OUTCOME OF MILD TRAUMATIC BRAIN INJURY IN THE COURSE OF 24 HOURS OF OBSERVATION AMONG PATIENTS ADMITTED AT MUHIMBILI ORTHOPAEDIC INSTITUTE (MOI).

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A Dissertation Submitted In Partial Fulfillment of the Requirements for the Award of the Degree Of Masters Of Medicine In Orthopaedics and Traumatology of Muhimbili University Of Health And Allied Science.

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CERTIFICATION.

The undersigned certify that he has read and hereby recommend for acceptance by the Muhimbili University of Health and Allied Sciences a dissertation entitled:

Outcomes of Mild Traumatic Brain Injury In the Course of 24 hours of Observation Among Patients Admitted at Muhimbili Orthopedic Institute. This is in partial fulfillment of the requirement for Master Degree in Orthopaedics and Traumatology.

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DECLARATION AND COPYRIGHT.

Declaration.

I, Shadrack SCHOCK declare that ,this dissertation is my own original work and that it has not been presented and will not be presented to any other for a similar or any other degree award.

Signature: ----------

Copyright

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DEDICATION

My late mother Anna Seba ,who died during the study period.

ABSTRACT:

Background: Traumatic brain injury is a global health problem with significant mortality and morbidity. The study aimed at determining the outcomes of mild traumatic brain injured patients during 24 hours of observation among patients admitted at Muhimbili Orthopedic, Trauma and Neurosurgical Institute (MOI) - Tanzania.

Study design: Prospective – Hospital base d observation study. All consecutive patients with mild traumatic brain injury meeting the inclusion criteria were recruited in the study and followed up in the ward for the first 24 hours after admission.

Methods: A total number of 424 patients were included in the study with GCS of 15, 14 and 13. The scores were strengthened by the presence or absence of neurological symptoms like; loss of consciousness, headache, pupillary dilatation, seizures and vomiting. The patients were followed up in the ward and progress was noted. After completion of 24 hours, final remarks of the outcome were noted. The target was three outcomes: 1.Good recovery where patients discharged home 2.Fair or no changes and patients needed more observations 3.Poor progress as the patients were deteriorating.

Also influence of long bone fractures on neurological outcomes were determined.

Results: Sixty three (14.8%) patients deteriorated, 88(20.7%) patients needed more observation and brain CT scan were inquired, 270(63.4%) recovered well and discharged on instructions of primonitory signs of impending neurological deterioration while 3(0.7%) died. The findings were statistically significant (P=000). Subdural hematoma was the leading cause of deterioration 23(39.7%) while normal CT Scan was found in 8(13.8%) patients despite of their deterioration. Presence of long bone fractures concomitantly with mild TBI has a significant role in neurological deterioration (P=0.043).

Conclusion: Inspite of a good score during admission of mild traumatic brain injured patients and high expectations of immediate recovery, still there is a significant possibilities of some of these patients deteriorating. Close monitoring and aggressive intervention measures are needed for the best outcome of patients.

CONTENTS:

ABBREVIATIONS:

B.P	Blood Pressure
C.P.P.	Cerebral Perfusion Pressure
cGMP	Cyclic Guanosine Monophosphate .
D.A.I	Diffuse Axonal Injury
E.M.D	Emergency Medicine Department
G.C.S	Glasgow Coma Scale
I.C.P	Intracranial Pressure
I.C.U	Intensive Care Unit
L.O.C	Loss of Consciousness
M.A.P.	Mean Arterial Pressure.
M.O.I	Muhimbili Orthopedic and Neurosurgical institute
M.T.B.I	Mild Traumatic Brain Injury
MUHAS	Muhimbili University of Health and Allied Sciences
P.R	Pulse Rate
P.S.V.	Persistent Vegetative State.
OT	Orthopedic and Trauma
R.R	Respiratory Rate
T.B.I	Traumatic Brain Injury

CHAPTER ONE

1.0 INTRODUCTION:

In this chapter, the background and pathophysiology of traumatic brain injury is enlighted. Also the primary ,secondary brain injury and an overview on the management approach is presented. The chapter ends by acknowledging Glasgow Coma Score, the tool which groups patients based on severity of traumatic brain injury.

1.1 BACKGROUND.

Traumatic brain injury(TBI) is frequently referred to as the silent epidemic because the problems that result from it often are not visible. Mild traumatic brain injury (MTBI) accounts for at least 75 percent of all traumatic brain injuries in the United States. However, it is clear that the consequences of MTBI are often not mild[1.]

According to the World Health Organization report 2003,TBI will surpass many diseases as the major cause of death and disability by the year 2020,currently ranking fifth while ischaemic heart diseases being the leading one [2]. Furthermore an estimated 10 million people are affected annually by TBI, the burden of mortality and morbidity that this condition imposes on society, makes TBI a pressing public health and medical problem.

According to Moore,TBI is defined as a damage to the brain resulting from external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetration by a projectile[3]. Moreover direction of force like angular, rotational, shear, and translational may contribute to different patterns of TBI.Brain function is temporarily or permanently impaired and structural damage may or may not be detectable with current technology.

Traumatic brain injury poses a public health importance due to its adverse outcomes; mortality and both long and short term morbidities. In developing countries road traffic crashes are the leading cause of traumatic brain injuries[4], mainly attributed to a disproportionate between

rapid increase in automobile than infrastructure support and inadequate users' education. Disabilities resulting from a TBI depend upon the severity of the injury, the location of the injury, and the age and general health of the individual[5]. Some common disabilities include problems with cognition, physical and behavioral changes.

Outcome from head injury is determined by two substantially different stages: the primary and secondary brain injury.

PATHOPHYSIOLOGY OF TRAUMATIC BRAIN INJURY.

The pathophysiology of TBI is highly complex. Several intracerebral inflammatory processes and cascades are initiated in the intracranial compartment as a consequence of head trauma.[6] Brain-derived proinflammatory mediators such as cytokines, chemokines, and complement anaphylatoxins induce chemotaxis of blood-derived leukocytes across the blood–brain barrier, which exacerbate the extent of neuroinflammation and neuropathology by releasing further inflammatory mediators. The inflammatory response is further aggravated by ischemia/reperfusion-related insults, development of cerebral edema, and intracranial hypertension.[7] Furthermore ,when patients with TBI present with additional musculoskeletal injuries, numerous systemic cascades and inflammatory reactions are activated in parallel to it.

Primary brain injury.

Primary brain damage occurs at the time of impact, produces its clinical effect almost immediately and is refractory to most treatment[8]. In treatment terms, this type of injury is exclusively sensitive to preventive but not therapeutic measures. The underlying pathology in this stage includes contusion, damage to blood vessels, and axonal shearing, in which the axons of neurons are stretched and torn. The blood brain barrier and meninges, undergo plastic deformation after exceeding threshold tolerance. In this stage cells death occurs in nonspecific manner [8.20].

