TRAUMATIC BRAIN INJURY IN DAR ES SALAAM

PATTERN AND OUTCOME IN THE DEPARTMENT OF ORTHOPAEDICS AND TRAUMA, NEUROSURGICAL UNIT

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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 Science a dissertation entitled:

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Declaration

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DEDICATION

To all victims of Traumatic Brain Injury

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LIST OF ABREVIATIONS

- ICP Intracranial cerebral pressure
- CPP Cerebral perfusion pressure
- CT Computer tomography
- ED Emergency department
- EDH Epidural haematoma
- EMS Emergency medical services
- GCS Glasgow coma scale
- GOS Glasgow outcome scale
- ICH Intracerebral haemorrhage
- ICP Intracranial pressure
- ICU Intensive care unit
- MAP Mean arterial pressure
- MUHAS Muhimbili University of Health and Allied Sciences
- MRI Magnetic resonance imaging
- RTA Road traffic accidents
- SAH Subarachnoid haemorrhage
- SDH Subdural haematoma
- TBI Traumatic brain injury

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ABSTRACT

Backgrond: Traumatic head injury is an important cause of mortality worldwide. Yet there are very few hospital or population based studies in Tanzania to initiate preventive measures. The objective of this study was to analysis the external cause, demographic pattern and outcome of potentially traumatic brain injured referred to the Institute of Orthopedics/Trauma Neurosurgical Unit-Muhimbili National Hospital in Tanzania.

Method: The study was based on cases of head injuries referred to the Institute of Orthopedics/Trauma-Neurosurgical Unit, in a period of twelve months, years 2003/ 2004.

Result: There were 489 cases whose ages ranged between 1and 85 years (mean=28.95 years). The average age of females (26.57 years, range: 1-85 years) being less than that of males (30.35 years, range: 2-78 years). There were 143 females and 333 males with missing data on 21 subjects making F:M ratio of 1:2. There was a strong association (p-0.001) between sex and type of Traumatic head injury .Majority of the head injuries (43.5%) were experienced by people who were below 30 years of age. The age-group 20-40 years (47.4%) had the highest number of head injuries. There was no significant relation ($p>0.05$) between age and the type of Traumatic head injury. Mild injuries (57.3%) were more frequently reported followed by severe head injuries (26.4%), moderate injuries (13.9%) while 2.7% of subjects had 'missing' data. Road Traffic associated brain injury presented a larger number of injuries. Pedestrian and cyclist related injuries (46.8%), assault (18%) and RTA (15.7%). Falls (8.6%), strucked (7.0%) whereas subjects whose data was missing accounted for 3.9%. Of the 489 patients, only 454(92.8%) had valid outcomes recorded, 162 (35.7%) died. There was a significant relationship between type of Traumatic head injury, hospital stay and outcome

Interpretation: The present study shows that TBI in Dar-es-Salaam Tanzania are motor/traffic related and males young adult males, are especially predominantly prone to fatal head injuries. However, the profile highlights the need for further research on potentially traumatic brain injury (Traumatic head injury)

1. INTRODUCTION

Traumatic brain injury (TBI) is a global health problem. Although the incidence varies geographically, TBIs are among the most likely types of injury to cause death or permanent disability in young people and thus causing considerable demand on economic resources and Health services [1, 2]

TBIs are frequently referred to as the "silent epidemic" since problems such as those of thinking and memory are often not visible and because the general public awareness of TBI is seemingly also limited.

TBI can be classified according to a combination of causes, mechanisms and consequences. Nevertheless, inconsistencies in definition and classification of TBI, along with discrepancies in data collection, have made the epidemiology of TBI difficult to describe and compare accurately and internationally [1].

The incidence of head and spinal injury is on the rise in developing countries including sub-Saharan Africa [2, 3]. The increase is partly due to young people migrating to large cities, rapid growth of motorized transport and to the expansion of industrial production without adequate safety precautions. The burden and pattern of TBI in Africa and other developing countries have not been well studied and are therefore poorly known.[1, 3]

Although, doctors' estimates of prognosis are still often unduly optimistic, unnecessarily pessimistic, or inappropriately ambiguous [4, 5, 6, 7] the management of TBI has evolved dramatically in the last twenty years, becoming increasingly governed by protocols and clinical pathway guidelines. [8, 9]. These have made it possible to be much more confident about what is likely to happen, and to consider prognosis in terms of probabilities rather than prophecies. However, it is likely that many patients with TBI are not treated according to current treatment principles in poor resource settings. [8, 9]

