail to improve or deteriorate after treatment or surgery and which also enable evaluation of chronic traumatic pathologies. [19]

Xenon CT was first used in the early 1970s and involves injecting xenon intracarotidly and then inhaling or injecting it. These scans were essentially maps of the brain's blood flow because xenon is spread throughout the circulation. [20]

Marion et al used this capacity to explore the heterogeneous nature of cerebral blood flow (CBF) and cerebral vasoreactivity. [20] This work changed the way in which CBF is viewed thereby giving insight to highly variable nature of CBF and wide variations of blood flow in relatively small areas of brain. Xenon CT thereby not only provides a graphic representation of perfusion status but also depicts the consequences of overlay aggressive hyperventilation and inadequate cerebral perfusion pressure (CPP).[20]

The tremendous impact of CT in the field of head injury was demonstrated when **Cordobes** et al in 1981 published a detailed comparison of results of observation in patients with EDH before and after the advent of CT scans.[21]

Acute, subacute and chronic stages of EDH were put forth by **Zimmerman and Bilaniuk** in 1982. They also described the 'Lucent Swirl Sign'. [22]

Baratham and **Dennyson** in 1972 reported the development of delayed intracerebral hematoma 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. [23]

Lipper et al put forth that both extra and intra axial delayed traumatic intracerebral hemorrhage (DTICH) were commoner than previously thought and were due to failure of regulation of blood flow. The delayed extradural collections are due to diffuse brain injury, relief of tamponade effect (postsurgical) and reaccumulation of fluid at site of original hematoma. These delayed collections, according to them were associated with poor outcome.[24]

The contribution of CT scanning in the early diagnosis of delayed traumatic

intracerebral hematomas was emphasized by **Diaz, Douglas et al** they said that the head is usually in motion at the time of trauma in patients who develop delayed traumatic intracerebral hematomas.[25]

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 CT hematoma size regardless whether lesion was intra or extra axial. [26]

In a comparative study of CT and MRI **Zimmerman et al** in 1988 classified head injury patients into three groups according to duration of examination from the time of injury - Acute (1 to 3 days), subacute (15 to 20 days) and chronic (one month to three years). In acute injuries, CT and MRI both demonstrated hemorrhagic lesions but only MR scan revealed coexisting chronic hematomas or hypothalamic infarctions. However, due to ease of use with other monitoring system, CT is the best technique for evaluating acute head injuries.[27]

Gentry LR, Godersky JC, Thompson B, Dunn VD in 1988 stated that 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. [28]

Vander Naalt J., Hew J.M. et al in 1999 agreed that CT was most frequently

used imaging technique in patients with acute head injury in which it provides accurate detection of parenchymal and subarachnoid hemorrhages. MRI on other hand is more sensitive in detection of smaller lesions / non-hemorrhagic contusions. [29]

NICE guidelines in 2003 recommended that patients who have sustained a head injury and present with GCS less than 13 at any point since the injury or GCS equal to 13 or 14 at 2 hours after the injury or with a suspected open or depressed skull fracture should have CT scanning of the head immediately requested. [30]

Susan Mayor in 2003 mentioned that CT imaging of the head is recommended as the primary investigation of choice for the detection of acute, clinically important brain injuries. Professor Yates explained: "We are recommending early diagnosis in a more proactive way, using CT scanning rather than skull x ray and observation. This means we won't miss clinically important head injury, and we can feel comfortable in sending patients with normal CT home".[31]

Suzanne Laughlin, Walter Montanera in 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. [32]

Reed MJ, Browning JG, Wilkinson AG, Beattie T in 2005 emphasized that Skull x-rays can be abandoned in children aged 1-14 without a significant increase in admission rate, radiation dose per head injury or missed intracranial injury. We suggest

that routine skull x-ray have no place in emergency department for these children aged 1-14 years. [33]

Besenski N in 2002 stated that Computed tomography is currently the first imaging technique to be used after head injury, in those settings where CT is available. Extra-axial hematomas, parenchymal injury, and scalp and bone injuries can all be seen on a CT scan. Computed tomography is rapid and easily performed even in monitored patients. It is the imaging technique that is most pertinent for surgical lesions. Computed tomography is a suitable method to follow the dynamics of lesion development, giving an insight into the corresponding pathological development of the brain injury. [34]

Lolli V et al (2016) a review was done to talk about the basic traits of primary and secondary brain injuries. In summary, he said that imaging is crucial to the treatment of individuals with TBI. In the case of acute head trauma, CT is the imaging method of choice because it allows for the precise detection and subsequent treatment of vascular injuries, hydrocephalus, mass effect, and extra- and intra-axial hemorrhage. CT is effective in identifying secondary injuries as well, making it crucial for follow-up. Despite the absence of structural brain damage on CT, individuals with severe neurological impairment are only given MRIs in the acute situation. In subacute and chronic TBI, MRI is the preferred imaging technique, and it appears to be more accurate than CT at predicting outcome.[35]

Arfat M et al (2017) used a multi-detector computed tomography scanner to conduct a study to assess the epidemiological trends and severity of traumatic brain injury (TBI), at the Uttar Pradesh University of Medical Sciences in Saifai, Etawah, between December 2015 and May 2016, 61 patients with a mean age of 36 and a history

of serious head trauma visited the emergency room. Each individual was evaluated using 64 MDCT. This study found that people with various kinds and degrees of brain damage can benefit from MDCT. This study found that 65.57% of TBIs result from automobile accidents involving young adult men.[36]

Nithesh N and Ravindranand (2021) carried out a study to evaluate how multi-detector CT is used to evaluate patients who have suffered craniocerebral trauma. The authors came to the conclusion that MDCT is helpful in a wide range of fractures, traumatic consequences, and therapeutic choices.[37]

A retrospective observational study was conducted by **Kumar Cs et al (2017)** which included 100 patients admitted in King George hospital, Andhra Medical College, Visakhapatnam, Andhra Pradesh. A detailed clinical history, physical examination and CT scan was performed in all patients. Operative and post-operative findings for individuals who underwent surgery were recorded. The study demonstrated that early presentation with mild to moderate GCS has good clinical outcome with minimal disability.[38]

Repeat head CT scans are frequently performed on traumatic brain injury (TBI) patients to monitor any potential injury progression. With this insight **Nagesh M et al (2019)** conducted a retrospective analysis with the goal of determining whether patients with mild to moderate head injuries and an initial positive abnormal CT scan required routine repeat exams. The study comprised patients who presented to the emergency room between January 2016 and December 2017 with a Glasgow Coma Scale (GCS) score > 8 and an initial abnormal CT scan and underwent a repeat CT during their in-hospital medical treatment. The study's findings led researchers to draw the inconclusive conclusion that the utility of routine repeat head CT in medically treated head injury patients is debatable. The authors have made an effort to research the

numerous elements that may contribute to neurological decline, radiological decline, and/or the requirement for neurosurgical intervention. Lower GCS score at admission, abnormal INR, the presence of midline shift, effaced basal cisterns, and multiple lesions on first CT were all observed in this study to be substantially linked with the outcomes mentioned above.[39]

A prospective Study was carried out by **Jagdish P et al (2017)** Patients with clinically suspected head injuries who visited or were referred to Sardar Patel Medical College & Associate Group of Hospitals, Bikaner, between March 2016 and November 2016. 100 patients with clinically suspected head injuries participated in this study. They underwent multi-detector computed tomography (PHILLIPS BRILLIANS 64 SLICE MDCT SCAN) evaluation, and findings were, when appropriate, correlated with clinical data. The authors came to the conclusion that computed tomography is one of the comprehensive diagnostic techniques for precisely localizing the site of injury in acute craniocerebral trauma based on the study's findings. The precise lesion's early and fast detection by CT not only had a significant impact on the implementation of suitable treatment and timely surgical intervention, but it also assisted in predicting the final result.[40]

CT is single primary modality in evaluation of patients with acute head injuries. With these, a cross-sectional study was taken by **Ramanana Rao DV et al (2020)** to identify different clinico-radiological patterns of head injuries and to compare CT characteristics to clinical and surgical findings in head injury patients. The study covered the head injury and craniofacial trauma patients who had CT scanning. Patients on a ventilator and those scoring below a 6 on the Glasgow Coma Scale were eliminated. Siemens Somatom Emotion Duo dual slice CT was used to scan the patients. According to the study's findings, males between the ages of 21 and 40

experience head injuries most frequently, and patients older than 61 have a higher fatality risk. The most common kind of hematomas are Multiple Intracranial Traumatic Lesions (MICTLs).[41]

PHYSICS OF COMPUTED TOMOGRAPHY

A pencil-thin X-ray beam is used to evaluate the head's thin tomographic slice from various angles. The scintillation detector was used to count the radiation that was transmitted, and the data was then sent into a computer for processing by a mathematical procedure and reconstruction as a Tomographic Image. The following categories can be used to discuss the physics. [42]

Data Acquisition[42]

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

1) First generation: Translate - Rotate, one detector

2) Second generation: Translate - Rotate, numerous detectors

3) Third generation: Rotate - Rotate

4) Fourth generation: Rotate - Fixed

5) Other geometries

First Generation (Original EMI Scanner)

It used a single detector and an X-ray pencil beam. Both linear and rotatory movements were made by the X-ray tube detector (usually termed translate-rotate motion). It took about 25 to 30 minutes to analyze the head from five angles.[42]

Second Generation (Translate-Rotate)

- A beam with the shape of a fan replaced the X-pencil ray's beam.

- Number of detectors increased from 1-30.
- The number of detectors allowed for a reduction in the overall linear movements.

Hence the time required for the CT Slice ranged from 10-90 seconds. [42]

Third Generation (Rotate - Rotate)

- Here, only rotational motion is necessary; all linear motion is totally disregarded.
- Scan time is 4-9 seconds.
- Multiple detectors, upto 700 are aligned along the arc of a circle, whose centre is the X-ray tube focal spot.
- Scintillation crystal detectors and Xenon ionization chambers are both employed.
- Number of scan slices in each projection are equal to the no. of scan lines. [42]

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 than 2000.
- When the X-ray tube is on, the exposed detectors are read.
- Fan-primary beam's drawback is that it emits scattered radiation. This can be controlled by the collimators of individual detectors, which absorb scatter radiation.[42]

OTHER SCAN CONFIGURATIONS

Spiral CT

Recently, commercial scanners using the Slip Ring technology have been made accessible. With the help of this technology, the X-ray tube and detector assembly can rotate continuously in one direction without having to be wound up again. Data collection over an entire patient volume can be done in one breath if the patient is moved concurrently through the gantry. This was introduced in 1989 and is known as "Spiral or Helical Scanning." Its benefits include reducing issues caused by motion streaks, varying levels of inspiration from scan to scan, and the subsequent missed anatomic levels caused by motion.[43]

Additionally, lengthy examination times caused by breathing in between scans and interscan delays that prevent the completion of extended volume and contrast enhanced studies during the vascular enhancement phase of dynamic scanning will be resolved. Quantitative CT number investigations like determining the size of lung nodules and measuring bone mineral density are two more uses for spiral scanning in addition to dynamic scanning. A 3D image can be created with sophisticated software employing surface and volume rendering techniques. Another advantage is the availability of multidetector spiral CT scans. [44]

To achieve scan time of less than one second, one proposed solution is to use multiple X-ray tubes (about 20) positioned in a semicircular gantry. This way the scan time can be reduced to "16 m sec".[44]

Advantages of Spiral CT[45]:

- Ability to minimize motion artefacts.
- Decreased incidence of misregistration between consecutive axial slices.

- ❖ Reduced patient dose.
- ❖ Improved spatial resolution in Z-axis.
- ❖ Enhanced multiplanar or three dimensional renderings.

Disadvantage

- ❖ High cost
- ❖ Mechanical motion not completely eliminated.

Multi-slice Spiral CT[46]:

This transcends spiral CT's limitations. They utilize the rotate-only third-generation geometry with the additional dimension of several detector arcs. The dual-arc detector was part of the technology's initial implementation. [46]

Each rotation can yield several slices thanks to the multiple detector rows. Additionally, the gantry rotation speed is accelerated, increasing the overall scan speed. [46]

The dramatic reduction in scan time allows larger volumes to be scanned in the same time and the same volume in a much reduced time at narrower collimation leading to higher axial resolution. [46]

It has a promising application in CT angiography, CT chest, abdomen and also in cardiac CT with virtually motion free, high resolution images. [46]

Cardiovascular Computed Tomography (CVCT)

Here magnetic focusing and deflection of an electronic beam replaces X-ray tube motion. Scan time is "50 m sec".[46,47]

X-ray tubes

The diagnostic X-ray tube in modern fan beam devices has a revolving anode

and a significantly smaller focal point (0.6 mm). The substantial heat loading and heat dissipation characteristics of these tubes allow them to survive the extremely high heat loads produced when several slices are obtained quickly one after the other.[46,47]

Collimators

The X-ray beam is collimated at two points, one close to the X-ray tube and the other at the X-ray tube detectors. Perfect alignment between the two is essential. Collimator width varies from 1 mm to 10 - 15 mm. [46,47]

Detectors

The types of detectors used in CT scanner are:

1) Scintillation crystals

2) Xenon gas ionization chambers.

Image Reconstruction

A cross sectional layer of the body is broken up into multiple tiny blocks in computed tomography, and each block is given a number that is proportional to how much it attenuated the X-ray beam. The individual building components are known as "Voxels" for the full volume and "Pixels" for a single plane. [46,47]

It is necessary to calculate the amount of X-ray attenuation (mu) before each pixel may be given a number. Because there are thousands of pixels, the calculations get difficult and must be resolved using computer techniques (An algorithm is a mathematical method for solving a problem). These algorithms make an effort to solve the equations as quickly and accurately as they can. [46,47]

The following are three mathematical methods of image reconstruction,

1) Back projection

2) Iterative methods

3) Analytical methods.

CT Numbers

The CT scanner calculates, from the collected data, the linear attenuation coefficients of each pixel. After the CT computer calculates a value for the linear attenuation co-efficient of each pixel, the value is converted to a new number called "CT number" (Table 1).[46,47]

To honor Hounsfield, CT numbers based on magnification constant of 1000 are called Hounsfield units (H.U.).

Table-1: Attenuation coefficients and CT Numbers for Biological Tissues [47] (AT 60 KeV)

Tissue	Attenuation Coefficient U(Em *)	CT Numbers
Bone	0.400	+ 1000
Blood	0.215	+ 100 (approx)
Brain matter	0.210	+ 30 (approx)
CSF	0.207	+ 5 (approx)
Water	0.203	0
Fat	0.185	- 100 (approx)
Air	0.0002	- 1000

Image Display[47]

- A CT image is usually displayed on a television monitor for immediate viewing and recorded on a film.
- Original EMI scanner used 80 x 80 matrix but newer scan matrix have 512x 512 or 1024 x 1024 sizes.
- The basic shades of gray are 256.

Window Level

The CT number which is at the center of the window width along with Hounsfield scale is called window level or window center. [47]

Window Width

- This is the total number of CT numbers selected on the Hounsfield scale for a given scan.
- Thus, radio-opaque materials appear white and radio - lucent appear black.
- The range can be varied by changing the gate or window width at will so that tissue in a wide range or narrow range can be evaluated. [47]

Figure 1: SIEMENS® SOMATOM EMOTION 16 CT machine

NORMAL AXIAL CT

CT scans are examined from the caudal to the cephalic levels, and they are taken at an angle of 15 to 20 degrees from the canthomeatal line. These scores are broken down into supratentorial and posterior fossa cuts in 3mm and 5mm increments, respectively.[47, 48, 49, 50]

Posterior Fossa Cuts:

Four slices from the foramen magnum to the suprasellar region are viewed.

