PROFILE AND OUTCOME OF TRAUMATIC BRAIN INJURY PATIENTS IN A TERTIARY CARE CENTRE IN NORTH KARNATAKA:

(A PROSPECTIVE HOSPITAL-BASED STUDY)

By

DR. BINDUMADHAV YENDIGERI

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Under the guidance of

DR. UDAYKUMAR C NUCHHI

PROFESSOR & HOD, DEPARTMENT OF FORENSIC MEDICINE AND TOXICOLOGY

CO GUIDANCE OF

DR. BASAVARAJ BADADAL, MS, MCh (NEUROSURGERY)

ASSISTANT PROFESSOR, DEPARTMENT OF SURGERY

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B. L. D. E. (Deemed to be university)
SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR, KARNATAKA
B. L. D. E. (Deemed to be university)
SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, VIJAYAPUR.

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DATE – 31/7/2024
PLACE – Vijayapura

DR. BINDUMADHAV YENDIGERI

B. L. D. E. (Deemed to be university)

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

CERTIFICATE BY THE GUIDE

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DR UDAYKUMAR C NUCHHI
PROFESSOR & HOD
Department of Forensic Medicine and Toxicology
B. L. D. E (DU) Shri B. M. Patil Medical College
Hospital & Research Centre,
Vijayapura.

Date: 31/7/2024
PLACE – Vijayapura

B. L. D. E. (Deemed to be university)
SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL &RESEARCH CENTRE, VIJAYAPURA.

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DR BASAVARAJ BADADAL
ASSOCIATE PROFESSOR
Department of Surgery
B. L. D. E (DU) Shri B. M. Patil Medical College
Hospital & Research Centre,
Vijayapura.

Date: 31/7/2024
PLACE – Vijayapura

B. L. D. E. (Deemed to be university)

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

ENDORSEMENT BY THE HOD AND PRINCIPAL

This is to certify that the dissertation entitled **"PROFILE AND OUTCOME OF TRAUMATIC BRAIN INJURY PATIENTS IN A TERTIARY CARE CENTRE IN NORTH KARNATAKA: A PROSPECTIVE HOSPITAL-BASED STUDY"** is a bonafide research work done by Dr. Bindumadhav Yendigeri Under the guidance of Dr. Udaykumar C Nuchhi, Professor & HOD, Department of Forensic medicine and Toxicology, at BLDE (Deemed to be University) Shri. B. M. Patil Medical College Hospital and Research Centre, Vijayapura.

Dr. UDAYKUMAR C NUCCHI

Professor & HOD

Department of Forensic Medicine and Toxicology

BLDE (DU)

Shri. B. M. Patil Medical College Hospital and Research Centre, Vijayapura.

Dr. ARAVIND V PATIL

Principal & Dean Faculty of Medicine

BLDE (DU)

Shri. B. M. Patil Medical College Hospital and Research Centre, Vijayapura.

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DR. BINDUMADHAV YENDIGERI

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DR. BINDUMADHAV YENDIGERI

ABSTRACT

Background

Traumatic Brian injury (TBI) has been one among the significant Public Health problems. It remains one of the leading causes of Mortality, morbidity, disability in developed and developing nations. It was estimated that TBI would be the third main cause of death and disability by 2020 according to World Health organization1. It is a leading cause of disability and death among young adults in the world, with devastating impact on patients and their families.

Objective

The objective of study is to assess the Epidemiological profile of patients with Traumatic brain injury attending tertiary care hospital in North Karnataka and to follow them up for six months, document the outcome and estimate the outcome predictors for the same

Methods

Over the course of three months, 172 cases of Traumatic Brain Injury at the Shri B.M. Patil Medical College, Hospital & Research Centre, Vijayapura, were studied as a part of an observational study. The TBI cases with or without Loss of consciousness and/or CT scan findings of Traumatic Brain Injury were selected. The data collection has been done in six different Google formats, which included initial assessment and care, details of hospital stay and follow up after 3rd and 6th month separately and autopsy details, in case of death.

Results

The study revealed, male preponderance (87.8%), the distribution was more in early adulthood (age group (18-45 years) (82%) for TBI than other age group. The area distribution was Rural (71%), semi urban (12%) and Urban (17%),. RTA(85%) was cause of most TBI cases followed by fall from height (10%) and Assault (5%). Most of the cases presented within first hour (37.9%) and subsequent 6 hours (41%) of time since injury. The presentation symptoms were LOC (94%), Vomiting (66%), Seizures (1%). 11 were brought dead cases, and rest presented as Mild TBI (55.3%), Moderate TBI (24.3%), Severe TBI (20.5%). On CT Scan, Skull fractures (63.4%), Traumatic SAH (36.6%), Epidural Hematoma (15.7%), Subdural Hematoma (30.8%), Contusions (44.2%), Diffuse axonal Injury (9.3%) were found. Out of 172 cases, 126 cases were admitted and treated conservatively (72.2%), Underwent neurosurgery (18.3%), other surgical procedures (7.9%) and observation (1.6%), during admission 5.6% of cases suffered non-neurological complications. 8.1% of cases treated at the hospital on OPD and IPD basis, succumbed to death, and 85.8% had upper good recovery on GOS-E score. Out of 24 cases that

underwent neurosurgery, only 4 cases succumbed to death. Age, gender, and delay in presentation were not significantly associated as outcome predictor of TBI in this study with p value >0.05. GCS score at initial presentation was associated with high significance, as outcome predictor at patient's discharge, 3rd month and 6th month follow ups with p value <0.001.

Conclusion

TBI is a major health problem with RTA being the most common cause of TBI. The occurrence is highest and prognosis is better of Mild TBI, though those suffering from Moderate and severe TBI suffer disabilities of various degree and/or death. Neurosurgery is having better outcome in those who are operable. GCS at initial presentation is an outcome predictor for TBI cases, at discharge, as well, at the end of 3rd and 6th month follow up.

LIST OF ABBREVIATION:

RTA: Road Traffic Accident

SDH: Subdural Haemorrhage

SAH: Subarachnoid Haemorrhage

MLC; Medico legal Case

OPD: Outpatient Department

IPD: Inpatient department

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INTRODUCTION

ANATOMY OF SKULL AND BRAIN

SKULL

In an adult individual, the skull is made up of 22 bones that are joined together by cranial sutures. The skull serves as both a defensive and structural support structure. During development, the skull hardens and fuses to protect the brainstem, cerebellum, and orbits, which are situated within the skull. Along with protecting neurovascular structures, housing several sinuses to handle pressure increases, and providing muscular and tendinous attachments for support, it also supports the muscles of the face and scalp.

CALVARIA AND SKULL BASE

The cerebral cortex, cerebellum, and contents of the orbits are shielded by the calvaria, the topmost portion of the skull. The frontal bone, parietal bone, temporal bone, and occipital bone make up this structure. The transverse mid-anterior connection of the frontal bone and the two parietal bones is known as the coronal suture. The squamosal sutures connect the parietal bones to the temporal bones inferiorly, and the lambdoid suture connects the parietal bones to the occipital bone posteriorly. The sagittal suture, which connects the two parietal bones, is located along the anterior-posterior axis. The articulation of the sphenoid, frontal, parietal, and temporal bones just superior to the pinna is known as the pterion.The parietal, temporal, and occipital bones articulate at the asterion. Finally, a variety of neurovascular systems can enter through the base of the skull. It is made up of portions of the frontal, temporal, and occipital bones as well as the sphenoid and ethmoid bones, which have air sinuses connected to them.

The frontal bone forms the superior aspect of the orbits from the anterior direction. One important frontal bone feature in the midline is the glabella. It is situated between the superciliary ridges and above the nasion. Deep to the brow ridges are the frontal sinuses. The coronal and sagittal sutures meet at the bregma, while the lambdoid and sagittal sutures meet at the lambda. There are four divisions of the temporal bones: petrous, squamous, zygomatic, and mastoid. The inner ear is located in the petrous part. The bony protrusion known as the mastoid, which has a sinus attached to it, is located behind the ear. The most posterior part of the skull is called the occipital bone.

INTRACRANIAL FOSSAE

Three cranial fossae exist, each with a unique set of structural markers. The frontal, sphenoid, and ethmoid bones combine to form the anterior cranial fossa. The sphenoid bone and two temporal bones combine to produce the middle cranial fossa. Ultimately, the occipital bone and two temporal bones combine to form the posterior cranial fossa. The following is a list of each fossa's important anatomic landmarks.

- Anterior Cranial Fossa (contains frontal lobe of the brain)
    - Cribriform plate
- Middle Cranial Fossa (contains temporal lobe of the brain)
    - Optic canal
    - Superior orbital fissure
    - Foramen spinosum
    - Foramen rotundum
    - Foramen ovale
- Posterior Cranial Fossa (contains the cerebellum)
    - Internal auditory meatus
    - Jugular foramen
    - Foramen magnum
    - Hypoglossal canal

FACIAL BONES

There are fourteen face bones, each with distinct embryologic growth mechanisms and anatomical landmarks. These consist of the jaw, the vomer, the two nasal conchae, the two nasal bones, the two maxilla bones, the two palatine bones, the two lacrimal bones, the two zygomatic bones, and the mandible. There are air sinuses connected to the maxillae. An important landmark for efficient mastication is the temporomandibular joint (TMJ).

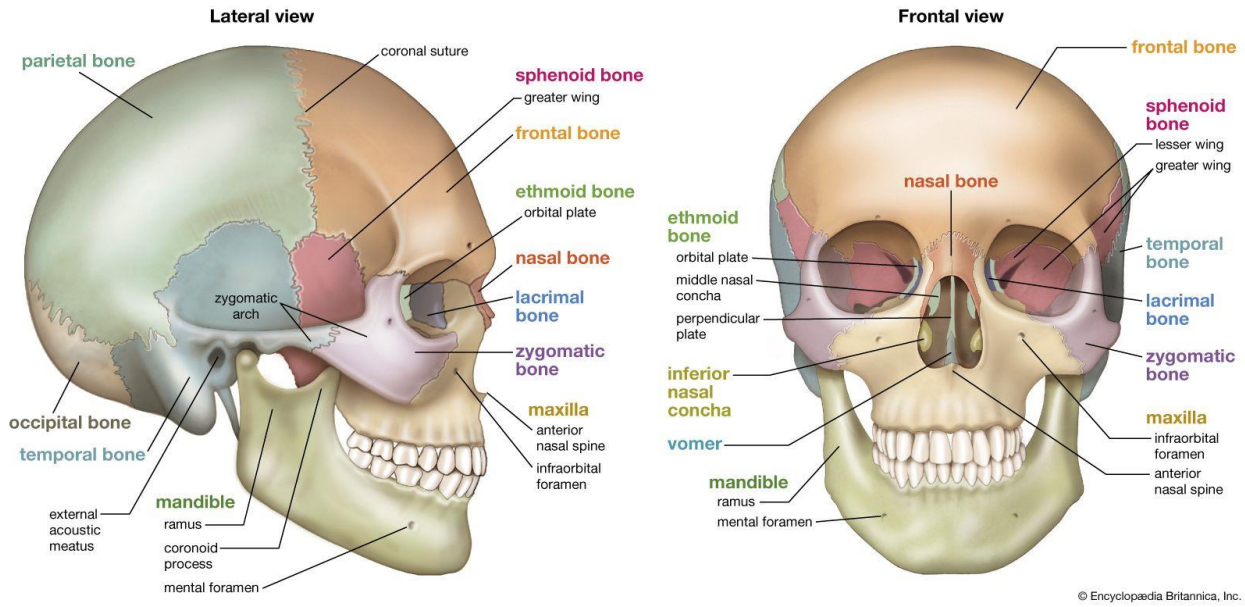


Fig:1 Skull bones

BRAIN

THE MENINGES

The bone covering known as the cranium contains the brain. The brain is shielded from injuries by the skull. The skull is the collective term for the cranium and face-protecting bones. The meninges, a three-layered covering of tissue that covers and shields the brain and spinal cord, are located between the skull and the brain. The dura mater, arachnoid, and pia mater are arranged inward from the outermost layer.

Dura Mater: Two layers of white, nonelastic film or membrane make up the dura mater in the brain. The periosteum is the term for the outer layer. The dura, the inner layer that lines the inside of the skull, forms tiny folds or compartments that serve as safe havens for various brain regions. The falx and the tentorium are the names of two unique dura folds found in the brain. The

tentorium divides the brain's upper and lower regions, and the falx divides the brain's left and right halves.

Arachnoid: The arachnoid is the second layer of the meninges. The entire brain is covered by this delicate, thin membrane. There is a space between the dura and the arachnoid membranes that is called the subdural space. The arachnoid is composed of blood veins of different diameters and delicate, elastic tissue.

Pia Mater: The piamater is the layer of meninges that lies closest to the brain's surface. Numerous blood veins that extend deeply into the brain's surface are found in the pia mater. The pia, which encompasses the whole brain, traces the contours of the brain. The pia receives its blood vessels from the main arteries that supply the brain. The subarachnoid space is the area between the arachnoid and the piamater. The cerebrospinal fluid flows in this region.

CEREBROSPINAL FLUID

The fluid that surrounds the brain and spinal cord is called cerebrospinal fluid, or CSF. It is a transparent, aqueous material that aids in protecting the spinal cord and brain from harm. This fluid is continuously absorbed and replaced as it travels via pathways surrounding the brain and spinal cord. The fluid is created in the brain's ventricles, which are hollow canals. The majority of CSF production occurs in the choroid plexus, a specialized tissue found within each ventricle. Normal brain function involves maintaining a balance between the amount of CSF generated and absorbed.

THE VENTRICULAR SYSTEM

The ventricular system consists of four cavities called ventricles that are joined by foramen.

The term "lateral ventricles" refers to the first and second ventricles, which are located within the cerebral hemispheres. Through individual apertures known as the Foramen of Munro, they are able to communicate with the third ventricle. The thalamus and hypothalamus make up the walls of the third ventricle, which is located in the middle of the brain. The Aqueduct of Sylvius is a lengthy conduit that joins the third and fourth ventricles.Via a further set of apertures, CSF that enters the fourth ventricle travels around the brain and spinal cord.

BRAIN COMPONENTS AND FUNCTIONS

CEREBRUM

The term "gray matter" refers to the grayish-brown color of the cerebral cortex. The brain seems to have wrinkles on its surface. Small grooves called sulci, wider grooves called fissures, and bulges between the grooves called gyri are all present in the cerebral cortex. The depressions and furrows found on the surface of the brain have particular names among scientists. The precise roles of the different brain areas have been identified after decades of scientific investigation. The white region known as "white matter" is made up of fibers that connect neurons beneath the cerebral cortex, or surface, of the brain.

There are multiple discrete fissures in the brain hemispheres. The brain can be divided into pairs of "lobes" by placing these markers on its surface. Simply said, lobes are large areas of the brain. There are pairs of frontal, temporal, parietal, and occipital lobes in the cerebrum, or brain. There are frontal, temporal, parietal, and occipital lobes in each hemisphere. Again, each lobe can be further subdivided into regions with highly specific tasks. The brain's lobes interact with one another in incredibly intricate ways to perform various functions; they do not work independently.

Within the brain, messages can be conveyed in numerous ways. Pathways are the routes by which the signals are transferred. Loss of function, including the capacity to read, speak, and obey simple verbal directions, will be the outcome. Communications can move from one brain bulge to another (gyri to gyri), between lobes, from one side of the brain to the other, between lobes and deep brain structures (thalamus, for example), or between deep brain structures and other parts of the central nervous system.

THE CEREBRAL LOBES

FRONTAL LOBES

Of the four lobes, the frontal lobes are the biggest and perform a wide range of activities. These encompass cognitive and behavioral abilities, voluntary movement, communication, and motor skills. The precentralgyrus or primary motor cortex contains the regions responsible for generating movement in different body parts. Memory, intelligence, focus, temperament, and personality are all significantly influenced by the prefrontal cortex.

Adjacent to the primary motor cortex lies, the premotor cortex. It directs head and eye motions as well as an individual's perception of direction. The Broca's region in the frontal lobe, which is crucial for language creation, is often located on the left side.

OCCIPITAL LOBES

People are able to receive and analyse visual information. They affect how people perceive shapes and colours. For the left visual space, the right occipital lobe interprets visual information, and for the right visual space, the left occipital lobe does the same.

PARIETAL LOBES

These lobes concurrently process information from the brain's other regions, including vision, hearing, motor, sensory, and memory.

TEMPORAL LOBES

These lobes, which are separated into two sections, beneath the temporal bones. Each hemisphere has two parts: a ventral part located at the bottom and a lateral part located on the side. The right side of the brain is linked to visual memory and aids in facial and object recognition. The left side of the brain is linked to verbal memory, which aids with language comprehension and remembering. People are able to decipher the feelings and responses of others due to the temporal lobe.

BRAINSTEM

Positioned anterior to the cerebellum and attached to the spinal cord, the brainstem is the inferior projection of the brain. The medulla oblongata, pons, and midbrain make up its three structural components. It acts as a communication hub, relaying information from the cerebral cortex to different areas of the body. This is where a lot of basic or archaic functions that are necessary for survival are found.

While the pons is involved in facial sensibility, hearing, balance, and coordination of eye and facial motions, the midbrain is a key hub for ocular motility.

Blood pressure, cardiac rhythms, respiration, and swallowing are all regulated by the medulla oblongata. The pons and the brainstem transmit messages from the cortex to the spinal cord and

the nerves that emerge from the spinal cord. "Brain death" will result from damage to these brain regions. Humans cannot survive without these essential processes.

The medulla, pons, midbrain, and a portion of the thalamus contain the reticular activating system. It regulates wakefulness, makes it possible for humans to focus on their surroundings, and affects sleep cycles.

Ten of the twelve cranial nerves, which regulate hearing, eye movement, taste, swallowing, and movements of the muscles of the face, neck, shoulder, and tongue, have their origins in the brainstem. The cerebrum is the source of the cranial nerves responsible for vision and smell. The pons is the source of four pairs of cranial nerves, numbered five to eight.

CEREBELLUM

Beneath the occipital lobes in the rear of the brain is the cerebellum. It is isolated from the cerebrum by the dura's tentorium. The cerebellum adjusts motor function or movement, such as the precise movements of fingers during surgery or painting. By regulating the tone of muscles and limb position, it aids in the maintenance of posture, balance, or equilibrium. Playing video games or other fast-paced repetitive tasks requires the use of the cerebellum. Right-sided abnormalities in the cerebellum cause symptoms on the same side of the body.

CRANIAL NERVES

The brain itself is the source of 12 pairs of nerves. Very specific functions are controlled by these nerves.

HYPOTHALAMUS

The pituitary gland receives information from the little structure known as the hypothalamus, which is home to nerve connections. The autonomic nerve system sends signals to the hypothalamus. It affects the regulation of bodily temperature, emotions, hormone secretion, mobility, and sexual and eating behaviours, among other things.

LIMBIC SYSTEM

The amygdala, which is involved in the production of aggressive behaviour, the hippocampus, which aids in the retention of new knowledge, and the hypothalamus, a portion of the thalamus, are all included in this system.

PINEAL GLAND

The posterior, or back, region of the third ventricle gives rise to this gland. It regulates how some mammals react to light and darkness. Although its precise involvement in sexual maturation is unknown, the pineal gland plays a role in human development.

PITUITARY GLAND

Located in the pituitary fossa, also known as the sellaturcica, the pituitary is a little gland that is linked to the base of the brain, which is behind the nose. The pituitary is frequently referred to as the "master gland" because to its ability to regulate hormone output. The following are under the pituitary's regulation and coordination:Development and growth; the operation of other body organs, such as the kidneys, breasts, and uterus; and the operation of other glands, such as the thyroid, gonads, and adrenal glands

POSTERIOR FOSSA

This is a recess in the back of the skull that houses the brainstem, cranial nerves 5–12, and cerebellum.

THALAMUS

Almost all information that travels to and from the cortex is relayed via the thalamus. It affects awareness, attentiveness, and pain perception. The hypothalamus, epithalamus, ventral thalamus, and dorsal thalamus are its four constituent regions. The thalamus is surrounded by nerve cell clusters called basal ganglia.

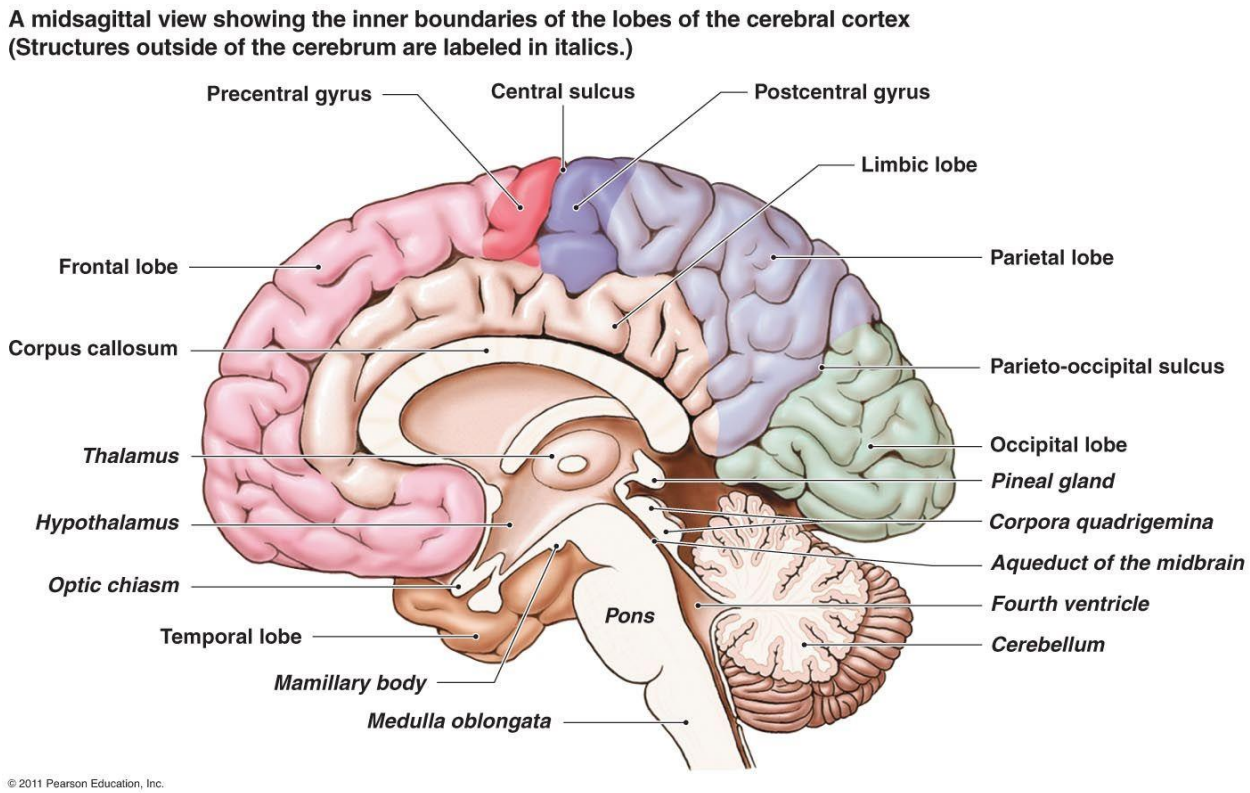


Fig:2 Components of Brain

TRAUMATIC BRAIN INJURIES

INRODUCTION:

The term "traumatic brain injury" (TBI) refers to brain damage caused by an outside mechanical force. TBI is generally divided into two categories: penetrating and closed. The latter is identified by breaching the dura mater and skull. Closed head injury (CHI) is the more prevalent of the two. Concussion, contusion, diffuse axonal damage, and intracranial hematoma (which includes intraparenchymal hemorrhage, subdural hemorrhage, subarachnoid hemorrhage, and epidural hematoma) are examples of CHI types. The terms TBI and CHI are usually used interchangeably.

TBI is frequently categorized using the Glasgow Coma Scale (GCS) as mild, moderate, or severe. Over 80% of TBI cases are classified as mild TBI, sometimes known as "concussions," and are characterized by a GCS of 14 to 15. About 10% of TBIs fall into the category of moderate TBI, which is indicated by a GCS of 9 to 13. An index of severe TBI is a GCS of 3 to 8.

EPIMEDIOLOGY

Traumatic Brian injury (TBI) has been one among the significant Public Health problems. It remains one of the leading causes of Mortality, morbidity, disability in developed and developing nations. It was estimated that TBI would be the third main cause of death and disability by 2020 according to World Health organization[1]. It is a leading cause of disability and death among young adults in the world, with devastating impact on patients and their families.

Traumatic brain injury is defined as an alteration in brain function, or any other evidence of brain pathology, caused by an external force, as according to the Brain Injury Association of America.

The age standardized incidence rate of TBI is around 369 per 100000 population, and global age standardized prevalence of TBI is around 759 per 100000 population. The age standardized prevalence rates and YLD rates increased by 8.4% and 8.5% respectively from 1996 to 2016. Falls were the main cause of TBI, followed by road traffic accidents[2].

According to a meta-analysis study, the prevalence of TBI in general adult population is 12.1%, (16.7% in males, 8.5% in females)[3].