It is reported that TBI accounts for about one quarter to one third of all accidental deaths and for two thirds of trauma deaths in hospital. They are also the main cause of lifelong disability after trauma. [1, 10]

Given the impact on morbidity, mortality and economic importance of TBI, there is a need to acquire more epidemiological information on the management and outcome of TBI of all grades

of severity [11]. However, reliable statistics are difficult to extract from routinely collected data. The best sources are therefore research surveys, of which there have been several from different countries in the past 10-20 years. [2, 16, 12]

One problem with these surveys is the variation in the definition of TBI and its severity. Furthermore,incidence studies in developing countries present epidemiologists with unique methodological challenges. These challenges arise from archival inadequacies, hospital overcrowding, high violence rates, and limited research funding that necessitates low cost epidemiology. [13]

A recent study conducted in Tanzania has revealed that injuries rank as the third major leading cause of death among the adult population only after tuberculosis and HIV/AIDS [14].The aim of this study however, was aimed to describe epidemiology of injuries among inpatient in four hospitals in Dar es Salaam.

Critical to any injury prevention strategy is a reliable surveillance system. Such a system may for instance be based on hospital registration of TBI. While community surveys may give a more complete picture, the estimates of severity are imprecise, and information on clinical care is lacking.

Dar es Salaam, the commercial capital of Tanzania is served by several district hospitals, health centers, and private hospitals. Muhimbili Orthopaedic Institute (MOI) is the only centre which offers specialized neurosurgical care in the city and indeed in the country. The registry at MOI is a vital source of information on the pattern of injuries and quality of clinical management

2. STATEMENT OF THE PROBLEM

TBI is and will remain a formidable challenge and major cause of hospital admission. In a study to analyze patients admitted to the Intensive Care Unit (ICU) of Muhimbili National Hospital (MNH) Dar es Salaam over one year in 1997 there were 330 admissions of whom 21.6% were due to TBI. TBI was also the leading cause of in-unit mortality accounting for 44.2% of ICU mortality, while the hospital mortality rate during this time was 7.8%. Generally ICU records reflect the magnitude of mortality and morbidity patterns in the patient population at large. [15]

In the MOI annual report for 2002/2003 TBI accounted for 16% of total admissions. The male: female ratio was 2:1. The mean crude death rate (CDR) was 4.2% and the leading cause of death was TBI. (Tables 1 and 2).

Table 1- Death rate September – October 2002			
Month	Admission	Death	Rate
September	547	20	3.7
October	535	25	4.7
Total	1082	45	4.2

Table 2 Causes of Death September – October 2002		
TBI	25	
Poly Trauma	22	
Spinal injury	3	
Isolated injuries	3	
Infections	3	
Others i.e. tumor, DM	4	

It is projected that in the next 2 decades the risk of traumatic brain injuries will be on the increase due to rapid urbanization and motorization in developing countries (WHO). [16]

Statistics obtained from the Ministry of Home Affairs (Tanzania Traffic Police) indicate that there is an alarming increase in the umber of traffic accidents annually. In the period from 1977 to 2002 there was an increase of 141.6% of road traffic accidents (RTA) and a rise of RTA related injuries of 150.4%.and RTA-related death by 127.1%.

The magnitude and consequence of TBI and its contribution to the disease burden in the country is not well documented [11-17, 18-20]. Traumatic Brain Injuries resulting in admission to hospital provide an important basis for determining priorities, emerging issues and trends in TBI. To my knowledge no study has specifically addressed this problem.

The aim of this study was to evaluate available hospital records for the purpose of describing the epidemiology of TBI in a hospital setting among inpatients referred to a Neurosurgical Unit in Dar es Salaam.

This study is an attempt to address this potentially growing problem. A hospital based study analyzing the pattern and outcome associated with TBI have a potential to making a contribution in the management and prevention initiative.

3. REVIEW OF LITERATURE

3.1 Epidemiology

Traumatic brain injury is a non-degenerative, non-congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions with an associated diminished or altered state of consciousness.