Above the foramen magnum:

On the side of the medulla, the cerebellar tonsil is visible. If a large window setting is employed, the foramen ovale and spinosum in the middle fossa can be seen. The cerebellar hemisphere's posterior aspect is defined by the inferior section of the cisterna magna. [48, 49, 50]

Above the fourth ventricle level:

The superior vermis, which divides the two hemispheres, reveals the superior cerebellar surface. The transverse sinuses can be seen converging in the torcula with contrast investigations. The sylvian fissure divides the temporal lobes from the frontal lobe in the middle fossa. In patients over the age of 50, the temporal horn, which is visible as a comma-shaped structure in the middle of the temporal lobes, is simple to visualize. The internal carotid artery, the optic chiasm, the infundibulum, the mammillary bodies, and the top of the basilar artery are all located in the medial side of the temporal lobes, which also surrounds the suprasellar cistern. The interhemispheric fissure divides the frontal lobes most inferior portion, which is visible in the anterior fossa. [48, 49, 50]

Tentorial Level:

The V-shaped enhancement of the tentorial notch outlines the superior vermis and junction of the pons to the midbrain. [48,49,50]

Supratentorial Cuts:

Third Ventricular Level:

It is possible to see the posterior inferior interhemispheric fissure, which is partly medial to the frontal lobes. The third ventricle is a midline feature that resembles a slit. Typically, it shouldn't have a transverse diameter bigger than 5 mm. The sylvian fissure, which extends medially, separates the frontal lobe from the temporal lobe. Medial to the medial aspect of the sylvian fissure, the putamen, globus pallidus, external capsule, and insular cortex are all discernible. The quadrigeminal plates and cistern, which are a part of the brainstem, can be seen behind the third ventricle. [48, 49, 50]

Low Ventricular Level:

The head of the caudate nuclei can be seen encircling the superior part of the frontal horns. The genu of the corpus callosum has an indentation that shapes the frontal horns anteriorly. The corpus callosum can be observed separating from the cingulate gyrus by the cingulate sulcus.[48, 49, 50]

The pineal gland's calcification is located behind the third ventricle. On rare occasions, there may be some habenula calcification visible anterior to the pineal gland calcification. The Galen vein and its relationship to the straight sinus can be observed behind the pineal gland on a contrast examination. The lateral ventricle contains the choroid plexus that is most frequently calcified. The atrium, lateral ventricles, posterior horns are evident at this level.[48, 49, 50]

Mid Ventricular Level:

The superior temporal gyrus and the sylvian fissure's expansion are visible. The frontal lobe and parietal lobe are divided by the central sulcus, which lies somewhat anterior to the sylvian fissure. The anterior component of the corpus callosum lines with

the most superior aspect of the frontal horn anteriorly, and the most superior aspect of the caudate nucleus connects it laterally. White matter fibres of the corpus callosal splenium can be observed encircling the posterior medial aspect of the occipital horns. It is possible to see how the posterior interhemispheric fissure connects to the cingulate gyrus from the occipital lobe. [48, 49,50]

Above the Ventricular Level:

The frontal, parietal, and a little piece of the occipital lobes make up the majority of the scan. The central sulcus is visible in the middle of the scan because it is deep. Motor and sensory cortices are outlined by pre- and post-central sulci. With falx in between, the interhemispheric fissure is visible along its whole length. [48, 49, 50]

PATHOPHYSIOLOGY AND MECHANISMS OF TRAUMA

Understanding the pathophysiology and mechanics underlying trauma offers insight into the body's reaction to harm, and this understanding in turn creates a framework for logical therapy. All injuries to the skull, calvarium, and brain are collectively referred to as head injuries. Therefore, it is crucial for the practicing radiologist to comprehend the mechanisms producing brain damage, their fundamental pathophysiology, and their imaging manifestations.[51]

The size and location of a traumatic brain lesion are determined by the shape of the object that caused the damage, the force of the hit, and whether or not the head was moving when the injury occurred. Last but not least, the total harm caused by impact depends not only on the mechanical damage but also on the intricate interaction of the subsequent pathophysiological events. [51]

PATHOPHYSIOLOGY:

Primary and secondary lesions can be distinguished among the signs and symptoms of head trauma. Primary lesions are those that develop right away as a direct result of the trauma. As a result of primary lesions, secondary lesions develop, typically as a result of mass effect or vascular impairment. Secondary lesions are frequently preventable, but primary injuries have already happened by definition by the time the patient enters the emergency room, making this distinction clinically significant. [51]

There are two types of primary injuries: focal and diffuse.

Focal brain injuries include cerebral contusions, intracranial hemorrhages, epidural hematomas and subdural hematomas. [52]

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. [52, 53] DAI is more prevalent than previously believed and is associated with neurological deficit. [54]

Secondary insults are physiological alterations that take place after an injury and speed up the deterioration process. Following first injury, neural cells are sensitive to ischemia events.[55] There is proof that initial damage impairs both global and local blood flow in the brain. [56, 57] Cell death and secondary damage are the results of physiological stressors like hypoxia and hypotension. The rise in mortality and morbidity is separately correlated with these two factors.[58]

Another mechanism of secondary injury is by free radical formation. Both the superoxide radical (0_2^-) and hydroxyl radical (OH-) contribute to the progression of injury. Injured parts of the brain suffering from hypoperfusion and cellular ischemia are unable to eliminate free radicals, despite the fact that they are often created during

aerobic metabolism. Lipid peroxidation is caused by the buildup of these radicals.[59, 60,61]

Another cause of subsequent harm in brain injury patients is excitotoxins. Following trauma, levels of excitatory neurotransmitters like glutamate rise. The main injury-induced cellular ischemia is made worse by the elevated neurotransmitter levels induction of hyper metabolism.[62]

Epidural / Extradural Hemorrhage:

An epidural hematoma, often referred to as an extradural hematoma (EDH), is a collection of blood that develops between the endosteal layer of the dura, which is the outer layer of the dura, and the inner surface of the skull. The second most frequent extra axial lesion in severe head injuries is an epidural hematoma.[63]

These represent collection of blood located between the inner skull table and dura due to;

a) Laceration of middle meningeal or posterior meningeal artery (Arterial EDH).

b) Damage to meningeal emissary veins or venous sinuses (Venous EDH).

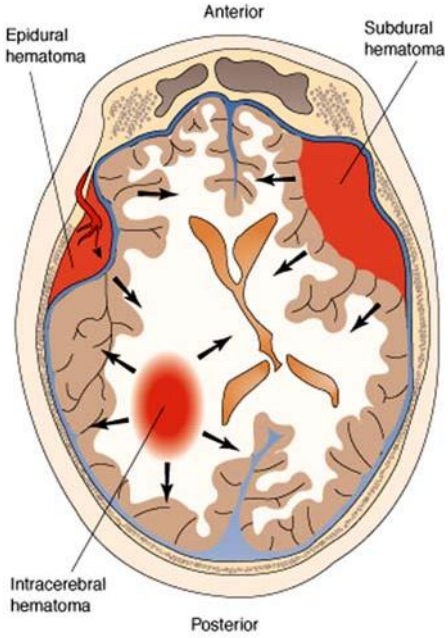


Figure-2: Location of epidural, subdural and intracerebral hematomas

They are frequently accompanied with a skull fracture and are typically associated with a history of head trauma. The source of bleeding is usually arterial, most commonly from a torn middle meningeal artery.[64]

EDHs often have a biconvex form and can herniate, which can have a mass effect. Normal cranial sutures serve as their boundary; venous sinuses do not. EDHs can be assessed using CT as well as MRI. The prognosis of EDHs is typically favourable when the blood clot is rapidly removed (or managed conservatively when tiny). [64]

A blood collection extra-axially within the possible space between the dura mater's outer layer and the inner table of the skull is known as an epidural hematoma (EDH). The lateral sutures, particularly the coronal sutures where the dura enters, contain it. It is a potentially fatal illness that may call for prompt treatment and, if

neglected, is linked to severe morbidity and mortality. For a positive outcome, quick diagnosis and evacuation are essential.[65, 66, 67]

Etiology

10% of traumatic brain injuries (TBI) requiring hospitalization have this complication. An epidural hematoma can be brought on by both traumatic and non-traumatic processes. Most cases involving traumatic mechanisms are the consequence of brain injuries brought on by car accidents, physical attacks, or unintentional falls.[69,70]

Non-traumatic mechanisms include the following:

- Infection/Abscess
- Coagulopathy
- Hemorrhagic Tumors
- Vascular Malformations

Epidemiology

Extradural hematomas are typically found in young individuals who have experienced head trauma, frequently in conjunction with a skull fracture. In 2% of all head injuries and up to 15% of all fatal head traumas, an epidural hematoma develops. Males are affected more frequently than females. Additionally, the incidence is higher among young people and teenagers. Patients who are affected on average are between 20 and 30 years old, and it is uncommon after the age of 50 to 60. The dura mater adheres to the underlying bone more firmly as a person ages. This lessens the possibility that a hematoma will form in the region between the dura and the skull.[70]

About 2% of people who suffer from head trauma have epidural hematomas, which are responsible for 5% to 15% of fatal brain injuries. Between 85% and 95% of epidural hematomas have a skull fracture on top of them.[70]

Clinical presentation

Extradural bleeding typically results from clearly defined head trauma, unlike subdural haemorrhage, in which a history of head trauma is frequently challenging to pinpoint. Young patients who have suffered a head injury—either in a sporting event or as the result of a car accident—typically arrive with temporary loss of consciousness. After the injury, patients return to a normal state of consciousness (lucid interval), but they typically still have a persistent, frequently quite bad headache. They begin to gradually lose consciousness over the following few hours. The sixth cranial nerve (abducens nerve, CN VI), which has a lengthy cisternal course, is frequently engaged as downward herniation starts, typically on the side of the haemorrhage, and it can, in an emergency, direct exploratory burr holes. [70]

Pathophysiology

The frontal region, middle fossa floor, and temporal region are typically affected by arterial EDH. Transtentorial herniation might ensue. A ruptured meningeal artery, most frequently the middle meningeal artery (75%), is the usual cause of haemorrhage. In over 75% of cases, a skull fracture is also present. The removal of the dura from the bone by the growing bleeding results in pain (sometimes a very bad headache). Traumatic injury to the posterior fossa, including EDH, is uncommon overall.[72,73]

Venous EDH has the unusual ability to cross over distinct compartments and occurs just exterior to the attachment of the falx or tentorium, or both. A single bi-

lentiform hematoma will result from the tear in the superior sagittal sinus, covering both cerebral hemispheres and inserting itself between the falx and calvarium. A torn torcula can result in a hematoma that concurrently compresses the posterior fossa and supratentorial compartments and rupture of the transverse sinus often results in a lesion with both infratentorial and supratentorial mass impact. Even a modest mass might impair essential brainstem function, causing fast degeneration. Extra-axial hematoma in the posterior fossa are uncommon. Multiple artery sites may bleed, resulting in a confluent multilobar mass or a number of distinct lesions. [72, 73]

The fact that young patients are affected is related to changes in the dura in older patients as well as the demographics of patients with head injuries, as the dura is considerably more adherent to the inner surface of the skull in older patients. It's vital to understand that extradural hematomas can cross sutures when there is sutural diastasis because the parietal (periosteal) component of the dura, which typically prevents dissemination, is also likely to be disrupted. [72,73]

Blood that dissects into the possible space between the dura and inner table of the skull causes epidural hematomas. This happens most frequently (85% to 95%) following a skull fracture. An arterial or venous vessel may sustain injury, allowing blood to bleed into the possible epidural area and cause an epidural hematoma. The middle meningeal artery beneath the temporo- parietal area of the skull is the vessel that sustains damage most frequently. [72,73]

Arterial Injury

The majority of epidural hematomas are caused by middle meningeal artery branch-related arterial haemorrhage. It may involve the dural arteriovenous (AV) fistula at the vertex or the anterior meningeal artery.[74,75]

Venous Injury

Venous haemorrhage caused by the laceration of a dural venous sinus accounts for up to 10% of EDHs. Up to 75% of EDHs in adults take place in the temporal region. However, they are equally common in the temporal, occipital, frontal, and posterior fossa regions in youngsters. The majority of EDH patients have a skull fracture. These hematomas frequently develop below a fracture of the temporal bone's squamous portion. This condition is known as a spinal epidural hematoma if it develops within the spine. A classification system based on the hematoma's attenuation properties was proposed by Zimmerman and Bilaniuk. [72,73]

Type –1: Acute - Swirling pattern of mixed density, related to the presence of clotted and non-clotted blood, usually occurs on day 1.

Type – II: Sub-Acute, occurring between days 2 to 4- Hyperdense and Homogeneous

Type – III Chronic, occurring between days 7 to 20- Mixed densities.

History and Physical Examination

Following a traumatic event, the typical presentation is an initial loss of consciousness followed by a full recovery (commonly referred to as a lucid interval), which is followed by a rapid escalation of neurological deterioration. Patients with epidural hematomas describe past focused head trauma, such as blows from a hammer or bat, falls, or car accidents. An epidural hematoma typically presents with loss of consciousness right after the injury, a brief period of lucidity, and then a decline in neurologic function. 14% to 21% of patients with an EDH experience this. These patients, however, might not lose consciousness at all, might lose awareness for a short time and then regain consciousness. As a result, there are many presentations, from a brief loss of consciousness to a coma. A patient who has other growing mass lesions may have the lucid interval, which is not pathognomonic for an EDH. Pure EDHs that

are quite big and show a CT scan indication of active bleeding experience the classic lucid interval. The speed at which the EDH is forming inside the cranial vault determines how quickly the symptoms may manifest. It is uncommon for a patient with a modest EDH to be asymptomatic. Additionally, an EDH may also manifest itself gradually. [74]

An EDH of the posterior fossa is unusual. About 5% of all posttraumatic cerebral mass lesions may be this type of EDH. Patients with posterior fossa EDH may be cognizant up until a late stage in the hematoma's development, at which point they may abruptly lose consciousness, develop apnea, and pass away. By removing the dura over the transverse sinus, these lesions frequently spread into the supratentorial compartment, which causes a large quantity of cerebral haemorrhage. [74]

This growing hematoma eventually causes an increase in intracranial pressure, which can be seen in a clinical environment by looking for ipsilateral pupil dilatation (caused by an uncal herniation and oculomotor nerve compression), high blood pressure, a slowing heartbeat, and erratic breathing. The "Cushing reflex" is a name for this trio. These results can point to the necessity of urgent intracranial therapy to avoid central nervous system (CNS) depression and demise.[75]

Evaluation

Radiographic features

Reviewing extradural hematomas' connections to the bone and dura will help you better understand their morphology. An extradural hematoma is actually a sub periosteal haemorrhage that is situated inside the skull, between the dura mater's parietal layer and the inner table of the skull (which is the periosteum). As a result of the periosteum crossing through the suture that is continuous with the outer periosteal layer, EDHs are typically constrained in their extent by the cranial sutures. This makes

it easier to tell EDHs from subdural hematomas, which are not constrained by sutures. [75]

But since venous sinuses are situated between the parietal and visceral layers of the dura, extradural bleeding might cross and raise them if there is no suture there. Unfortunately, these guidelines are not always accurate, and extradural hematomas occasionally cross sutures. One study discovered that up to 11% of EDHs in kids cross sutures. [76]

This happens in numerous circumstances:

- skull fracture across the suture [76]
- sutural diastasis[76]
- vertex extradural hematomas, usually due to venous extradural hemorrhage, often cross the midline elevating the superior sagittal sinus [77]

The computed tomography (CT) scan is one of the imaging investigations that forms the foundation of diagnosis. To determine whether there is an elevated risk of bleeding or underlying coagulopathies, laboratory tests such the INR, partial thromboplastin time (PTT), thromboplastin time (PT), and liver function test (LFT) may be performed. [78]

CT Scan

CT Features

Extradural hematomas are typically seen on brain CT images. They generally lie below the squamous portion of the temporal bone and are bi-convex (or lentiform) in shape. The vertex and posterior fossa are relatively seldom affected, while the temporo-parietal region and middle cranial fossa are the most frequent locations. EDHs are quite dense, a little diverse, and clearly defined. Secondary mass effect features, such as midline displacement, subfalcine herniation, and uncal herniation, may be

present, depending on their magnitude. Over the damage site, a skull fracture that is associated can be detected. Epidural hematomas, which most frequently affect the temporo-parietal region, are uncommonly linked to depressed fractures. An epidural hematoma may contain air pockets that are likely the result of a fracture but may also be connected to gas exchange across membranes. [78]

Fresh blood that has not clotted at the time of the CT scan is often less hyperdense, and a swirl indication may be visible. [79] In cases of acute EDH, postcontrast extravasation may be infrequently detected, while patients with chronic EDH may experience peripheral enhancement from granulation and neovascularization. The most popular imaging technique for evaluating cerebral haemorrhage is the CT scan. Its ubiquitous accessibility in emergency rooms explains its appeal. On a CT scan, the majority of EDHs are discernible. Due to blood's constrained ability to swell inside the dura's fixed attachment to the cranial sutures, the traditional presentation of brain tumours appears as a biconvex or lens-shaped mass on brain CT scans. Suture lines are not crossed by EDHs.[79] Radiologists typically employ a standard calculation to determine how much blood is present in an EDH. It goes like this:

ABC/2

A: The biggest area of haemorrhage on the CT slice, as measured by the maximum haemorrhage diameter.

B: On the same CT slice, the largest diameter at 90 degrees to A.

