"ASSOCIATION OF ISOLATED LOSS OF CONSCIOUSNESS AND CT FINDINGS IN TRAUMATIC BRAIN INJURY."

BY

Dr. HARI KRISHNAN G

DISSERTATION SUBMITTED TO

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR – 586103

IN PARTIAL FULFILLMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE IN EMERGENCY MEDICINE,

Under the Guidance of

DR. RAVI B PATIL

PROFESSOR AND HOD,

DEPARTMENT OF EMERGENCY MEDICINE,

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL

MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE

VIJAYAPUR

DOI 10.5281/zenodo.15493807
https://zenodo.org/records/15493808

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled "**ASSOCIATION OF ISOLATED LOSSOF CONSCIOUSNESS AND CT FINDINGS IN TRAUMATIC BRAIN INJURY.**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. RAVI B PATIL** Professor and HOD, Dept of Emergency Medicine, Shri B.M. Patil Medical College, Vijayapura, Karnataka.

Dr Hari krishnan g

Post Graduate student**,**

Department of Emergency Medicine.

BLDE (Deemed to be University)

Shri B M Patil Medical College, Hospital

And Research Centre, Vijayapura

Date:

Place: Vijayapura

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &

RESEARCH CENTRE VIJAYAPUR

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled "**ASSOCIATION OF LOSS OF CONSCIOUSNESS AND CT FINDINGS IN TRAUMATIC BRAIN INJURY.".**" is a bonafide and genuine research work carried out by Dr. Hari krishnan. In partial fulfillment of the requirement for the degree of MD in Emergency Medicine.

Date:
Place:- Vijayapura

DR RAVI PATIL
GUIDE
PROFESSOR AND HOD
DEPARTMENT OF EMERGENCY
MEDICNE

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

ENDORSEMENT BY THE HOD & PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled "**ASSOCIATION OF ISOLATED LOSS OF CONSCIOUSNESS AND CT FINDINGS IN TRAUMATIC BRAIN INJURY."** is a bonafide research work done by **Dr. Hari Krishnan** under the guidance of **Dr. Ravi B Patil** Professor, Department of Emergency Medicine, Shri B.M. Patil Medical College, Vijayapura.

Seal & Signature of HOD of Emergency Medicine	Seal & Signature of The principal
DR Ravi B Patil **M.D (Emergency Medicine)** BLDEU's Shri B.M. Patil Medical College, Hospital & Research Centre, Vijayapura	**DR Aravind V Patil** **M.S (General Surgery)** BLDEU's Shri B.M. Patil Medical College, Hospital & Research Centre, Vijayapura
Date: 31-JUL-2024	Date: 31-JUL-2024
Place: Vijayapura	Place: Vijayapura

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE VIJAYAPUR

COPYRIGHT DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE University, Karnataka shall have the right to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic / research purposes.

Dr. Hari Krishnan
Post Graduate Student,
Department of Emergency Medicine,
Shri B.M. Patil Medical College, Vijayapura.

Date: 31-JUL-2024

Place: Vijayapura

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE VIJAYAPUR

ACKNOWLEDGEMENT

I have got wordless to express my deep sense of gratitude and regards to my guide **Dr. RAVI B PATIL,** Professor and HOD of Emergency Medicine, under whose inspiring guidance & supervision, I am studying and continuing to learn the art of medicine. His deep knowledge, devotion to work, and zeal for scientific research make him a source of inspiration not only for me but for others too. It is because of his generous help, expert and vigilant supervision, that has guided & helped me to bring out this work in the present form.

My sincere thanks are due to **Dr. ARVIND PATIL,** Principal, Shri B.M. Patil Medical College, Vijayapura for permitting me to conduct this study.

I wish to acknowledge and express my gratitude to **Dr. BABU KATTIMANI,** MS Professor, Department of Emergency Medicine and **Dr. UDAYKUMAR KHASAGE** MD Associate Professor, Department of Emergency Medicine sincere thanks to them for their expert and vigilant supervision and timely advice who have enriched me with their knowledge and experience.

My sincere thanks to all the staff members of Department of Emergency Medicine, Shri B M Patil Medical College Hospital and Research Centre, Vijayapura. I am thankful for the support extend by my senior and junior colleagues.

I would be failing in my duty, if I would not acknowledge my thanks to all the patients and their attenders who were kind enough to help for this study.

I would also like to thank my parents without their constant encouragement & moral support, my studies would have been a distant dream.

I would like to express appreciation to my Seniors and Juniors who spent time and were always present for my support and encouragement during the course of this dissertation and for excellent cooperation at all times.

Finally, I would like to thank the **Almighty** who gave me the energy, skill, and enthusiasm to complete this as well as the other tasks in my life & also for continuing to shower their blessings upon me.

Dr. Hari Krishnan.

ABSTRACT

INTRODUCTION: Traumatic brain injury can present in various ways, with high rates of energy transfer leading to more damage. Routine CT imaging for mild head trauma with only loss of consciousness may not be necessary. Falls are the most common cause of TBI, and a GCS score of less than 15 and LOC are strong predictors of CT abnormalities. The study emphasizes the importance of prioritizing GCS scores and significant predictors in clinical decisions to reduce unnecessary radiation exposure. Future research should focus on tailoring prevention and treatment strategies for different population subgroups within the study cohort.

OBJECTIVES: is to study corelation between isolated loc in traumatic brain injury

TYPE OF STUDY: This is a observational study to be conducted in the EMERGENCY DEPARTMENT of BLDE(DU), Shri B.M Patil Medical College Hospital, Vijayapur , Karnataka.

STUDY PERIOD: Period of 21 months (August 2022 to April 2024).

STUDY POPULATION: Patients coming to Emergency Department BLDE, Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura from August 2022 to April 2024 who met the inclusion Criteria.

METHODOLOGY:

1. Classification: Patients with Traumatic Brain Injury (TBI) are divided into two groups:
    - LOC Group: Patients who experienced Loss of Consciousness (LOC).
    - NON-LOC Group: Patients who did not experience LOC.
2. . Later CT Scan Positivity was assesed
    - For each of these two groups (LOC and NON-LOC), you identify the CT scan positivity rates.
    - You calculate the p-value and odds ratio to determine the association between LOC and CT scan positivity.
3. Classification on basis of GCS:
    - Patients are reclassified based on their Glasgow Coma Scale (GCS) status:
        - GCS < 15 with LOC: Patients with a GCS score less than 15 who experienced LOC.
        - GCS 15 with LOC: Patients with a GCS score of 15 who experienced LOC.
        - For each of these GCS-based subgroups, you calculate the p-value and odds ratio for CT scan positivity.
4. Comparison:
    - You compare the p-values and odds ratios from the second classification (based on GCS and LOC) with those from the first classification (based only on LOC).

CONCLUSION: This study highlights the associations between Glasgow Coma Scale (GCS) scores, loss of consciousness (LOC), and positive CT scan findings in 145 participants with head trauma. Lower GCS scores and the presence of LOC were linked to higher rates of CT abnormalities. However, the overall prevalence of positive CT findings was low (3.5%), with only 7.4% of participants with LOC showing abnormalities. Symptoms like vomiting and headache, and demographic factors such as age, were not significant predictors of CT abnormalities. Given the low rate of positive CT findings and significant radiation exposure from CT scans, the study suggests that routine CT imaging for patients with mild head trauma and only LOC may not be necessary. Prioritizing GCS scores and other significant predictors in clinical decisions can reduce unnecessary radiation exposure and improve patient safety and resource utilization in emergency settings. This evidence-based approach ensures CT scans are reserved for higher-risk patients, enhancing diagnostic accuracy and patient care.

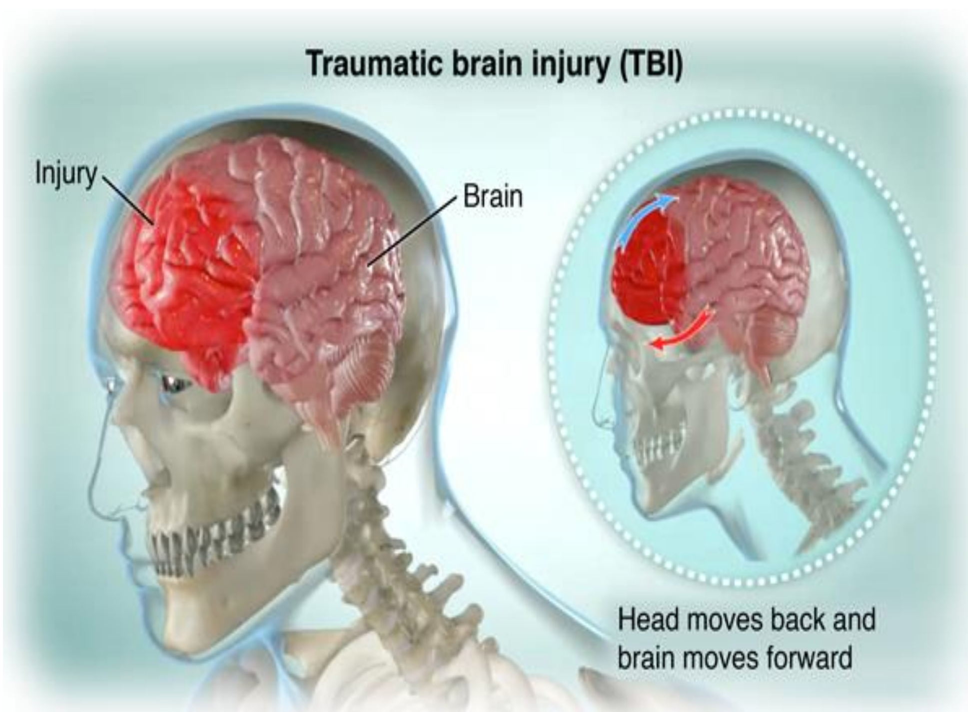
TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Traumatic brain injury (TBI) can manifest in a variety of ways, from slight changes in consciousness to a persistent state of unconsciousness and even death. In the most severe kind of traumatic brain injury, there is widespread damage and oedema throughout the brain. [1] Head injuries are a frequent cause of visits to the emergency room. Traumatic brain injury is a possibility for many individuals who experience blunt head trauma (TBI). [2]