The definition of TBI has not been consistent and tends to vary according to specialties and circumstances. Often, the term head injury is used synonymously with TBI, which may or may not be associated with neurological deficits. The definition has also been problematic on account of variations in exclusion and inclusion criteria. [10]

It is generally very difficult to interpret international data on the epidemiology of TBI. Even the definition of TBI remains unclear in almost all research papers [21]. In many reports, it is not clear whether the authors intend to focus on pure TBI, or on patients with skull and brain injuries [22-25]. Some reports deal only with brain injuries due to specific external causes, such as vehicular traffic accidents, while others fail to mention the external causes of the brain injury at all [15]

3.2 Age and Sex

Men are approximately twice as likely as women to sustain TBI [26]. This ratio approaches parity as age increases because of the increased likelihood of TBI caused by falls, for which members of both sexes have similar risks in later life [15,26-]. Several studies have identified age as a strong prognostic indicator following injury to the brain [27-30]. The male-to-female mortality ratio for TBI is 3.4:1. [31]

There are discrepancies in the literature when defining the age point where prognosis significantly worsens.[15,27-, 29,32-346] Several large pediatric TBI series have reported that children have a lower mortality than adults, while others report that the primary mortality rate does not differ between children and adults. Additionally, some investigations reported better outcomes below the age range of 40-50 years. [34, 35-37] while other studies reported outcome as a continuous function of age without threshold values. [29, 21,40-] In general age distribution shows that there is an increase in the absolute number of fatal injuries with advancing age and the critical age threshold for worsening prognosis appears to be above 60 in a review of Class I and II studies in brain injured patients. [36,41, 42].

In the developed nations the trend over 10 years (from 1989 to 2000) shows that fatal TBI in the age group 15-40 years are decreasing, with the greatest decrease in the group of 20–25 years. Classification of the incidences shows that the mortality among older persons with intracranial injuries in1996 was highest in the group of over-65s. This was mostly caused by falls of all kinds. [10, 31,)]

3.3 Incidence and Mechanism of the Cause of TBI

Common causes of fatal injuries vary according to gender, age, race, and geographical location. While various mechanisms may cause TBI, the most common causes are road traffic accidents (e.g. collisions between vehicles, pedestrians struck by motor vehicles, bicycle accidents), falls, assaults, sports-related injuries, and penetrating trauma.

The incidence varies from 67 to 317 per 100,000 and mortality rates are in the range of near 1% for minor injury, 18% for mild injury, and 48% for severe TBI. It is the main cause of death and disability in people under 40 years old. [(2)]

In the early 1980s, two large-scale epidemiological investigations for neurological trauma were conducted in the People's Republic of China [(43).] In the six-city study, the most common cause of traumatic brain injury was vehicular accidents (31.7%), followed by assaults (23.8%), falls (21.8%), stumbles (15.4%), and others (7.3%). In rural and minority areas, the most common causes was falls (33.5%), followed by vehicular accidents (33.0%), stumbles (15.8%), assaults (12.0%), and others (7.0%) in that order. Firearm injuries were less common both in the six cities (1.3%) and the rural and minority areas in the 21 provinces (1.4%).

In the United States, an estimated 1.5 million people sustain a TBI each year. This is 8 times the number of people diagnosed with breast cancer and 34 times the number of new cases of HIV/AIDS. (CDC) It is estimated that 50,000 people die from TBI, which accounts for one-third of all injury deaths (CDC). During 1979-1992, TBI-related death rates declined by 22%, from 24.6 to 19.3 deaths/100,000 population. [(44-46)]

In a report describing the epidemiology and trends in TBI-related mortality during 1989-1998, an annual average of 53,288 deaths (range: 51,848--54,501) among U.S. residents were associated with TBI. TBI-related death rates declined 11.4%, from 21.9 to 19.4/100,000 population. The major causes of TBI-related deaths were firearm-related (40%), motor-vehicle--related (34%), and fall-related (10%). The leading causes of TBI-related deaths differed among age groups.

Among youths aged upto19 years, motor-vehicle-related TBIs were the leading cause; among persons aged 20-74 years, firearm-related TBIs were the leading cause; and among persons aged >75 years, fall-related TBIs were the leading cause. Comparing rates in 1989 with rates in 1998, motor-vehicle-related causes declined by 22%; the majority of this decline occurred during the first 5 years of the period. During 1989-1998, firearm-related TBI-related deaths declined by 14%. In contrast, fall-related TBI-related death rates increased by 25% during the period. [46].