C: The number of CT slices with hemorrhage multiplied by the slice thickness in centimetres

However, while assessing EDH, additional CT findings may need to be considered. An indication of ongoing bleeding, for instance, can be patches of low density or a "swirl-

sign." The latter can be used to predict outcomes and frequently shows that surgical intervention is required. If the CT scan is not carefully scrutinised, it may be disregarded if the EDH abuts haemorrhagic or contused brain tissue because it appears shallow. [79]

Several factors may lead to a non-diagnostic CT scan. These are as follows:

- A collection of low density blood could be a sign of severe anemia (thus leading to misinterpretation).
- Severe hypotension may cause a reduction in arterial extravasation.
- A sufficient amount of blood must collect for visualization in order for a CT scan to be positive. If the CT is obtained too soon after trauma, there may not be sufficient accumulation for appropriate interpretation.
- Blood buildup could be delayed if venous bleeding is the cause of the EDH. This could potentially result in difficulty with CT interpretation. [79]

Magnetic resonance imaging (MRI)

When looking for EDH in the vertex, brain MRI is more sensitive than a CT scan. In order to separate the displaced dura from a subdural hematoma, MRI may clearly show the displaced dura, which shows as a hypointense line on T1 and T2 sequences.[80]

On a T1 scan, acute EDH looks isointense and on a T2 sequence, it varies in intensity from hypo- to hyperintense. While late subacute and chronic EDH are hyperintense on both T1 and T2 sequences, early subacute EDH appears hypointense on T2. [80]

Intravenous contrast may demonstrate displaced or occluded venous sinus in case of the venous origin of EDH. [80]

It should be obtained when there is high clinical suspicion for EDH, accompanying a negative initial head CT scan. [80]

In the situation of a suspected spinal EDH, a spinal MRI is the preferred imaging modality, as it affords higher resolution versus a spinal CT. [80]

Angiography

The healthcare provider should check for the presence of a dural arteriovenous (AV) fistula that may have developed from the middle meningeal artery when assessing EDHs in the vertex. To accurately assess the presence of such a lesion, angiography could be needed. It can be utilised to assess EDH's nontraumatic aetiology, such as AVM. Rarely, angiography can show middle meningeal artery laceration and the "tram track sign," which is the extravasation of contrast from the middle meningeal artery into its associated middle meningeal veins.[81]

Treatment / Management

EDH is a neurosurgical emergency. It immediately needs to be surgically evacuated in order to prevent death or lasting neurological damage due to hematoma growth and herniation. It is crucial to act within one to two hours of presentation; hence it is necessary to seek out neurosurgical consultation.[52,53,54]

Priority should be given to stabilising the patient, which includes attending to the ABCs (airway, breathing, and circulation).

Surgical intervention is recommended in patients with[52,53,54]:

- Acute EDH
- Independent of Glasgow coma scale (GCS) score; GCS less than 9 with pupillary abnormalities like anisocoria;
- Hematoma volume larger than 30 ml.

Operative Management

Craniotomy and hematoma evacuation are the main treatments for patients with acute and symptomatic EDHs. If more advanced surgical knowledge is not accessible, "trephination" (or burr hole evacuation) is frequently a necessary sort of surgery, according to the data that is currently available; it even has the potential to reduce mortality. However, if it is possible, doing a craniotomy can result in a more complete evacuation of the hematoma. [55]

Non-Operative Management

There is a dearth of research comparing nonsurgical treatment to surgical intervention in EDH patients. However, if a patient has minor symptoms, acute EDH, and fits all of the criteria given below, a non-surgical strategy may be taken into consideration[55]:

- EDH volume less than 30 ml,
- Clot diameter less than 15 mm,
- Midline shift less than 5 mm,
- GCS higher than 8, and no focal neurological complaints on physical examination.

If it is decided to treat acute EDH non-surgically, careful monitoring with frequent neurological exams and ongoing brain imaging monitoring are necessary due to the possibility of hematoma extension and clinical worsening. Following a brain injury, it is advised to get a follow-up head CT scan within 6 to 8 hours. [55]

Differential Diagnosis

- Intracranial abscess
- Intracranial mass
- Seizure

- Transient ischemic attack

Prognosis

As long as the clot is removed as soon as possible, the prognosis is often pretty excellent, even with a hematoma that is relatively large. Smaller hematomas without swirl or mass effects can be treated conservatively, albeit this may occasionally lead to dura calcification. [56]

When it is quickly diagnosed and removed, patients with pure EDHs have a great prognosis for a functional outcome following the surgical evacuation. Morbidity and death rise as a result of delayed diagnosis and treatment. [56]

EDHs brought on by arterial bleeding grow quickly and are easy to spot. But those brought on by a dural sinus tear take longer to manifest. Clinical signs may therefore take longer, which could delay identification and evacuation. A worse neurological prognosis and hence higher mortality are often the results of an EDH volume more than 50 cm prior to evacuation. [56]

Factors that may influence the outcome are as follows:

- Patient age
- Time lapsed between injury and treatment
- Immediate coma or lucid interval
- Presence of pupillary abnormalities
- GCS/motor score, on arrival
- CT results (hematoma volume, degree of midline shift, presence of signs of active hematoma bleeding, or associated intra-dural lesions)

Postoperative intracranial pressure (ICP)

Several markers that correlate with a poor prognosis of EDH include the following:

- A low GCS before surgery, or on arrival
- Examination of unusual pupils, in particular those who are not reacting (unilateral or bilateral)
- The interval between neurological symptoms and operation;
- Advanced age;
- An increase in ICP following surgery

A bad prognosis can be correlated with specific head CT findings:

- A midline shift more than 10 to 12 mm;
- Hematoma volume greater than 30 to 150 ml;
- "Swirl sign" indicating an active bleed
- Associated intracranial lesions (such as contusions, intracerebral hemorrhage, subarachnoid hemorrhage, and diffuse brain swelling) [56,57]

Complications

- Mass effect: compression of brain if bleeding is significant
- Herniation
- Seizures

Skull fracture, scalp hematoma / laceration.

a) Scalp injury:

The exposed position of the human scalp makes it vulnerable to harm. It aids in absorbing the power of head trauma, especially glancing hits. A tangential blow can land with only a laceration of damage due to the gale's movement. It is helpful to start by looking at the extracranial tissues for signs of scalp injury or radio opaque foreign bodies when evaluating CT scans for head trauma. Often, the only conclusive sign of

the impact location is the swelling of the soft tissues of the scalp.[63]

Type of scalp wounds

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

Sub Galeal Hematoma:

The skull typically moves more quickly upon impact than the scalp. These movements could cause the blood vessels that connect the scalp and the skull to tear. Even in the absence of visible bruising or scalp laceration, this incongruity of movement can cause subcutaneous or subgaleal hematomas.[63]

On CT the subgaleal hematoma appears as a well-defined soft tissue swelling of the scalp, located between the galea aponeurotica and the pericranium. [63]

b) Skull fractures[64]:

The distinctive skull morphology changes the brain injury in a number of ways. *First,* the skull's distinctive structure, which consists of a layer of cancellous bone between an inner and an outer table of compact bone, is best suited to guard against mechanical loading. [64]

Second, it can be said that the skull combines the properties of a hemisphere and a sphere, a configuration that is crucial for the pressure response at impact. [64]

Third, the final brain injury is influenced by how smooth the inner surface of the skull is. The inner table's irregularity determines how probable it is that the brain may sustain damage when it rubbed against the rough surface. [64]

Fourth, the thickness of the skull plays a part in protecting the brain, just as the thickness of the scalp does. The skull is often narrow where the scalp muscle covers the skull, such as in the temporal area. The parietal region, which is visible, is one of the thicker, denser areas of the skull. Both thin and unreliable describe the orbital surface of the anterior cranial fossa. As a result, the cortical surface of the brain is more easily deformed, fractured, and injured when tension is concentrated in one region. [64]

1. Linear fracture:

A significant amount of force deforming the skull vault yet dispersed over a larger surface area is implied by linear or fissure fractures of the skull vault. About 80% of all skull fractures are linear fractures, with 50% of them occurring in the middle. The related rupture of the dura and the adjacent scalp laceration are the two key factors that determine the development of significant infectious consequences. An epidural hematoma, which develops as a result of the underlying meningeal arteries being torn, is the most severe side effect of a linear fracture. [64]

The object must be both sufficiently large (more than 2 square inches) to prevent skull penetration and sufficiently tiny (less than 2 square inches) to prevent contact phenomena from being extensively dispersed throughout the surface of the head. [64]

2. Depressed fracture:

A depressed fracture occurs when one portion of the skull has been forced inside to the point that its outer table is at or below the level of the surrounding inner table. On a smaller impact surface, there are more concentrated and powerful contact forces. This causes the skull's flexibility to be exceeded, allowing for skull perforation. Inability to rebound causes bone fragments to be moved into the brain from the calvarium. Typically, the inner table is offset more than the outer table. [64]

These fractures could be complex or simple. Simple fractures, in which the scalp covering is unharmed, are rare and typically affect children. A compound fracture occurs when the skull breaks into multiple pieces that are pushed into the cranial cavity. This frequently occurs in conjunction with a dural rip, which serves as an infection entrance point. The frontal region is where half of the depressed fractures occur. [64]

3. Basilar fracture:

These result from direct skull base impact (occiput, mastoid, supraorbital), energy transmission from facial or mandibular trauma, or distant effects of skull impact. In the latter scenario, stress waves that radiate from the point of impact or changes in the form of the skull as a result of impact are common. [64]

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
- ☞ Transverse fractures

As a result of ruptured tympanic membranes, longitudinal fractures are more frequent and typically accompanied by CSF otorrhoea and CSF rhinorrhea (through the Eustachian tube). This may cause sensorineural hearing loss and ipsilateral facial nerve palsy.[64]

- Fractures of orbital roofs results in bilateral periorbital hematomas, black eye (raccoon eyes). [64]

4. Diastatic Fractures:

A so-called diastatic fracture happens when the cranial sutures are traumatized and torn apart. Diastatic skull fractures are rather typical, particularly in young children. Normal sutures come in varied widths for different people. The highest limit of normal is typically accepted to be a width of 2 mm, which is most frequently observed in the lambdoid region. Asymmetry between the two sides or between two separate sutures is the main radiologic finding of a diastatic fracture. Even while asymmetry can be evident when the coronal and lambdoid sutures are broken, the sagittal suture is not comparable. Sutural diastasis can happen either with or without a fracture present. It frequently signifies the continuation of a fracture line that began far from the suture. [64]

5. Comminuted Fractures:

The majority of depressed skull fractures are connected to comminution to some degree (i.e. multiple bone fragments). Comminuted fractures are typically caused by severe blunt or penetrating trauma, in which a significant amount of energy is lost quickly and within a constrained area. The enhanced calvarial flexibility of a child's

skull makes comminuted fractures (and fractures in general) rare. Fractures that are comminuted frequently are. [64]

SECONDARY EFFECTS OF CRANIOCEREBRAL TRAUMA[51]:

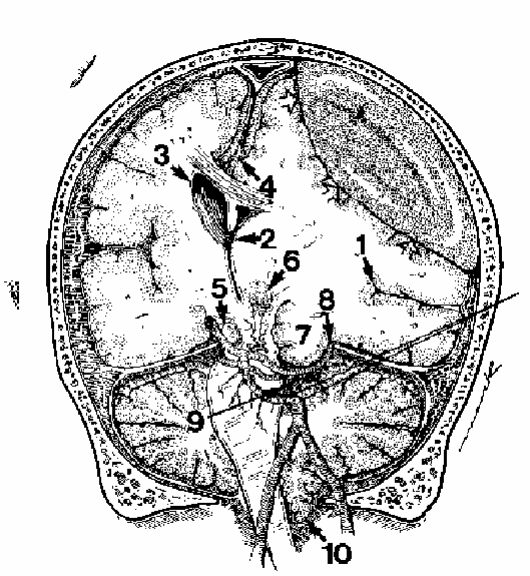


Figure-3: Secondary effects of a large epidural hematoma

1. Inferior displacement of sylvian fissure, middle cerebral artery branches.
2. Subfalcine herniation of lateral ventricles with compression of ipsilateral ventricle.
3. Contralateral lateral ventricle dilates secondary to functional obstruction at the foramen of Monro.
4. Anterior cerebral arteries (cut across), shifted across the midline by the mass effect, return to midline under the falx cerebri (may cause secondary ACA infarct).
5. Midbrain contusion (Kernohan notch) produced by displacement from the mass effect causing the cerebral peduncle to strike the opposite edge of the tentorial incisura.
6. Midbrain hemorrhage (duret hemorrhage) caused by downward displacement.
7. Medial temporal lobe herniates over the tentorial incisura (descending transtentorial herniation).
8. Ipsilateral posterior cerebral artery (PCA) is compressed against the tentorial incisura (may cause secondary PCA infarct).

9. Ipsilateral cerebellopontine angle cistern is widened as the brainstem is displaced by the herniating temporal lobe.
10. Descending tonsillar herniation.

CT Findings

The herniating medial temporal lobe's mild effacement of the lateral side of the suprasellar and ambient cisterns is the earliest discovery. Sometimes, as time goes on, the cerebral peduncle contralateral to the cerebral mass becomes crushed against the tentorium's free edge (Kernohans notch). The transverse diameter of the mesencephalon narrows due to bilateral compression, whereas the antero-posterior diameter increases. [51]

ISCHEMIA / INFARCTION

Raised intracranial pressure, embolization from a vascular dissection, direct mass effect on the cerebral vasculature from brain herniation, or an overlaying extra axial collection can all lead to post-traumatic ischemia or infarction. Additionally, patients may experience hypoxemia brought on by respiratory stoppage or status epilepticus, abrupt reductions in cerebral blood flow, or diffuse ischemia damage. [52]

Anterior cerebral artery infarction from subfalcine herniation, posterior cerebral artery infarction from uncal herniation, and posterior inferior cerebral artery infarction from tonsillar herniation are examples of patterns of infarction from focal mass impact. [52]

DIFFUSE CEREBRAL EDEMA:

Massive cerebral edema with intracranial hypertension is among the most life-threatening of all secondary traumatic lesions. [52]

While severe cerebral edema typically takes 24 to 48 hours to develop, substantial swelling of one or both hemispheres may happen hours after the acute

injury. Diffuse posttraumatic brain swelling is brought on by an increase in the amount of intravascular blood, the amount of water in the brain, or both. In these situations, the mortality rate is close to 50%.[52]

Cytotoxic, interstitial, or vasogenic causes of cerebral edema are possible. Cerebral edema that is vasogenic in the early aftermath of trauma indicates that the blood-brain barrier has broken down, allowing intravascular contents to flow into and around areas of injured tissue.[52]

Although the pathophysiology is unclear, it seems to be caused by a loss of normal brain autoregulation. Usually unharmed, the brainstem and cerebellum can appear hyperdense in comparison to the cerebral hemispheres. The falx and cerebral vessels frequently have an appearance that mimics acute subarachnoid hemorrhage. [52]

CT Findings:

- A generalized mass effect that causes the ventricular system to be compressed as well as the effacement of sulci, suprasellar, and quadrigeminal plate cisterns.
- Decrease attenuation of the brain parenchyma with loss of gray-white differentiation.
- A transtentorial herniation may be imminent if the brainstem cisterns are completely destroyed, which would imply a significant mass impact.