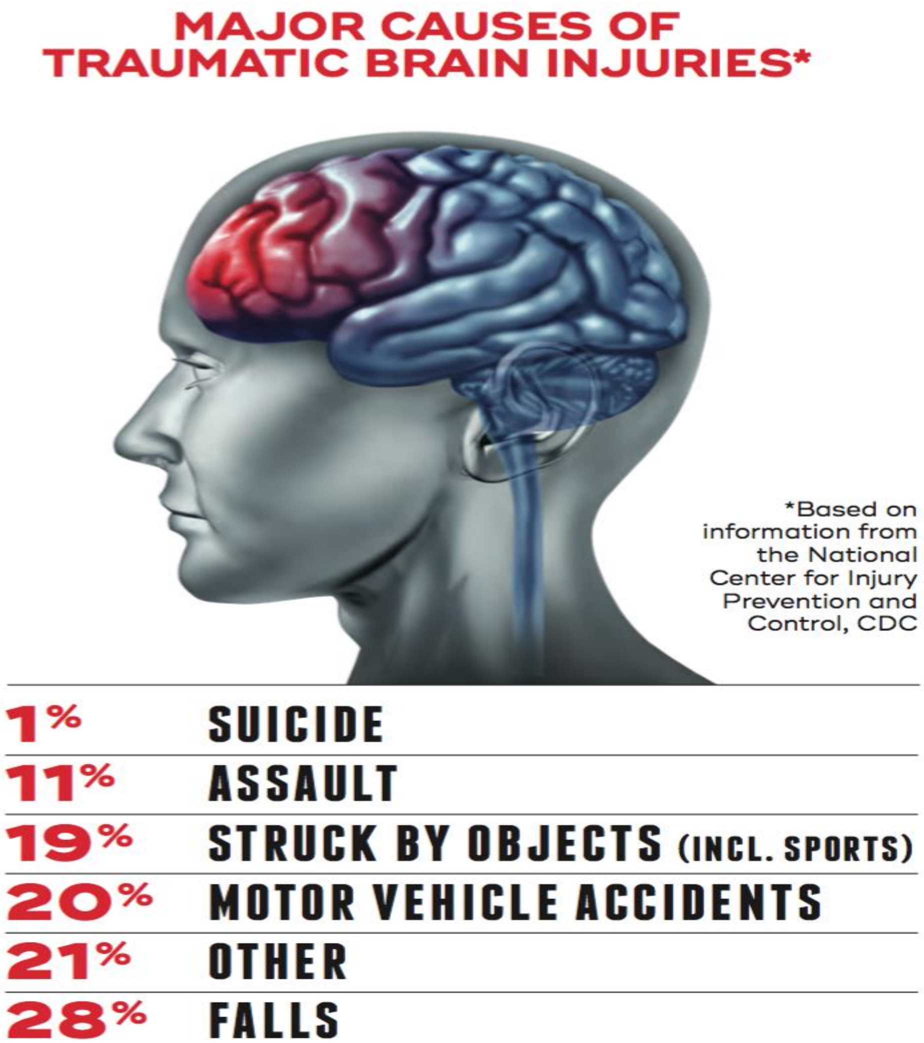
Traumatic brain injury still affects millions of people worldwide each year. The Centres for Disease Control report that during the years 2001–2010, there was a rise in the total combined rates of TBI-related ER visits, hospital stays, and fatalities. [3] All things considered, nevertheless, there have been fewer TBI-related deaths throughout this same time span. This is probably due in part to growing public awareness, formalising management and guidelines, and making substantial technological breakthroughs in treatment protocols. [4] The age groups with the highest rates of TBI are typically 0–4 years old, as well as teenagers and young adults (15–24 years old). Incidence peaks again in the elderly (>65 years of age). Car crashes and falls are the two main causes of traumatic brain injuries nationally. [5] Considering that there are often more TBIs but fewer deaths associated with them. When evaluating patients who have suffered blunt head trauma, reports of loss of consciousness is often taken into consideration as a sign that a brain CT scan should be performed. [6-8] For patients with aberrant Glasgow Coma Scale (GCS) scores and/or LOC, clinicians regularly order CT scans.

This study assessed whether getting a brain CT scan is necessary if a patient with blunt trauma reports having LOC but otherwise has a normal physical assessment.

Criteria used to classify TBI severity [9]

Criteria	Mild	Moderate	Severe
Structural Imaging	Normal	Normal or abnormal	Normal or abnormal
Loss of Consciousness	< 30 minutes	30 minutes to 24 hours	>24 hours
Alteration of Consciousness/Mental State	A moment to 24 hours	>24 hours	>24 hours
Post-traumatic Amnesia	0–1 day	>1 and <7 days	>7 days
Glasgow Coma Scale (best available score in 24 hours)	13–15	9–12	3–8

TBI = traumatic brain injury.



Traumatic Brain Injury (TBI) is a growing public health concern in India, with a significant impact on individuals and healthcare systems. Here are some key statistics and facts related to TBI in India. India has one of the highest rates of TBI in the world. It is estimated that approximately 1.5 to 2 million people suffer from TBI each year. TBI is a leading cause of death and disability in India. About 10% of all trauma-related deaths in the country are due to TBI. The most common cause of TBI in India, accounting for about 60% of cases. India has a high rate of road traffic accidents, with over 150,000 deaths annually, and many more sustaining injuries. Males are more commonly affected than females, with a male-to-female ratio of about 4:1. Young adults (ages 15-40) are the most affected group, primarily due to their higher involvement in road traffic accidents and sports activities. Urban areas report higher incidence rates due to denser traffic and greater exposure to road accidents, whereas rural areas face challenges in access to timely medical care, contributing to higher mortality rates. TBI places a substantial burden on the healthcare system, with a significant portion of emergency and trauma care resources dedicated to its management. The economic burden of TBI is considerable, including direct costs (medical expenses, hospitalization, rehabilitation) and indirect costs (loss of productivity, long-term disability).

Mechanism of injury

Traumatic brain injury can be caused by a number of different methods, and it can lead to structural or physiologic brain damage. Blast injury, penetrating injury, and blunt, non-penetrating traumatic brain injury.

1) Blunt non-penetrating TBI

Rapid head acceleration or deceleration without impact or a direct hit to the head can both cause blunt, non-penetrating traumatic brain injury. There are two stages to this mechanism's brain damage. The traumatic incident that started the process causes the first phase, which lasts for a few weeks to months. The second phase is a series of neuropathologic processes that follow the initial injury.

The main injury phase occurs right away, and by the time emergency care is provided, the damage which has the potential to be fatal practically instantly has usually been fully done. Neural injury is caused by the brain's direct contact with the bony cranial vault and the shearing of neurovascular systems. The brain is housed in a fluid-filled compartment, so when you decelerate quickly, the movement of its cellular components lags behind the skull. As a result, the brain will impact the inner aspect of the skull from both the anterior and posterior directions, resulting in a coup-countercoup lesion. [27] Intracranial structures will twist and torque if a rotational component is present, which is almost always the case with blunt traumatic brain injury (TBI), leading to excessive shear strain (i.e., stretch). [28,29] The tissues most vulnerable to extreme strain are blood vessels and neural axons because of their elongated architecture. As a result, axon loss and blood vessel damage occur during the first injury phase of traumatic brain injury (TBI) (haemorrhage). Since motor vehicles decelerate suddenly, they can cause severe injuries. [30]

Axonal injury progresses during the secondary injury phase, which follows the main phase. Changes in ionic flux cause axonal swelling, a reduction in axonal transport, and modifications to neurotransmission. [31] A neuron experiences an energy crisis as a result of mitochondrial failure, which causes apoptosis, or programmed cell death, and a loss of neural function. This secondary phase may also include demyelination of the neurons and necrosis. Within hours after the damage, a neuroinflammatory reaction including microgliosis begins, and it may last for months or even years. Peripheral immune system components can take part in this process due to blood-brain barrier disruption (BBBD) brought on by traumatic brain injury (TBI). Diffuse microvascular damage leads to both hyper- and hypo-perfusion, which exacerbates ischemia and cerebral edoema when paired with BBBD and a loss of autonomic control. The breakdown of intra-axonal structures can lead to aberrant build-ups of neurotoxic proteins such tau phosphorylation and beta-amyloid. Early-onset neurodegeneration or dementia may be influenced by post-TBI accumulations of such proteins in conjunction with chronic aberrant neuroinflammation. [29,32]

Penetrating TBI

Both open and closed TBIs are possible (non-penetrating). When an object penetrates the brain tissue and exerts physical force on the brain, a penetrating traumatic brain injury (TBI) results. A closed head injury, or non-penetrating head injury, results from an external force that causes movement of the brain inside the skull. TBI is frequently caused by missile injuries, such as gunshot wounds, which are categorised as either penetrating or perforating based on how the projectile passes through the brain. When something penetrates an injury, it goes into the cranium and becomes lodged there. When an object enters the cerebral cavity and exits through an exit wound, it can cause perforating injuries. The missile's mass, shape, direction, and velocity all affect how much damage it does. The amount of energy released as the missile flies through the brain also affects damage. [27,28]