Epidemiologic Data of traumatic brain injury in Johannesburg (1991) indicated an overall annual incidence of TBI of 316 per 100,000. Data for Africans and Whites showed a marked contrast. Among Africans, the incidence was 355/100,000, with a male-female ratio of 40:1 and 419/100,000 for men aged 15-24. Unfortunately no comparison data was shown for the white counterparts. However, the overall incidence of fatal TBI was 80/100,000, with a case fatality ratio of 0.20. Interpersonal violence accounted for 51% of nonfatal TBI among Africans, as against 10% for Whites, while motor vehicular accidents caused 27% of African nonfatal TBI and 63% among Whites. [13]

Reviews of statewide hospital separations' summaries and medical record data from a major teaching hospital were conducted to describe the epidemiology of traumatic brain injury (TBI) in South Australia (SA), and to document the demographics of the population affected and the nature of their injuries. The results indicate that SA experiences a high incidence of TBI. At 322 per 1000,000 head of population annually, it exceeds studies (with comparable methodologies) in communities in the United States and Europe. The causes, nature and severity of the injuries were similar to those found in the international literature, as were the profiles of the population most at risk. Specifically, young males living in the country and working in manual trades showed the highest incidence, and were most likely to have sustained TBI whilst driving a motor vehicle. [47]

In Germany with a population of 82 million people there had been 279,029 TBI admitted to hospital in 1996. TBI caused the deaths of 9415 patients in 1996, which accounted for a mortality of 11.5 per 100,000 inhabitants. Of all fatal cases, however, 68.4% died before reaching hospital. [51] The age distribution showed that there was an increase in the absolute number of fatal injuries with advancing age [12].

The Japanese Data Bank Committee for Traumatic Brain Injury began its formal activity in 1998. Traffic accidents on the road, injured victims and all other losses due to these accidents

have been increasing since around 1980. Nevertheless, the number of deaths due to traffic accidents has been gradually declining over the last six years. In this study, in 23% of traffic accidents and 12% of non- traffic accidents, consumption of alcohol led indirectly to TBI. [48]

Traumatic brain injuries (TBIs) are a leading cause of morbidity, mortality, disability and socioeconomic losses in India and other developing countries .[18, 19, 20, 22 48, 50, 54] It is estimated that nearly 1.5 to 2 million persons are injured and 1 million succumb to death every year in India. Road traffic injuries are the leading cause (60%) of TBIs followed by falls (20-25%) and violence (10%). The influence of alcohol is known to be present among 15%-20% of TBIs at the time of injury. [50]

In Pakistan a study was conducted from July 1, 1995 to June 30, 1999 to determine the socio-demographic characteristics, morbidity, and mortality of patients with head and spinal injury admitted to various neurosurgical centers. A total of 260,000 patients were admitted with TBI over a 4-year period. The majority of patients presented during the second decade (i.e., 33.2%) followed by first and third decade. There were 195,000 (75%) males, and 65,000 (25%) females with a ratio of 3:1 Road traffic accident was the commonest cause of TBI. Of these, 780 patients (29%) had complete spinal cord injury. [51]

In a study of TBI in Salvador city, 555 medical files from victims with TBI attended at the General Hospital of Bahia during 2001 showed 82.9% of all victims were male and 17.1% female. The most frequent age group was 21 to 30 years; the most relevant cause of TBI was traffic accidents (40.7%), followed by physical violence with or without weapons (25.4%) and falls (24%). [52]

A study was done to highlight the pattern and distribution of trauma deaths in a Nigerian teaching hospital. Prospective data collection was done for 24 months. There were 129 deaths but 84 (65.1%) had sufficient data for trauma scoring. Male: Female ratio was 60:24 = 2.5:1. The age range was 2-95 years, mean 36.8 +/- 15.2 years for males and 45.5 +/- 23.0 years for females. Three quarters of the deaths (75.0%) were in patients involved in road traffic accidents (RTA) followed by violent trauma (10.7%), falls 9.5% and burns 4.8%. The trauma deaths include TBI (31.0%), multiple injuries (30.0%), fractures (13.1%), cervical spine injury (10.7%), gunshot injuries (8.3%), burns (4.8%) and others (5.0%). Sixty-two patients (73.8%) reached the hospital within 6 hours of the injury. The pattern and distribution of trauma revealed that a

typical trauma death is a male aged below 40 years, who is a trader/businessman involved in RTA or violent trauma.[53]

3.4 Classification of TBI

Head trauma can be classified according to a combination of causes, mechanisms and consequences, both structural and functional. Classification by causes is of epidemiological interest or mechanistically into blunt or penetrating injury.

(i). Skull fractures (linear, depressed, basilar).

(ii) Focal injuries. Contusions (coup, contrecoup, intermediate), Hematomas (extradural, subdural, intracerebral)

(iii). Diffuse injuries Concussion (mild, classical), Diffuse axonal injury (mild, moderate, severe).