TBI is most commonly due to Road traffic accident in Low and middle income countries[4].

Young generation (18-29 age group) is at maximum risk for head injury due to Road traffic accidents, due to a high incidence of alcohol and drug abuse, poor judgment and decision making ability[5].

In a study at India, Only 13.4% two wheeler vehicular patients were wearing helmets and 7.5% of four wheeler vehicular patients were wearing seatbelts at the time of accident, in spite of legislation and many public awareness programs. About 17.6% of all injured patients had history of Alcohol consumption prior to driving[5].

Recent studies have shown that there is lack of data and lack of evidence in India on TBI, with data being published only 20,000 patients with TBI over 27 years. This accounts to only less than 1,000 TBI cases studied/year. This hardly would represent 0.1% of the population with TBI[6].

An increasing incidence of Traumatic Brain Injury in the productive age group is of major concern. Both the extremes of age group are more susceptible to severe injury and poor outcome. Quantitative analysis of injuries and outcomes of TBI patients shows a better impact on health in the economically productive population and in patients in the extreme age group[7].

Some studies have been stressing on the development of high quality, standardized epidemiological data in India that would help reduce the growing economic and social burden due to TBI. There is necessity of designing Trauma systems which link rural and next level centres for trauma. These mid-systems should have guidelines to help the local doctors in immediate management and referral to the nearest trauma centre[8].

There is a scope for recognizing best practices by comparative effectiveness research on TBI. The identified risk factors by prognostic analyses might help to develop steps to assess quality of care[9].

ETIOLOGY:

Over 35% of CHI cases are caused by falls, making them the most common cause of CHI. Motor vehicle/traffic injuries come in second. Acts of aggression, workplace accidents, and sports-related injuries are additional causes. When assessing a patient with CHI, the potential for non-accidental trauma, elder abuse, and marital violence should be taken into account.

PATHOPHYSIOLOGY:

Determining cerebral blood flow is a technically challenging task. Brain blood flow is mostly determined by cerebral perfusion pressure. By monitoring intracranial pressure (ICP) and mean artery pressure (MAP), one may estimate cerebral perfusion pressure (CPP) using the formula CPP=MAP-ICP. The relative concentrations of blood, brain parenchyma, and cerebrospinal fluid (CSF) in the inelastic space of the skull determine the intracranial pressure (ICP). According to the Monro-Kellie doctrine, an intracranial increase in blood, brain parenchyma, or CSF volume requires a corresponding drop in another compartment's capacity, or the intracranial pressure will rise. ICP can rise in the event of a head injury, as will be covered below.CPP drops to levels that are dangerously low when ICP gets close to the MAP. In response, the body raises MAP and dilates cerebral blood vessels, which raises ICP even more. This vicious loop can diminish CPP and result in long-term damage and brain ischemia.

The two types of brain lesions that influence TBI outcomes are primary and secondary. The direct impact force that causes damage to the brain parenchyma is known as primary brain injury. Diffuse axonal injury (DAI), hematomas, contusions, direct cellular destruction, disruption of

neurochemical and electrochemical function, and loss of the blood-brain barrier are examples of primary brain traumas.Subsequent neuronal damage brought on by neurotransmitter release, the presence of inflammatory mediators, and apoptosis is known as secondary brain injury. It's important to separate secondary brain injury from secondary insult, which encompasses a variety of conditions including hypoxia and hypotension that can hasten brain damage and exacerbate consequences.

Three distinct substances make up the intracranial compartment's volume: cerebrospinal fluid (CSF, 11%), blood (6%), and brain parenchyma (83%). For the skull to maintain a homeostatic environment, each of these components needs the other. But when the volume of the brain surpasses the sum of its regular parts, a series of compensatory processes happen. In the damaged brain, venous congestion, cytotoxic and vasogenic edema, and the mass effect of blood can all result in an increase in intracranial volume.Brain tissue cannot be compressed. Therefore, CSF will initially extrude into the spinal compartment due to edematous brain tissue. Blood eventually comes out of the brain as well, particularly venous blood. The compensatory processes fail in the absence of appropriate intervention, and in certain cases even with maximal intervention; the consequence is pathological brain compression and death.

ENLISTING THE TRAUMATIC BRAIN INJURIES

CONCUSSION

Since they result from a nonpenetrating TBI, concussive injuries are frequently considered to be mild TBIs without any noticeable structural damage. They typically occur after direct hits to the head, causing forces to accelerate and decelerate. After suffering a concussion, a person usually experiences varied degrees of momentary changed mental status, from mild bewilderment to momentary unconsciousness.Standard neuroradiographic imaging methods, such as magnetic resonance imaging (MRI) and computerized axial tomography (commonly known as computed tomography [CT] scan), do not reveal any abnormalities right away. On the other hand, more recent MRI imaging methods including functional MRI and diffusion tensor imaging could lead to an earlier concussion diagnosis. It has been suggested that even in the case of a moderate traumatic brain injury, there may be some degree of axonal damage.Second impact syndrome is an extremely uncommon ailment that primarily affects sportsmen. Concussions are frequently the initial injury; however, a player may return to play too soon and get another concussion while still

recovering from the first one. The mechanism usually entails the quick development of malignant cerebral edema, which arises shortly after the second injury, which frequently occurs on the playing field. Between 50% and 100% is the range of the death rate.

CHRONIC TRAUMATIC ENCEPHALOPATHY(CTE):

Repeated mild traumatic brain injury (TBI) can cause CTE, a delayed manifestation. This phenomenon has garnered media attention due to the unpleasant side effect of chronic fatigue injury (CTE) being psychological disorders, which in turn cause suicidal conduct in a number of prominent professional sportsmen. Dysarthric speech, tremors, attention problems, memory and executive function impairments, incoordination, and pyramidal symptoms are some additional clinical indicators of CTE. The progression of progressive neuronal degeneration is probably the cause of CTE.

EXTRA-AXIAL HEMATOMAS

Subdural hematomas (SDH) and epidural hematomas (EDH) are the two types of extra-axial hematomas. EDH are typically caused by a direct hit to the temporal region, which can occasionally result in a fractured skull and the interruption of the middle meningeal artery. However, more posteriorly oriented EDH has also been explained by venous injuries, such as disruption of the transverse sinus. When a threshold level of intracranial pressure (ICP) is reached, EDH can rise fast, resulting in virtually normal mentation at initial presentation, followed by deterioration down the cascade of herniation symptoms.

While SDH appear differently depending on the patient's age and the chronicity of blood products, EDH are nearly always observed in the acute environment. When trauma occurs, shearing lesions to the bridging veins are frequently caused by the acceleration and deceleration of the brain's surface against the skull's underside. In the context of trauma, acute SDH can be extremely harmful for the patient since they typically involve a significantly higher degree of underlying brain injury than does an EDH. If untreated, the underlying cerebral edema frequently plays a major role in the subsequent midline shifting of structures and the development of herniation syndromes. Certain SDH remain hidden from the patient for a while. Subacute and chronic SDH may occur as acute blood in the subdural space liquefies over time. The clinical manifestation of subdural hematoma in the subacute and chronic settings is not nearly as fast and progressive as it is in the acute phase. Elderly people are more likely to experience persistent SDH, and this is often the case when they are using antiplatelet or anticoagulant medications. Subacute and chronic SDHs typically manifest more subtly, with headaches, hemiparesis, speech difficulties, disorientation, and abnormal

mentation being the most common symptoms. Untreated unilateral or bilateral chronic SDH can occasionally cause a patient to present with severe neurological impairment.

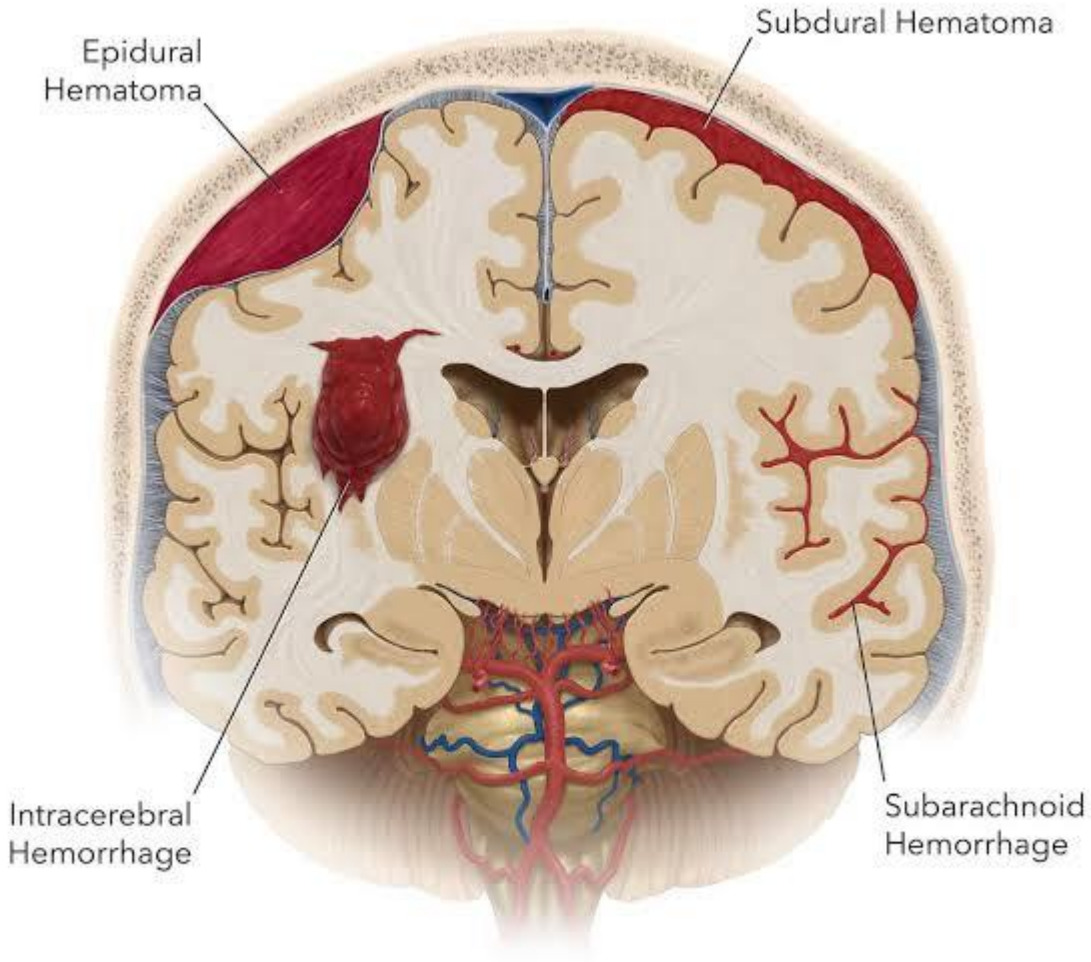


Fig:3. Extra Axial Haemorrhages

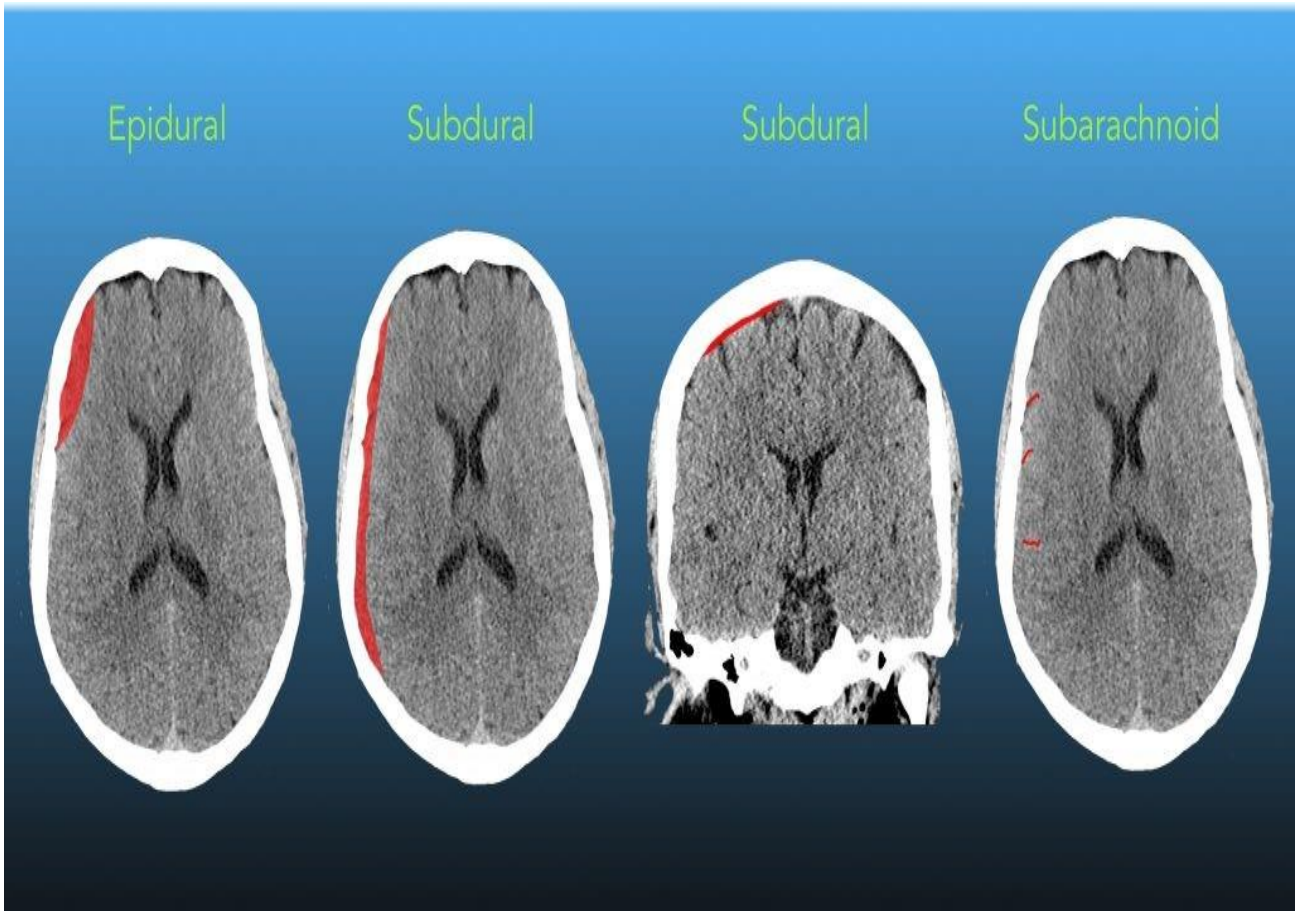


Fig:4. CT scan Extra Axial Haemorrhages

CONTUSIONS

In most cases, coup and contrecoup forces result in contusions. Contralateral side of impact is where contrecoup injuries usually occur, most frequently resulting in damage to the frontal and anterior temporal lobes. Coup injuries happen at the location of impact.

TRAUMATIC SUBARACHNOID HEMORRHAGE

Trauma is the most common cause of subarachnoid hemorrhage, which occurs when tiny capillaries rupture and temporarily leak blood into the subarachnoid region. Because spontaneous aneurysmal subarachnoid hemorrhage projects blood into the subarachnoid space under artery pressure, traumatic subarachnoid hemorrhage typically causes less severe brain damage than spontaneous aneurysmal subarachnoid hemorrhage.

DIFFUSE AXONAL INJURY (DAI)

The most severe type of axonal shearing injury is DAI. In most cases, significant rotational acceleration or deceleration forces are necessary for this kind of injury to occur. When visible radiographically, it is frequently detected as faint hemorrhagic foci in regions such the brainstem, thalamus, internal capsule, corona radiata, and corpus callosum on T2 and gradient echo sequences. The degree of clinical presentation in patients can vary depending on the location of the axonal shearing. Some people with DAI may exhibit altered consciousness for a few days, whilst other patients may show signs of internal capsule involvement and hemiparesis. Others never come to because certain areas of the reticular activating system have lost their axonal integrity.

HISTORY AND CLINICAL EXAMINATION:

It is recommended to adhere to the ATLS (Advanced Trauma Life Support) protocol for these patients, as they frequently suffer from multisystem trauma. EMS, the patient (if able to give a coherent narrative), the patient's family, and any witnesses should be contacted to get their history. Getting a history of the injury's mechanism, any recognized focal neurologic abnormalities, seizures, vomiting, or shifts in consciousness since the injury is crucial. Intoxication, the use of anticoagulant or antiplatelet drugs, and other concomitant conditions are further significant questions.

The Glasgow coma scale (GCS), which will be trended during the hospital stay, is a crucial component of both the history taken from EMS and the physical examination. Based on eye-opening, verbal response, and motor reaction, GCS is graded on a range of 1 to 15. A single fixed and dilated pupil on a follow-up physical examination of the head-injured patient in a coma or disturbed mental state raises the possibility of an uncal herniation. Bilateral uncal herniation, hypoxemia, or a markedly elevated ICP with inadequate perfusion can all be indicated by bilateral fixed and dilated pupils. Examine how the order and/or unpleasant sensations cause the upper and lower extremities to move. Upper extremity flexion and lower extremities extension, or decorticate posturing, point to a serious damage most likely above the midbrain region. A more caudal brain damage is indicated by decerebrate posture, which involves internal rotation and extension of the upper and lower limbs along with bending of the wrist and fingers. Examine the brainstem reflexes

in comatose patients that are helpful for diagnosis and prognostication, such as the respiratory pattern, pupillary response, corneal reaction, gag reflex, and cough.

Glasgow Coma Scale

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1

Fig:5. Glasgow Coma Scale

Regular repetition of the physical examination is recommended since any alterations could point to an approaching herniation or elevated ICP. Systolic hypertension, bradycardia, agonal respirations (often referred to as Cushing's triad), intense headache, altered vision, nausea, vomiting, lethargy, focal weakness/paresthesias, or coma are indications of rising ICP. Hemiparesis, unilateral or bilateral pupillary dilatation, gradual neurologic deterioration, and aberrant posturing might all be indicators of an imminent brain shift or herniation.

INVESTIGATIONS

When it comes to acute intracranial bleeding, CT is incredibly sensitive. When a patient's potential for severe traumatic brain damage is suspected, a CT scan should be performed as soon as it is safe to do so. There are many guidelines to help determine which TBI patients require a head CT scan in an effort to reduce the number of needless CT scans. These guidelines are intended to help identify patients who will need neurosurgical care. They don't identify those who may experience transient or permanent neurologic effects from traumatic brain injury. The Canadian Head CT Rule and the New Orleans Criteria are the two most often applied guidelines for adults. While the Canadian Head CT Rule is more precise, both are 100% sensitive for clinically severe traumatic intracranial lesions.Notably, these trials did not include individuals on anticoagulants or antiplatelets. The PECARN Pediatric Head Injury/Trauma Algorithm, which is also almost 100%

sensitive for clinically significant intracranial injuries, is the most widely utilized technique for patients under the age of eighteen. Additionally, C-spine imaging warrants careful consideration, particularly in patients who are unconscious. While MRI has not been proven to be of substantial help in the early diagnosis of acute CHI, it is not a standard element of the first evaluation. MRI may be able to further clarify lesions found on CT or discover subtle lesions not picked up on CT. An MRI could reveal further details regarding persistent bleeding.

MANAGEMENT OF TBI

There are two main categories for TBI: primary and secondary brain injuries. Primary brain injury is the outcome of kinetic energy transfer causing damage to brain tissue. The phrase "secondary brain injury" refers to the worsening of traumatic brain injury (TBI) over the course of minutes to hours due to a variety of circumstances, including hypoxemia, hypotension, hypo- or hypercarbia, hypo- or hyperglycemia, hypo- or hyperthermia, and seizures. The main goal of therapeutic measures after traumatic brain injury (TBI) is to prevent further brain injury, which is best handled by an anesthesiologist—ideally a neuroanesthesiologist.

In order to protect the patient's airway and ensure proper breathing and circulation, care for a patient with a traumatic brain injury (TBI) should start at the scene of the accident. As soon as feasible, patients with moderate or severe TBI should be sent to a tertiary care facility equipped with neurosurgical capabilities. Transport techniques, the length of the transit, and whether a doctor or a paramedic leads the responding team have all been demonstrated to affect the outcomes for TBI patients. Since even one episode of hypotension has been linked to a doubling of mortality and an increased risk of morbidity, the main objectives of therapy are to prevent hypoxia and hypotension.

1. Airway Control and Ventilation

Hypoxemia with unfavourable results has been linked in a number of research studies. Studies have shown that TBI patients who were intubated at the scene of trauma had worse outcomes, even though airway control may be our main priority in these patients. In patients whose airway was secured in the emergency room, intubation by less competent clinicians was associated with a considerable risk of worse functional outcomes and a four-fold increase in death. In 2013,

Sobuwa et al. proposed that competent prehospital basic airway care could be substantially superior to incompetent prehospital intubation.

2. Blood Pressure and Cerebral Perfusion Pressure (CPP)

Varied expert bodies have provided varied management guidelines, therefore there is no general consensus on resuscitation goals despite agreement on the concepts of early management. At first, it was advised to maintain CPP above 70 mmHg and, if necessary, use vasopressors. Subsequent research, however, revealed that results were better with a significantly lower CPP, presumably as a result of a decreased incidence of acute respiratory distress syndrome brought on by a decrease in the use of vasopressors.

3. Fluid Management

4. Sedation and Analgesia

5. Osmotherapy

"Mannitol (0.25–1 gm/kg) is useful for preventing hypotension (Level I) and controlling elevated ICP."

6. Anticonvulsant Therapy

Convulsive activity after traumatic brain injury (TBI) raises intracranial pressure and modifies the flow of oxygen to the wounded brain. Many studies have attempted to investigate the potential benefits of seizure prophylaxis in order to prevent subsequent brain injury. According to Temkin et al., phenytoin medication was successful in reducing the frequency of posttraumatic seizures within the first seven days following an accident, but it had no discernible effect on preventing posttraumatic seizures beyond the first week following an injury.

There is no discernible difference in the frequencies of early posttraumatic seizures between individuals treated with phenytoin and those treated with levetiracetam in clinical assessments of the two medications' efficacy in preventing early posttraumatic seizure prophylaxis. Within seven days of an accident, anticonvulsant medication is advised according to the most recent BTF Guidelines. To yet, no randomized controlled study has been conducted to establish the superiority of one antiepileptic medication over another in this particular context.

7. Glycemic Control

Following traumatic brain injury, there is a notable increase in catecholamines, cortisol release, and glucose intolerance that results in significant hyperglycemia. Cerebral edema and neuronal dysfunction may result from the anaerobic metabolism of glucose and the ensuing acidity in the brain. Hyperglycemia after traumatic brain injury has also been linked to impaired cerebrovascular control and poor outcomes. Avoiding glucose-containing liquids and keeping an eye on your blood sugar levels to keep them between 4 and 8 mmol/L are recommended.

8. Decompressive Craniectomy

The most common reasons for operating on TBI patients are to remove intracerebral hematomas (ICH), cerebral contusions, subdural hematomas (SDH), and epidural hematomas (EDH) that are big enough to create a noticeable mass effect on the brain. These hematomas need to be surgically removed as soon as feasible. If a big hematoma is found to be the source of the coma, TBI patients who arrive in the ED in a coma should be brought to surgery right away. In order to stop their neurological condition from getting worse, admitted patients who experience delayed hematoma development or enlargement need to have their hematomas removed as soon as possible.

To achieve appropriate resection, a formal craniotomy is required; burr-hole drainage of these solid clots is not a viable option. Although evidence-based surgical guidelines have been developed, their applicability is limited by the dearth of high-caliber randomized trials in this field. Generally speaking, CT evidence of elevated ICP, such as a midline shift of ≥5 mm and/or compression of the basal cisterns, indicates that a traumatic mass lesion needs to be surgically removed.

A large traumatic hematoma should be removed before neurological impairment arises from hematoma expansion or brain swelling, even in patients with relatively high GCS scores. Lesions in the posterior fossa may be subject to a reduced threshold for surgical intervention.

The procedure known as a decompressive craniectomy (DC), in which a sizable bone flap is removed intentionally or is not rebuilt, has become increasingly common in recent years. The surgeon may elect to forego the bone flap in anticipation of substantial cerebral edema, or he may do so because massive cerebral swelling arises following hematoma evacuation. In other situations, if ICP starts to rise, patients who would not typically require surgery could be brought to the operating room for DC.

The clinical utility of a DC in individuals with high ICP who are unresponsive to medicinal treatment is called into question by a recent study. The results of the randomized controlled DECRA trial showed that while craniectomy patients were able to effectively drop their

intracranial pressure, their neurologic outcomes at six months were inferior than those of patients who were randomized to receive maximal medical therapy. Critics of the experiment have, however, cited the following issues as evidence against the study's conclusions: imbalanced treatment groups, inconsistent medical care for the control group, a high rate of crossover to the surgery arm, and a brief (six-month) follow-up period. Decompressive craniectomy as a treatment for severe traumatic brain injury is still hotly debated.