Secondary brain injury.

Secondary injury is an indirect result from processes initiated by the trauma[9]. It occurs in the hours and days following the primary injury and plays a major role in the brain damage and death that result from TBI. Unlike in most forms of trauma a large percentage of the people dying from brain trauma do not die right away but rather days to weeks after the event.[10]

In addition, rather than improving after being hospitalized as most patients with other types of injuries do, about 40% of people with TBI deteriorate[11].

Hypoperfusion due to reduced cerebral blood flow following primary brain injury explain initial predisposing cause of deterioration Although the total ischaemic brain volume may be less than 10% on average, the presence of cerebral ischaemia is associated with poor ultimate neurological outcome, that is, dead or vegetative state[12,13].

Imbalance between cerebral oxygen delivery and cerebral oxygen consumption has high correlation with patient deterioration, due to the final common endpoint of brain tissue hypoxia[14]. Measurements of brain tissue oxygen pressure in patients suffering from TBI have identified the critical threshold of 15–10 mm Hg Pt_{O2} below which infarction of neuronal tissue occurs[14,15]. As a consequence of this, the incidence, duration, and extent of tissue hypoxia correlate with poor outcome.Syndrome of inappropriate ADH secretions,cerebral salt wasting and metabolic acidosi are the common end crisis which results into patients deterioration[16]. The most extreme consequences of severe and prolonged hypoxia are the persistent vegetative state (PVS) or death[17]. PVS may occur with preservation of brain-stem reflexes, but with loss of most of the cortex, although hallmarks of DAI are found in 77% of patients who die in PVS.

Cerebral metabolism as reflected by cerebral oxygen and glucose consumption, and cerebral energy state as reflected by tissue concentrations of phosphocreatine and ATP or indirectly by the lactate pyruvate ratio are frequently reduced after TBI[18].Furthermore,

the degree of metabolic failure relates to the severity of the primary insult, and outcome is worse in patients with lower metabolic rates compared with those with minor or no metabolic

dysfunction. The reduction in post-traumatic cerebral metabolism relates to the immediate (primary) insult leading to mitochondrial dysfunction with reduced respiratory rates and ATP-production, a reduced availability of the nicotinic co-enzyme pool, and intramitochondrial Ca^{2+}-overload[19].

Post-traumatic cerebral vasospasm is an important secondary insult that determines ultimate patient outcome[13]. Vasospasm occurs in more than one-third of patients with TBI and indicates severe damage to the brain. The mechanisms by which vasospasm occurs include chronic depolarization of vascular smooth muscle due to reduced potassium channel activity, release of endothelin along with reduced availability of nitric oxide, cyclic GMP depletion of vascular smooth muscle, potentiation of prostaglandin-induced vasoconstriction, and free radical formation[20].

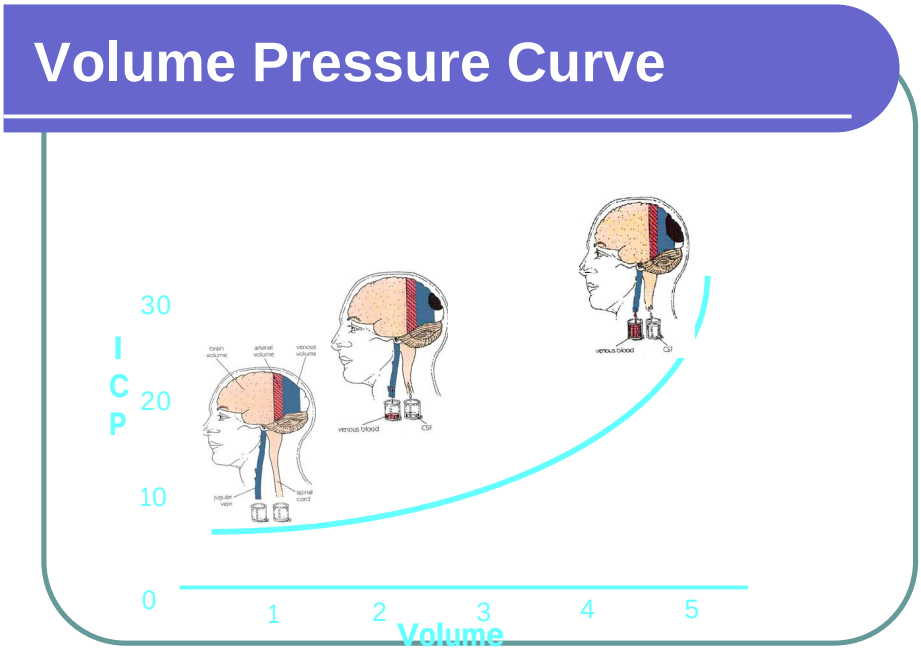
Raised intracranial pressure

Increased intracranial pressure is one of the most damaging aspects of brain trauma and other conditions which directly correlated with poor outcome.

If ICP rises too high it is likely to cause severe harm and if prolonged it becomes fatal. An increase in ICP occuring most commonly due to head injury leading to intracranial hematoma or cerebral edema can result in harmful consequences such as: crush to the brain tissues, shift of the brain structures due to the mass effect, brain herniation, restriction of blood supply to the brain and reflex bradycardia[20].

Since the cranium is rigid, any increase in any of its contents which are cerebral spinal fluid, brain and blood vessels can lead to increase in ICP if compensation does not occur. Any increase of one component must be at the expense of the two according to the Monroe-Kellie Doctrine[21].

Small increase in the brain volume, do not lead to immediate increase in ICP because the CSF will be displace into the spinal canal as well as the presence of slight ability to stretch the falx cerebri.



However, in an increase in ICP above 25mmHg, a small increase in brain volume can cause marked increase in ICP due to failure of intracranial compliance.

Herniation of the respiratory centre, results into respiratory arrest and death[20,21].

Common Types of Traumatic Brain Injury

Subdural hematomas

Subdural hematomas are collections of blood between the dura mater and the pia-arachnoid mater. Usually results from tearing of the bridging veins, particularly those adjacent to the superior sagittal sinus, in association with rapid acceleration or deceleration of the head, and does not require direct impact[20,21]. They often occur with head trauma from falls and motor vehicle crashes. Compression of the brain by the hematoma and swelling of the brain due to edema or hyperemia i.e. increased blood flow due to engorged blood vessels can increase ICP. When both these processes occur, mortality and morbidity increases[20].

Epidural hematomas.