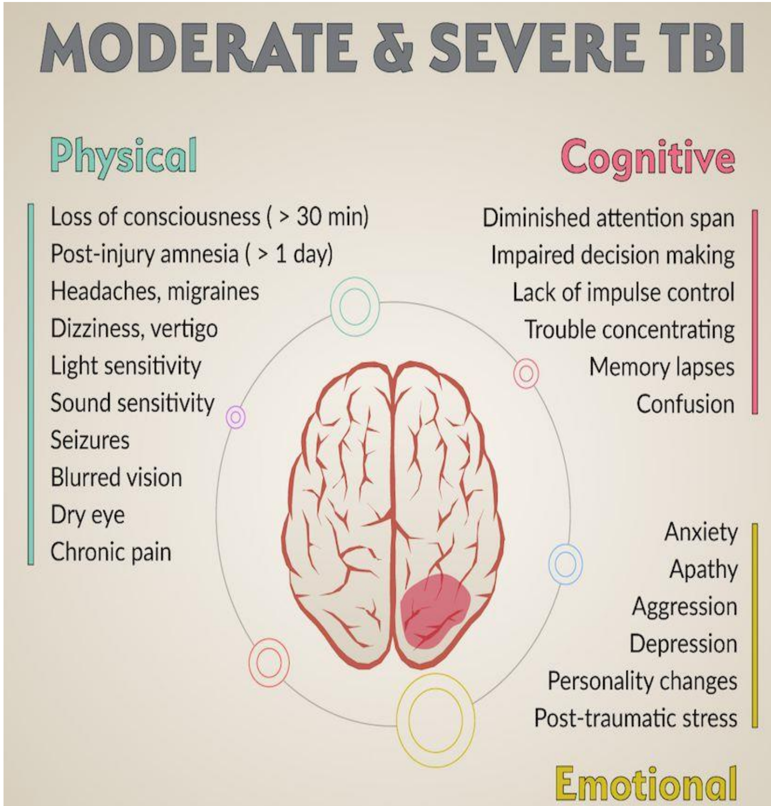
Blast induced TBI

While non-blast mechanisms are still widespread in both the military and civilian population (e.g., injuries from automobile and motorcycle accidents, athletic activities, and military physical training), blast-induced traumatic brain injury (bTBI) has become a common type of head injury . Both a direct shock wave effect and an indirect shock wave transmission to the brain via blood vessels and cerebrospinal fluid can cause neurologic harm from bTBI. Blast overpressure exposure sets off a series of cellular pathologic events in the brain, resulting in increased blood-brain barrier (BBB) permeability and damage to the microvasculature and BBB integrity. When the blood-brain barrier breaks down, it can lead to brain edoema, elevated intracranial pressure, and the onset of secondary brain damage due to reduced cerebral perfusion and oxygenation. Specifically, it has been demonstrated that in secondary brain injury following brain-traumatic brain injury (bTBI), the activation of oxidative processes and neuroinflammation lead to neurodegeneration and cell death. Like TBIs from other causes, bTBI can have two severity levels: mild, which is similar to or coexists with PTSD (discussed later in the chapter), and severe, which is frequently associated with polytrauma (i.e., multiple traumatic injuries, such as a TBI in addition to a serious burn or TBI and PTSD). The Department of Veterans Affairs (VA) and the military health care system face substantial short- and long-term challenges due to the epidemiologic scope and complexity of brain trauma and related neuropsychiatric disorders. [33]

Traumatic brain injury and Glassgow

Any traumatically caused structural injury or physiological disturbance of brain function brought on by an outside force is referred to as a traumatic brain injury (TBI). One or more clinical symptoms that appear right away, such as amnesia, a neurologic deficiency, an intracranial lesion, or a loss, diminished, or altered level of consciousness, are indicative of it. [15] External forces could be a blast injury, an object striking the head directly, or indirect forces from acceleration or deceleration. Traditionally, TBI has been categorised as mild (GCS 13–15), moderate (GCS 9–12), or severe (GCS 3-8) using the Glasgow Coma Score (GCS). A more modern TBI categorization system classifies TBI based on imaging results, post-traumatic amnesia (PTA), alteration of consciousness (AOC), and duration of loss of consciousness (LOC). [15]

A symptom-based classification categorises illnesses based on the description of symptoms found during a physical examination and history. This approach is inaccurate in that it frequently lumps together different pathophysiological processes as a single clinical entity. This is especially problematic in cases of moderate to severe traumatic brain injury (TBI), since there are frequently numerous damage processes present at the same time as shown by heterogeneous imaging findings. A more complex classification that takes into account anatomical, physiological logic, metabolic, immunologic, and genetic aspects develop as illness understanding grows and diagnostic capabilities advance. [16]



Biomechanics

When more energy from the surroundings is transferred to the tissue than can be absorbed without causing dysfunction, this is known as traumatic damage. The study of how forces interact with physical reactions in biological systems is known as biomechanics. Traumatic insults are also known as dynamic loading since they typically happen quickly. Direct or impact loading as well as impulsive loading, loading that happens without any physical contact are examples of dynamic loading. Following damage, the brain typically absorbs weights that are both linear and rotational, known as angular loads. Because loads delivered at high rates likely to result in more damage, the insult's rate and duration are crucial. [17] For example, you can apply the same force to a wall by punching it or by pressing your fist against it for a few minutes;

the former causes a boxer's fracture, while the latter does not. Contusions are examples of focal injuries that arise from direct stress and frequently happen without extensive damage. By contrast, the rotational acceleration that accompanies indirect loading frequently results in diffuse axonal damage (DAI). [18] Due of their enormous cranium and relatively weak neck muscles connecting it to the trunk, humans are especially vulnerable. Both the acceleration and deceleration phases of rotational acceleration cause significant and pervasive brain stresses. Diffuse stresses allow the brain to move differently in relation to the skull, which can result in bleeding. Following rotational injury, shear strain becomes more noticeable, and brain tissue is especially vulnerable to this kind of strain. [19] In animal studies, single linear acceleration resulted in contusions and subdural hematomas but not in loss of consciousness, whereas rotational acceleration is necessary to cause concussions. [20]

Pathophysiology

The initial traumatic insult causes mechanical damage, such as alterations in cerebral blood flow, ultrastructural damage to axons, and rupture of cellular and vascular membranes with release of intracellular contents. [21,22] Widespread release of excitatory neurotransmitters like glutamate, severe dysregulation of calcium homeostasis, energy deficiency owing to ATP depletion, production of free radicals, and necrotic and apoptotic pathways leading to cell death are examples of subsequent metabolic disruption. [22,23] Increased intracranial pressure, reduced cerebral blood flow, tissue ischemia, cerebral odema, and disruption of the blood brain barrier are other worldwide effects of the traumatic injury. [24,25]

After the initial insult, glial scar formation, cellular debris elimination, and plastic changes in neuronal networks initiate the healing and recovery processes. [26] The mechanisms outlined in this section were primarily obtained from animal research and, to a lesser extent, from humans with severe traumatic brain injury (sTBI), due to the challenges associated with investigating human mTBI. It is believed that similar processes take place in human mTBI.

Neuropathology

The neuropathology of traumatic brain injury (TBI) is comprised of two main injuries: a primary injury that arises directly from the traumatic insult and a secondary injury that is caused by a series of molecular and cellular events that are set off by the first injury. These events result in inflammation, axonal damage, and cell death. [34,35] When a tissue is damaged, cells release proteins into the extracellular space, which allows bodily fluids like blood to pass through. [35]

The main injury causes immediate reactions, which coordinate neuronal healing. Nevertheless, in a portion of the population, these biologic alterations are linked to symptoms and deficits that continue into the subacute and, in some cases, the chronic phases of recovery. The present focus of research is on the poorly known mechanisms that affect individual variability into recovery.

Military personnel frequently experience chronic neurologic symptoms after traumatic brain injuries, which might include psychosocial comorbidities, global disability, and neurobehavioral impairment.It is postulated that traumatic brain injury (TBI) and the ensuing pathogenic processes cause neurons, glial cells, and endothelial cells to release extracellular chemicals that enter the bloodstream.

Extracellular release of molecules can happen when cell membranes break down (neurodegeneration, for example) or when molecules are secreted as part of intercellular communication (cytokines, angiogenic factors, etc.). Both of these processes are likely to play a role in the emergence and maintenance of persistent symptoms and deficits that follow traumatic brain injuries. Additionally, there is proof that traumatic brain injury (TBI) is associated with neurodegenerative conditions such as multiple sclerosis, Parkinson's disease, amyotrophic lateral sclerosis, and various forms of dementia, such as Alzheimer's disease and chronic traumatic encephalopathy. [36-40]

Glassgow Coma scale

The Glasgow Consciousness Scale (GCS), which was first popularised in 1974 by a Glasgow working group, is a tool used at the bedside to objectively record a patient's degree of consciousness and neurological functioning in a form that allows healthcare professionals to easily share information. The GCS is employed in the majority of clinical settings and has a growing body of published research supporting its clinical utility.

According to several experts, individuals with GCS scores of 13 or 14 have more serious injuries than those with scores of 15, hence those with scores of 15 ought to be classified as having "minor head injuries" and be placed in a different group. [41-44]

For all types of acute medical and trauma patients, the Glasgow Coma Scale (GCS) is used to objectively characterise the degree of diminished consciousness. The three responsiveness categories that the scale utilises to assess patients' abilities are eye-opening, motor, and verbal responses. The total Glasgow Coma Score, which provides a less comprehensive explanation but can be a useful "shorthand" summary of the severity overall, can be obtained by adding the results of each scale component. [45]

Its use became widespread in the 1980s when the first edition of Advanced Trauma and Life Support recommended utilising the Glasgow Coma Scale for all trauma patients.

Furthermore, it was integrated into the World Federation of Neurosurgical Societies (WFNS) subarachnoid haemorrhage patient grading system created in 1988 [46]. Since then, the Glasgow Coma Scale and its total score have been included into numerous clinical guidelines and scoring systems for patients suffering from trauma or critical illness.[47] These include individuals of all ages and small children who are not yet verbal. The Glasgow Coma Scale, which is used in over 75 countries, is a required component of the NIH Common Data Elements and the ICD 11 revision for brain injury investigations. [48-50]

FUNCTION:

Scoring and parameter

Three parameters make up the Glasgow Coma Scale: best motor response (M), best verbal response (V), and best ocular reaction (E). The Glasgow Coma Scale's component reaction levels are "scored" from 1 (no response) to 6 (motor response), 5 (verbal response), and 4 (eye-opening response).