Like the "reversal" sign. When compared to the normal density of the thalamus, brainstem, and cerebellum, all of which are perfused by the posterior circulation, it is brought on by diffuse hypodensity of the cortex and deep white matter. [52]

SEQUELAE OF TRAUMA[43]:

Patients who recover from serious head injuries may still have terrible permanent neurological damage or only moderate curable abnormalities. Among the significant long-term effects of traumatic brain injury are:

1) ENCEPHALOMALACIA

Focal encephalomalacia, a common sign of remote head injury, consisting of tissue loss with surrounding gliosis. It might not show any symptoms or act as a seizure focus. [40]

Computed tomography shows areas of low attenuation with volume loss that are rather clearly delineated. The ventricular system's surrounding regions could enlarge. Porencephalic cysts can develop when encephalomalacia around a ventricle communicates with other nearby areas. [40]

2) CEREBRO SPINAL FLUID LEAK

A dural tear is necessary for the CSF leak, which can follow calvarial or skull base fractures. Following fractures, CSF rhinorrhea develops when contact between the paranasal sinuses and the subarachnoid area takes place. When the tympanic membrane is torn, there is connection between the middle ear and the subarachnoid area, which results in CSF otorrhea. Pneumocephalus, or an accumulation of air in the cranial cavity, is typically located at the cribriform plate of the ethmoid bone and may be accompanied by a cranio-nasal fistula with or without rhinorrhea, CSF otorrhea, or, less frequently, an open skull fracture with a dural tear. [41]

CSF extravasation is highly detectable by radionuclide cisternography41, but accurate anatomic localization of the defect requires CT scanning with intrathecal contrast. [41]

3) LEPTOMENINGEAL CYST

A traumatic tear in the dura, which results in the herniation of arachnoid material at the site of the suture or skull fracture, is the origin of leptomeningeal cysts or "growing fractures." This causes the skull defect or suture to gradually enlarge, most likely as a result of CSF pulsations. On a CT scan or plain skull film, the leptomeningeal cyst shows as a lytic skull defect that can grow over time.[46]

4) INFECTION:

Meningitis, cerebritis, and abscess may develop as a direct result of bacteria extending directly through a fracture site. A second CT scan may reveal hydrocephalus, effacement of the basal cisterns due to inflammatory exudate, and augmentation of the basal cistern and meninges with IV contrast material if the original scan is normal.[46]

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 has to do with how severe diffuse brain damage is, and it can last anywhere from a few seconds and several weeks. It also establishes whether the patient has become better or worse since the injury. If the patient's state of consciousness has progressively declined following the accident, there is likely an ongoing intracranial disease. [86]

2) *Period of post-traumatic amnesia:* Following the brain injury, there is a period of amnesia at this time. It is a reflection of the degree of injury and may last for several weeks in cases of serious injuries.[86]

3) *Cause and circumstance of injury:* A thorough narrative of what happened is crucial since the patient might pass out or crash his car as a result of an earlier intracranial incident. Subarachnoid hemorrhage, vertigo, epileptic seizures, a history of consuming alcohol, etc. are a few examples. The likelihood of related extracranial injuries increases with impact force. [86]

4) *Presence of headache and vomiting:* These are typical post-injury symptoms, and if they continue, the likelihood of cerebral bleeding and elevated intracranial pressure must be considered. [86]

5) *History of seizures:* This is crucial because seizures produce an increase in intracranial pressure, which could worsen the patient's condition. [86]

6) *ENT bleed:* These points to the presence of basal skull fractures. [86]

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
- Basal skull fracture sign

Level of consciousness:

Graham Teasdale and Bryan Jennett, professors of neurosurgery at the University of Glasgow, published the Glasgow Coma Scale for the first time in 1974.[85] All forms of acute illness and Using the Glasgow Coma Scale (GCS), the level of diminished consciousness in trauma victims can be assessed scientifically. The scale

rates patients based on their eye-opening, muscular, and verbal responses—the three components of responsiveness. Each of these should be reported separately to give a clear, understandable picture of the patient's condition. [85]

The results of each scale component can be combined to provide a total Glasgow Coma Score, which provides a valuable "shorthand" overview of the overall severity but offers a less detailed description. The score expression is made up of both the sum of the scores and each of the individual components. The formula for a score of 10 would be GCS10 = E3V4M3, for example. [86]

The Glasgow Coma Scale became widely used in the 1980s as a result of the initial version of the Advanced Trauma and Life Support manual's recommendation to use it on all trauma patients. In 1988, it was also included in the World Federation of Neurosurgical Societies (WFNS) scale for classifying patients with subarachnoid haemorrhage.[87] Since then, various clinical recommendations and scoring systems for those who have experienced trauma or a severe disease have included the Glasgow Coma Scale and its overall score.[88] These include preverbal children and patients of various ages. More than 75 nations use the Glasgow Coma Scale, which is a necessary part of the NIH Common Data Elements for research of brain injury and the ICD 11 revision.[88.89,90]

Scoring and Parameters

Best eye reaction (E), best verbal response (V), and best motor response (M) are the three components of the Glasgow Coma Scale (M).

***Best eye response*[88]**

1. No eye opening
2. Eye opening to pain

3. Eye opening to sound
4. Eyes open spontaneously

Best verbal response[88]

1. No verbal response
2. Incomprehensible sounds
3. Inappropriate words
4. Confused
5. Orientated

Best motor response[88]

1. No motor response.
2. Abnormal extension to pain
3. Abnormal flexion to pain
4. Withdrawal from pain
5. Localizing pain
6. Obeys commands

The Glasgow Coma Scale's component response levels are "scored" on a scale of 0 to 10. The score is calculated by adding the different parts' scores, which range from 1 for no response to 4 for an eye-opening response, 5 for a verbal response, and 6 for a nonverbal answer (Motor response). The formula for a score of 10 would be GCS10 = E3V4M3, for example. Thus, the overall Coma Score ranges from three to fifteen, with three being the worst and fifteen being the best. [88.89,90]

Mild head injury GCS of 13-15

Moderate head injury GCS of 9-12

Severe head injury GCS of < 8

Intubated patients are unable to talk, making it impossible to gauge their verbal score. They are only assessed for eye opening and motor function, and the suffix T is added to their score to denote the presence of an intubated patient. The maximum GCS score for intubated patients is 10T, while the lowest value is 2T. [88.89,90]

Clinical Significance

In order to assess responsiveness and guide early care for individuals who have experienced a head injury or another sort of severe brain impairment, the Glasgow Coma Scale is routinely utilised. For patients who are more badly impacted, immediate treatment decisions are taken, such as securing the airway and using triage to identify individuals who need to be transported. Patients with less severe impairments must decide whether they need neuroimaging, should be admitted for observation, or should be discharged. Serial Glasgow Coma Scale evaluations are essential for tracking a patient's clinical progress and directing therapy modifications. [90]

Across the responsiveness spectrum, the three Scale components' informational output vary. Changes in motor response predominate in patients with more severe disabilities, although ocular and linguistic capabilities are more beneficial in smaller doses. Therefore, the clinical results of the three components should be reported separately for each patient. The overall score conveys a helpful summary index, albeit with some information lost. [91]

The Glasgow Coma Scale is a reliable indicator of clinically significant traumatic brain injury in both preverbal and verbal paediatric patients (i.e., injury requiring neurosurgical intervention, intubation for more than 24 hours, hospitalisation for more than two nights, or injury resulting in death).[90]

Numerous standards and evaluation results have taken into consideration the Glasgow Coma Scale. These include the Brain Trauma Foundation's severe TBI

recommendations, trauma guidelines (such as Advanced Trauma Life Support), intensive care scoring systems (such as APACHE II, SOFA), and Advanced Cardiac Life Support.[91]

CLASSIFICATION OF HEAD INJURY

Glasgow Coma Scale (GCS) scores are used by the Traumatic Coma Data Bank (TCDB) to determine the severity of brain injuries. Patients who degenerate to that level within 48 hours of admission or who receive neurosurgical resuscitation with a GCS score of "8" or below are considered to have suffered a severe brain injury. Nearly 10% of head injury cases that are hospitalized are due to this.[91]

Patients with a GCS score of 9 to 12 have moderate head injuries. Numerous studies also include patients with higher GCS scores who have an intracranial hematoma surgically removed. All patients with a GCS score of 13 to 14 are considered to have mild head injuries. An average GCS score is considered to be 15. [91]

The TCDB has developed a new categorization of brain injury since some categories are not as precise as one would like. The fact that the intracranial pathology of the patient is described in terms of the CT scan results, despite the fact that this new nomenclature is not flawless, suggests that it represents a substantial breakthrough. This then categorises them in accordance with a number of pathological processes that are known to be significant in patients who have suffered brain injury. The degree of brain swelling, midline displacement, and the presence or absence of mass lesions of a particular size are a few of these. In order to fully define the disease process and the type of tissue structural damages, further advancement in the nomenclature of head injury is required. [91]

Table-2: TCDB Diagnostic Categories of Abnormalities Visualised on CT Scanning[91]

Diffuse Injury Grade	CT Appearance
Diffuse injury - Grade I	No visible intracranial pathology seen on CT
Diffuse injury - Grade II	Lesion densities and/or cisterns are evident with a midline displacement of less than 5 mm. tear in the brain parenchyma's tissue.
	No high/ mixed density region greater than 15 cc. May include bone fragments and foreign bodies
Diffuse injury - Grade III	Diffuse brain enlargement, compressed or missing cisterns, and a 5-mm shift in the midline. No lesion > 15 cc with high or mixed density.
Diffuse Injury - Grade IV	Diffuse brain swelling, cisterns are compressed / absent, midline shift > 5 mm, High / mixed density lesion of more than 15 cc.

Outcome

A relationship between assessments of the GCS (typically reported as the total GCS Score) and the outcome was shown clearly by Gennarelli et al.,[92] They showed that there was a consistent, gradual correlation between falling GCS Score from 15 to 3 and increasing mortality following a brain injury. Numerous later researches have confirmed this connection. Although the results for the visual, verbal, and motor responses also relate to the outcome, they do so in different ways so evaluating each one separately provides more information than the combined total score.[91]

Although the GCS score is one of the most potent clinical predictive indicators, neither it nor any other feature by itself should be used to predict the course of a particular patient. This is true because a number of variables have an impact on the prognostic implications of the score. These include the diagnosis, the cause of any extracranial injuries in cases of trauma, patient-related variables like age, and other clinical indices (like pupillary dysfunction and imaging findings). The GCS score is an important part of multifactorial models for outcome prediction, such as in the IMPACT and CRASH trials.[93,94]

Emergency imaging is used to find curable lesions before they cause additional neurological damage. In the immediate aftermath of head trauma, CT plays a crucial role in accurately identifying lesions that need prompt neurosurgical intervention. CT is effective in identifying secondary injuries as well, making it crucial for follow-up.[93,94]

Today, CT scans are also utilized to forecast patient outcomes and mortality due to their short turnaround times, ease of accessibility, and sensitivity to hemorrhage. Determining the role of MDCT in traumatic extradural hemorrhage with neurological correlation and follow-up is thus the goal of this sectional descriptive observational investigation.

MATERIALS &

METHODS

MATERIAL AND METHODS:

Study setting: The study was conducted in the Department of Radio-Diagnosis at" R.L. Jalappa Hospital and Research Center attached to Sri Devaraj Urs Medical College, Tamaka Kolar.

Study design: Cross sectional descriptive observational study was conducted over a period of eighteen months from January 2021 to July 2022.

Study duration: The data collection for the study was done between to over an 18 months period.

Sample size:

Udaykumar Ramrao Badhe et al[95] had reported that the about 70% of the subjects had good recovery according to Glasgow Outcome score. Assuming alpha error = 0.05 (95% Confidence Limit) and absolute precision of 12%,

The minimum required sample size was calculated to be 56 subjects with EDH.

The sample size was derived from the following formula:

$$\text{Sample size (n)} = \frac{Z^2(PXQ)}{d^2} \text{ where;}$$

Z is the value for Confidence Interval

D is the absolute precision

P is the expected proportion and q=1-p

The sample size was calculated using OpenEpi software version 3.01 (Open Source Epidemiologic Statistics for Public Health).

In our study, 62 study subjects were included.

Inclusion criteria:

Patients of adult age group with extradural hemorrhage due to craniocerebral injury.

Exclusion criteria:

- Patients with no extradural hemorrhage on CT.
- Patients who cannot be followed up.

Sampling method: All the study subjects were recruited consecutively till the sample size was reached.

Methodology:

A cross-sectional descriptive study was done at Department of Radio-Diagnosis at R.L. Jalappa Hospital and Research Center attached to SDUMC, Kolar. A total of 62 patients of adult age group with extradural hemorrhage due to craniocerebral injury were included in the study. All the patients satisfying the inclusion criteria underwent CT evaluation after giving consent. All the CT scans in this study was performed using SIEMENS® SOMATOM EMOTION 16 CT machine.

CT Protocol consisted of the following:

- Non contrast axial 16 slice helical series
- Beam collimation 4.8 mm.
- Detector configuration 16x0.625
- Pitch 0.8:1
- Tube current 270 mAs
- Voltage 130 kV

Along with axial images, coronal and sagittal images will be obtained and compared. The haemorrhage detected on CT examination were classified according to the region involved. The image findings of extradural hemorrhage on MDCT, correlation of the size of extramural hemorrhage and midline shift with neurological features and the prognosis of patients with the neurological deficits on follow up were evaluated.

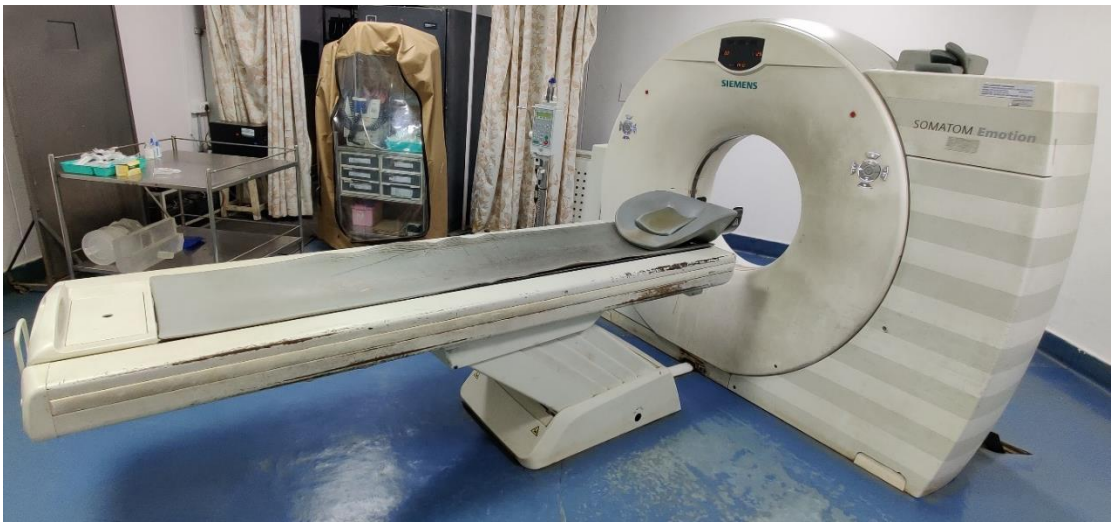


Figure 4: SIEMENS® SOMATOM EMOTION 16 CT machine

ETHICAL CONSIDERATIONS: The study was started after obtaining ethical clearance from institutional ethical committee meeting. Informed written consent was obtained from all the participants after providing detailed information on the objectives of the study, risks and benefits involved and the voluntary nature of participation. The confidentiality of the study participants was maintained throughout the study.

STATISTICAL METHODS:

Traumatic extradural hemorrhage was considered as the primary outcome variable. Study group considered as Primary explanatory variable. Age, gender, right side and left side were considered as other study relevant variables. Descriptive statistics were used to analyze data in accordance with the study's objectives. Data was also represented using appropriate diagrams like Error bar diagram, bar diagram, staked bar diagram and box plots. All Quantitative variables were checked for normal distribution. Continuous variables were analyzed by independent-sample T-tests and expressed as

the mean and standard deviation. Data was entered into Microsoft excel data sheet and analyzed using Stata version 12 software. Chi-square was used as test of significance for qualitative data and independent t test was used as test of significance for quantitative data. P value < 0.05 was considered statistically significant

OBSERVATIONALS &

RESULTS

Results

A cross-sectional descriptive study was done at Department of Radio-Diagnosis at R.L. Jalappa Hospital and Research Center attached to SDUMC, Kolar. The study aimed to evaluate the traumatic extradural hemorrhage patients by multidetector computed tomography with neurological correlation and follow up. A total of 62 patients of adult age group with extradural hemorrhage due to craniocerebral injury were included in the study.