If the depression is deeper than the surrounding inner table, depressed skull fractures are frequently elevated, particularly if they occur in a prominently visible place such as the forehead. In order to prevent infection, open depressed fractures are best treated surgically; nevertheless, nonoperative care may be tried in some cases, but only if there is no frontal sinus injury, dural laceration, or gross contamination or indications of infection. Surgery is often not recommended for a depressed skull fracture above the sagittal sinus due to the significant risk of uncontrollably large bleeding.

9. Nutrition

Better results are linked to early nutritional supplementation, and enteral feeding has been shown to be advantageous. After an injury, the BTF advises basal calorie replacement by the fifth day at the latest and the seventh day at the latest. Additionally, transgastricjejunal feeding may lessen the chance of pneumonia brought on by a ventilator. The use of opiates and elevated intracranial pressure (ICP) can cause abnormal gastric emptying, which in turn can lead to gastric feeding intolerance in patients with severe TBI. Metoclopramide is one example of a prokinetic drug that may increase feeding tolerance.

For patients who are not paralyzed, the BTF suggests replacing 140% and 100% of the resting metabolic expenditure, respectively; nevertheless, consuming fewer calories may also be advantageous.

10. Anitibiotic Therapy

TBI patients are more likely to be at risk for infection development because they are more likely to undergo intrusive monitoring and therapeutic treatments, such as mechanical breathing. It is necessary to locate possible infection sources and to start the right treatment.

Invasive ICP monitoring is a common source of infection. According to reports, the frequency of ICP device infections varies between 1% and 27%. The majority of research that examined prophylactic antibiotic coverage in TBI patients and was referenced in the BTF guidelines has not revealed many noteworthy variations in infection rates. An further investigation assessing individuals who underwent bacitracin flushes revealed a greater infection rate in the intervention

group. As a Level III suggestion, the current recommendations recommend using catheters impregnated with antibiotics to lower infection rates.

The use of antibiotic prophylaxis in TBI is not well supported by the data at this time, particularly given the possibility that these patients may become more susceptible to more serious infections as a result of the therapy. But after a penetrating TBI, there is strong evidence to support antibiotic medication, and treatment should be continued for at least 7–14 days.

11. Other Considerations

Thromboembolic events provide a serious concern to patients with traumatic brain injury. Pharmacological prophylaxis (low-dose or low-molecular-weight heparin) or mechanical prophylaxis (graduated compression stockings or intermittent pneumatic compression) are the two types of preventive options available. When there are no further contraindications, pharmacological thromboprophylaxis is often started 48–72 hours following neurosurgical surgery. Prophylactic treatment for gastric ulcers, physical therapy, and complete sanitary care are additional services. It is crucial for these patients to get both excellent perioperative critical care and rehabilitative therapy. Gupta et al. have demonstrated that appropriate physiotherapy and postdischarge treatment have been found to be an independent predictor of mortality and morbidity in this patient population.

AUTOPSY

Finding both primary and subsequent brain injury is the goal of autopsy. The popular bimastoid resection is used to make the scalp incision. According to the Rutty procedure, sectioning the skin and subcutaneous tissues of the splanchnocranium might be done if necessary to visualize the orbital cavities, as in the case of Shaken Baby Syndrome (SBS). Subdural hemorrhage, acute encephalopathy, retinal hemorrhage, optic nerve sheath hemorrhage, and little or nonexistent exterior damage indicators are all combined to cause SBS. The excision of the eyeballs can be helpful for later histological investigations in the search for these indicators. Furthermore, given the possibility of asymmetry in an individual's eyes, both eyeballs must to be gathered and examined.

Prior to dissection, the scalp is examined and described after the scalp strips have been turned over. Skin and bone pieces are retrieved for microscopic inspection in cases of gunshot wounds or hemorrhagic infiltration. Prior to the removal of the skull in cases of traumatic skull injuries, several forms of fractures are evaluated and described.

Following the removal of the skull, the brain needs to be examined and described. Macroscopic examination of the brain can reveal hematomas, contusions, and abrasions.

One can categorize subdural hematomas as acute (presenting symptoms within 72 hours), subacute (occurring between 3 days and 3 weeks), or chronic (occurring beyond 3 weeks post-injury). Although some studies suggest a dural origin for the subdural bleeding seen in young infants, this acceleration/deceleration injury is not linked to skull fractures, and it can occur bilaterally or on the ipsilateral or contralateral side of the impact area. It is caused by a shearing force acting upon the parasagittal bridging veins.

The amount of bleeding that persists till death depends on the type of vascular ruptured and the presence of necrosis.Hemorrhages are streak-like and can be solitary or many. When bleeding excessively, this region may enlarge and become intracerebralhemorrhage, extending into the subarachnoid space and white matter. Traumatic head injuries most frequently result in subarachnoid hemorrhage. It has been demonstrated that severe subarachnoid hemorrhage across the base of the brain results from injuries to the internal carotid, vertebral, or basilar arteries, and that these injuries are instantly deadly.

Postmortem subarachnoid hemorrhage may result from blood cell lysis, vascular deterioration, and subsequent blood leaking into the subarachnoid space. Moreover, there may be a little amount of subarachnoid bleeding during the brain's evisceration. Arachnoid membrane and cerebral veins are ruptured during the removal of the skullcap, causing blood to diffuse into the subarachnoid space in the posterior aspect (dependent region) of the cerebral hemispheres and cerebellum. Even though this type of bleeding is typically not severe, if the brain is not removed from the skull right away and is instead allowed to sit for some time, a significant amount of subarachnoid hemorrhage may build up.

Brain herniation, secondary brain stem bleeding, or the emergence of a severe condition of brain swelling over a specific period of time can all result in brain swelling after a major head injury. If this process advances too quickly, it may cause the brain to herniate tonsillarly or transtentorially, which can lead to necrosis, secondary infarction, and Durethemorrhages. The pons-medulla junction can sustain lacerations as a result of violent hyperextension of the head and neck.

Following the brain's evisceration, which in this instance must be done in toto, it is formalin-fixed for additional research, and basilar skull fractures, which are frequent due to the uneven shape and construction of the skull's base, may be seen: Ring fractures (circular fractures of the base of the

skull surrounding the foramen magnum; may be caused by impacts on the top of the head that drive the skull downward onto the vertebral column and impact the tip of the chin), hinge fractures (comprising of basilar fractures that completely bisect the base of the skull), anterior cranial fossae contrecoup fractures (single anterior cranial fossae fractures connected to brain contrecoup injuries, with the impact location on the opposing side of the skull).

Following evisceration, the brain can be examined and dissected using the Virchow or Ludwig technique; macroscopic injury, such as intra-parenchymal bleedings and contusions, will be seen.

Six different types of contusions exist: coup contusions, which cause tensile force injuries to the brain at the impact site; contrecoup contusions, which occur in the brain at locations directly opposite to the point of impact; fracture contusions, which are related to skull fractures; intermediary coup contusions, which are hemorrhagic contusions in the deep brain structures (the white matter, basal ganglia, and corpus callosum, usually observed in falls); gliding contusions, which are focal hemorrhages in the cortex and underlying white matter of the dorsal surfaces of the cerebral hemispheres, primarily in the frontal region (observed in falls and car accidents); Herniation contusions are generally brought on by impaction of the cerebellar tonsils against the foramen magnum or the medial portion of the temporal lobes against the tentorium's margin.

GLASGOW OUTCOME SCALE EXTENDED

In 1975, Jenkins and Bond1 published the Glasgow Outcome Scale (GOS), which is used to evaluate the overall result following a serious brain injury. While the GOS was being built, it was becoming more and more clear that traumatic brain injury (TBI) had long-term effects on the body and mind.The GOS was created to record how an injury affected one's ability to perform in key areas of life. Five potential result categories served as the basis for the initial scoring. The assessment process entailed assigning an outcome category based on the authors' defining text; no additional record was made except the final grade.At the time, the GOS's design was novel and acknowledged two important aspects of the effects of traumatic brain injury. The first is that after brain damage, problems with cognition and mental health play a significant role in impairment. Previously, the majority of the follow-up after.Acute injury concentrated on physical issues, especially those related to walking; the GOS covered more ground than this very limited approach.Secondly, the evaluation showed that global scales may be applied to summarize the results, obviating the requirement to enumerate the several types of impairment brought on by injury. By looking into how impairments ultimately affected the main facets of life following an

injury, the effects of impairment were captured. The Rankin Scale and other global outcome assessment scales are among the family of which the GOS is a member for cancer and the Rankin Scale used for stroke.

Jennett and colleagues subsequently proposed that categories of result may be split into upper and lower bands to produce an enlarged 8-point scale, which would increase the sensitivity of the GOS. Still, There were acknowledged issues with applying the GOS consistently, and this issue was made worse by the larger version.Six General practitioners were far more likely than psychologists who had completed a neuropsychological evaluation to score patients as having a Good Recovery (GR) on the GOS, according to research by Anderson and colleagues.

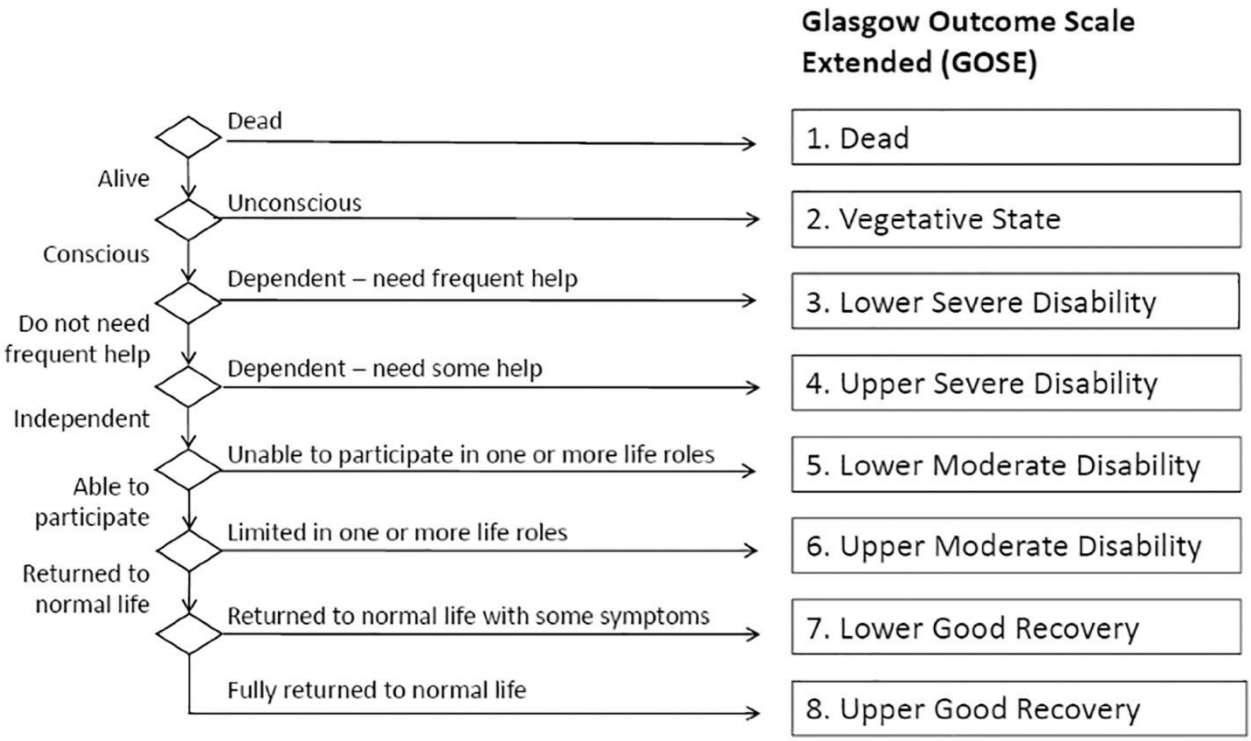


Fig 6. Glasgow Outcome Scale- Extended

To help standardize methods for scoring the GOS and GOSE, a structured version of the Glasgow Outcome Scale Extended (GOSE) interview was released in 1998.To evaluate the GOSE's domains or regions of operation, the interview offers a series of leading questions

The definitional guidelines for evaluating the result are still in place, but the interview questions provide the assessor the freedom to use their discretion to address discrepancies or delve deeper into the details when information is lacking.

Due to its unique format, which consists of a hierarchy of distinct categories, and the absence of a cumulative score derived from individual questions, the GOSE is not like other assessment tools. The technique of allocating a GOSE rating is straightforward in theory: The hierarchy's choice points are employed to determine a result. The GOSE interview schedule is made to help with this process by offering questions that help establish the boundaries between scoring categories and extract important information. As an illustration, there are

Three inquiries pertaining to the line dividing independence from dependence. Certain scenarios are easy to categorize.A person in the Lower Severe Disability (SD) group, for instance, would be someone who is conscious but requires full-time care; someone who is independent but unable to return to work; or forstudents with their schoolwork is Lower Moderate Disability (MD); an individual with no issues or symptoms that are limiting their ability is Upper GR. Independent agreement was demonstrated by seasoned interviewers on 78% of the GOSE outcomes. Nonetheless, there may be problems at borders and in some situations that make assessment difficult.The GOSE focuses on change post-injury, but does not itselfdistinguish changes attributable to injury to the brain from disability caused, for example, by injury to other parts of the body. TheGOSE can be used to assess the consequences of general trauma,including polytrauma, and in this case the effects of all kinds ofinjury are included. The decision about whether to assess theoverall impact of injury or focus on the effects of brain injury willdepend on the purpose of the study.

In TBI research investigations, the GOSE has gained widespread acceptance.

The Food and Drug Administration (FDA)regulators in the United States, have accepted it as the main clinical outcome assessment used to demonstrate the effectiveness of TBI clinical trials. It's the soleresult that is included in the Common Data Elements for TBI as "core." 17 different languages have translated the interview as part of the CENTER-TBI project. It's still difficult to apply the assessment consistently, especially in multi-center research with a large number of assessors.

OUTCOME PREDICTORS OF PATIENTS WITH TBI

One of the most common causes of neurological impairment and mortality is traumatic brain injury (TBI). An estimated 1.5 million people perish worldwide after a TBI. In large trauma centers,

traumatic brain injury (TBI) is also one of the main causes of death in the intensive care unit (ICU) and emergency department (ED).

Making decisions on the use of particular treatment methods, limiting nosocomial infections, providing patients and family with counseling, and determining the patient's individual rehabilitation needs all depend heavily on an accurate assessment of prognosis following traumatic brain injury.In large trauma centers, traumatic brain injury (TBI) is also one of the main causes of death in the intensive care unit (ICU) and emergency department (ED).

Making decisions on the use of particular treatment methods, limiting nosocomial infections, providing patients and family with counseling, and determining the patient's individual rehabilitation needs all depend heavily on an accurate assessment of prognosis following traumatic brain injury.

Since the majority of severe TBI patients are sedated, drugged, intubated, and unconscious, using clinical evaluation of theless accurate than using the Glasgow Coma Scale (GCS) to assess injury severity. As a result, the majority of the time, an emergent computed tomography (CT) scan is necessary as part of the ED's secondary survey in order to provide data on the patients' expected outcome.

Certain characteristics, including age, the presence of other injuries, a history of prior head injuries, alcohol and drug misuse, low socioeconomic and educational status, and previous head injuries, have been associated in prior research with increased mortality and worse outcomes following traumatic brain injury.According to the statistics available, the risk of traumatic brain injury (TBI) is highest between the ages of 15 and 24, declines in middle age, and then rises once more until age 70, when it mostlyas a result of falls. Males suffer traumatic brain injury (TBI) three to four times more frequently than females, while this ratio decreases with age. Motor vehicle collisions (MVCs) account for half of both fatal and non-fatal traumatic brain injuries (TBIs), with falls ranking as the second most common cause.

Less data is available from other societies, and these conclusions are frequently based on research done in wealthy nations.

The features of emerging and third-world cultures differ significantly from those of industrialized nations in areas like the kind and frequency of jobs, the healthcare system, the standard of cars,Large-scale research projects in these communities are therefore necessary. Our goal in doing this study was toassess the epidemiological features of TBI patients and look into the variables that influence the outcomes of TBI patients who are admitted to the emergency department.

The majority of those injured were middle-aged men. MVCs were the most common cause of TBI, occurring primarily in younger patients. Falls caused injuries, which were more noticeable in older patients, came in second. On CT scans, subarachnoid hemorrhage was the most commonly observed feature in patients. Our study patients' mortality rate was 11%. Compared to other patients, elderly patients experienced worse outcomes. As anticipated, there was a strong correlation between GCS at admission and death. The results of the patients with TBI were generally shown to be correlated with older age, lower GCS, the existence of abnormal CT scan findings, and hospital stays longer than five days, according to logistic regression analysis.

Previous research has shown that a patient's prognosis following a traumatic brain injury may be influenced by their demographics, the cause of injury, their GCS, and aberrant brain CT scan results. Men are more likely than women to suffer a traumatic brain injury (TBI), but gender does not statistically significantly affect the outcome. This has been linked to an increase in the number of male drivers and MVCs. With the exception of the damage mechanism, which did not demonstrate any correlation with the result, these are consistent with the results of the current investigation.

The overall mortality rate in this study is lower than in previous research, which typically ranges from 32-49%. The fact that the current investigation was carried out in a single center is one of the causes. More significantly, though, a register department made use of the recorded data. It is important to note that pre-hospital deaths and patients who passed away in the early hours of ED admission but were not yet transferred to inpatient departments are not included in this registry.

Teasdale and Jennett (1974) described the GCS score. Patients with traumatic brain injury had their level of unconsciousness evaluated. Research demonstrates that GCS is a highly reliable indicator of TBI outcome. But it can also be impacted by sedation, paralysis, or alcohol consumption, as well as by the presence of facial edema. As anticipated, the current study's findings also demonstrated that patients with lower GCSs at admission had a greater death rate than other TBI patients.

A brain CT scan is essential for the early evaluation of TBI patients. When intracranial pressure monitoring is not easily accessible, the results of a CT scan can be a valuable tool in predicting the prognosis of TBI patients in settings with limited resources. In the current investigation, cerebral bleeding was the most common imaging finding, whereas individuals with subdural hematoma findings had the highest death rate.

There are a number of factors to take into account while discussing the role and effects of hospital stay duration on the prognosis of traumatic brain injury patients. High-risk TBI patients are

anticipated to be admitted to the intensive care unit (ICU), and if they survive the critical phase, they will be moved to the ward units. Surgery may also be necessary for these patients, particularly if they were unconscious when they were brought to the emergency department (ED) or if their brain CT scan results were abnormal. However, extended hospital stays, particularly in intensive care units (ICUs), are linked to a number of complications, including the risk of hospital infections, pneumonia related to ventilator use in intubated patients, deep vein thrombosis, and numerous other problems that will lengthen the hospital stay for high-risk patients with traumatic brain injuries. Another crucial aspect is that high-risk TBI patients may not be able to be transferred rapidly to the ICU due to restrictions that are in place in some countries of the world, such as Iran. Instead, they will need to stay in the emergency department (ED) for a while, which could occasionally extend beyond a few days.This is the extent of care that is unavoidably possible, but we feel that the ED's level of care cannot be equated with the ICU's. Of course, this is just a hypothesis, and it would be worth doing research to demonstrate how waiting to move patients from the emergency room to the intensive care unit or other inpatient departments can impact the prognosis of patients with severe trauma.

In light of the findings of the current study as well as similar studies carried out in the same timeframe in other locations throughout the world, including developing nations, it could be necessary to create a specific evidence-based guideline or, at the very least, a national protocol to handle high risk TBI patients in ED.

As a conclusion, we advise conducting a thorough literature analysis to identify risk variables and then identify the most successful interventions that may be able to change the course of treatment for high-risk TBI patients. While raising people's awareness will undoubtedly help, creating and enforcing regulations would likely be very helpful for utilizing safety equipment and preventing injuries after MVCs.

AIM AND OBJECTIVES OF THE STUDY:

AIM OF THE STUDY:

To study the Epidemiological profile, Pattern and outcome of patients with Traumatic brain injury

OBJECTIVES OF THE STUDY:

TO STUDY:

1. To assess the Epidemiological profile of patients with Traumatic brain injury attending tertiary care hospital in North Karnataka.

2. To follow them up for six months, document the outcome and estimate the outcome predictors for the same.

******

REVIEW OF LITERATURE:

- Maas AI, in his study on Traumatic brain injury in India (2017)6, highlights the lack of data and lack of evidence in India on TBI, with data being published only 20,000 patients with TBI over 27 years. This accounts to only less than 1,000 TBI cases studied/year. This hardly would represent 0.1% of the population with TBI6.
- Agrawal A et al, in an overview of published literature from India on Pattern of reporting and practices for the management of TBI (2018)7, concludes that, An increasing incidence of

Traumatic Brain Injury in the productive age group is of major concern. Both the extremes of age group are more susceptible to severe injury and poor outcome. Quantitative analysis of injuries and outcomes of TBI patients shows a better impact on health in the economically productive population and in patients in the extreme age group.

- Reilly P, in his study on The role of methodological collection of epidemiological data in decreasing the burden of TBI in India(2017)8, has stressed on the development of high quality, standardized epidemiological data in India that would help reduce the growing economic and social burden due to TBI. There is necessity of designing Trauma systems which link rural and next level centers for trauma. These mid-systems should have guidelines to help the local doctors in immediate management and referral to the nearest Trauma Centre.
- Ţolescu RŞ et al, in a comparative study on Severe traumatic brain injury (TBI) at a Department of Forensic Medicine (2020)10, concluded that majority of TBI in adults was due to Falls (45%) followed by car accidents (31.61%), with most positive blood alcohol contents found in the adult age group; a high number of adults presented with 3rd and 4th degree of coma at admission(54.53%) and few deaths (5.41%) presented with GCS score of 15 on admission. 78% of patients with severe TBI presented with SDH; 50% of deaths due to TBI presented with brain lacerations.
- Bertozzi G et al, in a forensic literature review of Traumatic Brain Injury (2019)11, signifies an appropriate forensic approach as mandatory in the post mortem examination of a suspected TBI, and has stressed on proper dealing by means of forensic sciences in neuropathological studies, especially during Autopsy, radiological, and histological examination. It also considers the importance of genetic substrate study and the outcome of TBI; new Biomarkers and their use in clinical settings.
- Schwenkreis P et al, in a multi centric Prospective observational cohort study (2021) on management of traumatic brain injury at a group of Hospitals at Germany12 studied possible shift in TBI epidemiology and possible changes in TBI management and identified predictors of 1 year outcome especially in case of mild TBI; concluded that pronounced peak of TBI is around 20th year of age in males, severe TBI more commonly occurring in elderly patients. Non helmet wearing Bicyclists made up largest groups of RTA with TBI, CT scans were performed in 71% of patients, and 80% within 30 min of Severe TBI. GCS was being recorded in 94.5 % of cases at Emergency room. 12.4% of cases underwent immediate rehabilitation. 33% of patients participating in Telephonic interviews after 12 months still reported troubles related to TBI. The observed epidemiologic shift in TBI (ie, elderly patients, more falls and more bicyclists) calls for target based preventive measures. The heterogeneity behind the diagnosis 'mild TBI' stresses the need for defining different subgroups, not just on the basis of Glasgow Coma Scale.

- Gao G et al, in a prospective, multicenter, longitudinal, observational study on outcome of traumatic brain injury (2020)9, done in 56 centers across china, interpreted, differences in mortality between centers and regions across China. It also concluded that, there is a scope for recognizing best practices by comparative effectiveness research on TBI. The identified risk factors by prognostic analyses might help to develop steps to assess quality of care.
- Hagos A et al, in a Cross-sectional Hospital-based Study management of TBI at Addis Ababa, Ethiopia"(2022)13 concluded that The mortality rate of severe, moderate, & mild TBI were 25%, 8.0% & 2.0% respectively with an overall mortality of 5.6%. Road traffic injury was responsible for 45% of the cases, of which pedestrian struck accounts for 52.2% of the cases. Only 16.4% patients arrived below 02 hours.
- Prasad GL et al, in a tertiary care center study on Outcome of TBI in Elderly Population in India(2018)14, concludes that, poor prognostic factors in TBI includes Age more than 75 years, acute SDH and severe TBI. The benefit of surgery in these patients is not found significant, and surgery needs to be decided only after weighing with the economic and caregiving burden on the family.
- Munivenkatappa A et al, in a study of natural history of mild TBI by multidimensional approach (2017)15, showed the course of changes in cognition, symptoms and quality of life from the time of injury for the next 6-7 months.
- Wilson L et al, in a Manual for the Glasgow Outcome Scale Extended Interview 202116, have described GOSE as the most significant measure for the global outcome of TBI in research studies and clinical trials. GOSE-TBI scoring is based on 8 possible outcomes, which include Death, Vegetative state, Lower/Upper severe disability, Lower/Upper moderate disability and Lower/Upper good recovery.

MATERIALS & METHODS:

SOURCE OF DATA:

Data has been collected from the patients and their attenders attending BLDE(DU) SHRI BM Patil Medical College Hospital and Research Centre with a history of trauma, that are medicolegally registered, with or without Loss of consciousness and/or CT scan findings of Traumatic brain Injury.