With three being the lowest and fifteen being the highest, the total Coma Score consequently has values between three and fifteen.

The total of the constituent elements' scores makes up the score. For instance, GCS10 = E3V4M3 might be used to represent a score of 10.

Best eye response (4)

1. No eye opening
2. Eye opening to pain
3. Eye opening to sound
4. Eyes open spontaneously

Best verbal response (5)

1. No verbal response
2. Incomprehensible sounds
3. Inappropriate words

4. Confused
5. Orientated

Best motor response (6)

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

Concerning Matter

The following elements could impede the Glasgow Coma Scale evaluation:

1.Pre-existing conditions Obstacles in language Deficit in cognition or nervous system Speech difficulty or hearing impairment

2. Consequences of the current course of treatment Physical (e.g., intubation): A patient's score is marked with the suffix T to signify intubation if they are unable to talk and are only assessed on their motor and eye-opening responses. Pharmacological (e.g., sedation) or paralysis: Prior to administering sedation, the physician ought to, if at all feasible, ascertain the patient's score.

3. Consequences of further wounds or lesions cranial or orbital fracture injury to the spinal cord COLD-induced hypoxic-ischemic encephalopathy.Sometimes the Glasgow Coma Scale cannot be obtained even with the above-mentioned problems resolved. It is imperative that all components be tested and included before reporting the final score, as doing so will result in a low score and maybe misunderstanding.

Clinical significance:

When evaluating responsiveness and guiding the early management of individuals who have experienced a head injury or other severe brain injury, the Glasgow Coma Scale is widely utilised. Securing the airway and prioritising patients to determine which ones should be moved are crucial decisions in emergency treatment for patients with more severe impairments. Decisions are made about the need for neuroimaging, hospital for observation, and discharge in patients with less severe disability. Additionally crucial to monitoring a patient's clinical development and guiding therapy modifications are routine Glasgow Coma Scale assessments.

Different information is produced by the three Scale components based on an individual's position on the responsiveness spectrum. [51]. In individuals with more severe impairments, changes in motor response are the main contributing component, with ocular and verbal responses being more helpful to a lesser extent.

It is therefore appropriate to record the clinical findings in each of the three components individually for individual individuals. A valuable summary overall index is communicated by the total score, but with considerable information loss.

Loss of consciousness and CT-Scan

Patients with blunt head trauma frequently experience loss of consciousness, which plays a significant role in the decision to obtain a CT scan in the emergency department. 91% of patients in a sizable cohort study with moderate traumatic brain injury had a CT scan. [52]

The connection between a history of LOC and TBI has drawn criticism from numerous doctors [53,54]. In spite of this, CT has been a mainstay of diagnostic testing in the ED, and the procedure of getting CT scans for patients with LOC has not altered. When evaluating individuals who have suffered head trauma, the test that is most frequently ordered is a brain

CT scan [55,56]. For head trauma, more than a million head CT scans are performed annually [57,58]. According to a report, between 2006 and 2011, the rate of head CT utilisation increased by 35.7% (range = 34.5 to 46.8%) for all age groups. [59]

According to a New Orleans Criteria study, patients with minor head injuries who do not exhibit any of the following could benefit from not having a CT scan: headache, emesis, age greater than 60, drug or alcohol intoxication, posttraumatic seizure, physical evidence of trauma above the clavicle, and short-term memory deficits. [60]

We assessed whether, given the radiation exposure, a brain CT scan should be ordered in response to a report of LOC in a patient who has had blunt trauma but otherwise appears normal on physical examination. The goal of the study was to protect patients from unwarranted CT scan radiation.

REVIEW OF LITERATURE

Waseem et al (2017) carried out this investigation to lower CT radiation exposure while maintaining awareness of clinically meaningful damage. The study aims to ascertain whether there is a subgroup of patients for whom a CT scan is not necessary without missing clinically severe intracranial injuries, as well as the relationship between LOC status and brain CT scan results in patients with acute head trauma. This study was carried out retrospectively in an inner-city hospital's emergency room. Patients with traumatic head trauma between the ages of 13 and 35 who visited the emergency department (ED) between January 2010 and December 2013 comprised the patient population. Two groups of patients have been divided: one for "LOC" and the other for "no LOC." Each group's brain CT scan results were matched to their LOC status. "Clinically significant" was defined for research purposes as requiring interventions, an ICU stay of at least 24 hours, or an extended hospital stay. Chi-square computations were utilised to analyse the outcomes. A total of 494 patients were found to have experienced head trauma during the study period. Among them, 309 (62.5%) did not lose consciousness, while 185 (37.5%) experienced LOC. Significant CT findings were found in 15 (8.1%) of the LOC group and in 1.3% (4/309) of the non-LOC group ($p < .001$). Four patients with substantial brain CT abnormalities and no lymphocele (LOC) had positive physical findings indicative of head, neck, or facial trauma. With no outward indications of head, neck, or facial injuries and a normal GCS of 15, only 1/15 (6.7%) of the LOC group exhibited significant CT results. A tiny percentage of LOC patients had CT findings that needed to be addressed. Significant brain CT results were not seen in head trauma patients with a normal GCS and no physical head, neck, or facial injuries. This begs the question of whether individuals who have had blunt head trauma and have a normal GCS, no physical symptoms, and LOC should also get routine brain CT scanning.

Yue JK et al (2024) [11] sought to assess the features of isolated tSAH on CT and CT-occult intracranial injuries on brain magnetic resonance imaging (MRI) in traumatic brain injury (TBI) patients with Glasgow Coma Scale (GCS) scores of 13–15 upon presentation at the emergency department (ED). Participants who presented to the ED and got a clinically warranted head CT scan within 24 hours of a traumatic brain injury (TBI) were enrolled in the prospective, 18-center Transforming Research and Clinical Knowledge in Traumatic Brain Injury Study (TRACK-TBI; enrolment years 2014-2019). A portion of the TRACK-TBI subjects had research MRI two weeks after the injury, as well as venipuncture within 24 hours for plasma glial fibrillary acidic protein (GFAP) analysis. The present investigation examined plasma GFAP level, 2-week MRI data, isolated tSAH on initial head CT, and ED arrival GCS 13–15 in TRACK-TBI subjects ≥17 years of age. 52.6% of the 57 individuals had a median age of 46.0 years [quartile 1 to 3 (Q1-Q3): 34-57]. 12.3% of patients were sent home from the ED, 61.4% were sent to a hospital ward, and 26.3% were placed in an intensive care unit. 45.6% (26 of 57 participants) had MRI-identified CT-occult traumatic intracranial lesions (one additional lesion type: 31.6%; two additional lesion types: 14.0%); of these 26 participants, 65.4% had axonal injury, 42.3% had a subdural hematoma, and 23.1% had an intracerebral contusion. GFAP levels were linked to axonal injury (no: median 226.7 pg/mL [109.6-435.1], yes: 828.6 pg/mL [204.0-1194.3], p = 0.009) and were greater in persons with CT-occult MRI lesions compared with those without (median: 630.6 pg/mL, Q1-Q3: [172.4-941.2] vs. 226.4 [105.8-436.1], p = 0.049). The findings show that in GCS 13–15 TBI, isolated tSAH on head CT is frequently not the only intracranial traumatic injury. Out of 57 participants, 26 patients exhibited additional CT-occult traumatic lesions on MRI, representing 46% of the group. An essential biomarker for identifying further CT-occult damage, such as axonal injury, could be plasma GFAP. Given our limited sample size, these results should be interpreted with caution and should be confirmed by bigger investigations.

Madhok DY (2022) [12] explain the recovery results after two weeks and six months for a group of patients who had moderate traumatic brain injury (TBI) and a negative head CT scan with a GCS score of 15. This cohort study examined individuals who were enrolled in the Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) study between January 1, 2014, and December 31, 2018. TBI patients were the subjects of this prospective, observational cohort study, which was carried out in the emergency departments of eighteen level I trauma centres located in urban areas. 991 of the 2697 patients in the TRACK-TBI research met the eligibility criteria for this analysis since they had a negative head CT scan and a GCS score of 15. From September 1, 2021, to May 30, 2022, data were examined. The Glasgow Outcome Scale-Extended (GOS-E) score was the main outcome, and it was stratified at two weeks and six months following the injury based on functional recovery (GOS-E score, 8) vs partial recovery (GOS-E score, <8). The River-mead Post Concussion Symptoms Questionnaire (RPQ) total score was used to measure the severity of mild TBI- related symptoms, which was the secondary endpoint. There were 991 participants in total (mean [SD] age: 38.5 [15.8 years; 631 men, or 64% of the sample) in the study. 204 (27%) of these individuals had a GOS-E score of 8 (functional recovery), and 547 (73%) had a score of less than 8 (incomplete recovery). Of these participants, 751, or 76%, were monitored at two weeks following the injury. Six months after the injury, 287 (44%) of the 659 participants (66%) who were followed up showed functional recovery, whereas 372 (56%) showed incomplete recovery. The majority of individuals who did not fully recover stated that they were not back to their pre-injury or baseline lives (88% [479 of 546]; 95% CI, 85%-90%). When comparing participants with a GOS-E score of 8 to those with a score of less than 8, the mean RPQ score was 16 (95% CI, 14-18; $P < .001$) points lower after 2 weeks (7 vs 23) and 18 (95% CI, 16-20; $P < .001$) points lower at 6 months (4 vs 22).According to this study, after two weeks and six months following their accident, the majority of patients with a GCS score of 15 and a negative head CT scan reported incomplete recovery. The results imply that in order to identify patients who have not fully recovered and to aid in their rehabilitation, emergency department physicians ought to advise 2-week follow-up appointments for these patients.