3.4.1 Mechanisms of injury

The mechanism of how a force acts upon the head determines which of these injuries occurs following an accident.

Static Loading. These are Forces applied gradually over 200 ms or longer causing a squeezing effect which will lead to multiple skull fractures. After the skull has been crushed, the brain is affected by injury. These mechanisms in general are uncommon form of injury.

Dynamic Loading The injury force acts in less than 200 ms, often less than 50 ms.

1. **Impulsive Loading:** The brain is injured by inertial forces that result from acceleration, deceleration. Which occurs when the head is set into motion or when the moving head is stopped without the head being struck, e.g. child shaken by the shoulders, blow to the body so that head moves.

2. **Impact Loading:** This a frequent type of dynamic loading injury characterized by skull bending and shock waves. The inertial effects can be minimal when the head is prevented from moving.

Two main mechanisms are responsible for damaging the brain. These are referred to as inertial or contact mechanisms

Strain: This is the amount of **deformation** that the tissue undergoes as a result of mechanical loading. Biological tissues withstand strain better if they are deformed slowly rather than

quickly. There are three types of strain; compression, tension and shear. Brain damage is caused mostly by compression and shear strains.

Inertial injuries are commonly called "acceleration" or "deceleration" injuries. The brain is damaged by one or both of two mechanisms:

The type of damage produced depends on the type of acceleration, the amount of acceleration, the direction of head motion, and the duration of the acceleration. Three types of acceleration are described.

(i) Translational. Brain center of gravity (pineal) moves in straight line.

(ii) Rotational. Movement about the center of gravity (rarely seen).

(iii) Angular. Combined translational and rotational (commonest).

Acceleration and deacceleration results in compressive, tensile and shear strains that cause structural damage.

Differential **acceleration** of the skull and the brain, cause a strain on the subdural bridging veins. Laceration of these veins causes most subdural hematomas. The movement of brain away from the skull can also create low pressure (tensile strains) and cause contrecoup injuries.

Intraparenchymal strains from acceleration will cause **diffuse axonal injury**

Contact Injuries: A forces applied to the head in the absence of head motion. These injuries rarely occur in pure form. he impact usually sets the head in motion . A number of **contact phenomena** occur at site of impact, the degree to which is dependent on the size of the impacting device and the magnitude of the force of impact.

(i) Immediately beneath point of impact there is a localized **skull deformation** with in bending of the skull surrounded by out bending of the skull peripheral to the impact site.

(ii) If skull deformation is significant, then **penetration**, **fracture** or **perforation** of skull can occur.

(iii) **Shock waves** are also created which propagate through the skull and the brain and can cause local brain distortion and injury.

3.5 Unconsciousness

Loss of consciousness is the clinical hallmark of TBI and reflects both the amount and distribution of brain damage.

Unconsciousness is likely to be inactivation of a sufficient proportion of the cerebral cortex, but the precise mechanisms remain incompletely understood and debated both at the level of the whole brain and its component systems and at the level of the individual nerve cell and its connections. [54-60].

The term "concussion" is applied to an instant, but also transient, alteration in consciousness immediately after the injury. For many authorities, loss of consciousness is essential. Although there is no agreement on how long this should be, most concepts imply duration measured in minutes [61].

"Coma" is a more profound impairment of consciousness. Teasdale and Jennett defined coma as "not opening eyes, not uttering words, and not obeying commands." [62] This definition was used as a starting criterion to identify a severe TBI in a collaborative United Kingdom, Dutch, and American study. [63]

Post-traumatic amnesia is an index of severity of TBI useful when assessment has to be retrospective. Retrograde amnesia concerns events prior to the injury; it is almost always very much shorter than post-traumatic amnesia and is much less clearly related to severity. [64, 65]

The most widely used measure in assessing the severity of HI is the Glasgow Coma Scale introduced by Teasdale and Jennett in 1974. The patient's best motor, verbal, and eye opening responses determine the Glasgow Coma Scale. The severity of TBI according to the GCS score is as follows: Severe TBI = 8 or less, Moderate TBI = 9-12, Mild TBI = 13-15. (See Appendix I)

The various clinical, pathological, radiological, and neurophysiologic observations give rise to the hypothesis that the instantaneous loss of consciousness in a TBI represents a state of transient, but sometimes profound, inhibition of arousal mechanisms within the hypothalamic and diencephalic brain stem regions. This has been likened to a form of suprareticular "shock" similar to the loss of function in the distal segment of the spinal cord after transaction. [66-68]