TYPE OF STUDY: A Hospital-based prospective descriptive study

PERIOD OF STUDY: October 2022 to March 2024

SAMPLING:

TOTAL SAMPLE SIZE: 164

According to a meta-analysis by Brock frost R et al., the Prevalence of Traumatic Brain injuries is found to be 12.1% among the adult population. The sample size is calculated by the Statistical formula of Fisher for observational studies

$n= z2pq/E2$

Where $P=0.121$

$Z= 1.96$ taking 95% confidence limits

$d= 0.05$, taking 5% as absolute error

The calculated minimum sample size is 164.

INCLUSION CRITERIA

1. Age >18 years

2. Cases that are medicolegally registered, with H/O Trauma with Loss of Consciousness and/or CT findings of Traumatic Brain injury of any degree.

EXCLUSION CRITERIA

1. Brain haemorrhage due to any natural diseases like Hypertension; Stroke
2. Those who are not willing to participate.

METHOD OF COLLECTION OF DATA:

Data has been collected from the patients attending BLDE(DU) Shri BM Patil Medical College Hospital and Research Centre with a history of trauma, that are medicolegally registered, with or without Loss of consciousness and/or CT scan findings of Traumatic Brain Injury. The data collection has been done in six different Google formats, namely

Form no 1- Initial assessment and care

Form no 2- Hospital care

Form no 3- Autopsy findings, in case of Death (New cases)

Form no 4- Autopsy findings, in case of Death (Treated cases)

Form no 5- Follow up - End of 3rd Month

Form no 6- Follow up - End of 6th Month

STATISTICAL ANALYSIS AND OUTCOME:

1. Characteristics of participants are expressed descriptively, with Mean and SD for continuous variables and counts and proportions for categorical variables. The variables measured are the proportion of TBI with different Ages, Sex, Type of injury, Causes of Injury, and Proportion of Patients presenting with different degrees of severity of the disease.

2. Categorical variables like type of injury, the pattern of TBI, and grade of TBI are presented in proportions.

3. Association of Age, gender, and cause with the severity of TBI is measured using the Log-linear model.

4. Estimation of Outcome predictors, like the severity of disease, amount of time delay for the treatment, condition at presentation in terms of SpO2, Systolic BP, Pupillary reaction, GCS score and Injury severity score to be done by a Logistic regression model.

5. Statistical analysis of the collected Data has been done using JNP-SAS Software.

PROFILE AND OUTCOME OF TRAUMATIC BRAIN INJURY PATIENTS IN A TERTIARY CARE CENTRE IN NORTH KARNATAKA: A PROSPECTIVE HOSPITAL-BASED STUDY

Form No 1- Initial assessment and care

Registration details

1. Sl no;

2. Patient Hospital ID No;

3. Age

4. Sex

5. Date

6. Phone number

7. Address

History

1. Type of trauma

a. Domestic fall- Ground level fall/ Fall from Height

Details…….

b. Assault

c. Self-harm

d. Gun shot

e. Road traffic Accident

2. Place of trauma

3. Time and Date of Injury

Time and Date of Presentation

4. Referral type

a. Direct without referral

b. With referral

c. Details of treatment outside …….

5. H/o Loss of consciousness Yes/No

H/o Seizures Yes/No

H/o Vomiting Yes/No

H/o Amnesia Yes/No

6. H/o DM II/ Hypertension/ Asthma/ COPD/ Neurological deficit

7. Medico Legal Case recorded Yes/ No

Clinical Examination

1. Blood Pressure

2. Heart rate

3. SpO2

4. GCS at presentation, GCS Score

5. Pupils

6. Intubation

7. Focal Neurological sign

Investigations at ER

1. Any other organs, parts of body involved

2. CT findings

a. Skull Fractures; type and

Sites……

…….

b. Traumatic Subarachnoid Hemorrhage; Site

c. Epidural Hematoma; Site…… size……

d. Subdural Hematoma; Site…… size……

e. Brain Contusion; Site

f. Crush injury; Yes/ No

g. Diffuse Axonal Injury: Yes/ No

Treatment advised;

1. OPD Basis

2. Admission for observation

3. Admission for Medical Management

4. Admission for Surgical management

Patient choice

1. Agrees to the advice

2. Patient goes AMA

SIGNATURE: **DATE:**

Form No 2- Hospital Care

1. Sl. No

2. Patient Hospital ID No;

3. Date of Admission

4. Date of Discharge

5. Discharge status; Improved/ Referred/ Against medical advice/ Death

6. Duration of stay

7. Mode of treatment; Conservative/ Neurosurgical / other surgical/ Observation

In case of Surgery, Details of Surgery……

Date and Time of Neurosurgery

8. Other system involvement;

a. Details

b. Management details

9. Complications during hospital care

10. Glasgow Outcome Scale- Extended score

SIGNATURE:

DATE:

Form No 3.1 Autopsy, in case of Death (New)

1. Sl. No

2. PM no

3. Name

4. Age

5. Sex

6. Police station

7. UD/Cr No

8. U/S

9. Date

10. Address

History

1. Type of trauma

a. Domestic fall- Ground level fall/ Fall from Height

Details…….

b. Assault

c. Self-harm

d. Gun shot

e. RTA

8. Place of trauma

9. Time and Date of Injury

2. Other history furnished by the Police

3. Treatment details, if any;

Autopsy findings

1. External findings

2. Internal findings; Head, Brain

a. Skull Fractures; type and

Sites……

…….

b. Traumatic Subarachnoid Hemorrhage; Site

c. Epidural Hematoma; Site…… size……

d. Subdural Hematoma; Site…… size……

e. Brain Contusion; Site

f. Type of head injury; Open/ Close

g. Crush injury; Yes/ No

3. Details of other sites of injury, if involved

Conclusion

1. Time since death;

2. Cause of Death;

Signature: Date:

Form No 3.2. Autopsy, in case of Death (Treated cases)

1. Hospital ID no

2. PM no

3. Name

4. Age

5. Sex

6. Police station

7. UD/Cr No

8. U/S

9. Date

10. Address

Autopsy findings

1. External findings

2. Internal findings; Head, Brain

a. Skull Fractures; type and

Sites……

…….

b. Traumatic Subarachnoid Hemorrhage; Site

c. Epidural Hematoma; Site…… size……

d. Subdural Hematoma; Site…… size……

e. Brain Contusion; Site

f. Type of head injury; Open/ Close

g. Crush injury; Yes/ No

3. Details of other sites of injury, if involved

Conclusion

1. Time since death;

2. Cause of Death;

Signature: Date:

Form No 4. Follow up- End of 3 rd Month

(OPD basis/ Telephonic/ Video call basis)

1. Sl. no

2. Patient Hospital ID no;

3. Subjective symptoms;

4. Current therapy status;

5. Rehabilitation done; Yes/ No

If yes, Home/ Rehabilitation center

Details…….

6. Glasgow Outcome Scale- Extended score;

Signature: Date:

Form No 5. Follow up- End of 6th Month

(OPD basis/ Telephonic/ Video call basis)

1. 1. Sl. no

2. Patient Hospital ID no;

3. Subjective symptoms;

4. Current therapy status;

5. Rehabilitation done; Yes/ No

If yes, Home/ Rehabilitation center

Details…….

6. Glasgow Outcome Scale- Extended score;

Signature:

Date:

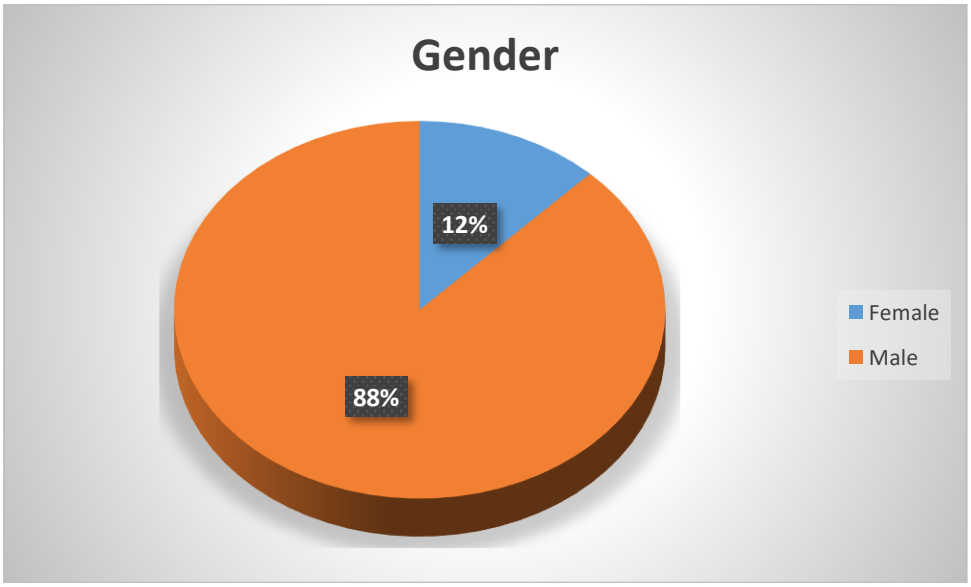
******

OBSERVATIONS & RESULTS:

Table 1- Gender disparity among total TBI cases

Gender	No. of patients	Percentage
Female	21	12.2
Male	151	87.8
Total	172	100.0

Chart 1- Gender disparity among total TBI cases

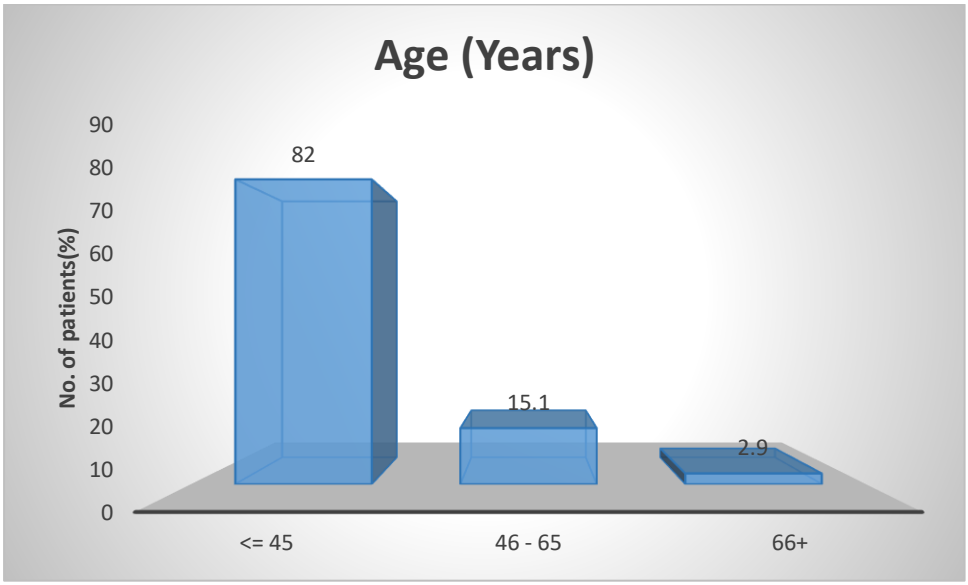


There is male preponderance among the total TBI cases, 87.8% of the total 172 cases were males and 12.2% were females

Table 2- Age distribution among Total TBI Cases

Age (Years)	No. of patients	Percentage
18- 45	141	82.0
46 – 65	26	15.1
66+	5	2.9
Total	172	100.0

Chart 2- Age distribution among Total TBI Cases

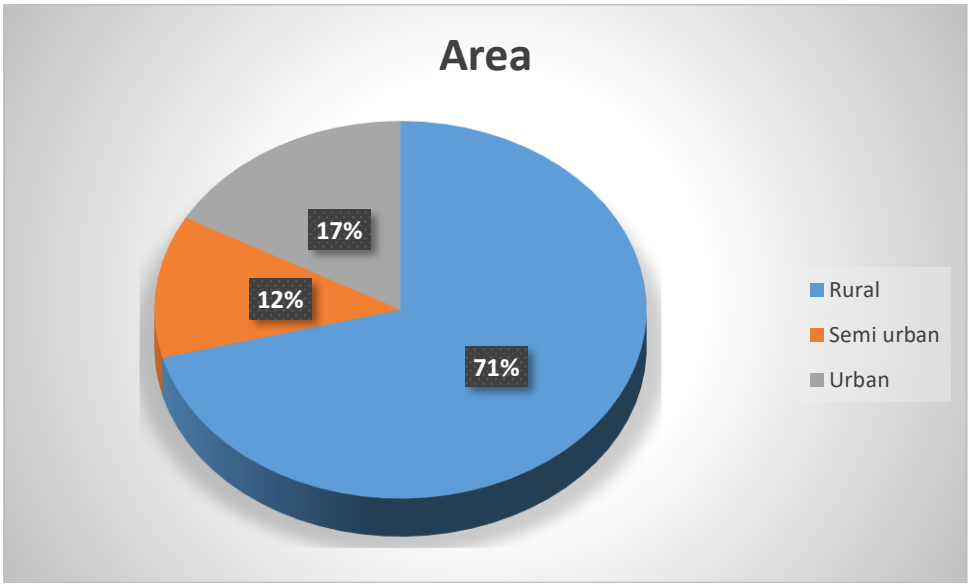


In the present study, Maximum number of TBI cases belong to the in the early adulthood, i.e. age group of 18-45 years (82%). 15.1% of cases belong to the middle adulthood, i.e. age group of 46-65 years and only 2.9% of cases belong to the older age group i.e. more than 65 years.

Table 3- Area distribution- Total TBI cases

Area	No. of patients	Percentage
Rural	122	70.9
Semi urban	21	12.2
Urban	29	16.9
Total	172	100.0

Chart 3- Area distribution- Total TBI cases

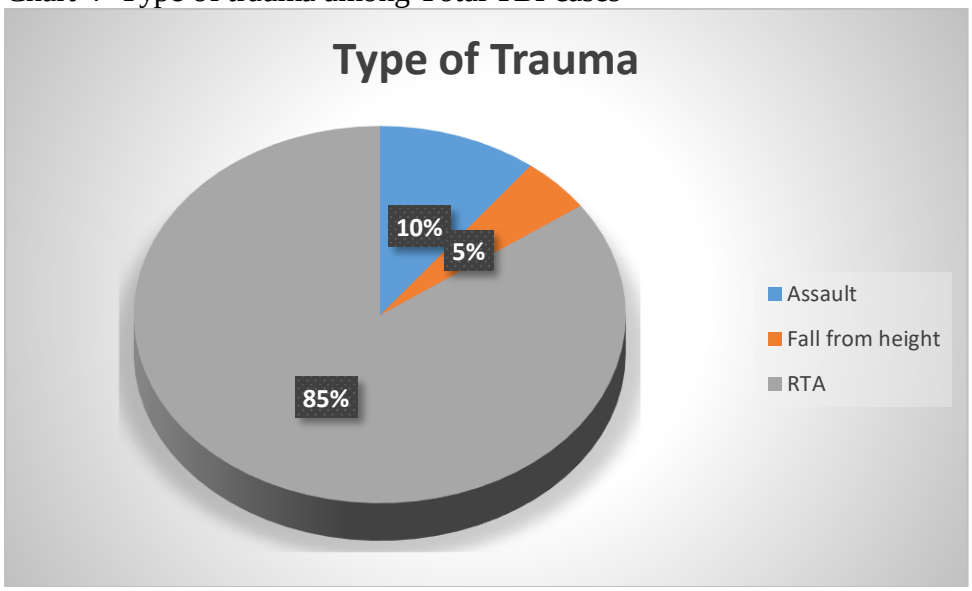


Among the total TBI cases that presented to our tertiary care center, 71% were from rural, 12% were from semi urban area, and 17% were from urban areas.

Table 4- Type of trauma among Total TBI cases

Type of Trauma	No. of patients	Percentage
Assault	18	10.5
Fall from height	8	4.7
RTA	146	84.9
Total	172	100.0

Chart 4- Type of trauma among Total TBI cases



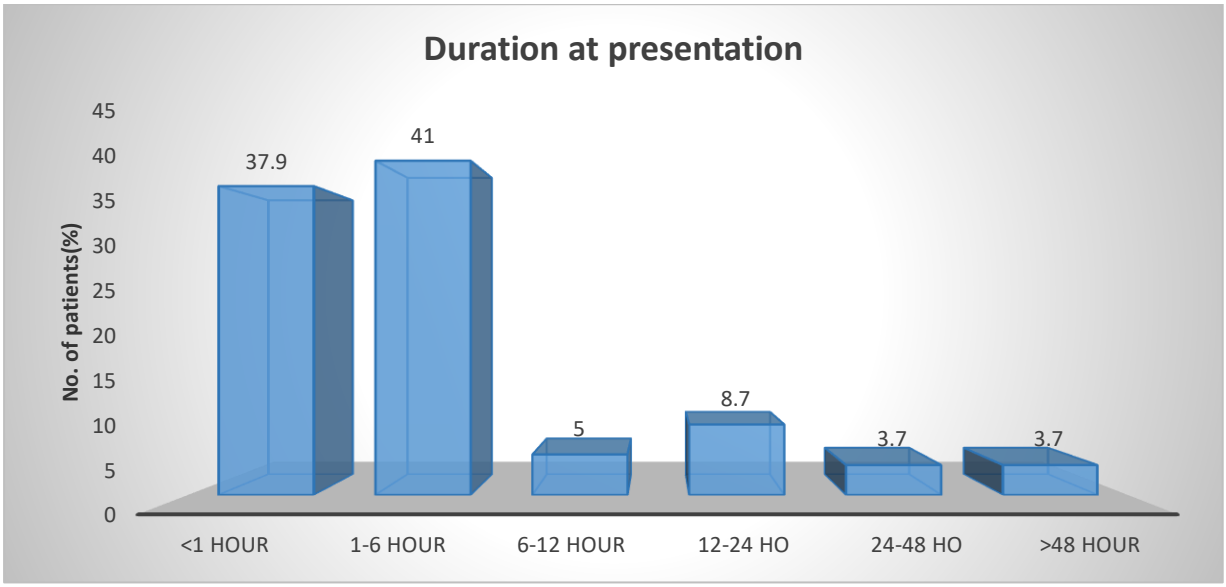
Among the Total TBI Cases attending Our tertiary care centre, 85% were due to Road traffic accident, 10% were due to fall from height, 5% due to Assault

Table 5- Duration of presentation from the time since injury

Duration at presentation from the time since injury	No. of patients	Percentage
<1 hour	61	37.9
1-6 hour	66	41.0
6-12 hour	8	5.0
12-24 ho	14	8.7
24-48 ho	6	3.7

>48 hour	6	3.7
Total	161	100.0

Chart 5- Duration of presentation from the time since injury

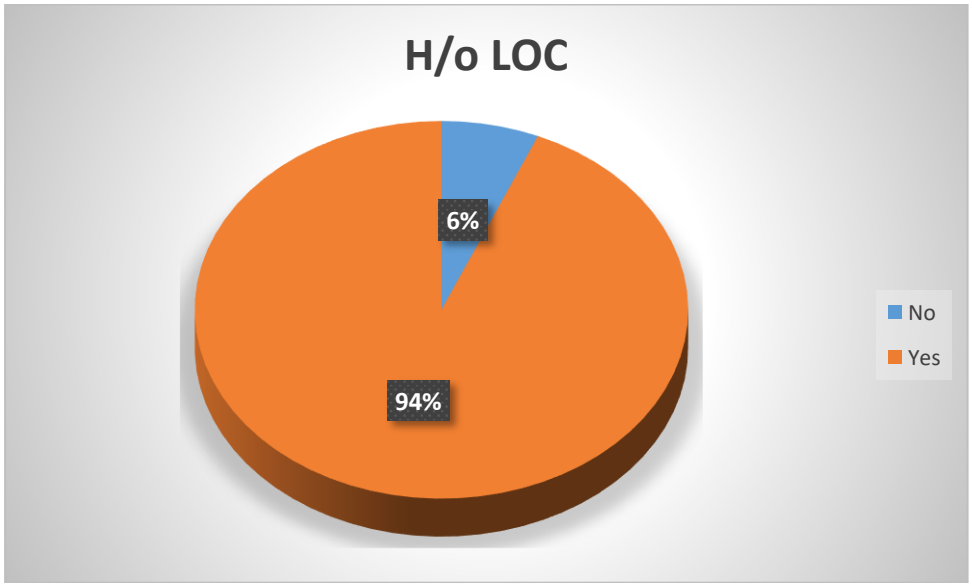


Among 172 total TBI cases, 161 cases presented to the casualty in the living condition. Among the 161 cases, 37.9% cases presented within one hour of the TBI event, 41% within 1 to 6 hours, 5% within 6 to 12 hours, 8.7% since 12-24 hours, 3.7% presented within 24-48 hours, 3.7% after 2 days. This concludes that 4 out 5 patients presented within 6 hours of Head injury.

Table 6- History of LOC among total TBI cases

H/o LOC	No. of patients	Percentage
No	11	6.4
Yes	161	93.6
Total	172	100.0

Chart 6- History of LOC among total TBI cases

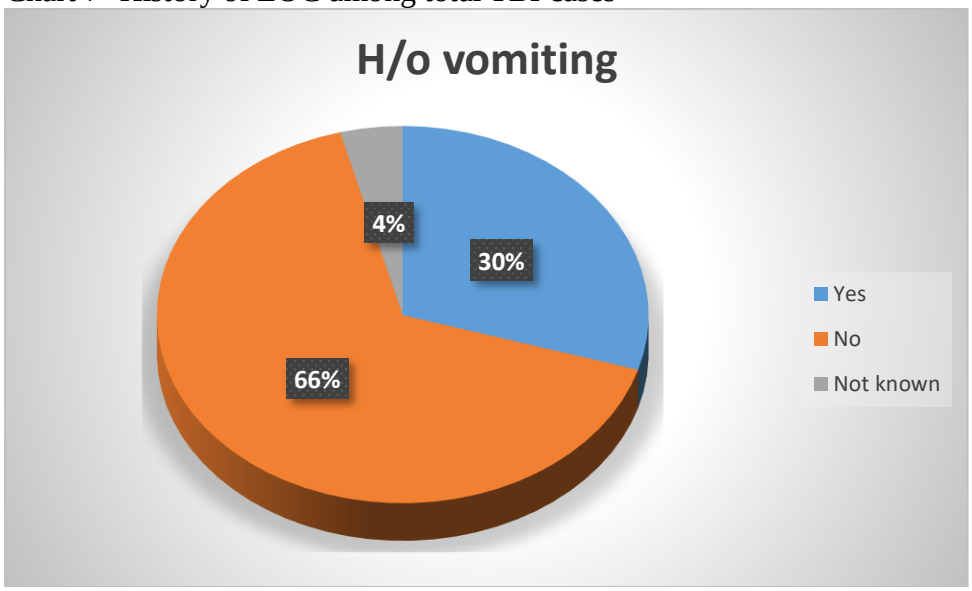


History of Loss of consciousness after the trauma was present in 94% of total TBI cases. The duration of loss of consciousness was very much varied from few minutes to few hours.

Table 7- History of Vomiting among total TBI cases

H/o vomiting	No. of patients	Percentage
Yes	51	29.7
No	114	66.3
Not known	7	4.1
Total	172	100.0

Chart 7- History of LOC among total TBI cases

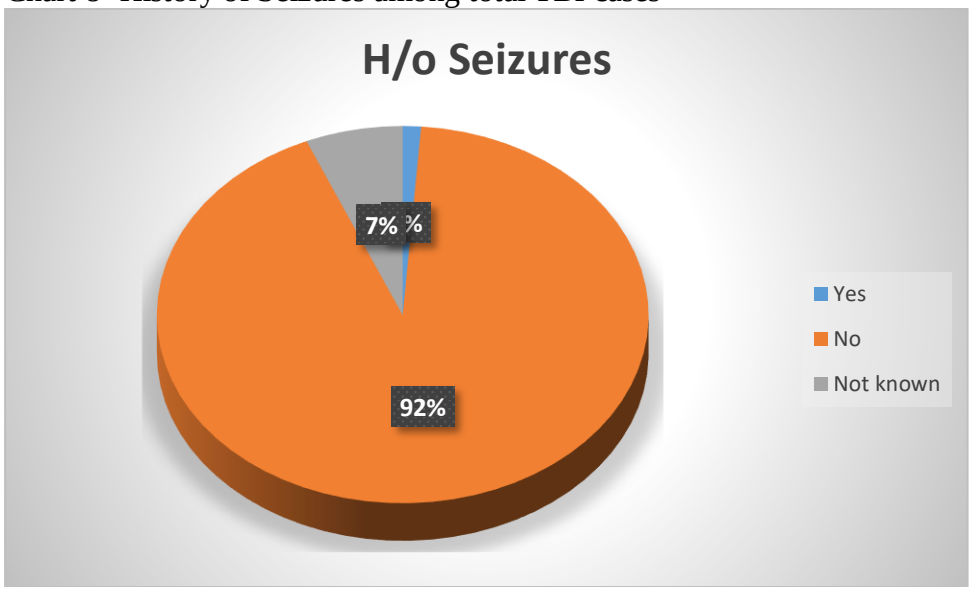


History of Vomiting was present in 66% of total TBI cases, and absent in 30% of cases. it was not possible to elicit the history of vomiting among 4% of cases.