Nayebaghayee H et al (2016) [13] The objective of the study was to evaluate the correlation between the results of a computed tomography (CT) scan and the Glasgow Coma Scale (GCS) score in order to establish the GCS scoring system as a viable substitute for CT scans for managing brain injuries in patients who have suffered head trauma. Hospitalised individuals who complained of head trauma were the subjects of this investigation. The GCS score was used to determine the severity of the head injury upon admission and classified it as mild, moderate, or severe.45% of the study individuals had GCS 15, and 80.5% of the subjects had GCS 13–15. Moreover, 9% had a GCS of less than 8, and 10.5% had a range of 9 to 12. 54.5% of the participants had abnormal CT results; of these, 77.1% were classified as having mild head injuries, 11.0% as having moderate head injuries, and 11.9% as having severe head injuries. Additionally, 41.0% of individuals with GCS 15 reported abnormal CT scan results.33.0% of all patients with abnormal CT results had surgery; of these, 61.1% were classified as having had a mild head injury, 13.9% as having a serious head injury, and 22.2% as having a severe head injury. Merely 27.0% of individuals with a GCS of 15 received surgery. Given, that CT findings are the gold standard for determining injury severity, combining the GCS score with other relevant scoring systems may be a more useful method of stratifying the degree of brain injury than using the GCS score alone.

Ravi M Godavarthi (2018) [14] The current study looked at the relationship between the Glasgow Coma Score (GCS) and computed tomographic (CT) results in head injury victims. With ethical committee approval, a two-year prospective study was carried out at a tertiary care hospital. Glasgow coma score scoring was applied to 330 TBI cases, and tomography of all the cases were completed, and conclusions were recorded. IBM Corp. produces SPSS statistics for Windows, version 20.0 (NY: IBM Corp), which was used for data analysis. 34.45% of the participants in this study were female, and 64.55% were male (M: F=1.8:1) and the study group's average age 30.12± 11.2 years old. Ages 31 to 40 were the most common age group for TBI. fall from a height comes next (24.85%). RTA accounted for 42.73% of all causes, with falls from height accounting for 24.85% of cases. According to the GCS score, 47.585 instances were classified as mild brain injuries, 30.91% as moderate, and 21.52% as severe. Vomiting and loss of consciousness were the most typical clinical manifestations. 89% of cases showed abnormal CT results, with intraxial bleeding (72.79%) and skull fractures (70.75%) being the most prevalent.Atypical CT results were present in every case with serious head injury. According to the study's findings, patients with low GCS scores were associated with a higher frequency of aberrant CT findings, which was regarded as a severity risk factor. Serious morbidity and devastation are experienced by patients with low GCS scores.

AIM:

1. To determine the correlation between LOC and CT findings in Traumatic Brain Injury Patients [Primary objective]

2. To assess the efficacy of Glasgow Coma Scale as an indicator for doing CT.

MATERIALS AND METHODS

This is a observational study to be conducted in the Emergency Department of BLDE(DU), Shri B.M Patil Medical College Hospital, Vijayapur , Karnataka.

INCLUSION CRITERIA

- For study purposes, we include only previously healthy patients above age group of 18 with isolated head injury.

EXCLUSION CRITERIA

- Patients with other medical conditions that may influence CT result, such as previous history of stroke, were excluded.
- patients who are less than 18 and those taking anticoagulant medications were also excluded.
- Patients who were suspected of substance abuse or intoxication were not included in this study.

SOURCE OF DATA

Data will be collected from the patients presenting to Emergency department of Shri BM Patil medical college and research centre Vijayapura for a period of 18 months who fullfill the inclusion criteria

SAMPLING

With anticipated head injury case within the inclusion criteria this study would require a sample size of 145 patients. With 95% level of confidence and 5% absolute precision. The data obtained will been entered in Microsoft excel sheet and statistical analysis will be performed using JNL software.

METHODOLOGY

For this study the data of patients with Traumatic Brain Injury and classify them as LOC group and NON-LOC group and CT scan positivity among 2 is identified and with that “p” value and ODDS ratio is calculated Same set of patients are classified based on GCS status and CT positivity. P value and ODDS ratio calculated and compared with first group Combining GCS status with LOC, Patients are classified as those with GCS less than 15 wih LOC and those

with GCS15 with LOC and ‘p’ value and odds ratio is calculated and compared with first group.

:

Results

TABLE 1: DEMOGRAPHIC AND INCIDENT CHARACTERISTICS OF THE STUDY POPULATION

S.NO	VARIABLE	VALUES
1	AGE (MEAN+ SD)	6.06+4.0
2	AGE 18-24	52 (35.9%)
3	AGE 25-35	23 (15.9)
4	AGE 35-45	36 (24.8)
5	AGE >45	34 (23.4)
6	SEX	
7	MALE	103 (71)
8	FEMALE	42 (29)
9	FALL	76 (52.4)
10	SHORT FALL	66 (45.5)
11	LONG DISTANCE	11 (7.6)
12	MOTOR VEHICLE	33 (22.8)
13	PEDESTRIAN	12 (8.3)
14	DIRECT IMPACT	21 (14.5)
15	PHYSICAL ASSAULT	2 (1.4)
16	PHYSICAL ABUSE	1 (0.7)

The study sample comprised 145 participants, with a mean age of 6.06 years (±4.0). The age distribution of the participants revealed that 52 individuals (35.9%) were aged between 18-24 years, 23 individuals (15.9%) were aged between 25-35 years, 36 individuals (24.8%) were aged between 35-45 years, and 34 individuals (23.4%) were aged over 45 years. This diverse age range highlights the variability in the affected population, which is critical for understanding the broad impact of the studied conditions across different life stages. The gender distribution was skewed towards males, with 103 males (71%) and 42 females (29%), indicating a potential gender-based difference in the incidence or reporting of injuries and symptoms.

In terms of the mechanism of injury, falls emerged as the most prevalent cause, accounting for 76 cases (52.4%). Within this category, short falls were the most common, reported in 66 cases (45.5%), while long-distance falls were noted in 11 cases (7.6%). These findings underscore the significance of fall-related injuries in the study population, prompting further investigation into preventive measures and risk factors associated with different types of falls. Motor vehicle accidents were responsible for 33 cases (22.8%), emphasizing the need for enhanced safety measures and interventions in traffic-related incidents. Among these motor vehicle accident cases, 12 participants (8.3%) were pedestrians, highlighting the vulnerability of pedestrians in traffic environments.

Direct impacts accounted for 21 cases (14.5%), suggesting a notable proportion of injuries resulting from blunt force trauma. Physical assaults were reported in 2 cases (1.4%), and child physical abuse was reported in 1 case (0.7%). Although these numbers are relatively low, they represent significant and concerning instances of intentional harm, warranting targeted interventions and support systems for victims. The overall statistics of the study, including the mechanisms of injury and demographic data, provide a comprehensive overview of the affected population, enabling a deeper understanding of the underlying factors and guiding future research and policy development to mitigate these risks. This detailed analysis of demographic variables and injury mechanisms is crucial for tailoring effective prevention and treatment strategies to address the specific needs of different population subgroups within the study cohort.

TABLE 2: COMPARISON OF CT SCAN RESULT BASED ON LOSS OF CONSCIOUSNESS

	CT POSITIVE	CT NEGATIVE	TOTAL	% POSITIVE	% NEGATIVE
LOC	5	50	55	7.4%	92.6%
NO LOC	1	89	90	1.1%	98.9%
TOTAL	6	139	144	3.5%	96.5%

The analysis of CT scan results in relation to loss of consciousness (LOC) revealed significant findings. Among the 144 participants, 6 (3.5%) tested positive for abnormalities on CT scans, while 138 (96.5%) tested negative. Specifically, of the participants who experienced loss of consciousness (LOC), 5 out of 55 (7.4%) had positive CT findings, whereas 50 (92.6%) had negative results. In contrast, only 1 out of 90 participants without LOC (1.1%) showed positive CT results, and 89 (98.9%) had negative CT scans. These results indicate a notable association between LOC and the likelihood of positive CT findings, with LOC being linked to a higher probability of detecting abnormalities on CT scans. The majority of participants, irrespective of LOC status, had negative CT findings, underscoring that abnormal CT results are relatively rare overall. This distribution highlights the importance of considering LOC as a significant factor when assessing the risk of CT abnormalities and informs clinical decision-making regarding the necessity of further imaging and evaluation.

TABLE 3: CT SCAN RESULTS BY GLASGOW COMA SCALE (GCS) SCORE

	CT POSITIVE	CT NEGATIVE	TOTAL	% POSITIVE	% NEGATIVE
GCS 15	4	132	136	2.9%	97.1%
GCS <15	2	7	9	22.2%	77.8%
TOTAL	6	139	145	4.1%	95.9%

The distribution of CT scan results in relation to Glasgow Coma Scale (GCS) scores revealed significant insights into the association between GCS levels and the presence of abnormalities on CT scans. Out of a total of 145 participants, 6 (4.1%) had positive CT findings, while 139 (95.9%) had negative results. Specifically, among participants with a GCS score of 15, which indicates full consciousness, only 4 out of 136 (2.9%) had positive CT findings, whereas 132 (97.1%) had negative results. In contrast, among participants with a GCS score of less than 15, indicating reduced consciousness, 2 out of 9 (22.2%) had positive CT findings, and 7 (77.8%) had negative results. This analysis demonstrates that a lower GCS score is associated with a higher likelihood of detecting abnormalities on CT scans. The majority of participants with a GCS score of 15 had negative CT scans, reinforcing that normal consciousness levels are correlated with fewer CT abnormalities. Conversely, the higher percentage of positive CT results among those with GCS scores less than 15 suggests that impaired consciousness is a significant predictor of CT abnormalities. These findings highlight the utility of GCS as an important clinical indicator when evaluating the need for CT imaging and further diagnostic investigation.