In the present study, out of 62 (100%), majority (27 (43.6%)) of patients were in the age group of 20 to 29 years followed by 30 to 39 years, 14 (22.6 %). (Table 3) (Figure 5)

Age group	Frequency	Percent
20-29	27	43.6
30-39	14	22.6
40-49	11	17.7
50 and above	10	16.1
Total	62	100.0

Table 3: Age wise distribution of patients

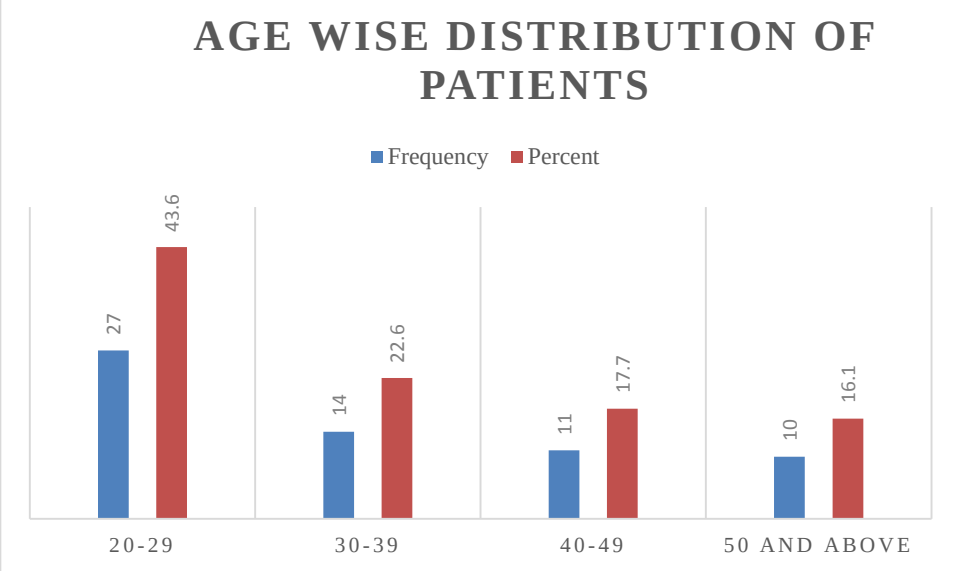


Figure 5: Cluster column chart of age wise distribution of patients (N=62)

Out of 62 (100%) patients, about 55 (88.7%) of patients were males and 7 (11.3%) were females showing male predominance. (Table 4) (Figure 6)

Sex	Frequency	Percent
Female	7	11.3
Male	55	88.7
Total	62	100.0

Table 4: Sex wise distribution of patients

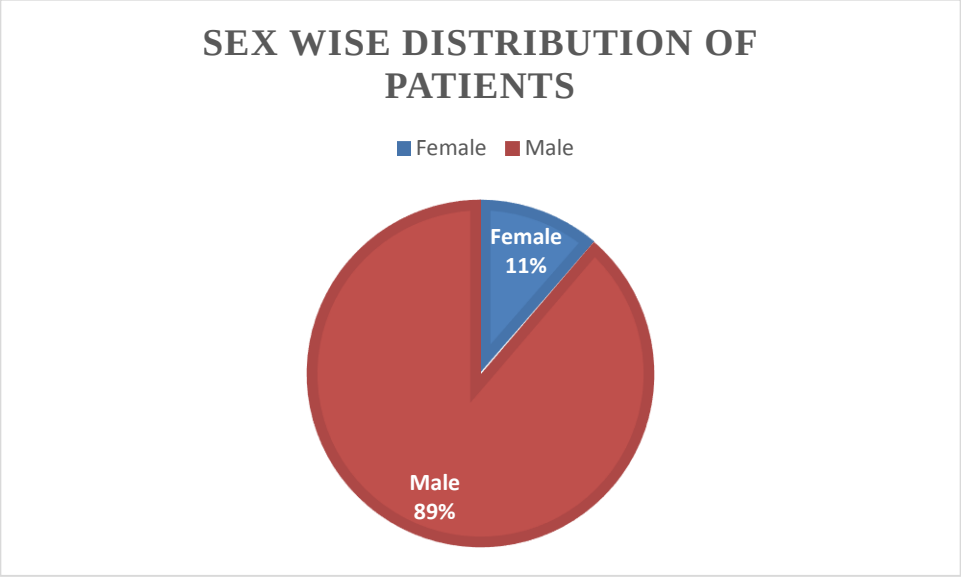


Figure 6: Pie chart of sex wise distribution of patients (N=62)

The clinical history of all 62 (100%) patients showed that all had history of head injury and the mode of injury for all patients was RTA. Even all were clinically diagnosed as head injury following RTA.

On radiological examination, out of 62 (100%) patients, majority of them, 17 (27.4%) patients had right temporal EDH followed by l3 (21.0%) left temporal EDH. (Table 5)

Radiological diagnosis	Frequency	Percent
LEFT FRONTAL EDH	5	8.1
LEFT OCCIPITAL EDH	1	1.6
LEFT PARIETAL EDH	7	11.3
LEFT TEMPORAL EDH	13	21.0
RIGHT FRONTAL EDH	11	17.7
RIGHT PARIETAL EDH	6	9.7
RIGHT OCCIPITAL EDH	2	3.2
RIGHT TEMPORAL EDH	17	27.4
TOTAL	62	100.0

Table 5: Distribution of patients based on their radiological diagnosis

Among all 62 (100%) patients, the commonest site of EDH was right temporal region, 17 (27.4%) followed by l3 (21.0%) left temporal region. (Table 6) (Figure 7)

Site of EDH	Frequency	Percent
LEFT FRONTAL	5	8.1
LEFT OCCIPITAL	1	1.6
LEFT PARIETAL	7	11.3
LEFT TEMPORAL	13	21.0
RIGHT FRONTAL	11	17.7
RIGHT PARIETAL	6	9.7
RIGHT OCCIPITAL	2	3.2
RIGHT TEMPORAL	17	27.4
Total	62	100.0

Table 6: Distribution of patients based on site of EDH

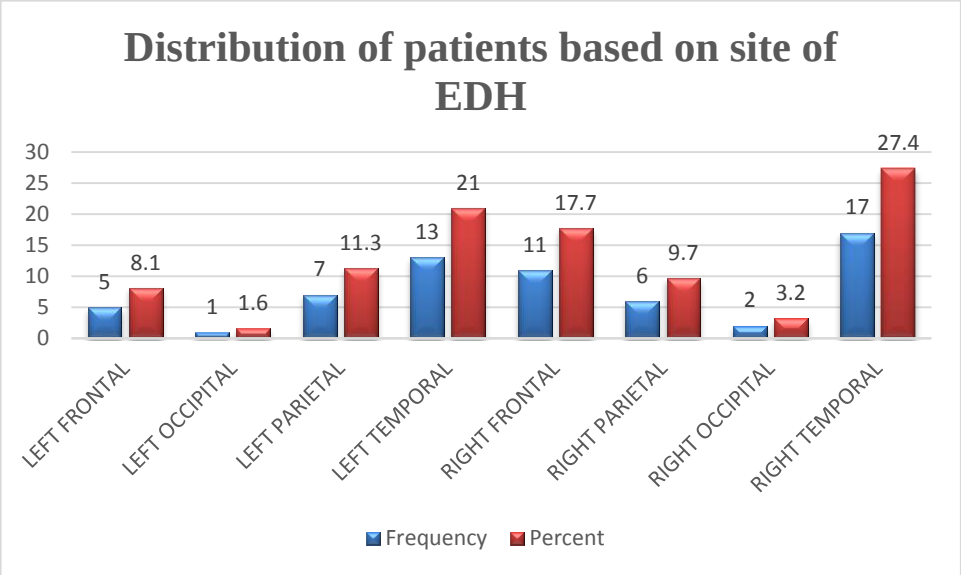


Figure 7: Cluster column chart of distribution of patients based on site of EDH (N=62)

Majority of the patients out of 62 (100%), had mild category of GCS score i.e., 26 (41.9%) of patients followed by 22 (35.5%) moderate and 14 (22.6%) had severe category of GCS. (Table 7) (Figure 8)

GCS category	**Frequency**	**Percent**
Mild	26	41.9
Moderate	22	35.5
Severe	14	22.6
Total	62	100.0

Table 7: Distribution of patients based on GCS score

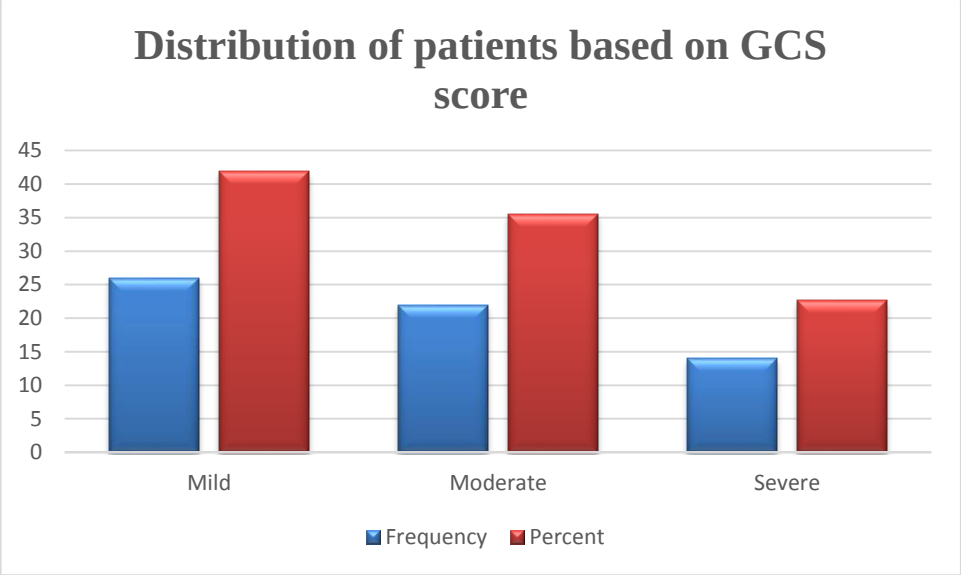


Figure 8: Cluster column chart of distribution of patients based GCS score (N=62)

The mean size of EDH among study patients was 8.25 ± 3.917 mm with minimum of 3mm to maximum of 18.2mm. (Table 8)

Measure	Size in mm
Minimum	3
Maximum	18.2
Mean	8.25
Std. Deviation	3.917

Table 8: Descriptive analysis of size of EDH

About 36 (58.0%) of study patients presented with midline shift out of 62 (100%) patients. (Table 9) (Figure 9)

Midline shift	Frequency	Percent
Present	36	58.0
Absent	26	42.0
Total	62	100.0

Table 9: Distribution of patients based on presence of Midline shift

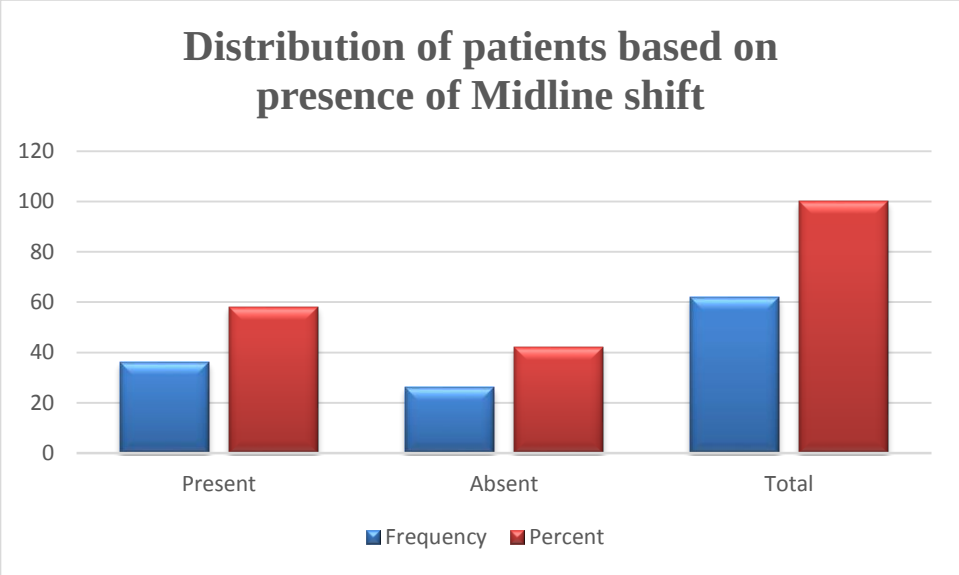


Figure 9: Cluster column chart of distribution of patients based on presence of midline shift (N=62)

Among 36 patients with midline shift, majority, 19 (52.7%) had it on left side and 17 (47.3%) had on right side. (Table 10) (Figure 10)

Midline shift	Frequency	Percent
Left	19	52.7
Right	17	47.3
Total	36	100.0

Table 10: Distribution of patients based on Side of midline shift

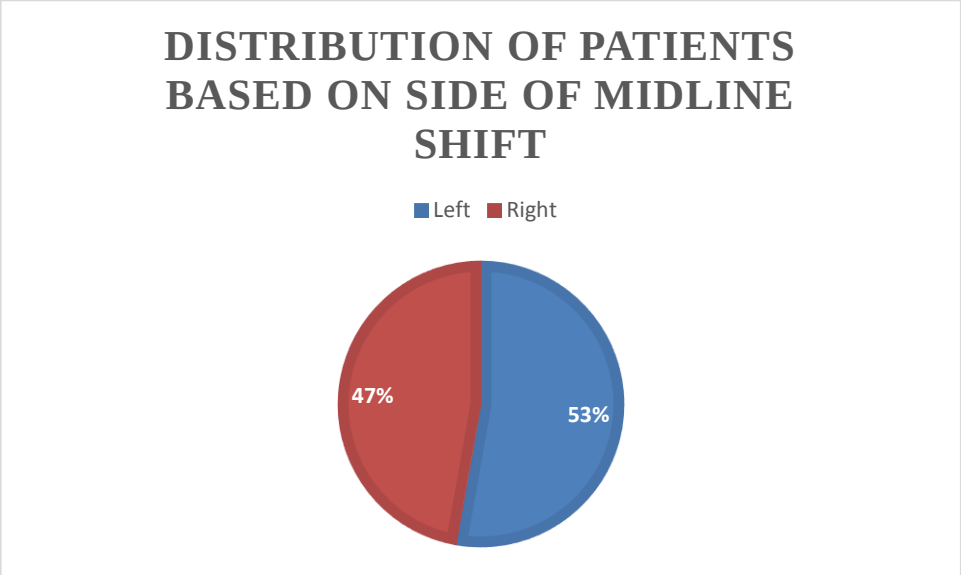


Figure 10: Pie chart of distribution of patients based on side of midline shift (N=62)

The commonest symptom among the patients was loss of consciousness 38 (61.3%) followed by vomiting 25 (40.3%) and hemiparesis 12 (19.3%). (Table 11) (Figure 11)

Symptoms	Frequency	Percent
Loss of consciousness	38	61.3
Vomiting	25	40.3
Hemi-paresis	12	19.3

Table 11: Distribution of patients based on Symptoms

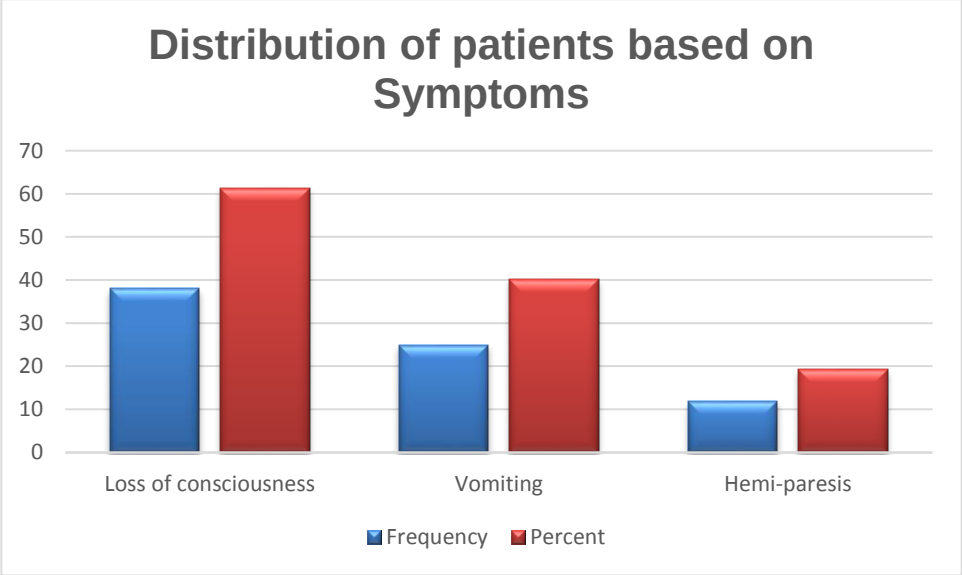


Figure 11: Cluster column chart of distribution of patients based on symptoms (N=62)

Out of 62 (100%) patients, majority 57 (92.0%) of patients had good recovery followed by 5 (8.0%) had moderate recovery. (Table 12) (Figure 12)

Recovery	Frequency	Percent
Good recovery	57	92.0
Moderate recovery	5	8.0
Total	62	100.0

Table 12: Distribution of patients based on Recovery

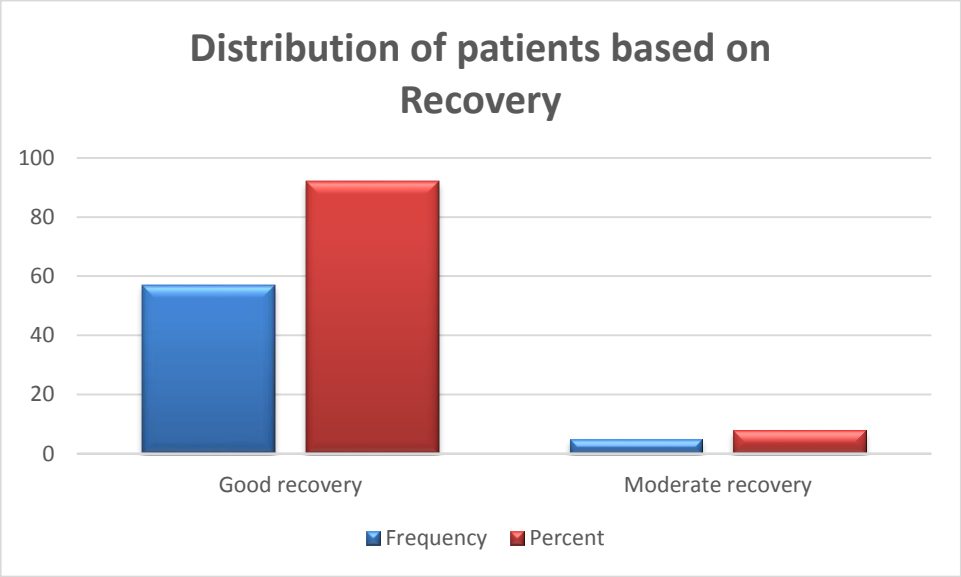


Figure 12: Cluster column chart of distribution of patients based on recover (N=62)