Are collections of blood between the skull and dura mater and are less common than subdural hematomas. Epidural hematomas that are large or rapidly expanding are usually caused by arterial bleeding, classically due to damage to the middle meningeal artery by a temporal bone fracture[21]. Classically they present with a "lucid interval" i.e. few hours of clinically stable state between an initial loss of consciousness and later loss of consciousness and rapid deterioration.

Subarachnoid haemorrhages.

Subarachnoid hemorrhages are small collections of subarachnoid blood and are fairly common after head injury, particularly in association with contusions and lacerations. Subarachnoid haemorrhage may also complicate intraventricular hemorrhage due to a leakage of blood through the exit-foramina of the fourth ventricle[21]. Occasionally, a massive subarachnoid haemorrhage may occur around the ventral aspect of the brainstem due to laceration of a vertebral artery, basilar artery or one of the smaller arteries. This type of haemorrhage often results from an impact to the head or neck in an assault, and causes immediate collapse, and is often fatal[20].

Brain contusions:

Contusions mean bruises of the brain that can occur with open or closed injuries and can impair a wide range of brain functions, depending on contusion size and location. Larger contusions may cause brain edema and increased intracranial pressure. Contusions may enlarge in the hours and days following the initial injury and cause neurologic deterioration[20].

Diffuse axonal injury:

Diffuse axonal injury (DAI) occurs when acceleration and deceleration causes shear-type forces that result in generalized, widespread disruption of axonal fibers and myelin sheaths. DAI is sometimes defined clinically as a loss of consciousness lasting > 6 h in the absence of a specific focal lesion[20]. Edema from the injury often increases ICP, leading to various manifestation of traumatic brain injury.

Managements of TBI.

It is obvious and poses no challenge on the way forward in management of moderate and severe head injury, close monitoring and intensive care are highly needed.

Since little can be done to reverse the initial brain damage caused by trauma, medical personnel try to stabilize an individual with TBI and focus on preventing further secondary brain injury. MILD traumatic brain injury poses great challenges and one among medical surprises happen in the field due to failure to establish common treatment regime[20]. It's no longer surprise a number of patients admitted for 24hours observation following mild traumatic brain injury deteriorate to severe head injury or death.

It poses further challenges when a patient admitted with a normal brain CT scan deteriorates to an extent of requiring intubation or surgical intervention[22,23].

Understanding the pathophysiology of the primary and secondary traumatic brain injury is of great value in anticipating subsequent outcome to patients.

It is important to begin emergency treatment within the so-called "golden hour" following the injury[24]. The main priority in the early management of head-injured patients is the maintenance of an adequate cerebral perfusion pressure [CPP = mean arterial

pressure (MAP) – intracranial pressure (ICP)] above 70 to 80 mmHg The initial assessment follows the normal Advanced Life Support Guidelines. Resuscitation of life threatening injuries accompanied with traumatic brain injury should be prioritized[21].

Complete neurologic examination is done as soon as the patient is sufficiently stable. This is followed by neuroimaging in which CT scan has replaced X-rays to diagnose most of the brain pathology. Magnetic Resonance Imaging is rarely used to diagnose diffuse axonal injuries but not in emergency basis. It's very expensive and inconvenient to most of neurotrauma patients.

The cornerstone of management for all patients is maintenance of adequate ventilation, oxygenation, and brain perfusion to avoid secondary brain insult. Aggressive early management of hypoxia, hypercapnia, hypotension, and increased ICP helps avoid secondary complications[61].

Craniotomy: Intracranial hematomas may require urgent surgical evacuation to prevent or treat brain shift, compression, and herniation; hence, early neurosurgical consultation is mandatory[25]. However, not all hematomas require surgical removal.

Patients with small subdural hematomas can often be treated without surgery.

Factors that suggest a need for surgery include a midline brain shift of > 5 mm, compression of the basal cisterns, and worsening neurologic examination findings[20].

Rehabilitation: When neurologic deficits persist, rehabilitation is needed. Rehabilitation is best provided through a team approach that combines physical, occupational, and speech therapy, skill-building activities, and counseling to meet the patient's social and emotional needs[26].

The Glasgow Coma Scale (GCS)

The Glasgow Coma Scale (GCS) is the standard measure used to quantify the level of consciousness in patients with head injuries. Rapid and accurate GCS scoring is essential for adequate assessment and treatment of critically sick and injured patients. Motor, Verbal and Eye opening responses classify head injuries into MILD (13-15), MODERATE (9-12) and SEVERE (3-8).

Glasgow Coma Scale.

Area Assessed	Response	Points
Eye opening	Open spontaneously	4
	Open to verbal command	3
	Open in response to pain applied to the limbs or sternum	2
	None	1
Verbal	Oriented	5
	Disoriented, but able to answer questions	4
	Inappropriate answers to questions; words discernible	3
	Incomprehensible speech	2
	None	1
Motor	Obeys commands	6
	Responds to pain with purposeful movement	5
	Withdraws from pain stimuli	4
	Responds to pain with abnormal flexion (decorticate posture)	3
	Responds to pain with abnormal (rigid) extension (decerebrate posture)	2
	None	1

*Combined scores < 8 are typically regarded as coma.

Adapted from Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. Lancet 2:81–84; 1974.

CHAPTER TWO

INTRODUCTION.

Literature review showing various studies pertaining to mild traumatic brain injury and the gap present on what is known and not known are highlighted. Problem statement ,rationale and objectives of the study are presented. The chapter ends in defining important term of the study, "the neurological deterioration ".

LITERATURE REVIEW:

Lee AJ et al study in epidemiological comparison of injuries in the school and senior club rugby in China 1996, prospectively followed 1812 patients who were discharged from hospital with GCS-15, at 3 days, 7days, 30 days and 60 days. Overall 28 patients deteriorated. Of the patients that deteriorated 57% (16patient) deteriorated in the first 24 hours post discharge.Twenty three patients of the 28 that deteriorated needed neurosurgical intervention. Most of these patients did not have CT-scan on discharge[27, 28.]This explains the golden period present in the first 24 hours post trauma and hence the need to observe these patients closely.

In another study Dacey et al performed a retrospective study on delayed neurological deterioration between January 2005 and December 2009. Two hundreds and ninety two patients admitted with mild traumatic brain injuries were evaluated. Fifteen cases (5.1%) were immediately taken to theatre following neurological deterioration. These patients did not have CT- scan on admission, epidural and subdural hematomas were the main indication for surgical intervention[4]. Presence of intracranial hematoma whether acutely or slowly and progressive accumulation of hematoma may result into neurological deterioration.

Moreover ,admitting patients with normal brain CT and clinical examination does not rule out the chance of poor progress.This was observed by Shackford SR et al in their study on clinical utility of computed tomographic scanning in the management of patients with mild head injuries.He reported on 933 patients with neurological examination normal and normal Brain

CT – scan who were admitted to the hospital for observation, 2% of these patients deteriorated and needed an intubation but no diagnosis was established on the cause of deterioration. The author did not provide the timing for deterioration.[22, 23].