TABLE 4: CT SCAN RESULTS BY GLASGOW COMA SCALE (GCS) AND LOSS OF CONSCIOUSNESS (LOC) STATUS

	CT POSITIVE	CT NEGATIVE	TOTAL	% POSITIVE	% NEGATIVE
GCS<15 AND LOC	5	53	58	8.6%	91.4%
GCS <15 AND NO LOC	1	86	87	1.1%	98.9%
TOTAL	6	139	145	4.1%	95.9%

The evaluation of CT scan results in relation to Glasgow Coma Scale (GCS) scores and loss of consciousness (LOC) provided valuable insights into the correlation between these factors and the likelihood of CT abnormalities. Among the total of 145 participants, 6 (4.1%) had positive CT findings, while 139 (95.9%) had negative results. Specifically, when examining the subgroup of participants with a GCS score of less than 15 and LOC, 5 out of 58 (8.6%) had positive CT findings, whereas 53 (91.4%) had negative results. In contrast, among participants with a GCS score of less than 15 but without LOC, only 1 out of 87 (1.1%) had positive CT results, and 86 (98.9%) had negative findings. These results suggest that the combination of a GCS score of less than 15 and the presence of LOC is associated with a significantly higher likelihood of detecting abnormalities on CT scans compared to cases where

LOC is absent. The majority of participants with a GCS score of less than 15 and no LOC had negative CT findings, indicating that the absence of LOC may mitigate the risk of CT abnormalities even when GCS is reduced. These findings underscore the importance of both LOC and GCS scores as critical factors in assessing the need for CT imaging and predicting the likelihood of detecting brain abnormalities.

TABLE 5: CORRELATION OF ODDS RATIO AND P VALUE FOR VARIOUS VARIABLES I STUDY POPULATION

S.NO	VARIABLE	OR	P VALUE
1	SEX	3.346	0.067
2	GCS (15)	5.961	0.051
3	GCS (14)	1.387	0.239
4	GCS (13)	4.413	0.036
5	VOMITING	1.069	0.301
6	HEADACHE	0.745	0.388
7	LOSS OF CONSIOUSNESS	0.167	0.683
8	AGE 18-24	1.373	0.712
9	AGE 25-35	0.384	0.535
10	AGE 35-45	0.105	0.746

11	AGE >45	0.179	0.673
12	OVERALL STATISTICS	13.898	0.126
13	MOTOR VEHICLEACCIDENT	0.527	0.468
14	PEDESTRIAN	0.026	0.872
15	DIRECT IMPACT	2.334	0.127
16	FALL	4.957	0.084
17	SHORT FALL	1.601	0.206
18	LONG DISTANCE FALL	4.379	0.036
19	OVER ALL STATISTICS	7.632	0.178

The logistic regression analysis of variables predicting abnormal brain CT findings revealed several notable associations. The odds ratio (OR) for sex was 3.346 with a p-value of 0.067, indicating a trend toward a higher likelihood of abnormal CT findings in one gender, though this result did not reach conventional statistical significance. Glasgow Coma Scale (GCS) scores showed significant variability in association with CT results. Specifically, a GCS score of 15 had an OR of 5.961 with a p-value of 0.051, approaching significance and suggesting that a higher GCS score might be linked to an increased probability of detecting abnormalities. Conversely, GCS scores of 14 and 13 showed ORs of 1.387 (p = 0.239) and 4.413 (p = 0.036), respectively, with the latter indicating a significant association between lower GCS scores and positive CT findings.

Symptoms such as vomiting and headache had ORs of 1.069 ($p = 0.301$) and 0.745 ($p = 0.388$), respectively, suggesting that these symptoms did not significantly predict CT abnormalities. Loss of consciousness (LOC) had a notably low OR of 0.167 with a p-value of 0.683, indicating no significant association with abnormal CT findings. Age categories, including 18-24 years (OR = 1.373, $p = 0.712$), 25-35 years (OR = 0.384, $p = 0.535$), 35-45 years (OR = 0.105, $p = 0.746$), and over 45 years (OR = 0.179, $p = 0.673$), also did not show significant associations with CT abnormalities.

Mechanisms of injury revealed some significant predictors. Motor vehicle accidents had an OR of 0.527 with a p-value of 0.468, while pedestrian injuries had a negligible OR of 0.026 ($p = 0.872$). Direct impacts had an OR of 2.334 ($p = 0.127$), suggesting a potential association with CT abnormalities, though not statistically significant. Falls, particularly long-distance falls, showed a significant association with CT abnormalities, with an OR of 4.957 ($p = 0.084$) for falls in general and an OR of 4.379 ($p = 0.036$) for long-distance falls specifically. Overall statistics indicated that, while some variables approached significance, others did not show a strong predictive value for abnormal CT findings. This analysis underscores the importance of specific clinical and injury-related factors in predicting CT results and highlights areas for further research to better understand these associations.

DISCUSSION

Loss of consciousness is prevalent among individuals who have forceful head trauma and is a significant factor that affects the decision to get a CT scan in the emergency department. A CT scan was acquired in 91% of participants in a significant cohort study [65].

A number of medical professionals have raised doubts regarding the correlation between a past occurrence of loss of consciousness (LOC) and traumatic brain injury (TBI) [66,67]. However, the practice of acquiring CT scans in patients with loss of consciousness (LOC) has not altered, and CT scans have been consistently used for diagnostic testing in the emergency department (ED). The brain CT scan is the primary diagnostic technique used to assess individuals with head trauma [68,69]. Annually, about 1 million cranial CT scans are performed for the purpose of diagnosing head trauma [70,71]. A recent report has shown that the utilization of head CT scans has risen by 35.7% (with a range of 34.5% to 46.8%) for all age groups from 2006 to 2011 [72].

This study reexamined the issue of whether a brain CT scan is necessary for patients with traumatic head trauma who experience loss of consciousness upon admission in the emergency department. While relying solely on clinical examination might decrease the need for CT scans, it may not completely rule out the possibility of diagnosing intracranial damage. For numerous medical practitioners, the notion of failing to identify any intracranial lesion is seen as intolerable. The fear of being sued may influence medical decision-making, and some healthcare professionals would not tolerate missing the diagnosis of any intracranial lesion, regardless of its importance. A research based on the New Orleans Criteria has proposed that the use of CT scans may not be necessary for patients with mild head injuries, as long as none of the following conditions are present: The factors associated with the condition include headache, vomiting, age over 60 years, drug or alcohol intoxication, seizures after a traumatic event, physical signs of injury above the collarbone, and difficulties with short-term memory [73]. The "New Orleans Criteria" were developed to encompass all types of cerebral injuries, regardless of whether they need neurosurgical surgery or not. In order to enhance the accurate identification of significant brain damage, the Canadian CT Head Rule permits patients with a Glasgow Coma Scale (GCS) score of 13-15 within 2 hours of the accident to forgo a head CT scan. However, it does mandate a brain CT scan for patients who meet additional criteria such as being under the age of 16 or having a particular cause of injury. The Canadian CT Head Rule incorporates amnesia as one of the criteria for identifying individuals with any clinically significant brain damage [74].

This study provides a comprehensive analysis of demographic characteristics, incident mechanisms, and clinical variables associated with positive CT scan findings among a cohort of 145 participants. The results offer valuable insights into the factors that may influence the likelihood of detecting brain abnormalities, which is critical for improving diagnostic accuracy and patient management strategies.

This study provides a comprehensive analysis of demographic characteristics, incident mechanisms, and clinical variables associated with positive CT scan findings among a cohort of 145 participants. The results offer valuable insights into the factors that may influence the likelihood of detecting brain abnormalities, which is critical for improving diagnostic accuracy and patient management strategies.

A small proportion, namely less than 5%, of individuals with mild head trauma exhibit aberrant brain CT results, and none of them needed neurosurgical surgery. The incidence of abnormal brain CT scans in cases of mild head injury is similar to the global average, which ranges from 7-10%. The rate of surgical intervention in such cases is reported to be between 1-5%.The given text is the list [61,62].

There was no correlation between age and a higher likelihood of intracranial damage with abnormal brain CT following forceful trauma. Out of the patients aged 18-24 years, only 2.9% were found to have abnormal brain CT scans, which is lower than the previously reported rates of roughly 3-10% in worldwide studies. (62 ,63) The cohort research indicated that younger individuals with mild head trauma do not have a distinct risk for intracranial damage. Nevertheless, our study did not primarily target this particular age-group.

The literature extensively documents the presence of gender disparity, with the majority of patients with mild head trauma being male. [64] The incidence of intracranial damage in mild head trauma was greater in males than in females; however, the difference in risk did not reach statistical significance. Regarding various injury processes, briefly

Our analysis found that distance falls and motor vehicle accidents were the most prevalent causes of injury. However, in the univariate analysis examining the risk correlation, it was found that 'fall' was not substantially linked to intracranial damage, perhaps due to the fact that most falls occurred over a short distance. The univariate analysis did not provide strong evidence supporting the link between the long distance fall and intracranial damage. The analysis did not find evidence to suggest a connection between intracranial damage and motor vehicle accidents (MVA) as a cause of mild head trauma. However, it was challenging in the study to categorize the MVA data into 'low' or 'high speed impact'. No correlation was seen between pedestrians and intracranial damage, possibly because the majority of cases classified as mild head injuries were low-speed impacts. Crush head injury refers to the direct impact of a heavy item on the head. Young children are most commonly injured at home by falling objects such as furniture, television sets, and ovens. Children who experienced a direct hit to the head had a twofold increase in the likelihood of sustaining an intracranial injury. However, it is important to note that this link did not reach statistical significance, which may be attributed to the limited size of the sample.

The analysis of CT scan results revealed a significant association between loss of consciousness (LOC) and positive CT findings. Participants who experienced LOC had a higher proportion of positive CT scans (7.4%) compared to those without LOC (1.1%). This indicates that LOC is a critical factor in assessing the risk of brain abnormalities and should be a key consideration in clinical evaluations. The overall prevalence of positive CT findings was relatively low (3.5%), suggesting that while LOC is a significant predictor, the majority of cases do not result in detectable CT abnormalities.