About 86.1% patients with and 100.0% of patients without midline shift had good recovery. The association between midline shift and recovery was found to be statistically significant. (Table 13)

Midline shift	Recovery		Total	Chi square	p
	Good	Moderate			
Present	31 (86.1)	5 (13.9)	36 (58.0)	3.928	0.047
Absent	26 (100.0)	0 (0.0)	26 (42.0)		
Total	57	5	62		

Table 13: Relationship between midline shift and recovery

The size of EDH was significantly larger among patients with midline shift compared to those who did not have midline shift. (Table 14)

Midline shift	Mean	Std. Deviation	t	p
Yes	10.95	2.53	6.775	0.001
NO	6.23	3.26		

Table 14: Relationship between midline shift and size of EDH

Patients with moderate recovery had significantly larger EDH compared to those with good recovery. (Table 15)

Recovery	Mean	Std. Deviation	t	p
Good	7.505	3.0967	6.576	0.001
Moderate	16.740	1.3145		

Table 15: Relationship between size of EDH and recovery

CASE IMAGES

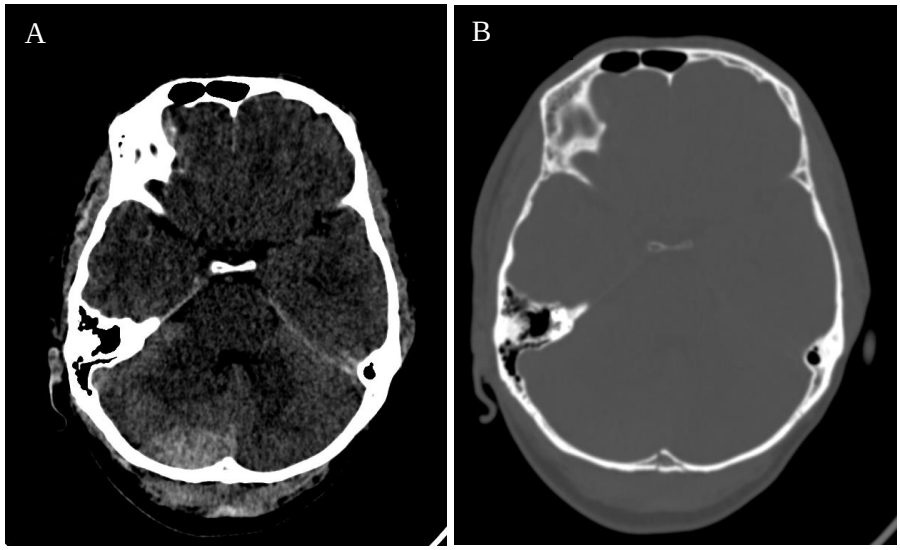


Figure 13: CT Brain plain study in a 45 years old male patient with history of Road traffic accident showing Extradural haemorrhage in right occipital region (A) and fracture of right occipital bone (B)

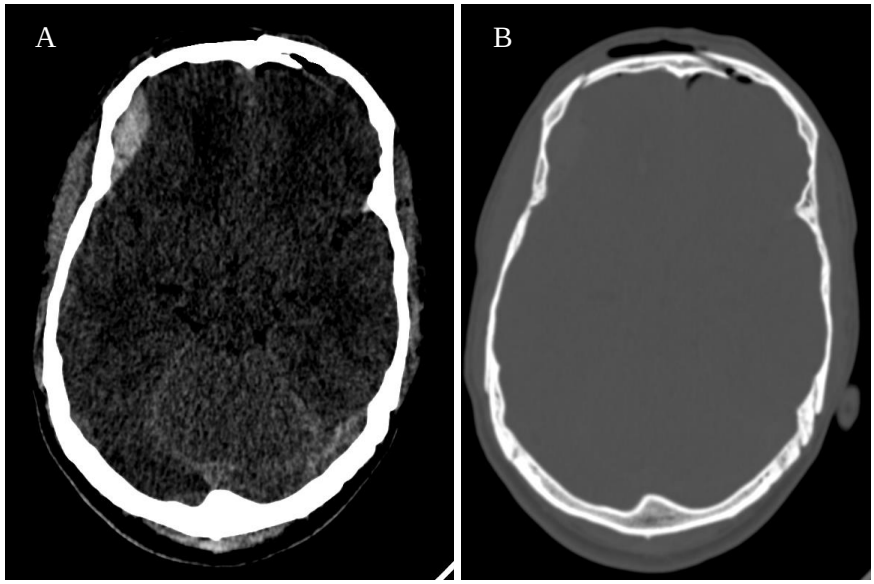


Figure 14: CT Brain plain study in a 31 years old male patient with history of Road traffic accident showing Extradural haemorrhage in right frontal region (A) and fracture of right frontal bone (B)

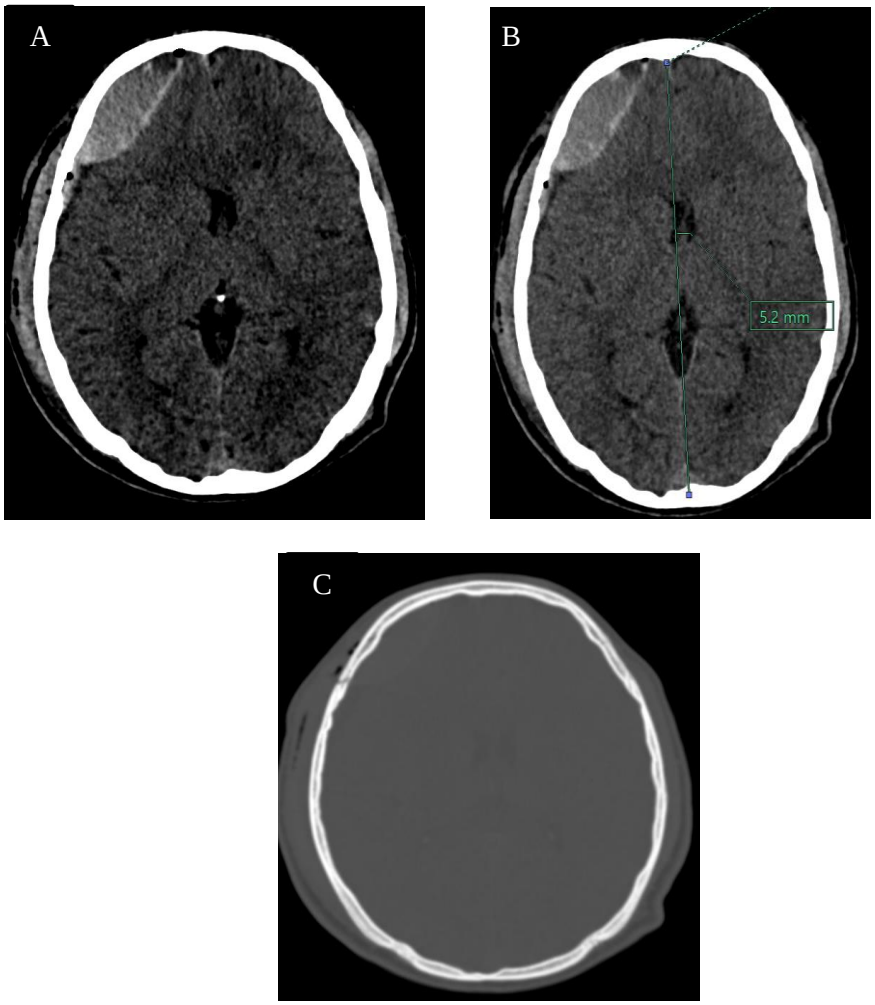


Figure 15: CT Brain plain study in a 21 years old male patient with history of Road traffic accident showing Extradural haemorrhage in right frontal region (A) with a midline shift of 5.2 mm towards left side (B) and fracture of right frontal bone (C)

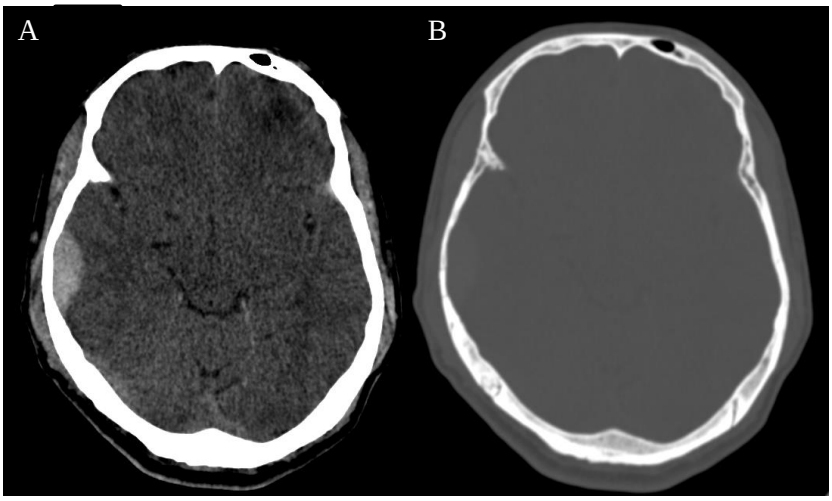


Figure 15: CT Brain plain study in a 28 years old male patient with history of Road traffic accident showing Extradural haemorrhage in right temporal region (A) and fracture of right temporal bone (B)

DISCUSSION

DISCUSSION:

Patients with traumatic brain injury are routinely diagnosed with MDCT imaging. MDCT scanning is useful for determining the severity of an injury, and multi-detector high-resolution scanners can be utilized to obtain its image. Soft tissue and bone windows might be used to view the pictures, and 3D CT sets could be used to identify skeletal and cerebral lesions.[96]

According to international surveys, middle-aged persons with head traumas made up the majority of reports of epidural bleeding. Out of 62 patients (100%) in the current study, 27 (43.6%) were in the 20 to 29 year age group, followed by 14 (22.6%) in the 30 to 39 year age group. A total of 460 individuals participated in the study by Amir et al., with a mean age of 19 years and a range of 2-55 9.59 years. Of these, 196 (42.60%) had epidural haemorrhages. The majority of the patients, 148 (75.51%), were between the ages of 15 and 44, with 40 (20.405) being under 14 years old.[97] The findings of a study carried out in India and Pakistan were comparable. This is because boys of this age group drive more than girls do in our traditional culture and society, making them more susceptible to head injuries. [98,99]

Car accidents are the most common cause of TBI, and as males participate more actively in daily activities, they are disproportionately affected. Out of 62 individuals (100%) in our study, about 55 (88.7%) were men and 7 (11.3%) were women, indicating a male predominance. This is consistent with the research by Amir et al. [97] According to the study, out of the 196 cases of epidural haemorrhage, 152 (77.55%) patients were male and 44 (32.3%) were female. 2 Studies have shown that men experience head trauma more frequently than women, which is consistent with our findings. [100 -102]

The clinical histories of all 62 (100%) patients revealed that all had a history of head injuries, with RTA being the most common mode of injury. Even all had a head injury diagnosis after RTA. Similar to our investigation, Amir et al. found that all brain injury patients had been involved in car accidents.[97] The enormous number of motor vehicles on our roads, as well as the drivers' propensity to disobey basic traffic laws and drive too fast, may be to blame for the high incidence of head injuries from road traffic incidents in this study. It is possible to hold passengers accountable for disregarding safety precautions like fastening their seatbelts. The low frequency of work-related brain injuries may indicate that employers and employees are both aware of the need of workplace safety. [97]

Since it is less expensive, quicker, and more widely accessible, CT is one of the most important modalities and is a crucial diagnostic tool for many brain hematomas.[103,104] Out of 62 (100%) patients in our study, radiographic examination revealed that 17 (27.4%) had right temporal EDH, followed by l3 (21.0%) had left temporal EDH. The most common kind of EDH identified by Kumar et al. in their study was frontal, which presented in 36% of cases, followed by temporal in 21% of instances, and temporal parietal in only 10% of cases. Compared to occipital, posterior fossa EDH manifestation was detected in 5% of cases.[105] According to Pathak A et al study, temporo-parietal EDH predominated in 43% of cases, followed by temporal in 21% of cases, and frontal in 17% of instances.[106]

In the current study, out of 62 patients (100%), 26 (41.9%) had a mild category of GCS score, followed by 22 (35.5%) for moderate, and 14 (22.6%) for severe. In 188 patients, 23 (12%) suffered serious injuries, whereas 165 (88%) had intermediate injuries, according to Tariq's research.[107] According to De Sousa[108] and M. Naisseri[109], 56.8% and 75% of their patients, respectively, had mild brain injuries. The type of

institution, private versus public and the availability of medical coverage, as well as the screening procedure utilised prior to requesting a CT scan could be responsible for this discrepancy. [107-109]

Patients in the study had an average EDH size of 8.25 3.917 mm, ranging from a minimum of 3 mm to a maximum of 18.2 mm. Mushtaq et al. also deduced from their research that the size and volume of the hematoma affect EDH result. A large volume is defined as 50 mL to 100 mL, and a small volume is 25 mL to 50 mL. [110] McKissock et al study's came to similar conclusions.[111]

Out of 62 study participants (100%) around 36 (58.0%) had midline shift. Most of the 36 individuals with midline shift, 19 (52.7%) on the left side and 17 (47.3%) on the right, had it on the left side. 17.5% of patients had midline shift, according to an earlier study.[100] Nitesh et al. found 12 cases with EDH; all of these individuals had skull fractures, and 94% of them had midline shift, which is a well-recognized finding.[112]

Loss of consciousness was the most frequent symptom among the patients, occurring in 38 (61.3%), followed by vomiting in 25 (40.3%) and hemiparesis in 12 (19.3%). In past research, similar findings were made.[108] Patients who presented with many symptoms had a tendency to have a higher diagnostic yield than patients who just had one symptom. This was more prevalent in patients who had at least one variable in their combination that was loss of consciousness. This was also observed in patients who had minor head injuries. According to Tariq et al., the parietal bone was involved in roughly 43% of fractures, the frontal bone in 24.6%, the occipital bone in 21.1%, and the temporal bone in 20.2% of patients. In 29.8% of patients with linear skull fractures, extradural hematoma was observed. In comparison to other skull bones, the parieto-temporal sections of the skull were home to 73.5% of extradural hematomas.[107] In Kumar et al study's frontal EDH predominately presented in 36% of cases, followed by

temporal EDH in 21% of cases, and only Temporal Parietal EDH in 10% of instances. Compared to occipital EDH presentation, posterior fossa EDH was present in 5% fewer cases.[105]

Out of 62 patients (100%) the majority (57, or 92.0%) recovered well, while 5 patients (8.0%) recovered moderately. Approximately 86.1% of patients with midline shift and 100.0% of patients without it made a satisfactory recovery. A statistically significant relationship between midline displacement and recovery was discovered. According to the research by Kumar et al, 79% of patients showed signs of a satisfactory recovery. 11% of patients had a moderate level of impairment. Only 10% of the cases resulted in death, a severe disability, or a vegetative state.[105]

Patients with midline shift had EDHs that were noticeably bigger than patients without midline shift. EDH was noticeably bigger in patients with intermediate recovery compared to those with strong recovery. This agrees with the research results of Tariq et al. Extradural bleeding size and volume are closely connected to the clinical and functional outcome. The prognosis deteriorates as the hematoma size grows.[107]

CONCLUSION:

From this study we conclude that neurological status of patient on presentation and the thickness of Extradural hemorrhage are the most important factors in management and outcome of Extradural hemorrhage. The size of EDH was significantly larger among patients with midline shift compared to those who did not have midline shift. Patients with moderate recovery had significantly larger EDH compared to those with good recovery.

Computed tomography is a simple, inexpensive, highly accurate and provides the ability to rapidly evaluate patients with acute head injuries. CT helps in surgical planning, result prediction, and recovery time estimation. It can demonstrate extradural hemorrhage, skull fractures and other intracranial haemorrhages.

Thus, it is justifiable to conclude that CT is and should be considered the first imaging of choice in acute head injury as it forms the cornerstone for rapid and accurate diagnosis. MDCT was found to be an accurate diagnostic tool in the evaluation of head trauma. The reduction in the scan delay time resulted in imaging more patients which greatly assisted the clinicians in instituting early appropriate therapy.