Table 8- History of Seizures among total TBI cases

H/o Seizures	No. of patients	Percentage
Yes	2	1.2
No	159	92.4
Not known	11	6.4
Total	172	100.0

Chart 8- History of Seizures among total TBI cases

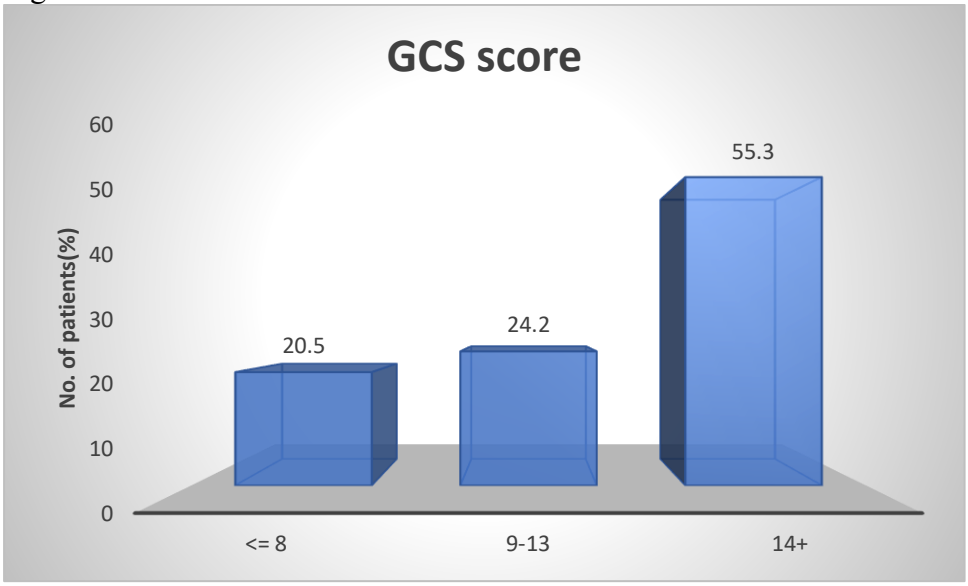


History of Vomiting was present in only 1% of total TBI cases, and was not present in 92% of cases. It was not possible to elicit the history of seizures among 7% of cases.

Table 9- GCS score of total live TBI cases

GCS score	No. of patients	Percentage
<= 8 (Severe TBI)	33	20.5
9 – 13 (Moderate TBI)	39	24.2
14-15 (Mild TBI)	89	55.3
Total	161	100.0

Figure 9- GCS score of total live TBI cases

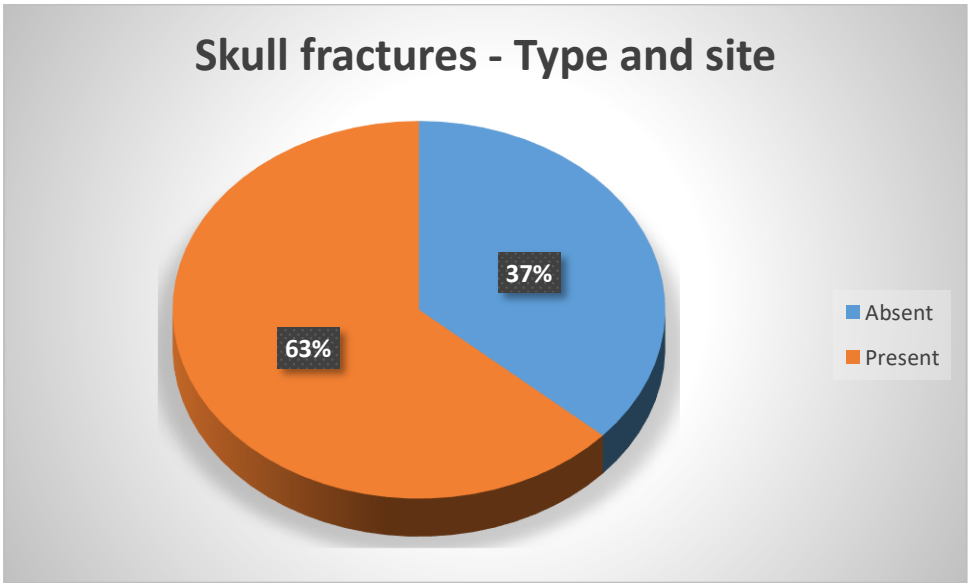


Among all the total 161 TBI cases that were brought live to the casualty, 55.3% of cases were having GCS score between 14-15 (Mild TBI), 24.3% of cases presented with a GCS score between 9-13 (Moderate TBI) and 20.5% of patients presented with a GCS score less than 8 (Severe TBI)

Table 10- Skull fractures among total TBI cases

Skull fractures - Type and site	No. of patients	Percentage
Absent	63	36.6
Present	109	63.4
Total	172	100.0

Chart 10- Skull fractures among total TBI cases

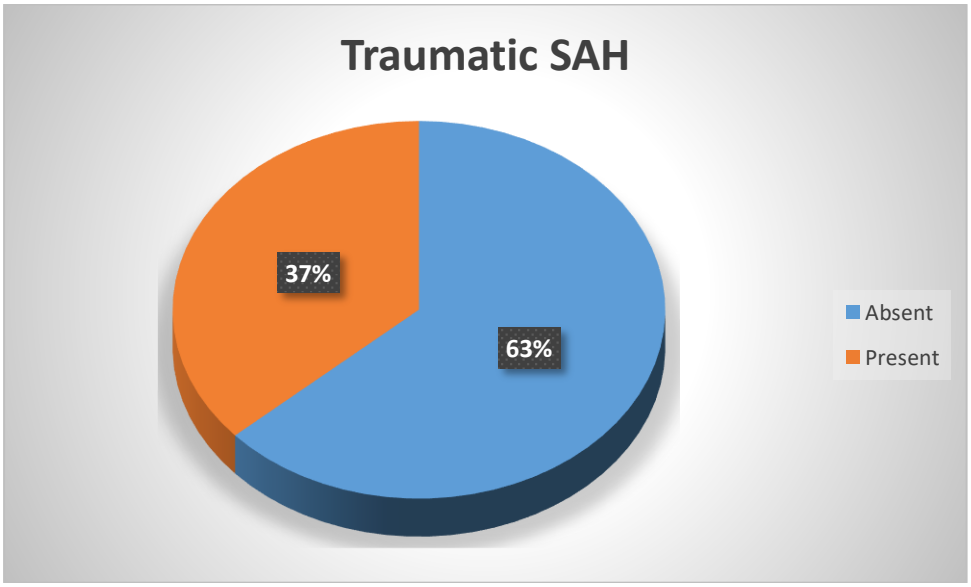


Skull fractures were present in 63.4% of total TBI cases, and absent in 36.6% of cases.

Table 11- Traumatic Subarachnoid hemorrhage among total TBI cases

Traumatic SAH	No. of patients	Percentage
Absent	109	63.4
Present	63	36.6
Total	172	100.0

Chart 11- Traumatic Subarachnoid hemorrhage among total TBI cases

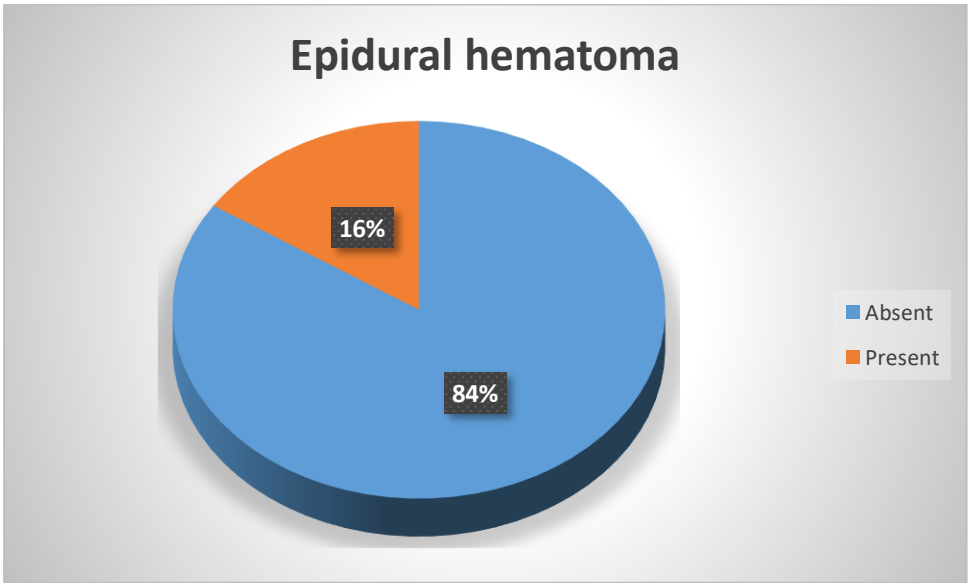


Subarachnoid hemorrhage was present in 36.6% of total TBI cases, and absent in 63.4% of cases.

Table 12- Epidural Hematoma among total TBI cases

Epidural hematoma	No. of patients	Percentage
Absent	145	84.3
Present	27	15.7
Total	172	100.0

Chart 12- Epidural Hematoma among total TBI cases

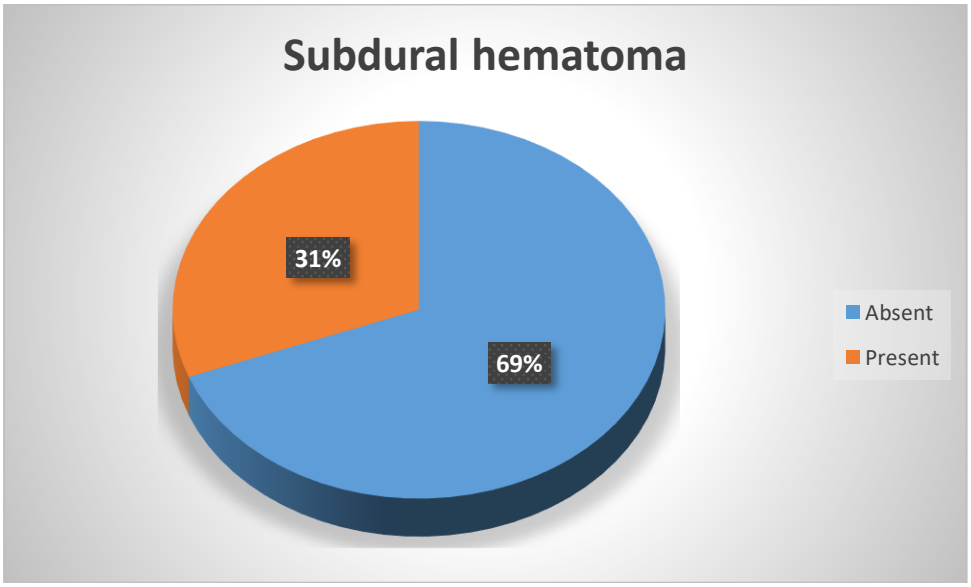


Epidural hemorrhage was present in 15.7% of total TBI cases, and absent in 84.3% of cases.

Table 13- Subdural Hematoma among total TBI cases

Subdural hematoma	No. of patients	Percentage
Absent	119	69.2
Present	53	30.8
Total	172	100.0

Chart 13- Subdural Hematoma among total TBI cases

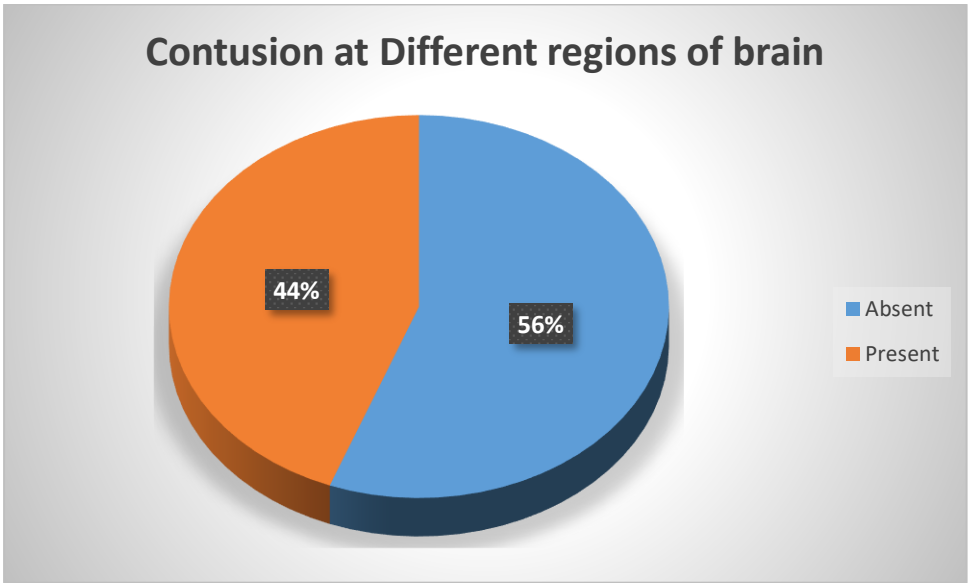


Subdural hematoma was present in 30.8% of total TBI cases, and absent in 69.2% of cases.

Table 14- Contusions in the Brain parenchyma among total TBI cases

Contusion at Different regions of brain	No. of patients	Percentage
Absent	96	55.8
Present	76	44.2
Total	172	100.0

Chart 14- Contusions in the Brain parenchyma among total TBI cases

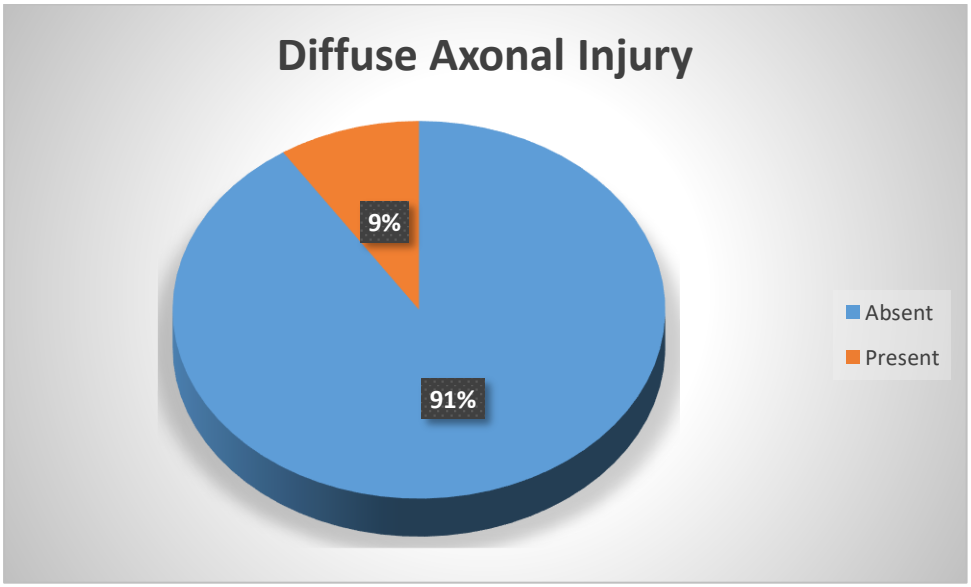


44.2% of total TBI cases suffered with contusions in various parts of brain parenchyma. The contusions varied from pin point hemorrhages to subcentimetric hematomas to massive hemorrhages at different parts of brain parenchyma.

Table 15- Diffuse axonal injury among total TBI cases

Diffuse Axonal Injury	No. of patients	Percentage
Absent	146	90.7
Present	15	9.3
Total	161	100.0

Chart 15- Diffuse axonal injury among total TBI cases

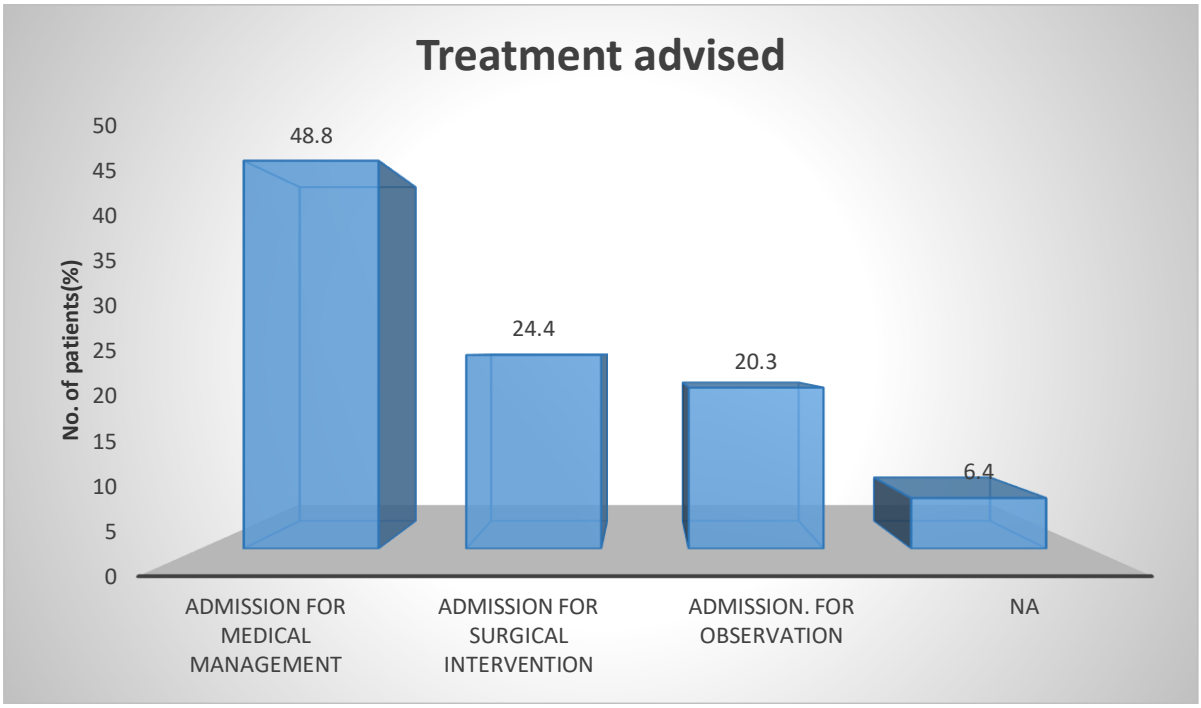


Among total 172 cases, Diffuse axonal injury couldn't be elicited in Autopsy of the brought dead cases. Among the total 161 live cases, 9.3% of cases were diagnosed with diffuse axonal injury.

Table 16- Treatment advised among total TBI cases

Treatment advised	No. of patients	Percentage
Admission for medical management	84	48.8
Admission for surgical intervention	42	24.4
Admission. for observation	35	20.3
NA	11	6.4
Total	172	100.0

Chart 16- Treatment advised among total TBI cases

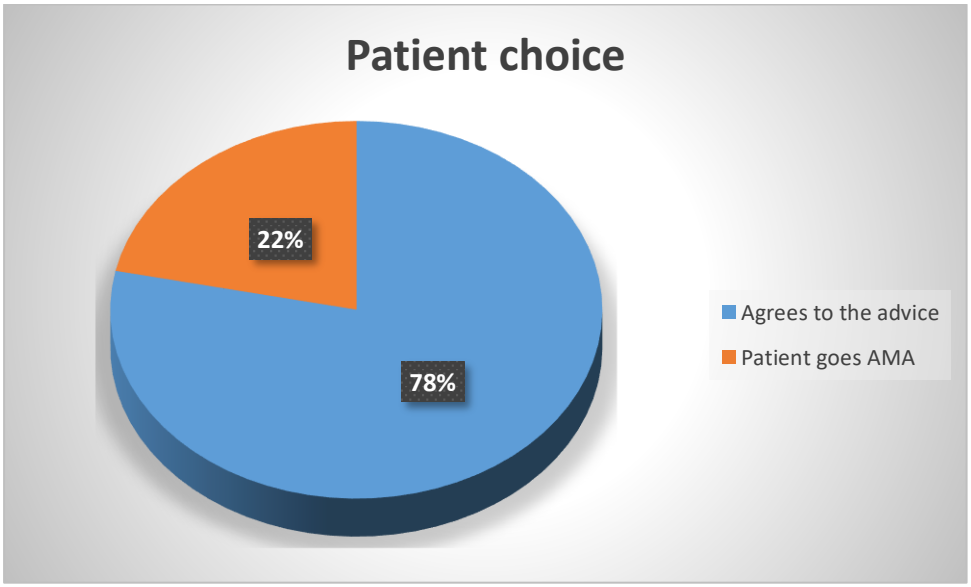


Among total 172 cases that presented to the casualty of the tertiary care centre, 48.8% of patients were adviced admission for medical management, 24.4% for surgical intervention and 20.3% for only a 24 hours observation for any untoward event following a TBI. Those who were adviced admission for medical management, included all such cases which could be managed conservatively, and those severe cases, which were not operable immediately after admission. 6.4% among the total cases were brought dead and could not receive any treatment.

Table 17- Patient choice for treatment among total live TBI cases

Patient choice	No. of patients	Percentage
Agrees to the advice	126	78.3
Patient goes AMA	35	21.7
Total	161	100.0

Chart 17- Patient choice for treatment among total live TBI cases

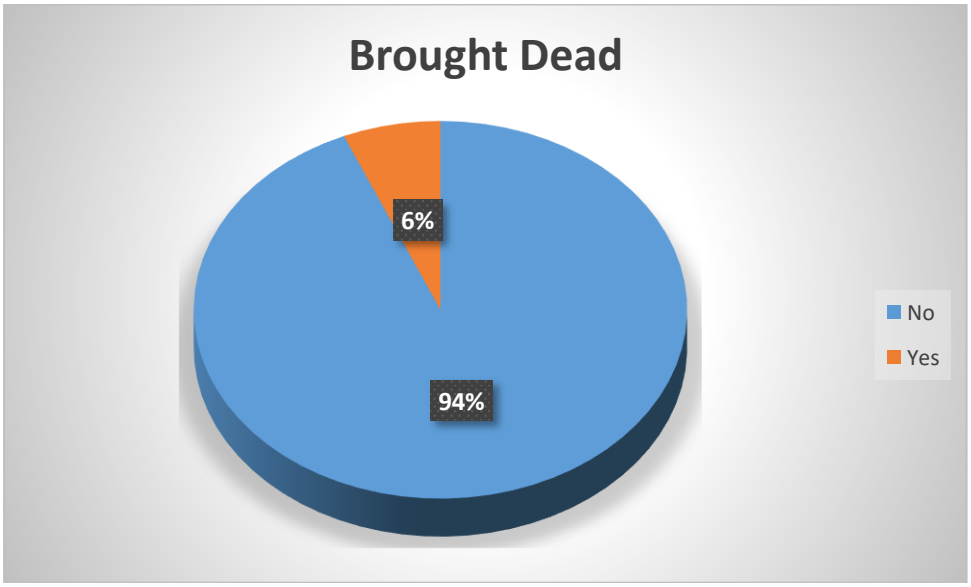


Among total 161 cases opf TBI, which presented to casualty in living condition, 78% of patients agreed to the doctors' advice and 22% went against medical advice.

Table 18- Brought dead cases among total TBI cases

Brought Dead	No. of patients	Percentage
No	161	93.6
Yes	11	6.4
Total	172	100.0

Chart 18- Brought dead cases among total TBI cases

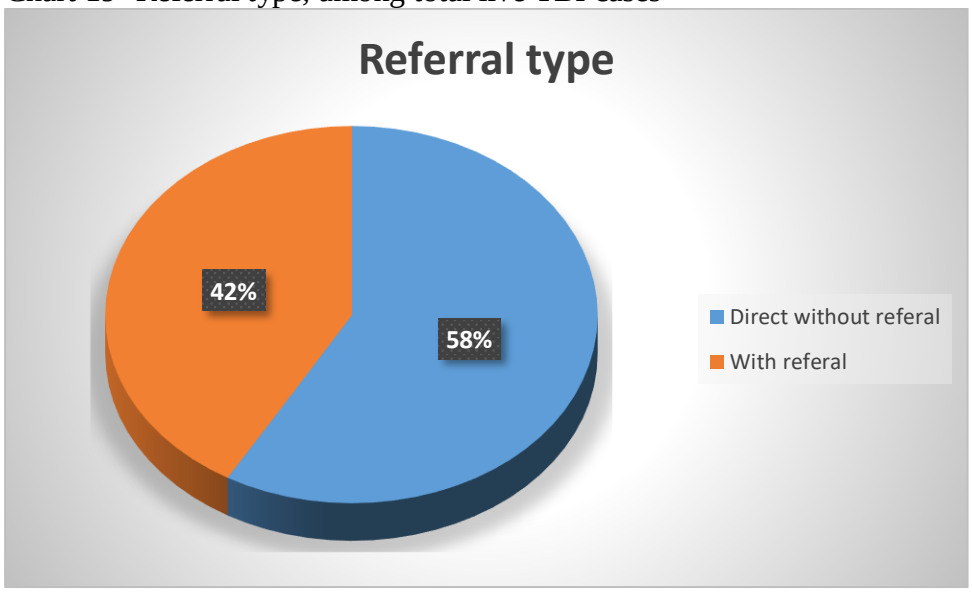


Among 172 total cases, 6.4% cases were brought dead, and 93.6% cases were in living condition.