Contrary to Shackford findings,Stein SC et al observed on the value of computed tomographic scans in the patients with low risk head injuries in 1990.In their retrospective study of 658 patients with an initial GCS of 13 to 15., Non of the 542 patients with normal CT scan showed deterioration or required surgery[29]. No single factor can explain the cause of deterioration.Brain CT is very helpful but does not grant physician to relax in normal findings.

In a series of case studies on patients with head injuries who sometimes talked before dying,,P. Reilly observed that, of 66 patients with head injuries who had talked at some time after injury, 25% did not have intracranial hæmatoma at autopsy. Most of these had raised intracranial pressure (I.C.P.), and the commonest finding was local swelling related to contusions. Almost half of the non-hæmatoma cases had ischæmic or hypoxic brain damage[30].

In Malyasia Ong et al ,observed the outcome of 151 children less than 15 years of age and admitted within 24 hours of head injury were studied in relation to clinical and computed tomography (CT) scan features. Thirty one (20.5%) had a poor outcome (24 died, 6 were severely disabled at 6 months after injury and 1 was in a persistent vegetative state) while 120 (79.5%) had a good outcome (89 recovered well and 31 were moderately disabled). Factors associated with a poor outcome were Glasgow Coma Scale (GCS) score 24 h following injury, presence of hypoxia on admission and CT scan features of subarachnoid haemorrhage, diffuse axonal injury and brain swelling. GCS scores alone, in the absence of other factors, had limited predictive value[31].

In assessment of coma and impaired consciousness, Teasdale G. Jennet B,compared depths of coma in Glasgow and the Netherlands showed remarkably similar outcomes at 3 months. Based upon observations made in the first 24 hours of coma after injury, data from 255 previous cases reliably predicted outcome in the majority of 92 new patients. The exceptions were patients with potential to recover who later developed complications: no patient did significantly better than predicted[27,32].

In the study done in Madrid Spain by Gomez PA on mild head injury differences in prognosis among patient with a GCS of 13 to 15 and analysis factor associated with abnormal CT findings. Retrospective study of 24 84 consecutive patients with mild head injury (GCS 13-15) were seen during period of 18 months, 2351(94.6%) patients scored 15 points, 88 (3.5%) scored 14 points and 45 (1.3%) 13 points. A multivariate analysis showed that advanced age, a lower GCS increase the incidence of abnormal CT-findings, patients with 13-14 GCS had a significantly higher incidence of initial LOC, skull fracture, abnormal CT findings and needed hospital admission. The study concluded that, patients with GCS of 13-14 with different category and needed CT-scan if didn't improve within 4 to 6 hours post injury[33].

During a 15-month study period, Hydel MJ et al,. 1,448 patients underwent CT scanning for mild head injury. Abnormalities resulting from the trauma were found in 119 (8.2%), and 11 patients (.76%) required neurosurgical intervention. Patients with higher GCS scores had a greater chance of having a solitary CT abnormality (P = .004).. High-risk clinical variables included the presence of cranial soft-tissue injury, a focal neurologic deficit, signs of basilar skull fracture and age older than 60 years [34].

According to Akang E.E et al on the underlying causes of deterioration and mortality Ibadan hospital 1991-2000 , 529 cases were recruited , 79.1% of dead persons had a GCS of 8 and less at first presentation.Subdural haematoma was a leading cause of death (62.4%) ,subarachnoid (24.6%),epidural (10.2%) . Skull fracture occurred in 38.2% while cerebral contusion occurred in 22.1%., where as 83.8% were due to RTA[35].

In the study done by McKee MD et al in Toronto on the effect of a femoral shaft fracture, and its treatment by early intramedullary nailing, on the neurologic outcome of patients with multiple injuries with a concomitant head injury identified 46 patients with closed head injuries and femur fractures, and matched as controls 99 patients with head injuries but without femur fractures for age, sex, mechanism of injury. No significant differences between the two groups in terms of early mortality , length of hospital/intensive-care unit stay, level of neurologic disability, or results of cognitive testing. This supports the continued early intra medullary nailing of femoral fractures for these patients[36].

Michael A. Flierl, MD et al in their study on femur shaft fracture fixation in head I njured patients: When Is the Right Time? They concluded that ‘‘early total care’’ principles may be considered in patients with isolated femur fractures and mild TBI and ‘‘damage control’’ approach may be applied to patients who sustained isolated femoral fractures with associated moderate or severe TBI (GCS 3–13) as well as to the severely poly traumatized patient with concomitant injuries to the chest and/or abdomen or multiple fractures[37].

In a study of predictors of functional recovery and hospital mortality in patients with traumatic intracranial hematomas, Dr. Kitunguu K.P and Kiboi G.J at Kenyatta hospital reported that 59.3% patients with pre operative GCS of 8 or less died, while only 11% and 3% death occurred in patients with moderate and mild head injure respectively.

Patients operated on more than 4 days, after initial trauma had a mortality of 42.1% as compared to 9.3% for patients operated within 24 hours[38].

Traumatic brain injury is a global problem and is a major cause of disability, death and economic cost to our society. From various researches done about traumatic brain injury, one central concepts emerge is that “most neurological damage from traumatic brain injury does not occur at the moment of impact, it evolves over the ensuing hours and days. From the research, it has been observed that, improved outcome results when these secondary brain damage have been prevented

PROBLEM STATEMENT:

MOI being a well established trauma and neurosurgical institute is experiencing an increased number of trauma patients seeking health services. In the six months survey from medical records, May 2010 to October 2010, 5009 patients attended at MOI emergency department. 2248(45%) of these patients were admitted. 1138(23%) patients were diagnosed to have traumatic brain injury regardless of their score, of these patients 552(25%) of the total admission were due to mild traumatic brain injury. Most of them were admitted for 24 hours observation; few were discharged home straight from the emergency department. There is no study conducted highligthing the outcomes of MTBI during 24 hours of observation on neurological recovery or deterioration. Those patients deteriorating or with good recovery, no data available addressing the magnitude of problem, prevalence and the underlying causes of patient deterioration after 24 hours of observation in the ward.

The ideal timing and modality of long bone fracture fixation in head-injured patients remains a topic of debate. Several groups advocate the immediate definitive fixation of femur fractures (‘‘early total care’’), whereas others support the concept of ‘‘damage control orthopaedics. This problem is also encountered at MOI. Inspite of these debates, no study has been conducted to address findings on the influence of long bone fractures and their early treatment on neurological outcomes of patients with concomitant mild traumatic brain injury.