SUMMARY:

- In the present study Out of 62 (100%) majority, 25 (40.3%) of patients were in the age group of 21 to 30 years followed by 31 to 40 years 15 (24.2%).
- Out of 62 (100%) patients, about 55 (88.7%) of patients were males and 7 (11.3%) were females showing male predominance.
- The clinical history of all 62 (100%) patients showed that all had history of head injury and the mode of injury for all patients was RTA. Even all were clinically diagnosed as head injury following RTA.
- On radiological examination, out of 62 (100%) patients, majority of them, 17 (27.4%) of patients had right temporal EDH followed by l3 (21.0%) left temporal EDH.
- Among all 62 (100%) patients, the commonest site of EDH was right temporal region, 17 (27.4%) followed by l3 (21.0%) left temporal region.
- Majority of the patients out of 62 (100%), had mild category of GCS score i.e., 26 (41.9%) of patients followed by 22 (35.5%) moderate and 14 (22.6%) had severe category of GCS.
- The mean size of EDH among study patients was 8.25 ± 3.917 mm with minimum of 3mm to maximum of 18.2mm.
- About 36 (58.0%) of study patients presented with midline shift out of 62 (100%) patients.
- Among 36 patients with midline shift, majority, 19 (52.7%) had it on left side and 17 (47.3%) had on right side.
- The commonest symptom among the patients was loss of consciousness 38 (61.3%) followed by vomiting 25 (40.3%) and hemiparesis 12 (19.3%).
- Out of 62 (100%) patients, majority 57 (92.0%) of patients had good recovery

followed by 5 (8.0%) had moderate recovery.

- About 86.1% patients with and 100.0% of patients without midline shift had good recovery and 100.0% without midline shift had good recovery. The association between midline shift and recovery was found to be statistically significant.
- The size of EDH was significantly larger among patients with midline shift compared to those who did not have midline shift.
- Patients with moderate recovery had significantly larger EDH compared to those with good recovery.

BIBLIOGRAPHY

REFERENCES:

1. Riegor J, Linesenmaier U, Pfeifer M. Radiological diagnosis in acute craniocerebral trauma. Radiol, 2002; 42 (7): 547-55.

2. Udsteun GJ, Claar JM. Imaging of acute head injury in the adult. Seminar in Ultrasound CT MR, 2001; 22 (2): 135-147.

3. Kido DK, Cox C, Hamill RW, Rothenberg BM, Woolf PD. Traumatic Brain Injuries:
   Predictive Usefulness of CT. Radiol 1992; 182 (3): 777-781

4. Ramesh S. Doddamania, Sunil K. Guptaa, Navneet Singlaa, Sandeep Mohindraa, Paramjeet Singh. Role of repeat CT scans in the management of traumatic brain injury. The indian journal of Neurotrauma. 2012; 9:33-39.

5. Roberson FC, Kishore PR, Miller JD, et al. The value of serial computerized tomography in the management of severe head injury. Surg Neurol.1979; 12:161-167.

6. Lee TT, Aldana PR, Kirton OC, et al. Follow-up computerized tomography (CT) scans in moderate and severe head injuries: correlation with Glasgow Coma Scores (GCS), and complication rate. ActaNeurochir (Wien). 1997; 39:1042-48

7. Servadei F, Murray GD, Penny K, et al. The value of the "worst" computed tomographic scan in clinical studies of moderate and severe head injury. European Brain Injury Consortium. Neurosurg.2000; 46:70-77.

8. Stein SC, Fabbri A, Servadei F. Routine serial computed tomographic scans in mild traumatic brain injury: when are they cost-effective? J Trauma.2008; 65:66-72.

9. Brown CV, Zada G, Salim A, et al. Indications for routine repeat head computed tomography (CT) stratified by severity of traumatic brain injury. J Trauma. 2007; 62:1339-45.

10. Sifri ZC, Homnick AT, Vaynman A, et al. A prospective evaluation of the value of repeat cranial computed tomography in patients with minimal head injury and an intracranial bleed. J Trauma. 2006; 61:862-67.

11. Jeret JS, Mandell M, Anziska B, Lipitz M, Vilceus AP, Ware JA, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurg,1993;32:9 –15.

12. Yoshiro Toyama, Takayu Kobayashi, Yoshihiro Nishiyama, Katashi Satoh, Matoomi Ohkawa, and Keisuke Seki. CT for acute stage of closed head injury. Radiation Medicine, 2005; 23 (5):309-316.

13. Maria Mogoseanu, Magda Pascut, Florin Birsasteanu, Sorin Motoi, Adrian Tutelca, Ana Maria Vesa, Cristian Socoliuc. Computed tomography (CT) Vs Magnetic resonance imaging (MRI) in Evaluation of head injuries. TMJ, Vol. 53; 3-4.
14. CT of head trauma. Zee C S, GO JL. Dept of radiology, US, School of med, LA California 90033, USA. Neuroimaging Clin N. Am.1998; 8(3): 525-539.
15. Minor Head Injury: Guidelines for use of CT. Marion Smits et al. RSNA 2007;Vol. 245: 234-58.
16. Lee F Rogers. Helical CT: The Revolution in Imaging. Am J Roentgenol. 2003; 180-883.
17. Udsteun GJ, Claar JM. Imaging of acute head injury in the adult. Seminar in Ultrasound CT MR, 2001; 22 (2): 135-147.
18. Riegor J, Linesenmaier U, Pfeifer M. Radiological diagnosis in acute craniocerebral trauma. Radiologe, 2002 ; 42 (7) : 547-55.
19. Gentry LR, Godersky JC, Thompson B, Dunn VD. Prospective comparative study of intermediate field MR and CT evaluation of closed head trauma. Am J Roentgenol. 1988; 150: 673-82.
20. Ambrose J, Hounsfield G. Computerized transverse axial tomography. Abstracts of 32nd Annual Congress of the British Institute of Radiology. Br J Radiol 1973 ; 46 (542): 148-149.
21. New PF, Scott WR, Schnur JA, Davis KR, Taveras JM. Computerized axial tomography with EMI scanner. Radiology 1974; 110(1): 109-123.
22. Dublin AB, French B.N., Rennick J.M. Computed Tomography in head trauma. Radiology, 1977; 122: 365-369.
23. Galbraith S.. Teasdale G. Predicting the need for operation in the patient with an occult traumatic intracranial hematoma. J. Neurosourg, 1981;55: 75-81.
24. Hayman LA, Evans RA, Hinck VC. Rapid high dose contrast computed tomography of isodense subdural haematoma and cerebral swelling. Radiology 1979 ; 131 (2): 381-83.
25. Miller A, Ericson K. CT'of isoattenauting subdural haematomas. Radiology 1979 ; 130(1): 149-52.
26. Markwalder TM. Chronic subdural haematomas : A review. J Neurosurg, 1981 ; 54 (5): 637-45.
27. Smith WP, Batnitzky S., Rengachary SS. Acute isodense subdural haematomas : A problem in anemic patients. Am J Roentgenol 1981 ; 136(3) : 543-46.
28. Barmier E, Dubowitz B. Gray white matter interface displacement: a new sign in the CT diagnosis of subtle subdural haematomas. Clin Radiol 1981; 32 (4): 393-96.
29. Reed D, Robertson WD, Graeb DA, Lapointe JS, Nugent RA, Woodhurst WB. Acute subdural haematomas : A typical CT findings. Am J Neuroradiol 1986 ; 7 (3) : 417-21.
30. Baratham G, Dennyson WG. Delayed traumatic intracerebral haemorrhage. J Neurosurg Psychiatry 1972 ; 35 (5): 698-706.
31. Upper MH, Kishore PR, Girevendulis AK, Miller JD, Bcker DP. Delayed intracranial haematoma in patients with severe head injury. Radiology 1979 ; 133 (3 Pt 1): 645-49.

32. Diaz FG, Yock DH, Larson D, Rockswoid GL. Early diagnosis of delayed posttraumatic intracerebral haematomas. J Neurosurg 1979; 50 (2) : 217-23.
33. Levi L, Guilburd JN, Lemberger A, Soustiel JF, Feinsod M. Diffuse Axonal Injury : Analysis of 100 patients with Radiological signs. J Neurosurg 1990 ; 27 (3) : 429-432.
34. Kido DK, Cox C, Hamill RW, Rothenberg BM, Woolf PD. Traumatic Brain Injuries : Predictive Usefulness of CT. Radiology 1992 ; 182 (3): 777-781.
35. Lolli V, Pezzullo M, Delpierre I, Sadeghi N. MDCT imaging of traumatic brain injury. Br J Radiol. 2016;89 (1061):201-89.
36. Arfat M, Zahiruddin MD, Yadav Y, Brig T, Prabhakar V, Gupta AK. Evaluation of epidemiological trends and severity of traumatic brain injury using multi-detector computed tomography scanner in Uttar Pradesh University of Medical Sciences Hospital. OMICS J Radiol. 2017;6(254):221-36.
37. Nithesh N, Ravindranand RC. Role of Multi Detector Computed Tomography in Evaluating Traumatic Brain Injuries. Annals of the Romanian Society for Cell Biology. 2021:874-83.
38. Kumar CS, Prasad KS, Rajasekhar B, Raman BV. A study on various clinical presentations of extradural hemorrhage, factors affecting treatment and early outcome. Int J Res Med Sci. 2017;5(4):1288-93.
39. Nagesh M, Patel KR, Mishra A, Yeole U, Prabhuraj AR, Shukla D. Role of repeat CT in mild to moderate head injury: an institutional study. Neurosurg Foc. 2019;47(5):239-46.
40. Jagdish P, Chaturbhuj S, Naima M, Meena GL. Role of Computerized Tomography in Craniocerebral Trauma. J med sci. 2017;5(03):185-96.
41. Ramanana-Rao DV, NVP D, Chandra TJ. Role of Computed Tomography (CT) in traumatic head injury evaluation–a cross-sectional study. Int J Res Med Sci. 2020; 7(3): 129-36.
42. Zimmerman RA, Bilaniuk LT, Hackney DB, Goldberg Hi, Grossman Rl. Head injury : Early results of comparing Ct and high field MR. Am J Neuroradiology 1986 ; 147(6) : 1215-22.
43. Gentry LR, Godersky JC, Thompson B, Dunn VD. Prospective comparative study of intermediate field MR and CT evaluation of closed head trauma. Am J Roentgenol. 1988; 150: 673-82.
44. Van der Naalt J, Hew JM, van Zomeran H, Sluter WJ, Minderhoud JM. CT and MRI in mild to moderate head injury : Early and late imaging related to outcome. Ann Neurol 1999 ; 46 (1): 70-78.
45. National Institute for Clinical Excellence (NICE).Head injury: triage, assessment, investigation and early management of head injury in infants, children and adults. London (UK); 2003 Jun. 248 p. [373 references].
46. Susan Mayor. NICE recommends greater use of CT imaging for head injuries. Br Med Journal. (2003); 326:141-54.
47. Suzanne Laughlin, Walter Montanera, When is CT more appropriate than MRI? Postgraduate Medicine. 1998; 104 (5): 39-54.

48. Reed MJ, Browning JG, Wilkinson AG, Beattie T. Archives of disease in childhood: 2005; 859-64.
49. Besenski N. Traumatic injuries: imaging of head injuries. Eur Radiol. 2002 Jun;12(6):1237-52.
50. Curry TS, Christensen's. Physics of Diagnostic Radiology: Computed Tomography. Philadelphia: Lea and Febiger; 1990.
51. Fishmar ED, Jeffrey RB. Basic principles of spiral CT: Spiral CT Principles, Techniques and Clinical Application. New York: Raven Press; 1995.
52. Reuben AD, Wattsmith SR, Dobson D, Golding SJ. A comparative study of evaluating of radiographs, CT and 3D reformatted CT in facial trauma: What is the role of 3D. Br J Radiol. 2005; 78: 198-201.
53. Theory of spiral CT. Marconi Medical Systems. 1994
54. Arenson JS, Levinson R, Freundlich D: Dual slice scanner. U.S. patent no 5 228069. 1993.
55. Goss C: Gray's anatomy. 29th edition. Philadelphia. Lea and Febiger, 1973.
56. Hanaway J, Scott W, Strother C: Atlas of the human brain and the orbit for computed tomography. St. Louis, Warren Green, 1997.
57. Masdeu JC, Grossman BC: Head and spine imaging. New York, John Wiley and Sons, 1985.
58. Matsui T, Hirano A: Atlas of the human brain for computed tomography. New York, Igaku-Shoin, 1978.
59. Osborn AG. "Craniocerebral trauma". In Diagnostic Neuroradiology; USA: Mosby Year Book, 1994: 199-246.
60. Povlishock JT. Pathobiology of traumatically induced axonal injury in animal and man. Ann Emerg Med 1993; 22 (6): 980-986.
61. Povlishock JT. Traumatically induced axonal injury Pathogenesis and pathobiological implications. Brain Pathology 199; 2 (1): 1-12.
62. Chestnut RM. Management of severe traumatic brain injury. Emerg Med Clin North Am 1997; 15 (3): 581-640.
63. Jenkins LW, Moszynski K, Lyeth BG, Leyth BG, Lewelt W, De Witi DS et al. Increased vulnerability of the mildly traumatized rat brain to cerebral ischaemia: The use of controlled secondary Ischaemia as a research tool to identify common or different mechanisms contributing to mechanical and ischaemic brain injury. Brain Res 1989: 477(1-2): 211-24.
64. Bouma GJ, Muizelaar JP, Choi SC, Newlon PG, Young HF. Cerebral circulation and metabolism after severe traumatic injury. The elusive role of ischaemia. J Neurosurg 1991; 75 (5): 685-93.
65. Kohli R, Chaturvedi S. Epidemiology and Clinical Manifestations of Immune Thrombocytopenia. Hamostaseologie. 2019; 39(3):238-49.
66. Chen SH, McCarthy DJ, Sheinberg D, Hanel R, Sur S, Jabbour P, Atallah E, Chalouhi N, Dumont A, Amenta P, Hasan D, Raper D, Liu K, Jane JA, Crowley RW, Aguilar-Salinas P, Bentley J, Monteith S, Mitchell BD, Yavagal DR, Peterson EC, Starke RM. Pipeline Embolization Device for the Treatment of Intracranial Pseudoaneurysms. World Neurosurg. 2019; 127:86-93.

67. Best JG, Bell R, Haque M, Chandratheva A, Werring DJ. Atrial fibrillation and stroke: a practical guide. Pract Neurol. 2019;19(3):208-24.
68. Babu JM, Patel SA, Palumbo MA, Daniels AH. Spinal Emergencies in Primary Care Practice. Am J Med. 2019;132(3):300-06.
69. Kanematsu R, Hanakita J, Takahashi T, Park S, Minami M. Radiologic Features and Clinical Course of Chronic Spinal Epidural Hematoma: Report of 4 Cases and Literature Review. World Neurosurg. 2018;120:82-9.
70. Joubert C, Gazzola S, Sellier A, Dagain A. Acute idiopathic spinal subdural hematoma: What to do in an emergency? Neurochirurgie. 2019;65(2-3):93-97.
71. Spetzler RF, McDougall CG, Zabramski JM, Albuquerque FC, Hills NK, Nakaji P, Karis JP, Wallace RC. Ten-year analysis of saccular aneurysms in the Barrow Ruptured Aneurysm Trial. J Neurosurg. 2019;132(3):771-76.
72. Angele MK, Schwacha MG, Smail N, Catania RA, Ayala A, Cioffi WG, Chaudry IH. Hypoxemia in the absence of blood loss upregulates iNOS expression and activity in macrophages. American Journal of Physiology-Cell Physiology. 1999;276(2):C285-90.
73. Swartz MC, Lewis ZH, Deer RR, Stahl AL, Swartz MD, Christopherson U, Basen-Engquist K, Wells SJ, Silva HC, Lyons EJ. Feasibility and Acceptability of an Active Video Game–Based Physical Activity Support Group (Pink Warrior) for Survivors of Breast Cancer: Randomized Controlled Pilot Trial. JMIR cancer. 2022;8(3):368-89.
74. Burjorjee JE, Rooney R, Jaeger M. Epidural hematoma following cessation of a direct oral anticoagulant: a case report. Regional Anesthesia & Pain Medicine. 2018;43(3):313-16.
75. Bonow RH, Barber J, Temkin NR, Videtta W, Rondina C, Petroni G, Lujan S, Alanis V, La Fuente G, Lavadenz A, Merida R. The outcome of severe traumatic brain injury in Latin America. World neurosurgery. 2018;111:82-90.
76. Baker DG. Multiple sclerosis and thermoregulatory dysfunction. Journal of Applied Physiology. 2002;92(5):1779-80.
77. Grusin H, Kincaid-Smith PS. Scurvy in adult Africans. A clinical, haematological, and pathological study. American Journal of Clinical Nutrition. 1954;2:323-35.
78. Flaherty BF, Moore HE, Riva-Cambrin J, Bratton SL. Repeat head CT for expectant management of traumatic epidural hematoma. Pediatrics. 2018;142-43.
79. Ma Y, Wang P, Kuebler JF, Chaudry IH, Messina JL. Hemorrhage induces the rapid development of hepatic insulin resistance. American Journal of Physiology-Gastrointestinal and Liver Physiology. 2003;284(1):107-15.
80. Osborn A. Trauma overview. In: Osborn's brain: imaging, pathology, and anatomy. 1st edn. Manitoba, Canada: Amirsys; 2013:1–8.
81. Le TH, Gean AD. Imaging of head trauma. Semin Roentgenol. 2006; 41: 177–89.
82. Bhorkar NM, Dhansura TS, Tarawade UB, Mehta SS. Epidural hematoma: vigilance beyond guidelines. Ind Jrnl of Crl Care Med. 2018;22(7):555-59.
83. Gutowski P, Meier U, Rohde V, Lemcke J, von der Brelie C. Clinical outcome of epidural hematoma treated surgically in the era of modern resuscitation and trauma care. World neurosurg. 2018;118:166-74.