The study further examined the relationship between Glasgow Coma Scale (GCS) scores and CT scan results. A GCS score of less than 15 was associated with a higher likelihood of positive CT findings (22.2%) compared to a GCS score of 15 (2.9%). This finding highlights the importance of GCS as an indicator of neurological impairment and its utility in guiding the decision-making process for imaging. Lower GCS scores correlate with a higher risk of brain abnormalities, reinforcing the need for prompt and thorough evaluation in such cases.

When combining GCS scores with LOC status, the results showed that participants with a GCS score of less than 15 and LOC had the highest proportion of positive CT findings (8.6%). In contrast, those with a GCS score of less than 15 but without LOC had a significantly lower proportion of positive CT findings (1.1%). These results suggest that the combination of reduced GCS and LOC is a strong predictor of CT abnormalities, whereas the absence of LOC in patients with reduced GCS may mitigate this risk. This combined assessment provides a more nuanced understanding of the factors influencing CT scan results and can enhance clinical decision-making.

The logistic regression analysis identified several variables with potential predictive value for abnormal CT findings. Gender (OR = 3.346, $p = 0.067$) and a GCS score of 15 (OR = 5.961, $p = 0.051$) approached statistical significance, suggesting trends that warrant further investigation. A GCS score of 13 (OR = 4.413, p = 0.036) showed a significant association with positive CT findings, indicating that even moderate reductions in GCS can be indicative of abnormalities.

Interestingly, symptoms such as vomiting and headache did not significantly predict CT abnormalities, nor did age categories. This finding suggests that while these symptoms and age groups are important clinical considerations, they may not be strong independent predictors of CT findings in this population.

The Glasgow Coma Scale (GCS) upon arrival at the hospital was the sole clinical observation that demonstrated a statistically significant correlation with intracranial damage in our research. Patients who presented to the emergency department with a Glasgow Coma Scale (GCS) score of 13 had a fivefold higher probability of having an intracranial injury compared to those with a GCS score of 14-15 upon presentation. Overall, the level of consciousness (LOC) with the specified time period in our investigation did not show any statistically significant results. However, it has been observed that prolonged durations of loss of consciousness (LOC), as described in prior investigations, are related with intracranial damage. [62].

The clinical signs discussed in the previous section were examined using a prediction model. During our study, we examined them as a collection of observable occurrences and discovered that individuals with a Glasgow Coma Scale (GCS) score of 13 at initial assessment are the sole significant clinical indicator of intracranial damage.

The study's capacity to detect very mild to moderate but relevant clinical manifestations that might potentially indicate cerebral damage may have been compromised due to the limited sample size. Considering the limited predictive rule generated from this analysis, it is recommended that the selection of which patients should have neuroimaging be based on clinical judgments. Considering the significant level of radiation exposure in relation to the limited occurrence of positive brain CT results, it is strongly advised to undergo a period of clinical surveillance for children who have experienced mild head injuries but are otherwise in good condition.

CONCLUSION

This study highlights the associations between Glasgow Coma Scale (GCS) scores, loss of consciousness (LOC), and positive CT scan findings in 145 participants with head trauma. Lower GCS scores and the presence of LOC were linked to higher rates of CT abnormalities. However, the overall prevalence of positive CT findings was low (3.5%), with only 7.4% of participants with LOC showing abnormalities. Symptoms like vomiting and headache, and demographic factors such as age, were not significant predictors of CT abnormalities. Given the low rate of positive CT findings and significant radiation exposure from CT scans, the study suggests that routine CT imaging for patients with mild head trauma and only LOC may not be necessary.

Prioritizing GCS scores and other significant predictors in clinical decisions can reduce unnecessary radiation exposure and improve patient safety and resource utilization in emergency settings. This evidence-based approach ensures CT scans are reserved for higher-risk patients, enhancing diagnostic accuracy and patient care.

REFERENCES

1. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, Zhao LR. Traumatic brain injury: current treatment strategies and future endeavors. Cell transplantation. 2017 Jul;26(7):1118-30.
2. Waseem M, Iyahen P, Anderson HB, Kapoor K, Kapoor R, Leber M. Isolated LOC in head trauma associated with significant injury on brain CT scan. International Journal of Emergency Medicine. 2017 Dec;10:1-6.
3. Centers for Disease Control and Prevention. Injury prevention & control: traumatic brain injury & concussion [accessed 2016 Jan 22]
4. Masel BE, DeWitt DS, Levin H, Shum D, Chan R. Traumatic brain injury disease: long-term consequences of traumatic brain injury. Understanding traumatic brain injury: Current research and future directions. 2014 Jan 28;28.
5. Rutland-Brown W, Langlois JA, Thomas KE, Xi YL. Incidence of traumatic brain injury in the United States, 2003. The Journal of head trauma rehabilitation. 2006 Nov 1;21(6):544-8.
6. Ng SM, Toh EM, Sherrington CA. Clinical predictors of abnormal computed tomography scans in paediatric head injury. Journal of paediatrics and child health. 2002 Aug;38(4):388-92.
7. RAMUNDO ML, McKNIGHT TI, KEMPF J, SATKOWIAK L. Clinical predictors of computed tomographic abnormalities following pediatric traumatic brain injury. Pediatric emergency care. 1995 Feb 1;11(1):1-4.

8. Schutzman SA, Greenes DS. Pediatric minor head trauma. Annals of emergency medicine. 2001 Jan 1;37(1):65-74.

9. Orman JAL, Kraus JF, Zaloshnja E, et al. Epidemiology. In: Silver JM, McAllister TW, Yudofsky SC, editors. Textbook of traumatic brain injury. 2nd ed. Washington, DC: American Psychiatric Pub.; 2011. pp. 3–22

10. Waseem M, Iyahen P, Anderson HB, Kapoor K, Kapoor R, Leber M. Isolated LOC in head trauma associated with significant injury on brain CT scan. International Journal of Emergency Medicine. 2017 Dec;10:1-6.

11. Yue JK, Yuh EL, Elguindy MM, Sun X, van Essen TA, Deng H, Belton PJ, Satris GG, Wong JC, Valadka AB, Korley FK. Isolated Traumatic Subarachnoid Hemorrhage on Head Computed Tomography Scan May Not Be Isolated: A Transforming Research and Clinical Knowledge in Traumatic Brain Injury Study (TRACK-TBI) Study. Journal of Neurotrauma. 2024 Apr 11.

12. Madhok DY, Rodriguez RM, Barber J, Temkin NR, Markowitz AJ, Kreitzer N, Manley GT, Badjatia N, Duhaime AC, Feeser VR, Ferguson AR. Outcomes in patients with mild traumatic brain injury without acute intracranial traumatic injury. JAMA network open. 2022 Aug 1;5(8):e2223245-.

13. Nayebaghayee H, Afsharian T. Correlation between Glasgow Coma Scale and brain computed tomography-scan findings in head trauma patients. Asian journal of neurosurgery. 2016 Mar;11(01):46-9.

14. Ravi M Godavarthi, Gayatri Manam Ramakrishna Baru, Sunitha, Naveen Kumar B, A Comparison of Glasgow Coma Score with Computed Tomographic Findings in

Cases of Traumatic Brain Injury – A Two Year Experience at a Tertiary care Hospital. International Journal of Contemporary Medicine Surgery and Radiology. 2018;3(2)

15. The Concussion/mTBI Working Group. VA/DoD Clinical Practice Guideline for Management of Concussion/mTBI. [October, 2009].

16. Saatman KE, Duhaime AC, Bullock R, Maas AI, Valadka A, Manley GT. Classification of traumatic brain injury for targeted therapies. Journal of neurotrauma. 2008 Jul 1;25(7):719-38.

17. LaPlaca MC, Simon CM, Prado GR, Cullen DK. CNS injury biomechanics and experimental models. Progress in brain research. 2007 Jan 1;161:13-26.

18. Gennarelli TA, Thibault LE, Adams JH, Graham DI, Thompson CJ, Marcincin RP. Diffuse axonal injury and traumatic coma in the primate. Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society. 1982 Dec;12(6):564-74.

19. Holbourn AH. Mechanics of head injuries. The Lancet. 1943 Oct 9;242(6267):438-41.

20. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Brain. 1974 Dec 1;97(4):633-54.

21. McIntosh TK. Neurochemical sequelae of traumatic brain injury: therapeutic implications. Cerebrovascular and brain metabolism reviews. 1994 Jan 1;6(2):109-62.

22. Werner C, Engelhard K. Pathophysiology of traumatic brain injury. British journal of anaesthesia. 2007 Jul 1;99(1):4-9.

23. Thompson HJ, Lifshitz J, Marklund N, Grady MS, Graham DI, Hovda DA, McIntosh TK. Lateral fluid percussion brain injury: a 15-year review and evaluation. Journal of neurotrauma. 2005 Jan 1;22(1):42-75.

24. Statler KD, Jenkins LW, Dixon CE, Clark RS, Marion DW, Kochanek PM. The simple model versus the super model: translating experimental traumatic brain injury research to the bedside. Journal of neurotrauma. 2001 Nov 1;18(11):1195-206.

25. Marklund N, Bakshi A, Castelbuono DJ, Conte V, McIntosh TK. Evaluation of pharmacological treatment strategies in traumatic brain injury. Current pharmaceutical design. 2006 May 1;12(13):1645-80.

26. Board on Population Health, Public Health Practice, Committee on Gulf War, Brain Injury in Veterans, Long-Term Health Outcomes. Gulf War and health: volume 7: long-term consequences of traumatic brain injury. National Academies Press; 2009 Mar 2.