Although the hospital cut-off point for neuro imaging is GCS of 12 and below a number of constrains arise on the impending injury deterioration. There are a number of cases who have deteriorated regardless of a good score during admission. Decision making on undergoing a brain CT scan poses a great challenge to the attending doctors. It is therefore pertinent that the above mentioned facts are studied.

RATIONALE:

The study will enlighten on the magnitude of mild traumatic brain injury patients admitted for 24 hours observation at the institute.

Moreover the study will help to determine the extent of mild traumatic brain injured patients who deteriorate and need other intervention such as craniotomy or intubation.

The challenge present due lucid interval in epidural hematoma, slow leaking bridging vein in acute subdural hematoma, gradual increase intracranial pressure and change of auto regulatory set point due traumatized brain cells with standard of care which have to meet these challenges will be explained and the inputs will help to improve the standard of care at the institute.

Results from this study will add information to the body of knowledge and the data used as a basis for further studies.

OBJECTIVES:

Broad Objective:

To determine the outcomes of mild traumatic brain injured patients in the course of 24 hours of observation among patients admitted at Muhimbili Orthopedic Institute between April 2011 and September 2011.

Specific Objectives:

1. To determine the proportion of mild traumatic brain injuries by demographic distribution by age and sex between April 2011 and September 2011.

2. To determine the level of deterioration of patients admitted for 24 hours observation following mild traumatic brain injury

3. To determine the common underlying radiological causes of patient's deterioration.

4. To determine the influence of long bone fractures on neurological outcome of mild traumatic brain injured patients.

Term Definition.

Deterioration following traumatic brain injury ;

Sustained decrease in conscious level of at least 1 point in the motor or verbal response or 2 points in the eye opening response of the GCS score.

Development of severe or increased headache or persisting vomiting.

New or evolving neurological symptoms or signs, such as pupil inequality or asymmetry of limb or facial movement.

CHAPTER THREE

METHODOLOGY

3.0 INTRODUCTION.

In this chapter the outlines of methods used to achieve the aim and objectives of the study are presented. A quantitative approach using the individual interview and clinical examination was used in data collection and analysed using SPSS programme. The various aspects of methodology as well as ethical considerations are presented.

3.1 Study design

Prospective observation study. Those consecutive patients with mild traumatic brain injury admitted for 24 hours observation were followed on their progress in the wards.

3.2 Study Setting.

The study was conducted at Muhimbili Orthopedics, Trauma and Neurosurgical Institute. MOI is a tertiary and well established trauma centre in Tanzania and East Central Africa delivering Orthopedic, Trauma and Neurosurgical services. It is well staffed hospital in those three specialties. . It is in Dar es Salaam, currently receiving about 800 and 2100 patients per months through EMD and OPD respectively from all regions of Tanzania and Indian Ocean Islands especially the Comoros. The hospital has 171 admitting beds.

Patients enrolled in the study were recruited from the emergency department during admission, after meeting the inclusion criteria and thereafter followed in the wards..

3.3 Study population

All consecutive patients admitted with mild traumatic brain injury through MOI emergency department from April 2011 and September 2011. These patients were then followed up for 24 hours being reviewed every 6hours interval and, based on Glasgow Coma Score, those who deteriorate; CT Scan was requested for further diagnosis and intervention.

Inclusion criteria.

- All patient age 13years onwards .This is the age in which patient is able to explain his condition and evaluate himself or herself on the progress.
- Mild traumatic brain injured patient admitted at the institute, arriving within 24hrs post injury.
- Patients with mild traumatic brain injury who meet the criteria for admission
- Glasgow coma score of 13 and 14
- Glasgow coma score 15 with the following features

History of LOC

Post traumatic Amnesia.

Persistent and worsening headache.

Vomiting.

Seizures.

Presence lateralizing sign **.**

Exclusion criteria

- Age below 13years.
- Patients arriving 24 hours post injury
- Patients in serious medical condition eg. in stroke
- Known epileptic patients.
- Patient in alcohol intoxication.
- Non consenting patients.

3.4 Sample Size

The sample size was determined by the following formulae based on finite population of 840 patients derived for Epi info 6 software.

$$n = \frac{Nz^2P(1-P)}{d^2(N-1) + z^2P(1-P)}$$

N= population size

n = sample size required

z = percentage point corresponds to 5% level of significant

P = expected mortality rate

Population (P = 4.8)

d = margin of error (1.5%)

Therefore minimum required sample size was 374. However a total number of 424 patients were enrolled in the study.

3.5 Research instruments

Pre- tested Questionnaires were used to collect information. In person type, face- to face guided by clinical examination.

One research assistant accomplished by filling the admitting part of the questionnaire at casuality and confirmed by the author.

3.6 Data collection and management.

Data collection was done through a structured questionnaire at Emergency Department and in the ward in the course of 24 hours. After admission, these patients were reviewed at 6 hours interval to assess progress. This was done with assistance by pre trained admitting staff, and assisted by other neuro surgery firm doctors in the ward.

The fully filled questionnaires were entered in the data base prepared in the SPSS programme version 17 by the researcher for analysis. Data were summarized in form of proportions, frequency tables, pie charts bar charts and Two by Two tables.

Deterioration of patients was determined by GCS during admission compared to the score after 24 hours of observation; the scores were strengthened by presence or absence of lateralizing sign, loss of consciousness, protracted vomiting and headache. Deteriorated patients were followed for further diagnosis through brain CT-scan.

3.7 Ethical consideration

Permission to carry out study was granted by MUHAS research ethical committee.

Before the study begun further permission was obtained from MOI to gain access to the research setting. Additionally, informed consent was requested from the participants. In line with consent, participants were explained the nature and purpose of the study.

3.8 IMPLEMENTATION PLAN

Man power

This involved the whole admitting team i.e. Doctors and Nurses All doctors in neurosurgery firm especially through ward round were also a vital role in remarking patient progress after 24 hours of observation. All ward nurses, who were the first to report any changes of condition of patients in the ward.

The author was always part of admitting, discharging team and any emergency observed among study cases in the ward.

Facilities

The study was conducted within hospital vicinity. Patients handling was as per routine.

3.9 MONITORING.

This started by training all assistants on how to fill the questionnaire on patient information. The questionnaire had two part. 1st parts was filled at the emergence department while the second part was filled in the ward after 24 hours of observation or in midist when patient was found deteriorating during serial progress monitoring.

When a patient enrolled in the study deteriorated, the researcher was informed together with the doctors on call for immediate intervention.

CHAPTER FOUR.

RESULTS

4.1.INTRODUCTION.