84. Basamh M, Robert A, Lamoureux J, Saluja RS, Marcoux J. Epidural hematoma treated conservatively: when to expect the worst. Cnd J Neurol Sci. 2016;43(1):74-81.
85. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet. 1974;2(7872):81-4.
86. Teasdale G, Murray G, Parker L, Jennett B. Adding up the Glasgow Coma Score. Acta Neurochir Suppl (Wien). 1979;28(1):13-6.
87. Teasdale GM, Drake CG, Hunt W, Kassell N, Sano K, Pertuiset B, De Villiers JC. A universal subarachnoid hemorrhage scale: report of a committee of the World Federation of Neurosurgical Societies. J Neurol Neurosurg Psychiatry. 1988;51(11):1457-61.
88. Teasdale G, Maas A, Lecky F, Manley G, Stocchetti N, Murray G. The Glasgow Coma Scale at 40 years: standing the test of time. Lancet Neurol. 2014;13(8):844-54.
89. Grinnon ST, Miller K, Marler JR, Lu Y, Stout A, Odenkirchen J, Kunitz S. National Institute of Neurological Disorders and Stroke Common Data Element Project - approach and methods. Clin Trials. 2012;9(3):322-29.
90. Borgialli DA, Mahajan P, Hoyle JD, Powell EC, Nadel FM, Tunik MG, Foerster A, Dong L, Miskin M, Dayan PS, Holmes JF, Kuppermann N., Pediatric Emergency Care Applied Research Network (PECARN). Performance of the Pediatric Glasgow Coma Scale Score in the Evaluation of Children With Blunt Head Trauma. Acad Emerg Med. 2016;23(8):878-84.
91. Reith FCM, Lingsma HF, Gabbe BJ, Lecky FE, Roberts I, Maas AIR. Differential effects of the Glasgow Coma Scale Score and its Components: An analysis of 54,069 patients with traumatic brain injury. Injury. 2017;48(9):1932-43.
92. Gennarelli TA, Champion HR, Copes WS, Sacco WJ. Comparison of mortality, morbidity, and severity of 59,713 head injured patients with 114,447 patients with extracranial injuries. J Trauma. 1994;37(6):962-8.
93. Steyerberg EW, Mushkudiani N, Perel P, Butcher I, Lu J, McHugh GS, Murray GD, Marmarou A, Roberts I, Habbema JD, Maas AI. Predicting outcome after traumatic brain injury: development and international validation of prognostic scores based on admission characteristics. PLoS Med. 2008;5(8):165-69.
94. MRC CRASH Trial Collaborators. Perel P, Arango M, Clayton T, Edwards P, Komolafe E, Poccock S, Roberts I, Shakur H, Steyerberg E, Yutthakasemsunt S. Predicting outcome after traumatic brain injury: practical prognostic models based on large cohort of international patients. BMJ. 2008;336(7641):425-29.
95. Badhe U, Bhamre S. Clinical Evaluation and Management Outcome of Extradural Haematoma. MVP Journal of Medical Sciences. 2018;5:49-54.

96. Coles JP (2007) Imaging Dier brain injury. Br J Anaesth 99: 49-60.
97. Amir S, Noman MA, Ayub S. Epidural Hematoma in Different Ages Due to Road Traffic Accidents on Computed Tomography Scan Brain. JGMDS. 2022;9(2):13-6.

98. Amir A, Khalil S, Huda H, Fak M. Road traffic injuries among drivers and pillion riders using two-wheeler motorized vehicle in Western Uttar Pradesh. J Clin Diagn Res. 2018;12(8)
99. Riaz MA, Riaz MA, Tabussam S, Rehman H. Frequency of subdural hematoma in different ages due to motor vehicle mishaps on computed tomography scan brain. Pak J Med Health Sci. 2018;12(4):1318-20.
100. Sidpra J, Ong J, Birch W, Mankad K. Skull fractures in abusive head trauma: a single centre experience and review of the literature. Child Nerv Syst. 2021;37(3):919-29.
101. Williams V, Jayashree M, Bansal A, Baranwal A, Nallasamy K, Singhi SC, et al. Spontaneous intracranial haemorrhage in children-intensive care needs and predictors of inhospital mortality: a 10-year single centre experience. Child Nerv Syst. 2019;35(8):1371-9.
102. Kulesza B, Mazurek M, Rams L, Nogalski A. Acute epidural and subdural hematomas after head injury: clinical distinguishing features. Indian J Surg. 2020;83(1):96-104.
103. Obanife HO, Nasiru IJ, Ogunleye OO, Ahmad M, Otorkpa EJ, Shehu BB. Severity and predictors of outcome of motorcycle-associated head injury: an experience from a regional neurosurgery centre in northern Nigeria. World Neurosurg. 2021.
104. Anuradha B, Pasam KL, Rani KR, Rani BS, Rao KS. Role of CT in localising the injury to the particular intracranial compartment in patients with craniocerebral trauma. JEBMH. 2016;3(91):4981-6.
105. Kumar CS, Prasad KS, Rajasekhar B, Raman BV. A study on various clinical presentations of extradural hemorrhage, factors affecting treatment and early outcome. Int J Res Med Sci. 2017;5(4):1288-93.
106. Pathak A, Desania NL, Verma R. Profile of road traffic accidents & head injury in Jaipur (Rajasthan). J Indian academy of forensic medicine. 2008;30(1):6-9.
107. Tariq M, ahmad Mian M, Ali M, Ayub S, Khan WD, Khan SD. Outcome of the Traumatic Extradural Hematoma on the Basis of Size of Extradural Hematoma. Pak Jrl Of NeuroSurg. 2021;25(2):134-42.
108. De Sousa John. Computerized Tomography in Acute Head Injury. The Nairobi Experience. MMed Thesis 1992.
109. Naseri M, Tomasian A, Moghaddas AR. Correlation of CT scan findings with the level of consciousness in acute head trauma. Iran J Rad. 2005;2(3&4).
110. Rehman R, Mushtaq M, Haq MI, Azam F, Khattak A. Skull fracture on x-ray skull as indicator of extradural hematoma in patient with head injury. Pak J Surg. 2012;28(2):106-9.
111. Mckissock W, Richardson A, Taylor J. Primary intracerebral haemorrhage: a controlled trial of surgical and conservative treatment in 180 unselected cases. The Lancet, 1961; 278 (7196): 221-6.
112. Nithesh N, Ravindranand RC. Role of Multi Detector Computed Tomography in Evaluating Traumatic Brain Injuries. Annals of the Romanian Society for Cell Biology. 2021:874-83.

ANNEXURES

STUDY PROFORMA

Demographic details:

Name:
Age:
Sex:
Address:

Clinical History:

Clinical Diagnosis:

Radiological diagnosis:

PROFORMA

HOSPITAL NO.	MODE OF INJURY	SITE OF EDH	SIZE OF EDH	GCS

	YES	NO
FRACTURE		
MIDLINE SHIFT		
LOSS OF CONSCIOUSNESS		
ANISOCORIA		
VOMITING		
HEADACHE		
CONVULSIONS		
FACIAL WEAKNESS		
HEMI-PARESIS		

Follow up:

GLASSGOW OUTCOME SCORE	
Good recovery	
Moderate disability	
Severe disability	
Vegetative state	
Death	

INFORMED CONSENT FORM

PG guide's name: Dr. DEEPTI NAIK

Principal investigator: Dr. AVS NIKHILENDRA REDDY

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is "MULTIDETECTOR COMPUTED TOMOGRAPHY EVALUATION IN TRAUMATIC EXTRADURAL HEMORRHAGE WITH NEUROLOGICAL CORRELATION AND FOLLOW UP " I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

PATIENT INFORMATION SHEET

Principal Investigator: Dr. AVS NIKHILENDRA REDDY.

STUDY SITE: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

This is to inform you that, you require CT brain plain for making treatment plan for you condition that is extradural hemorrhage. The CT Brain plain is required for the making the diagnosis of the disease extent of the disease and for planning of the treatment. The patient with history of trauma referred to department of Radiology at R.L Jalappa hospital and research Centre, Tamaka, Kolar to undergo CT Brain plain as a part of protocol and of those patients who meet the inclusion criteria will be taken for the study.

We are conducting this study to predict the onset and severity of this condition.

If you are willing you will be enrolled in this study and we will do CT Brain plain and other relevant investigations which are required for surgical procedures.

You will receive the standard care pre and post operatively.

This will facilitate identifying EXTRA DURAL HEMORRHAGE AND ITS COMPLICATIONS (if any)in an early stage and treating it. It will also benefit other patients with extradural hemorrhage undergoing surgery in future. You are free to opt-out of the study at any time if you are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study. The study will not add any risk or financial burden to you if you are part of the study. In case of any complication during surgery patient will be treated accordingly.

Your identity and clinical details will be confidential. You will not receive any financial benefit for being part of the study. You are free to contact Dr. ALURU

VENKATA SAI NIKHILENDRA REDDY or any other member of the above research team for any doubt or clarification you have.

Dr. ALURU VENKATA SAI NIKHILENDRA REDDY

Mobile no: 7829972849

E-mail id: Nikhil.endra01@gmail.com

MASTER SHEET

Sl. No	Hospital number	Age	Sex	Site Of EDH	Size of EDH	Midline shift	Loss of consciousness	Vomiting	Hemi-paresis	Good recovery	Moderate disability
1	892635	20 y	M	Left temporal	12 mm	6.0 mm	Yes	Yes	No	Yes	
2	889727	56 y	M	Left temporal	7 mm	4.5 mm	Yes	No	Yes	Yes	
3	893564	38 y	M	Right parietal	5 mm	No	Yes	No	No	Yes	
4	894121	35 y	M	Right temporal	5 mm	No	Yes	No	No	Yes	
5	897068	93 y	M	Left parietal	4.7 mm	No	No	No	No	Yes	
6	894346	24 y	M	Left parietal	6 mm	No	Yes	Yes	No	Yes	
7	908052	25 y	M	Left temporal	3.5 mm	No	No	No	No	Yes	
8	909682	40 y	M	Right temporal	12 mm	5.9 mm	Yes	Yes	No	Yes	
9	913879	28 y	M	Right temporal	6.8 mm	3.2 mm	Yes	No	No	Yes	
10	916039	35 y	M	Left temporal	13.7 mm	6.9 mm	Yes	Yes	Yes	Yes	
11	916764	45 y	M	Left frontal	6.0 mm	No	Yes	No	No	Yes	
12	920788	65 y	M	Right frontal	18.2 mm	9.1 mm	Yes	Yes	Yes		Yes
13	924697	39 y	M	Left parietal	6.4 mm	3.1 mm	Yes	Yes	No	Yes	
14	925751	29 y	F	Right parietal	6 mm	No	Yes	No	No	Yes	

15	928524	28y	F	Right temporal	3.3mm	No	No	No	No	Yes	
16	930336	30y	M	Right temporal	6mm	3.5 mm	Yes	No	No	Yes	
17	933010	23y	M	Right frontal	5.8mm	No	Yes	No	No	Yes	
18	933971	25y	M	Left frontal	11mm	5.8 mm	Yes	Yes	No	Yes	
19	944386	58y	M	Right parietal	15.2mm	6.5 mm	Yes	Yes	Yes		Yes
20	944901	26y	M	Left parietal	4.5mm	No	No	No	No	Yes	
21	944305	20y	M	Right parietal	4.9mm	No	No	No	No	Yes	
22	947490	21y	F	Left parietal	3.9mm	No	No	No	No	Yes	
23	41533	26y	M	Left temporal	5.9mm	No	No	No	No	Yes	
24	42026	23y	M	Right frontal	6.1mm	3.0 mm	No	No	No	Yes	
25	56645	20y	M	Left parietal	11mm	5.9 mm	Yes	Yes	No	Yes	
26	56668	36y	M	Left temporal	6mm	No	No	No	No	Yes	
27	59050	26y	M	Left temporal	7mm	3.9 mm	Yes	Yes	No	Yes	
28	53838	22y	M	Right temporal	12mm	6.9 mm	Yes	Yes	Yes	Yes	
29	13907	25y	F	Right temporal	6mm	3.1 mm	No	No	No	Yes	
30	61332	35y	M	Left parietal	5.3mm	No	No	No	No	Yes	
31	62263	26y	M	Right temporal	4.0mm	No	No	No	No	Yes	

32	62442	50 y	M	Left temporal	5.7 mm	No	No	No	No	Yes	
33	62691	27 y	M	Left temporal	12.0 mm	6.4 mm	Yes	Yes	Yes	Yes	
34	65232	35 y	M	Right occipital	4.5 mm	No	No	No	No	Yes	
35	945522	45 y	M	Left temporal	12 mm	6.5 mm	Yes	No	No	Yes	
36	945849	35 y	m	Right parietal	3 mm	No	No	No	No	Yes	
37	947094	24 y	M	Right temporal	10 mm	5.5 mm	Yes	Yes	Yes	Yes	
38	952547	50 y	F	Right frontal	4.7 mm	No	No	No	No	Yes	
39	44929	45 y	F	Right frontal	7.0 mm	3.6 mm	No	No	No	Yes	
40	42088	42 y	M	Right frontal	9.0 mm	4.9 mm	No	No	No	Yes	
41	50484	73 y	M	Right temporal	5.0 mm	No	Yes	Yes	No	Yes	
42	936189	21 y	M	Left frontal	6.0 mm	No	No	No	No	Yes	
43	918774	37 y	M	Left temporal	11.0 mm	6.5 mm	Yes	Yes	Yes	Yes	
44	924729	42 y	M	Right temporal	4.2 mm	No	No	No	No	Yes	
45	74617	29 y	M	Right temporal	13.3 mm	6.8 mm	Yes	Yes	No	Yes	
46	74905	22 y	M	Right temporal	18.0 mm	8.5 mm	Yes	Yes	Yes		Yes
47	75275	21 y	M	Right frontal	10.5 mm	5.2 mm	Yes	Yes	No	Yes	

48	75352	46 y	M	Left occipital	11.3 mm	5.8 mm	Yes	No	No	Yes	
49	76274	50 y	F	Left frontal	5.5 mm	No	Yes	No	No	Yes	
50	77763	28 y	M	Right parietal	8.7 mm	4.2 mm	No	No	No	Yes	
51	78652	40 y	M	Right temporal	14.7 mm	7.1 mm	Yes	No	No	Yes	
52	84153	30 y	M	Left frontal	10.0 mm	5.0 mm	Yes	Yes	No	Yes	
53	81688	35 y	M	Right temporal	5.7 mm	No	Yes	Yes	No	Yes	
54	100678	48 y	M	Left temporal	6.0 mm	No	No	No	No	Yes	
55	104256	40 y	M	Right frontal	7.0 mm	3.8 mm	Yes	No	No	Yes	
56	107995	52 y	M	Right temporal	7.9 mm	4.3 mm	Yes	Yes	No	Yes	
57	111284	20 y	M	Right frontal	16.4 mm	9.2 mm	Yes	Yes	Yes		Yes
58	111955	45 y	M	Right occipital	15.9 mm	9.5 mm	Yes	Yes	Yes		Yes
59	112682	37 y	M	Right frontal	12.2 mm	6.3 mm	Yes	Yes	No	Yes	
60	118999	18 y	M	Left temporal	11.5 mm	5.8 mm	No	No	No	Yes	
61	121535	31 y	M	Right frontal	14.5 mm	6.5 mm	Yes	Yes	Yes	Yes	
62	100349	24 y	M	Right temporal	8.0 mm	4.0 mm	No	No	No	Yes	