Table 19- Referral type, among total live TBI cases

Referral type	No. of patients	Percentage
Direct without referral	94	58.4
With referral	67	41.6
Total	161	100.0

Chart 19- Referral type, among total live TBI cases

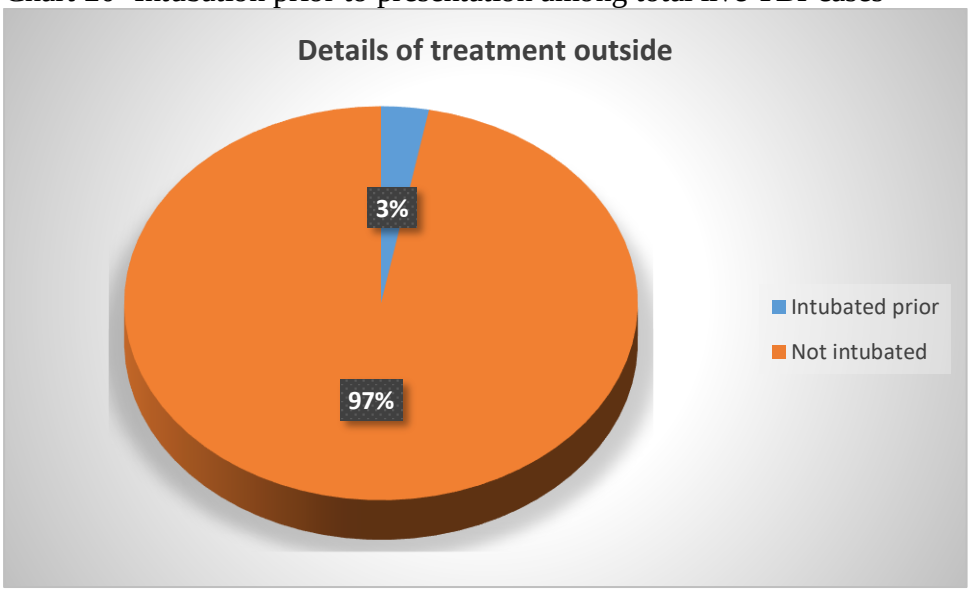


Among the 161 total live cases that presented at casualty, 58.4% of cases attended the casualty directly, without referral. 41.6% of total live cases were referred from various other health centres.

Table 20- Intubation prior to presentation among total live TBI cases

Prior intubation	No. of patients	Percentage
Intubated prior	5	3.1
Not intubated	156	96.9
Total	161	100.0

Chart 20- Intubation prior to presentation among total live TBI cases



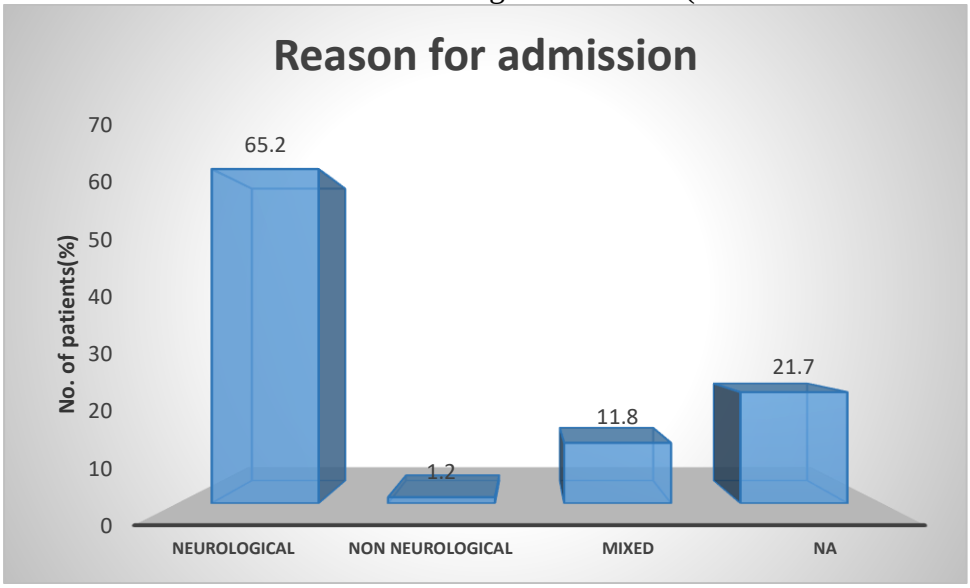
Statistical Analysis of Treated cases

Table 21- Reason for admission among treated cases (includes OPD and IPD cases both)

Reason for admission	No. of patients	Percentage
Neurological	105	65.2
Non neurological	2	1.2
Mixed	19	11.8

NA	35	21.7
Total	161	100.0

Chart 21- Reason for admission among treated cases (includes OPD and IPD cases both)

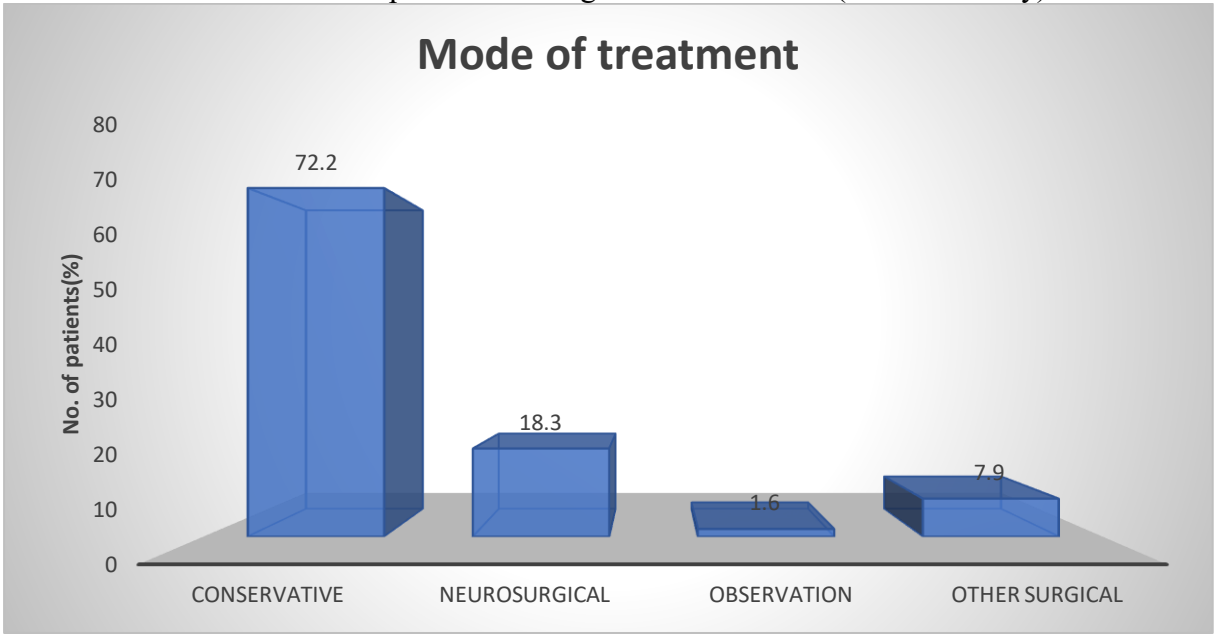


Among all the total 161 cases, which were treated at casualty, 65.2% were admitted for neurological reasons, only 1.2% of cases were admitted for non-neurological reasons and 11.8% of treated cases were admitted for both neurological and non-neurological reasons. Rest 21.7% of cases were treated at casualty, but denied admission and went against medical advice.

Table 22- Mode of Treatment provided among treated TBI cases (IPD cases only)

Mode of treatment	No. of patients	Percentage
Conservative	91	72.2
Neurosurgical	23	18.3
Observation	2	1.6
Other surgical	10	7.9
Total	126	100.0

Chart 22- Mode of Treatment provided among treated TBI cases (IPD cases only)

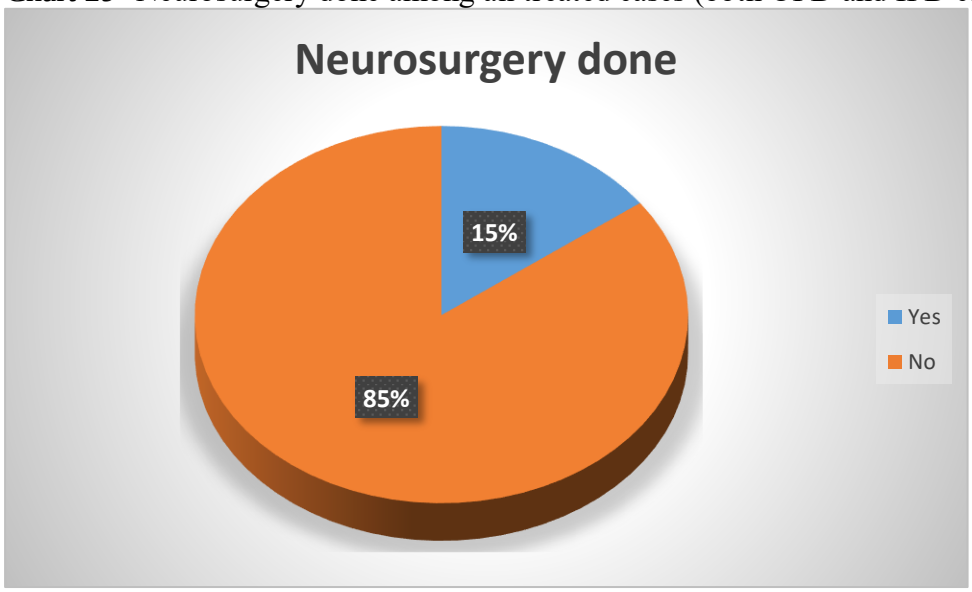


Among all the 126 IPD cases, 72.2% of patients were managed conservatively, 18.3% of cases underwent Neurosurgery, 7.9% of cases underwent other surgical procedures, and only 1.6% of patients were admitted for observation.

Table 23- Neurosurgery done among all treated cases (both OPD and IPD cases)

Neurosurgery done	No. of patients	Percentage
Yes	24	14.9
No	137	85.1
Total	161	100.0

Chart 23- Neurosurgery done among all treated cases (both OPD and IPD cases)

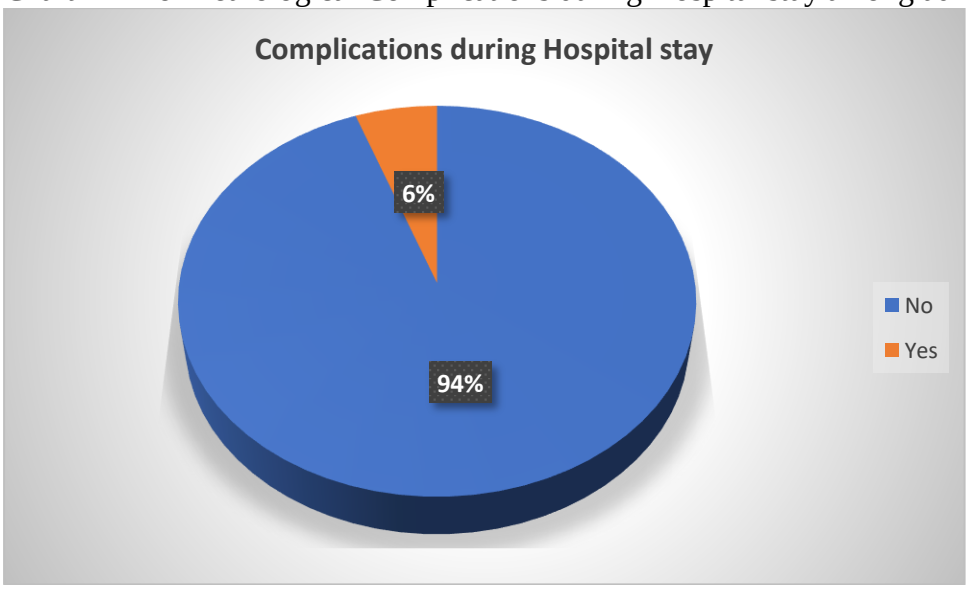


Of all the treated cases (both OPD and IPD cases), brought to casualty, 14.9% of cases underwent Neurosurgery and 85.1% of cases, no neurosurgery was being done.

Table 24- Non neurological Complications during Hospital stay among admitted cases

Non neurological Complications during Hospital stay	No. of patients	Percentage
No	119	94.4
Yes	7	5.6
Total	126	100.0

Chart 24- Non neurological Complications during Hospital stay among admitted cases



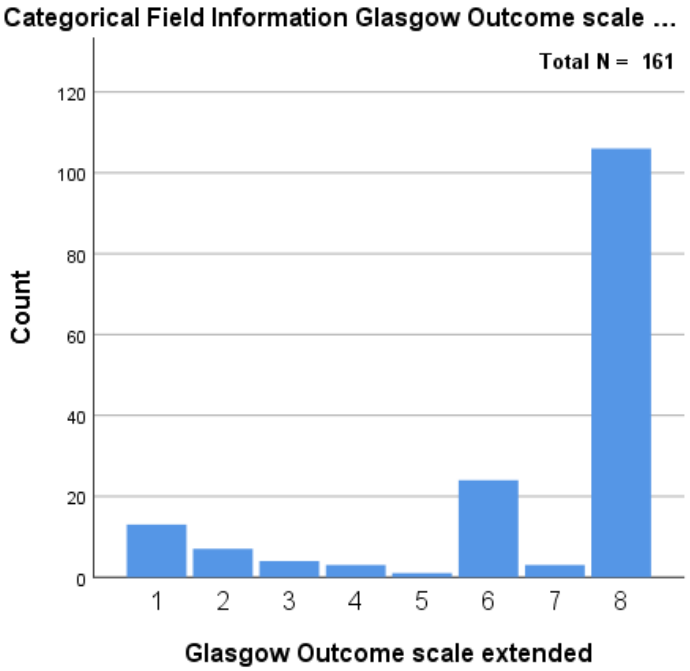
Among 126 admitted cases, 5.6% of cases suffered non neurological complications during the hospital stay

Table 25- Glasgow outcome scale-Extended, among the treated cases

Glasgow Outcome scale extended	No. of patients	Percentage
1	13	8.1
2	7	4.3
3	4	2.5
4	3	1.9

5	1	.6
6	24	14.9
7	3	1.9
8	106	65.8
Total	161	100.0

Chart 25- Glasgow outcome scale-Extended, among the treated cases



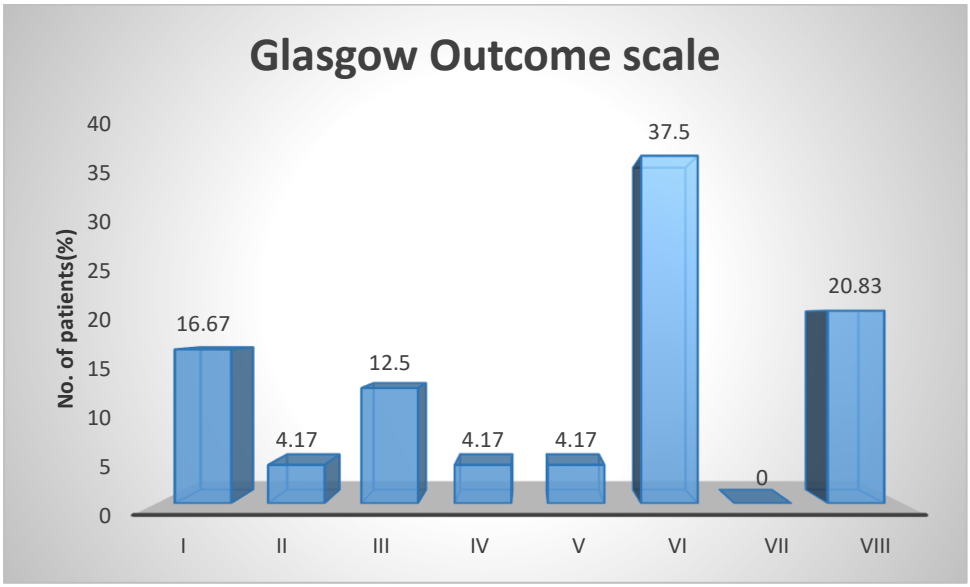
Among all the 161 treated cases, both IPD and OPD basis, at their discharge, 8.1% had a GOS-E score of 1, i.e. Death. 4.3% of cases were discharged in vegetative state (GOS-E score 2), 2.5% of cases were in lower severe disability (GOS-E score 3), 1.9% in upper sever disability (GOS-E score 4), 0.6% in lower Moderate disability(GOS-E score 5), 14.9% in upper Moderate disability (GOS-E score 6) and 1.9% of cases in Lower Good recovery(GOS-E score 7). Most of the cases, i.e. 85.8% of cases were having upper Good recovery, with a GOS-E score of 8.

Table 26- Glasgow outcome scale-Extended, among those who underwent Neurosurgery

Glasgow Outcome scale (Baseline)	No. of patients	Percentage
1	4	16.67
2	1	4.17
3	3	12.50
4	1	4.17
5	1	4.17

6	9	37.50
7	0	0.00
8	5	20.83
Total	24	100.00

Chart 26- Glasgow outcome scale-Extended, among those who underwent Neurosurgery

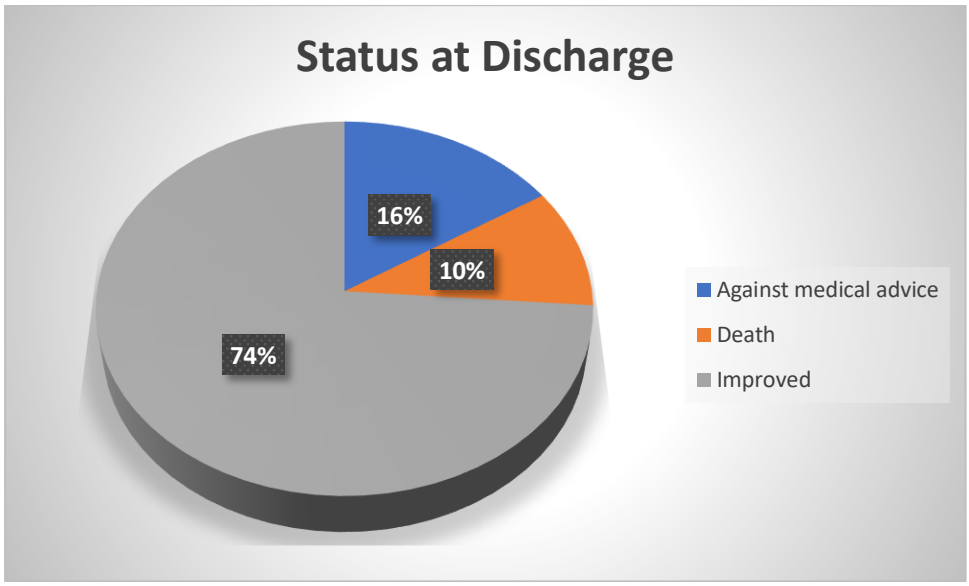


Among all the 24 cases, who underwent Neurosurgery, treated cases, both IPD and OPD basis, 4 cases succumbed to death (GOS-E score 1), 1 case was discharged in vegetative state (GOS-E score 2), 3 cases were in lower severe disability (GOS-E score 3), 1 case in upper sever disability (GOS-E score 4), 1 cases in lower Moderate disability(GOS-E score 5), 9 cases in upper Moderate disability (GOS-E score 6), no cases in Lower Good recovery(GOS-E score 7), and 5 cases were having upper Good recovery, with a GOS-E score of 8.

Table 27- Status at discharge among all admitted cases

Status at Discharge	No. of patients	Percentage
Against medical advice	20	15.9
Death	13	10.3
Improved	93	73.8
	126	100.0

Chart 27- Status at discharge among all admitted cases



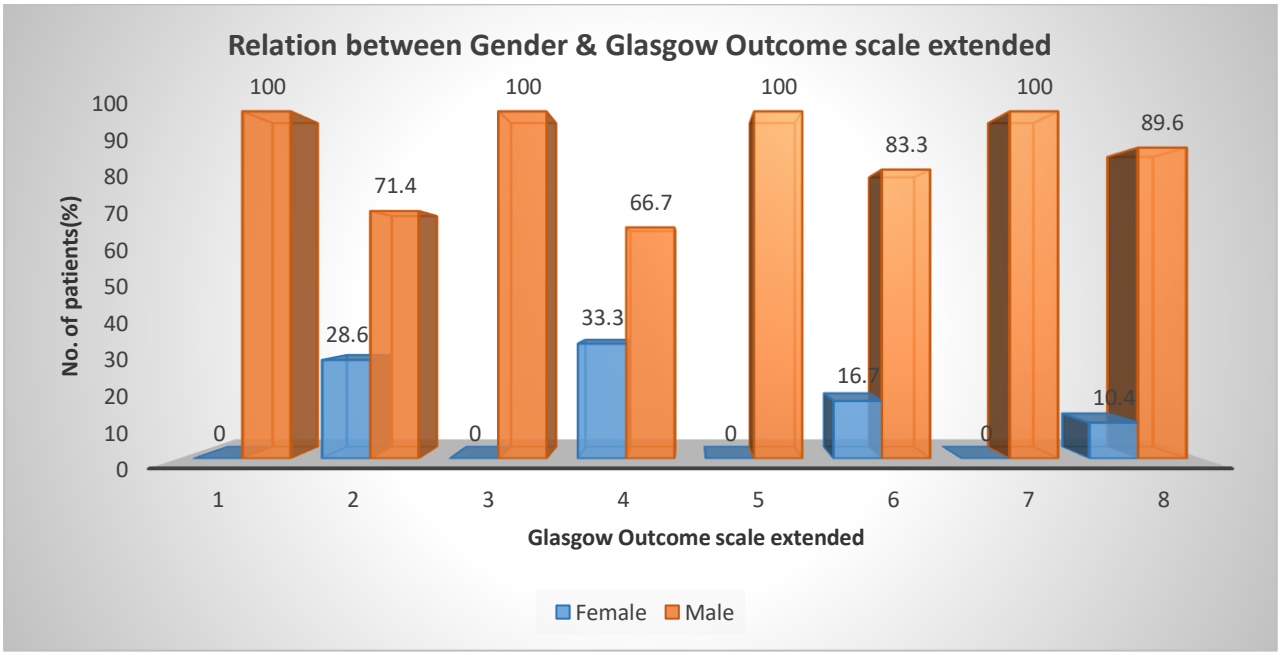
Among 126 admitted cases, 73.8% of cases were in an improved condition at discharge, 15.9% of cases went against medical advice and 10.3% of cases succumbed to death.

Table 28- Association between Gender and Glasgow outcome scale

Gender	Glasgow Outcome scale extended									Chi square test	Significant value
	1	2	3	4	5	6	7	8	Total		
Female	0	2	0	1	0	4	0	11	18	7.054	0.423

	0.0 %	28.6 %	0.0 %	33.3 %	0.0 %	16.7 %	0.0 %	10.4 %	11.2 %		
Male	13	5	4	2	1	20	3	95	143		
	100.0%	71.4 %	100.0%	66.7 %	100.0%	83.3 %	100.0%	89.6 %	88.8 %		
Total	13	7	4	3	1	24	3	106	161		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically Insignificant											

Chart 28- Association between Gender and Glasgow outcome scale



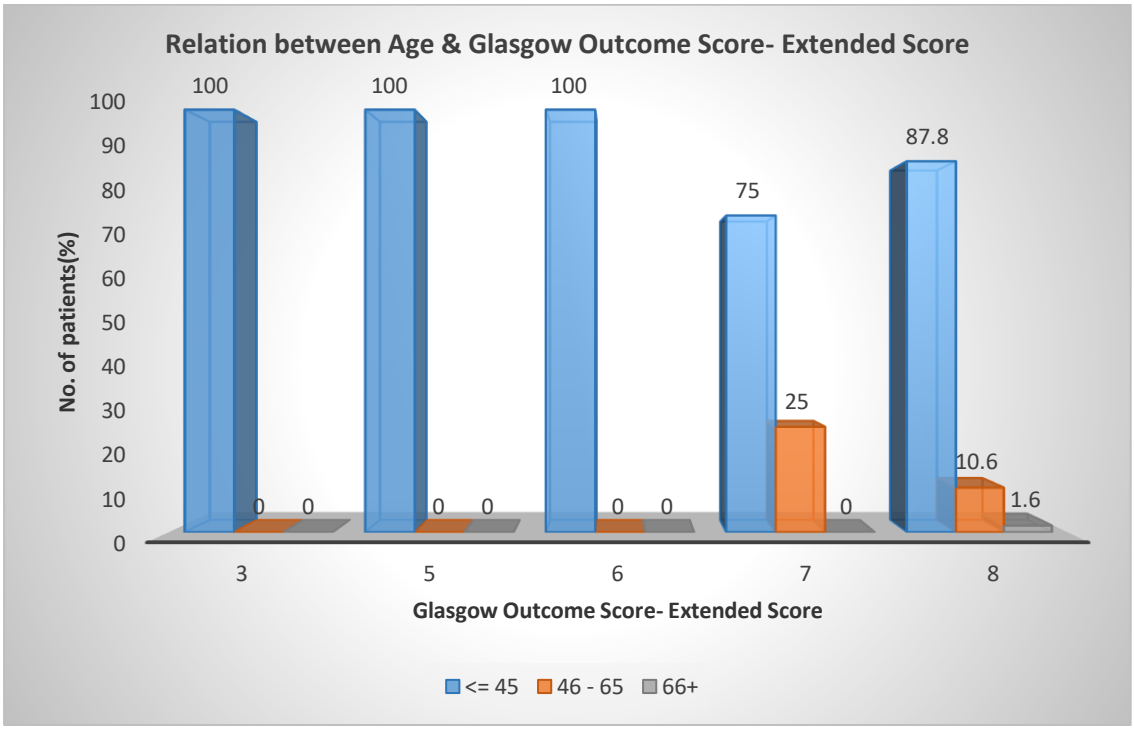
In the present study, we found no significant association between Gender and Glasgow outcome scale- Extended, since the p value is > 0.05.