The purpose of this chapter is to present the results obtained from the prospective cross sectional study which was quantitative in nature. An overview of the demographic profile of the participants in relation to age and sex have been highlighted. Level of neurological deterioration and their underlying causes are presented while statistical significance was determined through the Chi-square and *P value*. Tabulations and pi chart were used to present the characteristic features observed.

4.2.RESULTS. On proportion of mild traumatic brain injuries by their demographic distributions the findings revealed that, males were more affected 334(78.8%) than female 90(21.2%) by ratio of 3.7:1.The age group 25-34 years were at high risk of sustaining mild traumatic brain injury 140(33.0%) while age group <13 years was least group to be affected 4(0.9%).Mean age found to be 33.6 and SD 14.7.

CHARACTERISTICS	Number of patients	Percent
Age(years)		
<15	4	0.9
15-24	127	29.9
25-34	140	33.0
35-44	71	16.7
45-54	34	8.0
55-64	21	4.9
≥65	22	5.2 (N=424)
Mean 33.6 and SDV 14.7		
SEX		
Male	334	78.8
Female	90	21.2 (N=424)
Male:Female 3.7:1		

Table1. Socio-demographic characteristics of patients admitted with mild traumatic brain injury. (N= 424)

Motor traffic crash was the leading cause of mild traumatic brain injuries com

prising 77.8% of the total admission followed by assault.

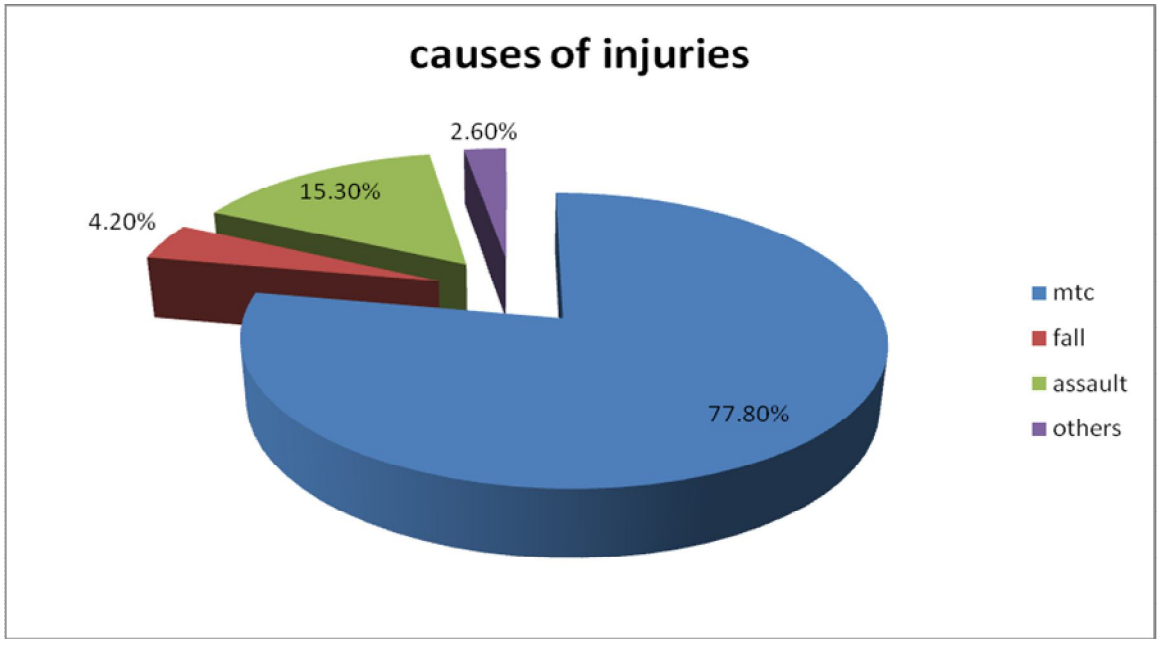


Figure 1: The underlying causes of injuries among patients admitted with mild TBI.

Determining the level of deterioration after 24 hours observation following mild traumatic brain injury, two hundred and sixty night(63.4%) of the admitted patients were discharged home after 24 hours of observation, eighty nine(20.7%) needed more observation while 63(14.8%) deteriorated and 3(0.7%) died. Level of consciousness (GCS) has direct impact towards patients' recovery or deterioration. These findings were found to be statistically significance (p = 0.000**).**

	OUTCOMES					
Level of consciousness (GCS)	N	Discharged home	More observation	Deteriorated	Dead	*P value*
15	309	239(77.6%)	29(9.4%)	39(12.7%)	2(0.6%)	**0.000**
14	85	30(35.3%)	41(48.2%)	14(16.5%	0(0.0%)	
13	30	0 (0.0%)	19(60.0%)	10(33.3%)	1(0.3%)	
TOTAL	424	269(63.4%)	89(20.7%)	63(14.8%)	3(0.7%)	

Table 2. Outcomes of mild traumatic brain injury after 24 hours of observation.

In the following table, the findings revealed that, initial loss of consciousness ,vomiting, seizures, dilated pupils, skull fracture, ear, nose and mouth discharge had statistically significant value on determining the outcome of mild TBI while headache showed no significant role.

Symptoms and Signs	N	Discharge Home	More observation	Deteriorated	*P - value*
LOC					
+	359	219(61.0%)	85(23.7%)	55(15.3%)	0.024
_	62	50(80.6%)	4(6.45%)	8(12.9%)	
Vomiting					
+	73	13(17.8%)	35(47.9%)	25(34.2%)	0.000
_	348	256(73.6%)	54(15.5%)	38(11.0%)	
Headache					
Normal	145	103(71.0%)	8(5.5%)	34(23.4%)	0.707
Mild headache	245	164(66.9%)	68(27.8%)	13(5.3%)	
Severe headache	51	2(3.9%)	13(25.5%)	36(70.6%)	
Seizure					
+	39	14(35.9%)	15(38.5%)	10(25.6%)	0.001
_	382	255(66.8%)	74(19.4%)	53(13.9%)	
Dilated pupils					
+	27	4(14.8%)	19(70.4%)	4(14.8%)	0.001
_	394	265(67.3%)	70(17.8%)	59(15.0%)	
Ear, nose, mouth discharge					
+	89	40(44.9%)	31(34.8%)	18(20.2%)	0.001
_	332	229(69.0%)	58(17.5%)	45(13.6%)	
Skull fracture					
+	70	35(50.0%)	20(28.6%)	15(21.4%)	0.015
_	351	234(66.7%)	69(19.7%)	48(13.7%)	
TOTAL	421	269(63.9%)	89(21.1%)	63(14.9%)	

Table3.Symptoms and signs observed and their prognostic role on outcomes of mild TBI.
Following deterioration , 58 (92.1%) of the patients deteriorated afforded to do brain CT scan. Subdural hematoma(39.7%) was the leading cause of deterioration while 8(13.8%) despite of their deterioration, brain CT scan did not reveal any abnormalities.