27. Graham DI, Lantos PL, Greenfield JG. Greenfield's neuropathology. (No Title). 1997 Jan.

28. Morales DM, Marklund N, Lebold D, Thompson HJ, Pitkanen A, Maxwell WL, Longhi L, Laurer H, Maegele M, Neugebauer E, Graham DI, Stocchetti N, McIntosh TK. Experimental models of traumatic brain injury: Do we really need to build a better mousetrap? Neuroscience. 2005;136(4):971–989

29. Smith DH. Diagnosing concussion. Canadian Medical Association Journal. 2013;185(18):1602

30. Johnson VE, Stewart W, Arena JD, Smith DH. Traumatic brain injury as a trigger of neurodegeneration. Advances in Neurobiology. 2017;15:383–400.

31. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75(Suppl 4):S24–S33

32. Giza CC, Kutcher JS. An introduction to sports concussions. Continuum. 2014;20:1545–1551

33. Papa L, Edwards D, Ramia M. Frontiers in neuroengineering. In: Kobeissy FH, editor. Brain neurotrauma: Molecular, neuropsychological, and rehabilitation aspects. Boca Raton, FL: CRC Press/Taylor & Francis © 2015 by Taylor & Francis Group, LLC; 2015.

34. McKee AC, Daneshvar DH. The neuropathology of traumatic brain injury. In: Grafman J, Salazar AM, editors. Handbook of clinical neurology. Vol. 127. Elsevier; 2015. pp. 45–66.

35. Taylor DD, Gercel-Taylor C. Exosome platform for diagnosis and monitoring of traumatic brain injury. Philosophical Transactions of the Royal Society B: Biological Sciences. 2014;369(1652)

36. Laskowitz D, Grant G. Frontiers in neuroscience. In: Laskowitz D, Grant G, editors. Translational research in traumatic brain injury. Boca Raton, FL: CRC Press/Taylor and Francis Group; 2016

37. DeKosky ST, Kochanek PM, Clark RS, Ciallella JR, Dixon CE. Secondary injury after head trauma: Subacute and long-term mechanisms. Seminars in Clinical Neuropsychiatry. 1998;3(3):176–185

38. Freeman LC, Ting JPY. The pathogenic role of the inflammasome in neurodegenerative diseases. Journal of Neurochemistry. 2016;136(S1):29–38

39. Gardner RC, Burke JF, Nettiksimmons J, Kaup A, Barnes DE, Yaffe K. Dementia risk after traumatic brain injury vs. nonbrain trauma: The role of age and severity. JAMA Neurology. 2014;71(12):1490–1497

40. Witcher KG, Eiferman DS, Godbout JP. Priming the inflammatory pump of the CNS after traumatic brain injury. Trends in Neuroscience. 2015;38(10):609–620

41. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. The Lancet. 1974 Jul 13;304(7872):81-4.

42. Hsiang JN, Yeung T, Ashley LM, Poon WS. High-risk mild head injury. Journal of neurosurgery. 1997 Aug 1;87(2):234-8.

43. Teasdale GM. Head injury. Journal of neurology, neurosurgery, and psychiatry. 1995 May;58(5):526.

44. Miller EC, Derlet RW, Kinser D. Minor head trauma: Is computed tomography always necessary?. Annals of emergency medicine. 1996 Mar 1;27(3):290-4.

45. Teasdale G, Murray G, Parker L, Jennett B. Adding up the Glasgow Coma Score. Acta Neurochir Suppl (Wien). 1979;28(1):13-6

46. 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 Nov;51(11):1457

47. 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 Aug;13(8):844-54.

48. 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 Jun;9(3):322-9

49. 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 Aug;23(8):878-84.

50. Reilly PL, Simpson DA, Sprod R, Thomas L. Assessing the conscious level in infants and young children: a paediatric version of the Glasgow Coma Scale. Childs Nerv Syst. 1988 Feb;4(1):30-3

51. 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 Sep;48(9):1932-1943.

52. Stead LG, Bodhit AN, Patel PS, Daneshvar Y, Peters KR, Mazzuoccolo A, Kuchibhotla S, Pulvino C, Hatchitt K, Lottenberg L, Elie-Turenne MC. TBI surveillance using the common data elements for traumatic brain injury: a population study. International journal of emergency medicine. 2013 Dec;6:1-7.

53. Seidel JS, Henderson D, Tittle S, Jaffe DM, Spaite D, Dean JM, Gausche M, Lewis RJ, Cooper A, Zaritsky A, Espisito T. Priorities for research in emergency medical services for children: results of a consensus conference. Annals of emergency medicine. 1999 Feb 1;33(2):206-10.

54. Lohr KN, Durch JS, editors. Emergency medical services for children. National Academies Press; 1993 Feb 1.

55. Stiell IG, Wells GA, Vandemheen K, Clement C, Lesiuk H, Laupacis A, McKnight RD, Verbeek R, Brison R, Cass D, Eisenhauer MA. The Canadian CT Head Rule for patients with minor head injury. The Lancet. 2001 May 5;357(9266):1391-6.

56. Jagoda AS, Bazarian JJ, Bruns Jr JJ, Cantrill SV, Gean AD, Howard PK, Ghajar J, Riggio S, Wright DW, Wears RL, Bakshy A. American College of Emergency Physicians; Centers for Disease Control and Prevention. Clinical policy: neuroimaging and decisionmaking in adult mild traumatic brain injury in the acute setting. Ann Emerg Med. 2008;52(6):714-48.

57. Raja AS, Andruchow J, Zane R, Khorasani R, Schuur JD. Use of neuroimaging in US emergency departments. Archives of Internal Medicine. 2011 Feb 14;171(3):260-2.

58. Korley FK, Pham JC, Kirsch TD. Use of advanced radiology during visits to US emergency departments for injury-related conditions, 1998-2007. Jama. 2010 Oct 6;304(13):1465-71.

59. Zonfrillo MR, Kim KH, Arbogast KB. Emergency department visits and head computed tomography utilization for concussion patients from 2006 to 2011. Academic Emergency Medicine. 2015 Jul;22(7):872-7.

60. Haydel MJ, Preston CA, Mills TJ, Luber S, Blaudeau E, DeBlieux PM. Indications for computed tomography in patients with minor head injury. New England Journal of Medicine. 2000 Jul 13;343(2):100-5.

ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 704/2022-23

30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "ASSOCIATION OF ISOLATED LOC AND CT FINDINGS IN TRAUMATIC BRAIN INJURY".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Hari Krishnan G

NAME OF THE GUIDE: Dr. Ravi Patil, Dept. of Emergency Medicine.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)

Dr. Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

RESEARCH INFORMED CONSENT FORM

BLDE (Deemed to be University)
Shri. B.M. PATIL Medical College, Hospital & Research Centre,
VIJAYAPURA-586103

TITLE OF THE PROJECT: ASSOCIATION OF ISOLATED LOSS OF CONSCIOUSNESS AND INTRACRANIAL INJURY ON CT BRAIN IN ISOLATED HEAD INURY CASES: OBSERVATIONAL STUDY.

GUIDE: Dr. RAVI B PATIL, MD
PROFESSOR AND HOD
DEPARTMENT OF EMERGENCY MEDICINE

PG STUDENT: Dr HARI KRISHNAN
PG DEPARTMENT OF EMERGENCY MEDICINE

PURPOSE OF RESEARCH:

I have been explained about the reason for doing this study and selecting me as a subject for this study. I have also been given free choice for either being included or not in this study.

PROCEDURE:

I am aware that in addition to routine care received, I will be asked a series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand there is no risk involved and that the patient may experience some discomforts due to panic situation during the examination. This is mainly the observational study and no risk is involved in the study. All the data collected would be kept safe and private.

BENEFIT:

I do understand that my participation in this study will have no direct benefits to me, other than the potential benefit of the research and education.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subjected to confidentiality. Any information about sensitive, personal nature will not be a part of the medical record but will be stored in the investigations research file. If any of the data are used for publication in the medical literature or for teaching purpose, no name will be disclosed, and other identifiers such as photographs will be used only with special written permission taken priorly. I also understand that I may visualize the photograph before granting permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask questions about the study at any time; Dr. HARI KRISHNAN at the department of Emergency Medicine is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr HARI KRISHNAN may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me, resulting directly for participation in this study; if such injury were reported promptly, the appropriate treatment would be available to the patient. But no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have been explained about the purpose of the research, the procedures required and the possible risks to the best of my ability.

______________________ ______________________

Dr. HARI KRISHNAN Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that DR HARI KRISHNAN has explained to me the purpose of the research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read the form and understand this consent. Therefore, I agree to give consent to participate as a subject in this research project.

____

____

Participant / Guardian Date:

____

Witness to signature Date:

ANNEXURE III

B.L.D.E (DEEMED TO BE UNIVERSITY)

SHRI B M PATIL MEDICAL COLLEGE,

VIJAYAPURA, KARNATAKA

SCHEME OF CASE TAKING

INFORMANT:

Name:

Age: IP NO:

Sex: DOA:

Religion:

Residence:

ESI criteria -

Diagnosis –

ICU admission – YES/NO

Need for Ventilation: Yes—if yes then, Non-invasive ventilation / Invasive Ventilation

No

48-hrs outcome: Improved/Deteriorated or Died iThenticate

Similarity Report ID: oid:3618:63684649

PAPER NAME

21BMEMM02-ASSOCIATION OF ISOLATED LOSS OF CONSCIOUSNESS AND CT FINDINGS IN TRAUMATIC BRAIN INJURY."

AUTHOR

HARI KRISHNAN G

WORD COUNT

12092 Words

CHARACTER COUNT

66114 Characters

PAGE COUNT

58 Pages

FILE SIZE

853.2KB

SUBMISSION DATE

Jul 31, 2024 9:58 AM GMT+5:30

REPORT DATE

Jul 31, 2024 9:59 AM GMT+5:30

9% Overall Similarity

The combined total of all matches, including overlapping sources, for each database.

- 9% Internet database
- 4% Publications database
- Crossref database
- Crossref Posted Content database

Excluded from Similarity Report

- Submitted Works database
- Bibliographic material
- Quoted material
- Cited material
- Small Matches (Less then 14 words)