Table 29- Association between Age (Years) and **Glasgow Outcome Score- Extended Score**

Age (Years)	**Glasgow Outcome Score- Extended Score**						Chi square test	Significant value
	3	5	6	7	8	Total		
<= 45	1	3	2	3	108	117		

	100.0 %	100.0 %	100.0 %	75.0%	87.8%	88.0%	1.766	P=0.987
46 - 65	0	0	0	1	13	14		
	0.0%	0.0%	0.0%	25.0%	10.6%	10.5%		
66+	0	0	0	0	2	2		
	0.0%	0.0%	0.0%	0.0%	1.6%	1.5%		
Total	1	3	2	4	123	133		
	100.0 %	100.0 %	100.0 %	100.0 %	100.0 %	100.0 %		
Statistically Insignificant								

Chart 29- Association between Age (Years) and **Glasgow Outcome Score- Extended Score**



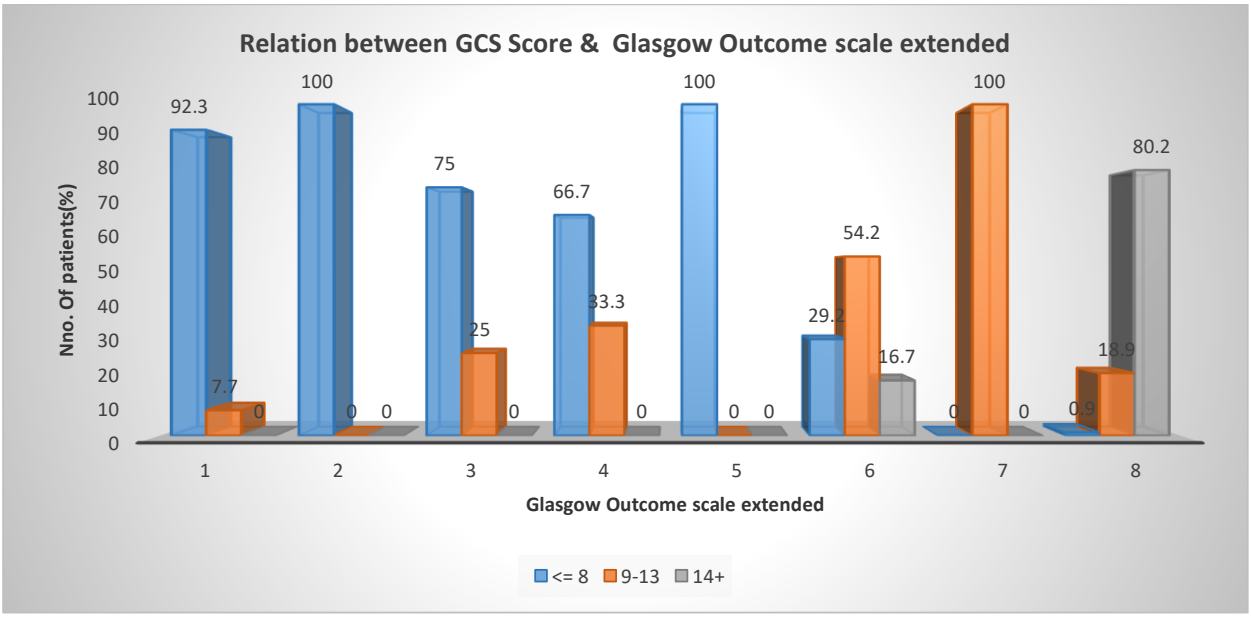
In the present study, we found no significant association between Age and Glasgow outcome scale- Extended, since the p value is > 0.05.

Table 30- GCS score (Binned) * Glasgow Outcome scale extended

GCS	Glasgow Outcome scale extended								Total	Chi square test	Significant value
	1	2	3	4	5	6	7	8			

score											
<= 8	12	7	3	2	1	7	0	1	33	143.827	0.001*
	92.3%	100.0%	75.0%	66.7%	100.0%	29.2%	0.0%	0.9%	20.5%		
9-13	1	0	1	1	0	13	3	20	39		
	7.7%	0.0%	25.0%	33.3%	0.0%	54.2%	100.0%	18.9%	24.2%		
14+	0	0	0	0	0	4	0	85	89		
	0.0%	0.0%	0.0%	0.0%	0.0%	16.7%	0.0%	80.2%	55.3%		
Total	13	7	4	3	1	24	3	106	161		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically significant											

Chart 30- GCS score (Binned) * Glasgow Outcome scale extended



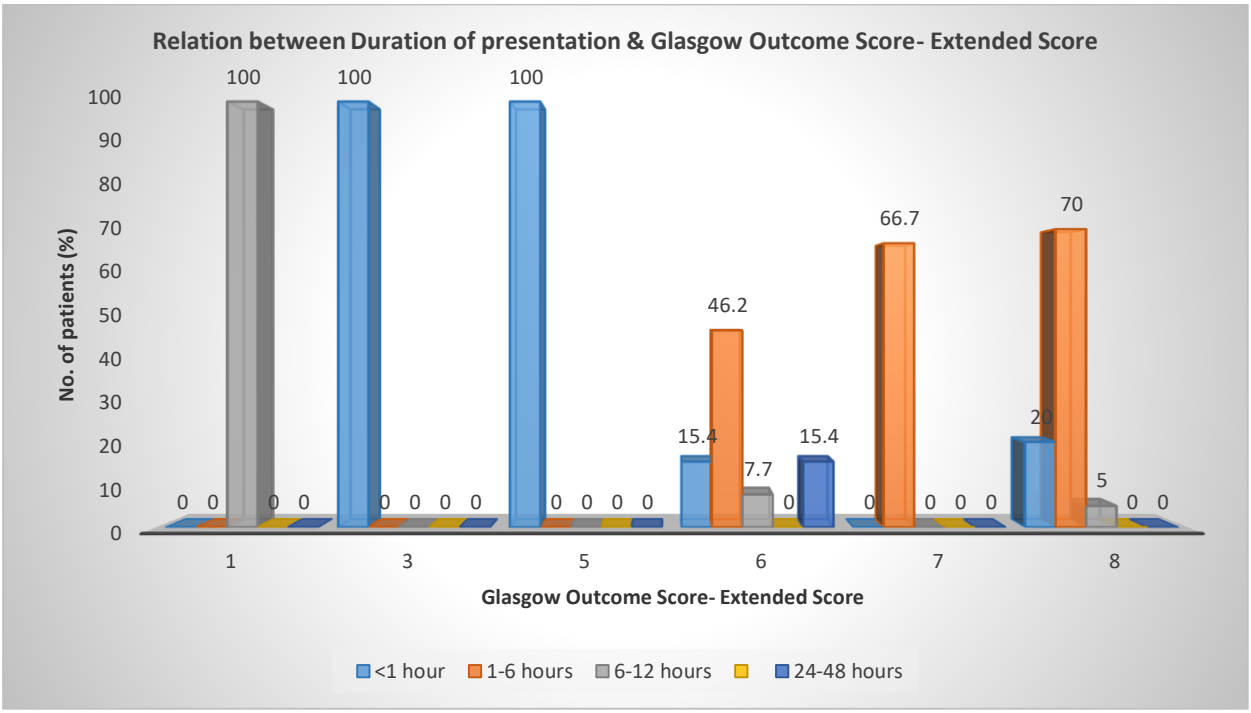
Statistically highly significant association was found with GCS score at presentation, and Glasgow outcome scale-Extended. Patients with mild TBI with GCS score of 14-15, the

outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8, the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor for TBI cases.

Table 31- Association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients

Duration at presentation	**Glasgow Outcome Score- Extended Score**							Chi square test	Significant value
	1	3	5	6	7	8	Total		
<1 hour	0	1	1	2	0	4	8	28.183	P=0.105
	0.0%	100.0%	100.0%	15.4%	0.0%	20.0%	20.5%		
1-6 hours	0	0	0	6	2	14	22		
	0.0%	0.0%	0.0%	46.2%	66.7%	70.0%	56.4%		
6-12 hours	1	0	0	1	0	1	3		
	100.0%	0.0%	0.0%	7.7%	0.0%	5.0%	7.7%		
12-24 hours	0	0	0	2	1	1	4		
	0.0%	0.0%	0.0%	15.4%	33.3%	5.0%	10.3%		
24-48 hours	0	0	0	2	0	0	2		
	0.0%	0.0%	0.0%	15.4%	0.0%	0.0%	5.1%		
Total	1	1	1	13	3	20	39		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically Insignificant									

Chart 31- Association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients

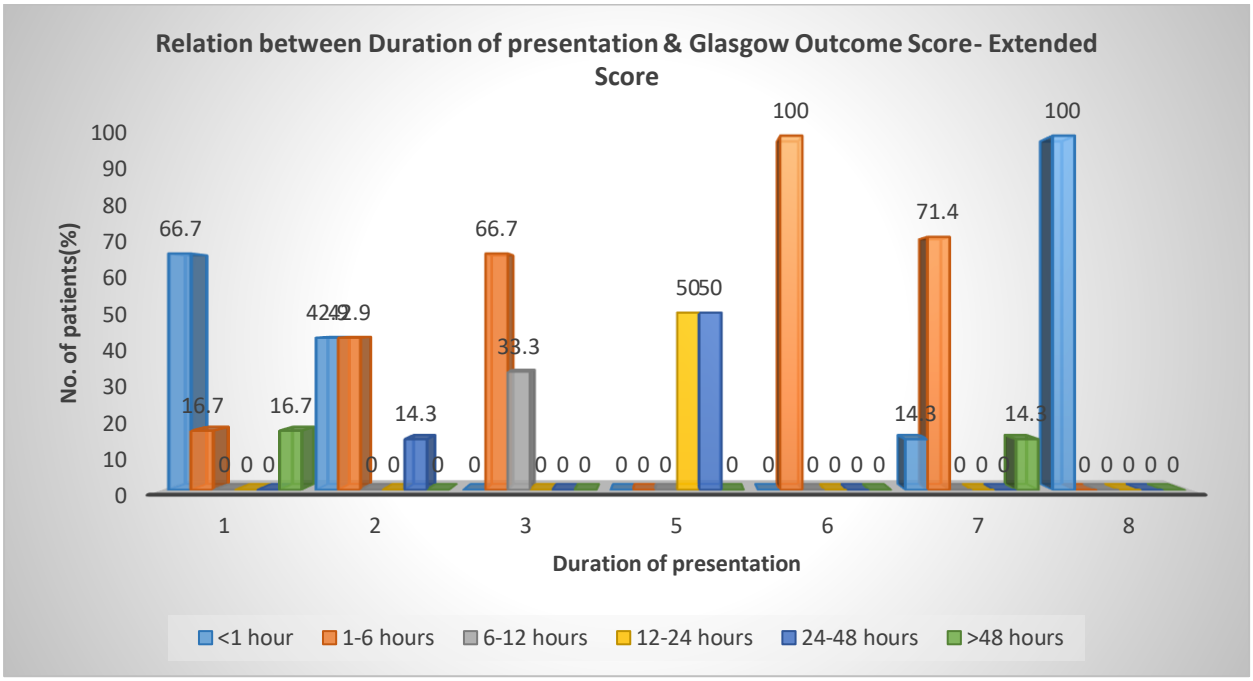


In the present study, we found no significant association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients, since the p value is > 0.05.

Table 32 - Association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients

Duration at presentation	**Glasgow Outcome Score- Extended Score**								Chi square test	Significant value
	1	2	3	5	6	7	8	Total		
<1 hour	8	3	0	0	0	1	1	13	49.147	P=0.015*
	66.7%	42.9%	0.0%	0.0%	0.0%	14.3%	100.0%	39.4%		
1-6 hours	2	3	2	0	1	5	0	13		
	16.7%	42.9%	66.7%	0.0%	100.0%	71.4%	0.0%	39.4%		
6-12 hours	0	0	1	0	0	0	0	1		
	0.0%	0.0%	33.3%	0.0%	0.0%	0.0%	0.0%	3.0%		
12-24 hours	0	0	0	1	0	0	0	1		
	0.0%	0.0%	0.0%	50.0%	0.0%	0.0%	0.0%	3.0%		
24-48 hours	0	1	0	1	0	0	0	2		
	0.0%	14.3%	0.0%	50.0%	0.0%	0.0%	0.0%	6.1%		
>48 hours	2	0	0	0	0	1	0	3		
	16.7%	0.0%	0.0%	0.0%	0.0%	14.3%	0.0%	9.1%		
Total	1		1	1	13	3	20	39		
	100.0%		100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically significant										

Chart 32 - Association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients



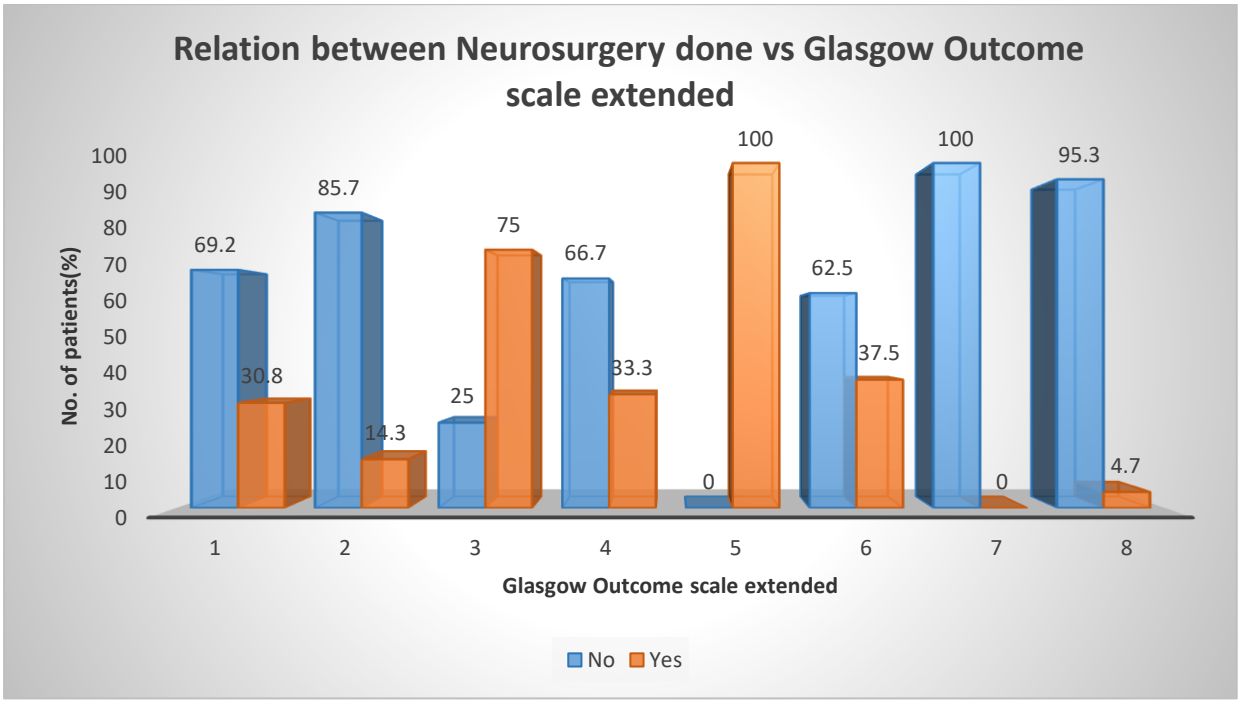
There appears to be a significant association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients, in this study, since the p value is < 0.05. There appears to be more deaths in patients who present to the casualty, within an hour and lesser in those who present late. But, this is not true, since the Outcome of severe cases is more dependent on the specific GCS score, than the range of GCS score. One more point to be noted is that, the number of samples in each of the duration range is varying, and the samples size is maximum for Less than 1 hour and 1-6 hour duration range, (13 each), than others, where the samples are between 0 to 4. Since the sample size for each duration range is varying and not equal, the samples are not comparable to each other. Hence, the test needs further study.

Table 33- Neurosurgery done * Glasgow Outcome scale extended

	Glasgow Outcome scale extended		

Neurosurgery done	1	2	3	4	5	6	7	8	Total	Chi square test	Significant value
No	9	6	1	2	0	15	3	101	137	143.827	0.001*
	69.2%	85.7%	25.0%	66.7%	0.0%	62.5%	100.0%	95.3%	85.1%		
Yes	4	1	3	1	1	9	0	5	24		
	30.8%	14.3%	75.0%	33.3%	100.0%	37.5%	0.0%	4.7%	14.9%		
Total	13	7	4	3	1	24	3	106	161		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically significant											

Chart 33- Neurosurgery done * Glasgow Outcome scale extended



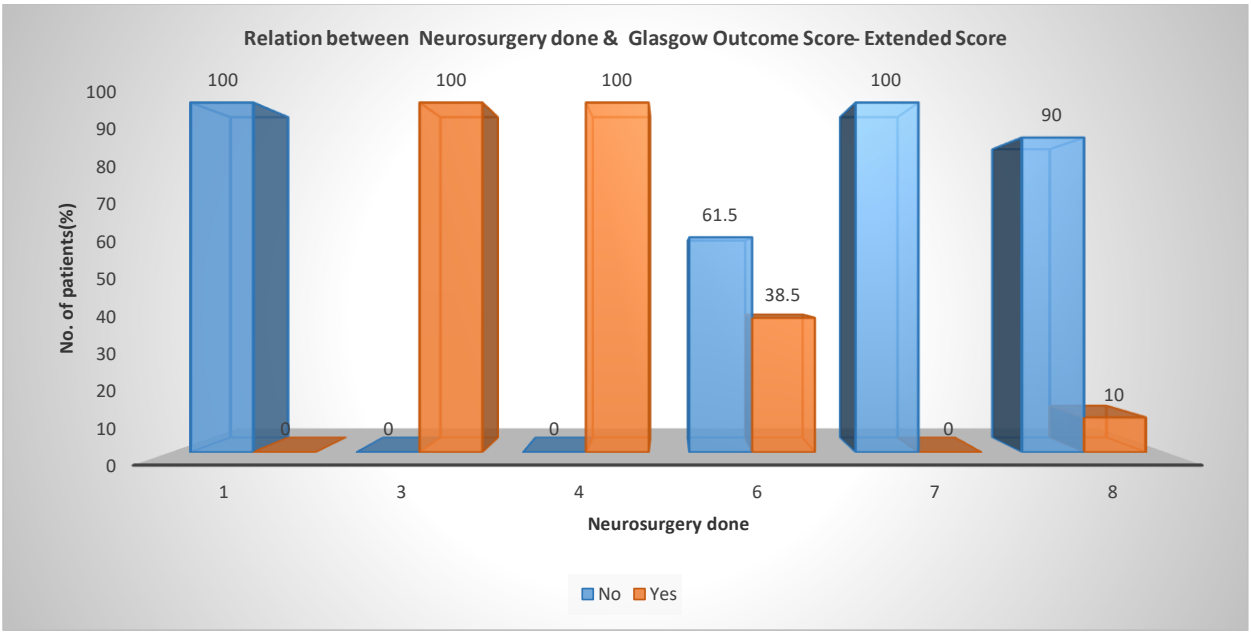
This table shows relation between Neurosurgery done Vs Glasgow coma Outcome scale. The P value is 0.001, indicating a highly significant association between the Neurosurgery done Vs Glasgow Outcome scale Extended. This table indicates better outcome for those who donot undergo Neurosurgery, and worst prognosis for those who undergo neurosurgery. But most of the sample size in this table is of mild TBI, not requiring Neurosurgery at all and hence limits the value of this association.

Let us calculate the association between Neurosurgery Vs Glasgow Outcome scale in moderate and Severe TBI separately and totally excluding mild TBI cases

Table 34: Association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients

Neurosurgery done		Glasgow Outcome Score- Extended Score						Chi square test	Significant value
	1	3	4	6	7	8	Total		
No	1	0	0	8	3	18	30	11.527	P=0.042*
	100.0%	0.0%	0.0%	61.5%	100.0%	90.0%	76.9%		
Yes	0	1	1	5	0	2	9		
	0.0%	100.0%	100.0%	38.5%	0.0%	10.0%	23.1%		
Total	1	1	1	13	3	20	39		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically significant									

Chart 34 Association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients



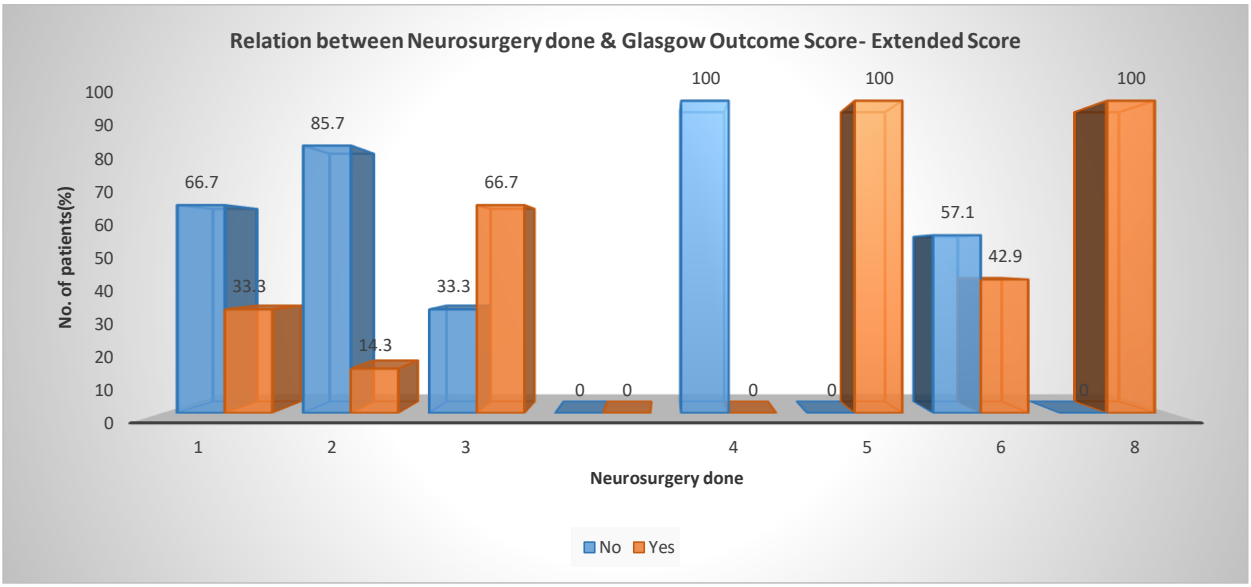
There appears to be a significant association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients, in this study, since the p value is < 0.05. There appears to be better outcome in cases who do not undergo Neurosurgery, in Moderate GCS scored cases. But, this may not be true, since the Outcome of moderate cases is more dependent on the specific GCS score, than the range of GCS score. Secondly, the number of samples in Neurosurgery done is only 9, as compared to the sample size of 30 for Neurosurgery not done cases. Since the sample size for each duration range is varying and not equal, the samples are not comparable to each other. Thirdly, the decision for neurosurgery

depends upon the composition of TBI, for example, Moderate SAH, may be decided to be treated conservatively, than by surgery. The test would have been apt, if operable cases were taken as sample size, and the neurosurgery done and not done status would have been compared. For all the above reasons, the test needs further study.

Table 35: Association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients

Neurosurgery done	**Glasgow Outcome Score- Extended Score**								Chi square test	Significant value
	1	2	3	4	5	6	8	Total		
No	8	6	1	2	0	4	0	21	7.483	P=0.278
	66.7%	85.7%	33.3%	100.0%	0.0%	57.1%	0.0%	63.6%		
Yes	4	1	2	0	1	3	1	12		
	33.3%	14.3%	66.7%	0.0%	100.0%	42.9%	100.0%	36.4%		
Total	12	7	3	2	1	7	1	33		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
Statistically Insignificant										

Chart 35. Association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients.