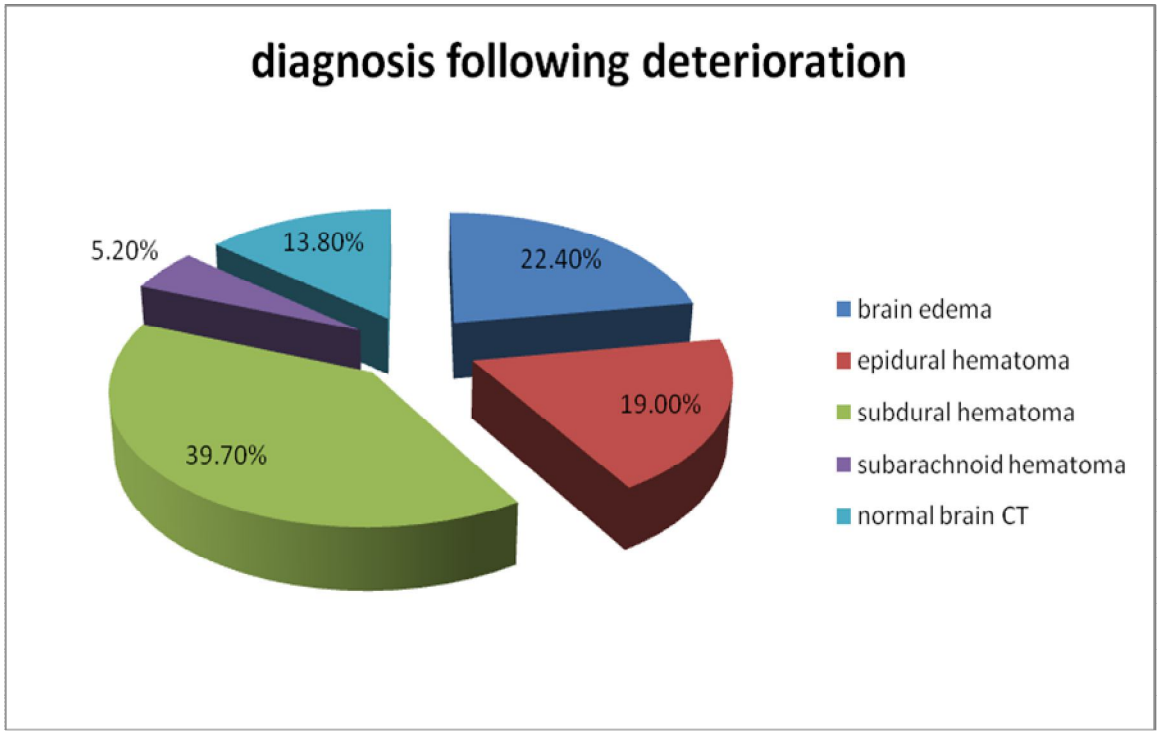


Figure2: The diagnosed underlying cause of patients deterioration after 24hours of observation

Patients failed to afford the cost of undergoing brain CT Scan, had to wait for assistance from social welfare.The logistics took longtime and hence these patients were discarded from the study for the next step of diagnosis.

On impact of long bone fractures on neurological outcomes, a high proportion of deterioration was observed in isolated TBI group 51(17.4%) than TBI and long bone fractures 12(9.3%). These findings were statistically significant. (p = 0.043).

OUTCOMES

PATTERNS OF TBI	N	Deteriorated	Not deteriorated	P value
				0.043
TBI+Long bone fractures	129	12(9.3%)	117(90.7%)	
Isolated TBI	292	51(17.4%)	241(82.5%)	
TOTAL	421	63(14.9%)	358(80.0%)	

Table4: Impact of long bone fractures on neurological outcomes of mild TBI**.**

CHAPTER FIVE.

DISCUSSION.

5.0. INTRODUCTION.

In this chapter the results of the study are discussed in relation to objectives and compared with literature on similar or closely related studies.The goal of this study was to determine the outcomes of mild traumatic brain injured patients after 24 hours of observations among patients admitted at Muhimbili Orthopedic Institute between April 2011 and September 2011.Guided by the results ,conclusion and recommendations have been highlighted accordingly.

5.1.DISCUSSION.

In this study the males female ratio was 3.7:1. This is a rapid increase in a gap between male and female for the past 5 years where a ratio of 2:1 was observed by Raymond Mwanga on his pattern of traumatic brain injury at MOI.[49.] Many studies shows men suffer twice as many TBIs as women do and have a fourfold risk of fatal head injury[40].

The age of the study population ranged from 13 years to 80 years. Age group between 25-34 years being most affected 140 (33%) with mean age 33.6 years and standard deviation 14.5 .Nearly similar findings were observed by Bruns Jr et al who concluded that male gender and young adults are at increased risk of succumbing traumatic brain injury compared to female and other age groups.[51,52]

The study findings revealed that 63 (14.8%) of the study population deteriorated within 24 hours of observation and 3(0.7%) died . As the level of consciousness on admission was decreasing; deterioration increased ie.12.7%, 16.7%,and 33.3% with GCS of 15,14 and 13 respectively. Likewise, as the level of consciousness increased, recovery improved. These findings were statistically significant ($P \leq 0.001$). Available data suggests that although between 5% and 13% of patients with a GCS score of 15 n

evaluated in the emergency department will have a traumatic lesion of some type on CT scan[41,42], < 1% of patients will require neurosurgical intervention[43]. This findings concurs with previously established studies by Kiboi GJ et al at Kenyata hospital who found a statistically significant difference in the proportion of patients who achieved functional recovery , with 65.2% in mild TBI versus 19.7% and 15.1% in moderate and severe head injury respectvely[31,38]. Moreover in this study , thirty nine patients 61.8% who deteriorated were admitted with GCS 15. These are the patients whom regardless of good score, talking during admission rapidly deteriorate and even die as a short term outcome i.e. talk and die syndrome[30]. 36(57.1%) of the patients that deteriorated had severe headache at final evaluation while 164(60.7%) of the patients who recovered well had mild headache at discharge, similar findings to those by Kitumka at Mulago hospital in Uganda[39].

In the present study, subdural haematoma was a leading cause of deterioration with about 39.7% rate. Similar findings were observed by Adeleye et al where subdural hematoma was the leading cause of mortality and disability[52].This is explained by its mechanism of injury which involves tearing of bridging veins which are easily torn even with minor injury, sudden acceleration and deceleration. Brain CT-scan was found to be normal in 8 (13.8%) despite of their deterioration. Shackford et al reported the same rate of normal brain CT among TBI group who deteriorated[22,23].Cause of deterioration can be explained by the presence of diffuse brain injuries due to second hit impact eg. hypoxia due to decreased cerebral perfusion pressure, diffuse axonal injuries etc. These kind of injuries are not evident in routine brain CT Scan.

On impact of long bone fractures on neurological outcomes of mild traumatic brain injury, the findings revealed that, the proportion of patients with head injury and long bone fractures who deteriorated was 12(9.3%) compared to 51(17.4%) of isolated mild traumatic brain injury. This is contrary to what was expected, because of blood loss due to fracture, increased metabolic and hormonal response due to trauma and the resultant inflammatory response which has direct negative impact on brain physiology. These complex inflammatory events

render the traumatized brain highly susceptible to secondary brain injury hence it was expected bring poor outcomes. These injuries probably did not have direct effect on brain physiology. Statistically the results were statistically significant (P = 0.043). Lining with these findings, Stahel et al concluded that, during the first 24 hours, any unnecessary surgical interventions, including intramedullary fracture fixation, may negatively alter the patient's MAP, resulting in deterioration of the CPP and aggravation of secondary brain injury[44], although in this study all patients with long bone fractures were included regardless whether earlier treated surgically or conservatively. On the contrary, McKee et al revealed different findings from the present study and concluded that, femoral fracture in a patient with a concomitant head injury does not increase mortality or neurologic disability, and supports the continued early intramedullary nailing of femoral fractures for these patients [36,45].

The exact GCS indicated for neuro-imaging is still under debate at the institute. The protocol cut off point of GCS 12 and below as an indication for brain CT is not valid due to a number of constrains which arises on the possible deterioration despite of good score. In this study of the 85 patients admitted with GCS of 14, only 30(35.3%) recovered well after 24 hours and needed discharge, the remaining 55(64.7%) either needed more observation or deteriorated and brain CT scan were required for all of them. This proportional is higher and statistically significant enough to warrant new cut off point of GCS 14 and below as an indicator of neuro imaging straight forward plus GCS of 15 with any of following features; loss of consciousness for more than 5 minutes, depressed or decreasing level of consciousness , focal neurological findings , seizure, failure of the mental status to improve over time in an alcohol-intoxicated patient , penetrating skull injuries , signs of a basal or depressed skull fracture, confusion or aggression on examination, severe headache, more than two episodes of vomiting and ≥65 years.[46,47]These symptoms were assessed and found to be statistically significant in predictive role on neurological outcomes. Therefore the established neuroimaging guidelines on traumatic brain injury proves superior and warrants adoption by the institute.

5.2.CONCLUSION:

The fact that patients with mild traumatic brain injury have no obvious brain damage makes a considerable impact on health care system and hospital policies.

Admitting patients for 24 hours observation should not mean damping them. Close follow-up is needed to rescue patients from impending injury deterioration due to second hit impact on brain physiology eg. the talk and die syndrome. To optimize outcomes on managing these patients; physician should be easily available for aggressive intervention in any case of any deterioration.

5.3.RECOMMENDATIONS:

All patients admitted with mild traumatic brain injury for 24 hours observations should be followed closely because they have potential risk of deterioration.

This study emphasized on early outcomes of mild traumatic brain injury within 24 hours of observation. As more mild TBI require prolonged hospitalization, long term outcome studies may give insight into better understanding and improve on treatment protocol.

5.4.Study Limitations.

There was a delay in getting immediate Brain CT scan and failure to afford the bearing cost of CT Scan. This was an obstacle of early intervention which has been shown to have better outcomes compared to delayed intervention.

The exact timing on when the patient starts to deteriorate was a problem because the six hours interval of examination was too long. Patients may deteriorate to death within few hours as compounded by constraints of resuscitative gears.

Reviewing the patient at 6 hours interval needs time devotion and commitment. Enough number of motivated staff was needed but fund was not enough to support.

CHAPTER SIX

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6.2.CONSENT FORM

My name is Dr. Shadrack SCHOCK. I have obtained permission from the MUHAS Ethics and Research Committee and MOI to conduct the study on:

OUTCOMES OF MILD TRAUMATIC BRAIN INJURY IN THE COURSE OF 24 HOURS OF OBSERVATION AMONG PATIENTS ADMITTED AT MUHIMBILI ORTHOPAEDIC INSTITUTE (MOI)

The main purpose of my study is to enlighten on the magnitude of mild traumatic brain injured patients admitted at the institute.

Also in my study, I will determine the extent of deterioration among patients admitted for 24 hours observation.

Being among patient meeting the criteria to enter in my study, I need your consent to participate. The advantage of your participation; is close follow-up which the doctor will offer you in your first 24 hours of admission and in any case of deterioration, the doctor will be there to help you to find out what is the cause of deterioration; and aggressive measure taken towards the identified problem.

Also the doctor will offer a discharge counseling on danger symptoms and signs of deterioration while at home.

Thanks for understanding my request.

Do you agree to participate in my study Yes/ No

……..

Patient signature.

6.3. QUESTIONNAIRE

PART 1 AT E.M.D

Registration number……

Time……

Age……..

Vital signs.;BP…PR….RR….T…

1.Sex…M/F….

2. Glasgow coma score

a) 15

b) 14

c) 13

3.Pupillary dilatation

a)Yes

b)No

4. History of LOC

a)Yes

b) No

5. History of vomiting

a)Yes

b)No

6. History of Seizures

a)Yes

b) No

7. History of headache

a) Mild Headache

b) Severe headache

c) No

8. Did Brain CT scan done?

a)Yes

b)No

9.If yes in part 7 above,

a)Normal

b) Abnormal

10.Presence of long bones fracture eg.fracture of femur

a) Yes

b)No

11.Ear ,Nose discharge ie.either fluid or blood

a)Yes

b)No

12.Presence of skull fracture,; either depressed or linear

a)Yes

b)No

13. Cause of injury

a) MTC

b) Fall

c) Assault

d)Others

PART 2.AFTER 24HOURS.

Vital signs.;BP….PR….RR….T….

Tme……

14. Glasgow coma score

a)15

b)14

c) 13

15. History of LOC in the ward

a)Yes

b)No

16. History of Seizures in the ward

a)Yes

b)No

17.History of vomiting in the ward.

a)Yes

b) No

18. Headache history

a)Mild Headache

b) Severe headache

c) No headache

19. Doctors remarks.,

a) Discharge home

b) More observation

c) Deteriorated

20.If deteriorated, is Brain CT Scan done?

a)Yes

b) No

21. Diagnosis after CT scan

a)Epidural haematoma

b) Subdural haematoma

c) subarachnoid hemorrhage

d) Brain edema/contusion

e) Normal CT,.ie.uknown