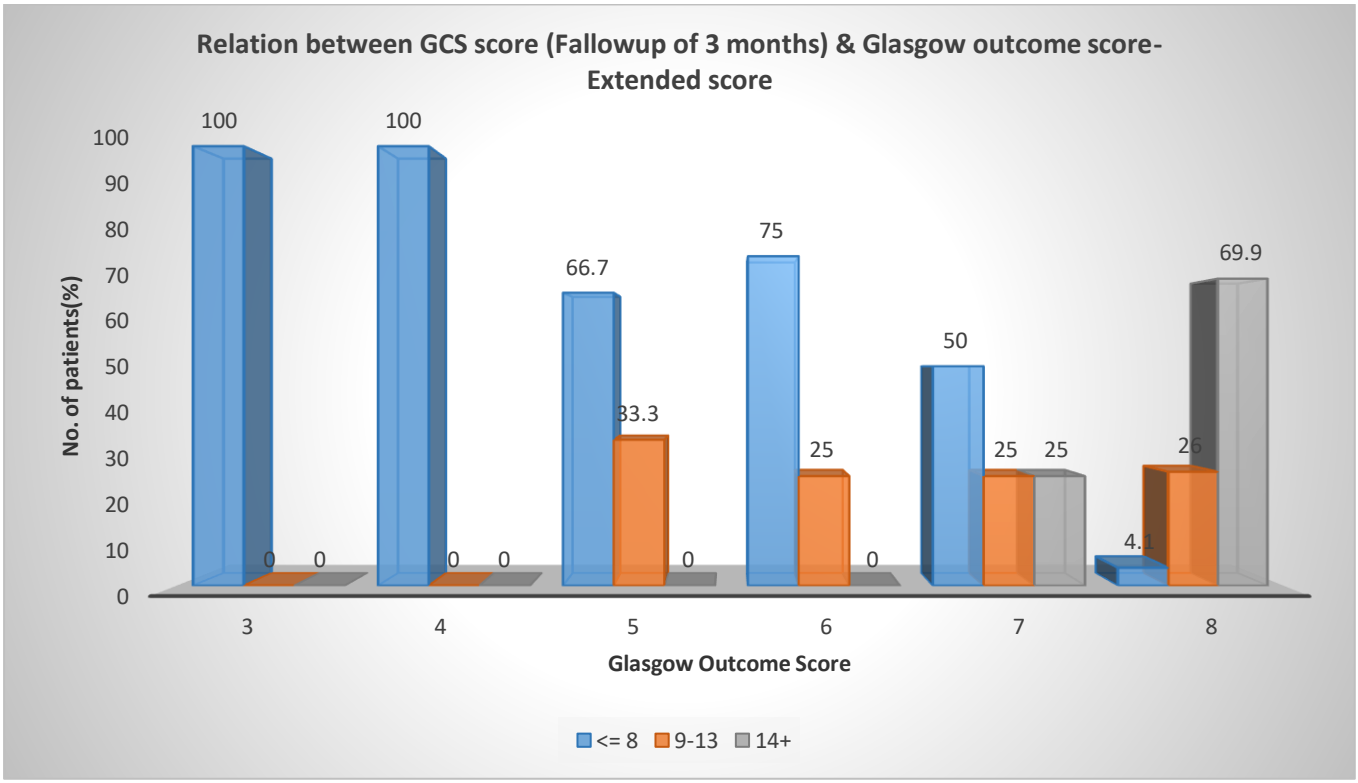


In the present study, we found no significant association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients, since the p value is > 0.05. For the same reasons, as stated above, i.e. the sample size and the varying nature of the sample, i.e. Operable, inoperable and need no surgery are to be separately compared with each other, to get better results. But one point to be noted, is that, deaths are lesser in Neurosurgery done cases (4), as compared to 8 deaths in neurosurgery not done cases. There appears to be a good outcome in terms of deaths, for cases who have undergone Neurosurgery.

Table 36: Association between GCS Score and Glasgow Outcome Score- Extended Score at 3rd month follow up, among all treated cases.

GCS score (FOLLOW UP OF 3 MONTHS	Glasgow Outcome Score- Extended Score							Chi square test	Significant value
	3	4	5	6	7	8	Total		
<= 8	1	1	2	3	2	5	14	59.902	P=0.001*
	100.0%	100.0%	66.7%	75.0%	50.0%	4.1%	10.3%		
9 - 13	0	0	1	1	1	32	35		
	0.0%	0.0%	33.3%	25.0%	25.0%	26.0%	25.7%		
14+	0	0	0	0	1	86	87		
	0.0%	0.0%	0.0%	0.0%	25.0%	69.9%	64.0%		
Total	1	1	3	4	4	123	136		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
*: Statistically significant									

Chart 36. Association between GCS Score and Glasgow Outcome Score- Extended Score at 3rd month follow up, among all treated cases.

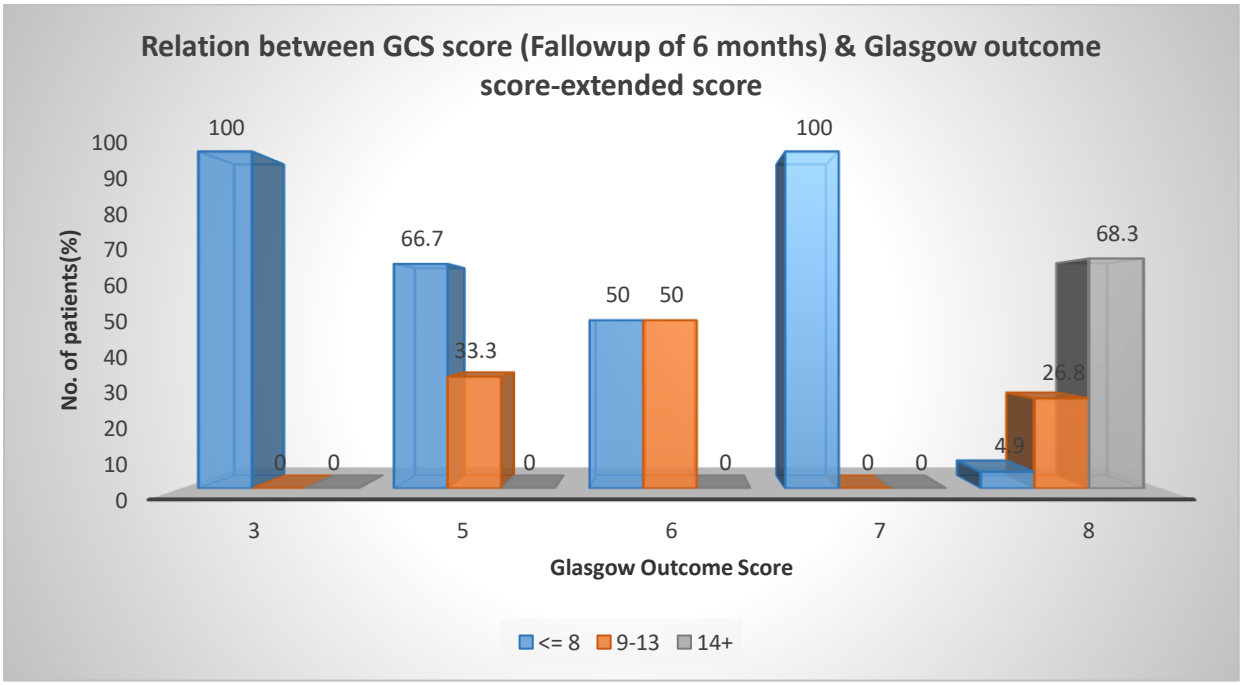


Statistically highly significant association was found with GCS score at initial presentation, and Glasgow outcome scale-Extended at 3rd month follow up. Patients with mild TBI with GCS score of 14-15, the outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8, the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor at 3rd month follow up, for TBI cases.

Table 37: Association between GCS Score and Glasgow Outcome Score- Extended Score at 6th month follow up, among all treated cases

GCS score (FALLOW UP OF 6 MONTHS	Glasgow Outcome Score- Extended Score						Chi square test	Significant value
	3	5	6	7	8	Total		
<= 8	1	2	1	4	6	14	62.337	P=0.001*
	100.0%	66.7%	50.0%	100.0%	4.9%	10.5%		
9 - 13	0	1	1	0	33	35		
	0.0%	33.3%	50.0%	0.0%	26.8%	26.3%		
14+	0	0	0	0	84	84		
	0.0%	0.0%	0.0%	0.0%	68.3%	63.2%		
Total	1	3	2	4	123	133		
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
*: Statistically significant								

Chart 37. Association between GCS Score and Glasgow Outcome Score- Extended Score at 6th month follow up, among all treated cases



Statistically highly significant association was found with GCS score at initial presentation, and Glasgow outcome scale-Extended at 6th month follow up. Patients with mild TBI with GCS score of 14-15, the outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8,

the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor at 6th month follow up, for TBI cases.

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DISCUSSION:

The present study was done in a tertiary care center, and the sample size taken was 172. There is male preponderance among the total TBI cases, 87.8% of the total 172 cases were males and 12.2% were females

According to Agrawal A et al, in an overview of published literature from India on Pattern of reporting and practices for the management of TBI (2018)[7], concludes that, An increasing incidence of Traumatic Brain Injury in the productive age group is of major concern. Both the extremes of age group are more susceptible to severe injury and poor outcome. In the present study, Maximum number of TBI cases belong to the in the early adulthood, i.e. age group of 18-45 years (82%). 15.1% of cases belong to the middle adulthood, i.e. age group of 46-65 years and only 2.9% of cases belong to the older age group i.e. more than 65 years

Among the total TBI cases that presented to our tertiary care center, 71% were from rural, 12% were from semi urban area, and 17% were from urban areas.

According to Hagos A et al, in a Cross-sectional Hospital-based Study management of TBI at Addis Ababa, Ethiopia"(2022)[13] . Road traffic injury was responsible for 45% of the cases. Among the Total TBI Cases attending Our tertiary care centre, 85% were due to Road traffic accident, 10% were due to fall from height, 5% due to Assault.

Among 172 total TBI cases, 161 cases presented to the casualty in the living condition. Among the 161 cases, 37.9% cases presented within one hour of the TBI event, 41% within 1 to 6 hours, 5% within 6 to 12 hours, 8.7% since 12-24 hours, 3.7% presented within 24-48 hours, 3.7% after 2 days. This concludes that 4 out 5 patients presented within 6 hours of Head injury

History of Loss of consciousness after the trauma was present in 94% of total TBI cases. The duration of loss of consciousness was very much varied from few minutes to few hours. History of Vomiting was present in 66% of total TBI cases, and absent in 30% of cases. it was not possible to elicit the history of vomiting among 4% of cases.

History of Vomiting was present in only 1% of total TBI cases, and was not present in 92% of cases. It was not possible to elicit the history of seizures among 7% of cases.

Among all the total 161 TBI cases that were brought live to the casualty, 55.3% of cases were having GCS score between 14-15 (Mild TBI), 24.3% of cases presented with a GCS score between 9-13 (Moderate TBI) and 20.5% of patients presented with a GCS score less than 8 (Severe TBI)

Skull fractures were present in 63.4% of total TBI cases, and absent in 36.6% of cases. Subarachnoid hemorrhage was present in 36.6% of total TBI cases, and absent in 63.4% of cases.

Epidural hemorrhage was present in 15.7% of total TBI cases, and absent in 84.3% of cases. Subdural hematoma was present in 30.8% of total TBI cases, and absent in 69.2% of cases. 44.2% of total TBI cases suffered with contusions in various parts of brain parenchyma. The contusions varied from pin point hemorrhages to subcentimetric hematomas to massive hemorrhages at different parts of brain parenchyma.
Among total 172 cases, Diffuse axonal injury couldn't be elicited in Autopsy of the brought dead cases. Among the total 161 live cases, 9.3% of cases were diagnosed with diffuse axonal injury.

Among total 172 cases that presented to the casualty of the tertiary care centre, 48.8% of patients were adviced admission for medical management, 24.4% for surgical intervention and 20.3% for only a 24 hours observation for any untoward event following a TBI. Those who were adviced admission for medical management, included all such cases which could be managed conservatively, and those severe cases, which were not operable immediately after admission. 6.4% among the total cases were brought dead and could not receive any treatment.
Among total 161 cases opf TBI, which presented to casualty in living condition, 78% of patients agreed to the doctors' advice and 22% went against medical advice.

Among 172 total cases, 6.4% cases were brought dead, and 93.6% cases were in living condition.
Among the 161 total live cases that presented at casualty, 58.4% of cases attended the casualty directly, without referral. 41.6% of total live cases were referred from various other health centres.
Among all the total 161 cases, which were treated at casualty, 65.2% were admitted for neurological reasons, only 1.2% of cases were admitted for non-neurological reasons and 11.8% of treated cases were admitted for both neurological and non-neurological reasons. Rest 21.7% of cases were treated at casualty, but denied admission and went against medical advice.
Among all the 126 IPD cases, 72.2% of patients were managed conservatively, 18.3% of cases underwent Neurosurgery, 7.9% of cases underwent other surgical procedures, and only 1.6% of patients were admitted for observation

Of all the treated cases (both OPD and IPD cases), brought to casualty, 14.9% of cases underwent Neurosurgery and 85.1% of cases, no neurosurgery was being done.
Among 126 admitted cases, 5.6% of cases suffered non neurological complications during the hospital stay
Among all the 161 treated cases, both IPD and OPD basis, at their discharge, 8.1% had a GOS-E score of 1, i.e. Death. 4.3% of cases were discharged in vegetative state (GOS-E score 2), 2.5%

of cases were in lower severe disability (GOS-E score 3), 1.9% in upper sever disability (GOS-E score 4), 0.6% in lower Moderate disability(GOS-E score 5), 14.9% in upper Moderate disability (GOS-E score 6) and 1.9% of cases in Lower Good recovery(GOS-E score 7). Most of the cases, i.e. 85.8% of cases were having upper Good recovery, with a GOS-E score of 8.

Among all the 24 cases, who underwent Neurosurgery, treated cases, both IPD and OPD basis, 4 cases succumbed to death (GOS-E score 1), 1 case was discharged in vegetative state (GOS-E score 2), 3 cases were in lower severe disability (GOS-E score 3), 1 case in upper sever disability (GOS-E score 4), 1 cases in lower Moderate disability(GOS-E score 5), 9 cases in upper Moderate disability (GOS-E score 6), no cases in Lower Good recovery(GOS-E score 7), and 5 cases were having upper Good recovery, with a GOS-E score of 8.

Among 126 admitted cases, 73.8% of cases were in an improved condition at discharge, 15.9% of cases went against medical advice and 10.3% of cases succumbed to death.
In the present study, we found no significant association between Gender and Glasgow outcome scale- Extended, since the p value is > 0.05.

In the present study, we found no significant association between Age and Glasgow outcome scale- Extended, since the p value is > 0.05.

Statistically highly significant association was found with GCS score at presentation, and Glasgow outcome scale-Extended. Patients with mild TBI with GCS score of 14-15, the outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8, the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor for TBI cases.

In the present study, we found no significant association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients, since the p value is > 0.05.

There appears to be a significant association between Duration of presentation and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients, in this study, since the p value is < 0.05. There appears to be more deaths in patients who present to the casualty, within an hour and lesser in those who present late. But, this is not true, since the Outcome of severe cases is more dependent on the specific GCS score, than the range of GCS score. One more point to be noted is that, the number of samples in each of the duration range is varying, and the samples size is maximum for Less than 1 hour and 1-6 hour duration range, (13

each), than others, where the samples are between 0 to 4. Since the sample size for each duration range is varying and not equal, the samples are not comparable to each other. Hence, the test needs further study.

This table shows relation between Neurosurgery done Vs Glasgow coma Outcome scale. The P value is 0.001, indicating a highly significant association between the Neurosurgery done Vs Glasgow Outcome scale Extended. This table indicates better outcome for those who donot undergo Neurosurgery, and worst prognosis for those who undergo neurosurgery. But most of the sample size in this table is of mild TBI, not requiring Neurosurgery at all and hence limits the value of this association.

Let us calculate the association between Neurosurgery Vs Glasgow Outcome scale in moderate and Severe TBI separately and totally excluding mild TBI cases

There appears to be a significant association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Moderate GCS scored patients, in this study, since the p value is < 0.05. There appears to be better outcome in cases who do not undergo Neurosurgery, in Moderate GCS scored cases. But, this may not be true, since the Outcome of moderate cases is more dependent on the specific GCS score, than the range of GCS score. Secondly, the number of samples in Neurosurgery done is only 9, as compared to the sample size of 30 for Neurosurgery not done cases. Since the sample size for each duration range is varying and not equal, the samples are not comparable to each other. Thirdly, the decision for neurosurgery depends upon the composition of TBI, for example, Moderate SAH, may be decided to be treated conservatively, than by surgery. The test would have been apt, if operable cases were taken as sample size, and the neurosurgery done and not done status would have been compared. For all the above reasons, the test needs further study.

In the present study, we found no significant association between Neurosurgery done and Glasgow Outcome Score- Extended Score among treated & Severe GCS scored patients, since the p value is > 0.05. For the same reasons, as stated above, i.e. the sample size and the varying nature of the sample, i.e. Operable, inoperable and need no surgery are to be separately compared with each other, to get better results. But one point to be noted, is that, deaths are lesser in Neurosurgery done cases (4), as compared to 8 deaths in neurosurgery not done cases. There appears to be a good outcome in terms of deaths, for cases who have undergone Neurosurgery.

Statistically highly significant association was found with GCS score at initial presentation, and Glasgow outcome scale-Extended at 3rd month follow up. Patients with mild TBI with GCS

score of 14-15, the outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8, the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor at 3rd month follow up, for TBI cases.

Statistically highly significant association was found with GCS score at initial presentation, and Glasgow outcome scale-Extended at 6th month follow up. Patients with mild TBI with GCS score of 14-15, the outcome was better, Patients with Moderate TBI with GCS score of 9-13, the outcome was moderate and the patients who suffered severe TBI with GCS score of less than 8, the outcome was poor. Hence GCS score at initial presentation could be used as outcome predictor at 6th month follow up, for TBI cases.

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SUMMARY

The study revealed, male preponderance (87.8%), the distribution was more in early adulthood (age group (18-45 years) (82%) for TBI than other age group. The area distribution was Rural (71%), semi urban (12%) and Urban (17%),. RTA(85%) was cause of most TBI cases followed by fall from height (10%) and Assault (5%). Most of the cases presented within first hour (37.9%) and subsequent 6 hours (41%) of time since injury. The presentation symptoms were LOC (94%), Vomiting (66%), Seizures (1%). 11 were brought dead cases, and rest presented as Mild TBI (55.3%), Moderate TBI (24.3%), Severe TBI (20.5%). On CT Scan, Skull fractures (63.4%), Traumatic SAH (36.6%), Epidural Hematoma (15.7%), Subdural Hematoma (30.8%), Contusions (44.2%), Diffuse axonal Injury (9.3%) were found. Out of 172 cases, 126 cases were admitted and treated conservatively (72.2%), Underwent neurosurgery (18.3%), other surgical procedures (7.9%) and observation (1.6%), during admission 5.6% of cases suffered non-neurological complications. 8.1% of cases treated at the hospital on OPD and IPD basis, succumbed to death, and 85.8% had upper good recovery on GOS-E score. Out of 24 cases that underwent neurosurgery, only 4 cases succumbed to death. Age, gender, and delay in presentation were not significantly associated as outcome predictor of TBI in this study with p value >0.05. GCS score at initial presentation was associated with high significance, as outcome predictor at patient's discharge, 3rd month and 6th month follow ups with p value <0.001.

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CONCLUSION:

TBI is a major health problem with RTA being the most common cause of TBI. The occurrence is highest and prognosis is better of Mild TBI, though those suffering from Moderate and severe TBI suffer disabilities of various degree and/or death. Neurosurgery is having better outcome in those who are operable. GCS at initial presentation is an outcome predictor for TBI cases, at discharge, as well, at the end of 3rd and 6th month follow up.

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SAMPLE INFORMED CONSENT FORM

INFORMED CONSENT FORM

TITLE OF RESEARCH : PROFILE AND OUTCOME OF TRAUMATIC BRAIN INJURY PATIENTS IN A TERTIARY CARE CENTRE IN NORTH KARNATAKA: A PROSPECTIVE HOSPITAL-BASED STUDY

GUIDE : DR UDAYKUMAR C NUCHHI

CO GUIDE : DR BASAVARAJ BADADAL

P.G.STUDENT : DR BINDUMADHAV YENDIGERI

PURPOSE OF RESEARCH:

I have been informed that the purpose of this research is to study the Profile and Outcome of Traumatic Brain Injury patients in a Tertiary Care Centre in North Karnataka: A Prospective Hospital-based study

PROCEDURE:

I understand that I will undergo a detailed history and clinical examination, and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved in this study, and I may experience some pain during the above-mentioned procedures.

BENEFITS:

I understand that my participation in this study will help to study the Profile and Outcome of Traumatic Brain Injury patients in a Tertiary Care Centre in North Karnataka: A Prospective Hospital-based study

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital records and will be subjected to confidentiality and privacy regulation of the hospital. If the Data is used for publication, the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary, and I may refuse to participate or withdraw from the study at any time.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me during the study, I will get medical treatment but no further medical compensation.

_____

(Signature of the patient)

STUDY SUBJECT CONSENT FORM:

I confirm that Dr. Bindumadhav Yendigeri has explained to me the purpose of this research, the study procedure that I will undergo, and the possible discomforts and benefits that I may experience in my own language.

I have been explained all the above in detail in my own language, and I understand the same. I agree to give my consent to participate as a subject in this research project.

SIGNATURE OF PARTICIPANT DATE:

P. G. GUIDE BIODATA

NAME : DR.UDAYKUMAR C NUCCHI

DATE OF BIRTH : 22/05/1972

AGE : 50 YEARS

DESIGNATION : PROFESSOR

DEPARTMENT OF FORENSIC MEDICINE & TOXICOLOGY

BLDE(DU) SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE VIJAYAPURA 586103.

CONTACT : 9845700469

EDUCATION

QUALIFICATION : MBBS (BLDEA'S SBMPMCH&RC, VIJAYAPUR)-1999

MD. (FORENSIC MEDICINE AND TOXICOLOGY)

(MRMC, GULBARGA) –2007

WORKING EXPERIENCE : AS UG TEACHER SINCE 2007 -15 YEARS

AS PG TEACHER SINCE 2013 – 9 YEARS

PG GUIDE : SINCE 2022

RESIDENTIAL ADDRESS : DR. UDAYKUMAR C NUCHHI

LIG 10,

KHB COLONY

SOLAPUR ROAD

VIJAYAPUR

P. G. CO-GUIDE BIODATA

NAME : DR. BASAVARAJ BADADAL

DATE OF BIRTH : 14/02/1982

AGE : 40 YEARS

DESIGNATION : ASSISTANT PROFESSOR

DEPARTMENT OF GENERAL SURGERY

BLDE(DU) SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE VIJAYAPURA – 586103.

CONTACT : 8277221455

EDUCATION

QUALIFICATION : MBBS (BLDEA'S SBMPMCH&RC, VIJAYAPUR), 2006

MD. (GENERAL SURGERY), 2012

(COIMBATORE MEDICAL COLLEGE, CHENNAI)

MCh (NEUROSURGERY), 2018

(NIZAM INSTITUTE OF MEDICAL SCIENCE,

HYDERABAD)

WORKING EXPERIENCE : AS UG TEACHER SINCE 2012 - 10 YEARS

AS PG TEACHER SINCE 2012– 10 YEARS

PG CO-GUIDE : SINCE 2020

RESIDENTIAL ADDRESS : WARD NO 9,

SHARAN NAGAR, VIJAYAPUR

BIODATA: CANDIDATE

NAME	:	DR. BINDUMADHAV YENDIGERI
DATE OF BIRTH	:	13-12-1979
AGE	:	42 years
QUALIFICATION	:	MBBS BLDEA's SHRI BM PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE
DESIGNATION	:	POST GRADUATE STUDENT DEPARTMENT OF FORENSIC MEDICINE AND TOXICOLOGY, BLDE(DU) SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYPURA, KARNATAKA
ADDRESS	:	"SHRI SATYASANKALP" MANJUNATH NAGAR, DARGA JAIL ROAD, VIJAYAPUR
CONTACT NO.	:	9611490021

Signature

(Dr.BINDUMADHAV YENDIGERI)

******

ETHICAL COMMITTEE CLEARANCE:

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/ 689/2022-23 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: "PROFILE AND OUTCOME OF TRAUMATIC BRAIN INJURY PATIENTS IN A TERTIARY CARE CENTRE IN NORTH KARNATAKA: A PROSPECTIVE HOSPITAL-BASED STUDY".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Dr Bindumadhav Yendigeri.

NAME OF THE GUIDE: Dr Udaykumar C Nuchhi, Professor, Dept. of Forensic Medicine.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

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 Scrutiny:

- 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

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