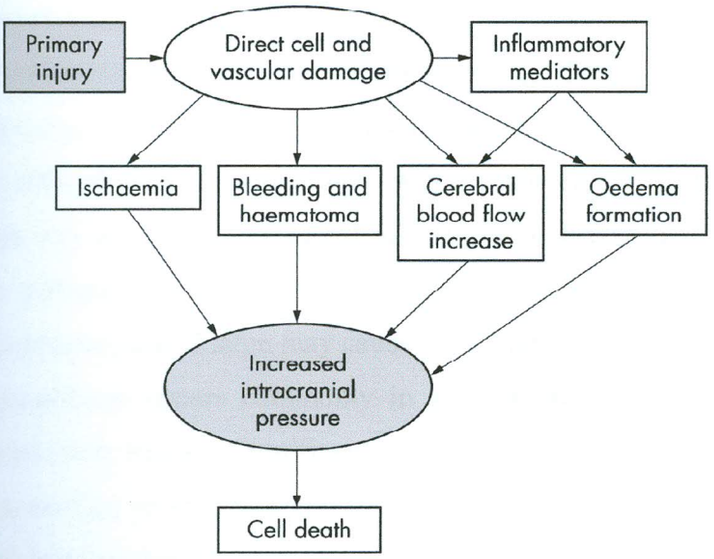
3.6 Pathophysiology

3.6.1 Primary TBI

By definition, "primary" TBI occurs at impact and may involve neural or vascular elements of the brain. Injury to the scalp and cranium may be included in this category, as well as parenchymal damage inflicted by stab and missile wounds of the head. Focal injuries impact morbidity and mortality based on their location, size, and overall progression.

The fundamental processes occurring at the molecular and cellular level following brain injury culminate in cell death. [69] These processes include the release of excitotoxic quantities of the amino acids, glutamate and aspartate, production of free radicals, and increased production of lactate and hydrogen ions. One of the final common pathways of these processes is the entry of calcium ions into cells, which results in cell swelling. This swelling, within the confines of the rigid cranium, results in an increase in intracranial pressure (ICP), reduction in cerebral perfusion pressure (CPP), cerebral ischaemia and reduced delivery of oxygen to the tissues. This further provokes acidosis, and glutamate and free radical release to potentiating the above cycle.

Figure 1 Processes in traumatic brain injury.



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The recognition that diffuse axonal injury is responsible for acute loss of consciousness and for severe late sequelae in the human head has stimulated much research aimed at discovering the mechanisms responsible at the level of the individual axon and neuron. [66, 70]. Diffuse axonal injury is caused by shearing forces affecting axons that traverse large areas of the brainstem, leading to dysfunction of the reticular activating system. [70, 71]. It is believed that axons are not torn at the moment of injury but rather undergo sequential, focal changes that lead to swelling and disconnection over multiple hours after injury. As a consequence of this disconnection, the

downstream disconnected fibers degenerate leading to diffuse de-afferentation of target sites. [71-773]

Diffusion-weighted MRI may identify diffuse axonal injury. [74] Characteristic lesions include, Hemorrhage or necrosis of the corpus callosum, hemorrhage or necrosis of the dorsolateral quadrant of rostral pons, focal contusions in adjacent structures (fornix, cingulate gyri, septum pellucidum, caudate nuclei, dorsal thalamus).

Epidural hematomas are located outside the dura but within the skull, and are typically biconvex or lenticular in shape. They are most often located in the temporal or temporoparietal region, and often result from laceration of the middle meningeal artery caused by a fracture. In many cases, but not always, there is loss of consciousness followed by a period of lucency, followed by neurological deterioration.

Subdural haematoma result most frequently from tearing of a bridging vein between the cerebral cortex and a draining venous sinus. The force of impact is often transmitted to the brain itself. In approximately 80% of subdural hematomas, it is the underlying brain injury that determines the patient's course and outcome. A subdural haematoma will appear on a CT scan as a crescent-shaped blood collection between the brain and the dura. There is frequently an adjacent parenchymal contusion, and if large may cause a midline shift.

Intracranial hemorrhage occurs commonly in association with moderate and severe TBI and usually produces mass lesions. Significant SAH is always associated with cortical contusions. Veins, venules, arteries or arterioles can be lacerated. SAH may block arachnoid villus causing communicating hydrocephalus.

Brain Contusions may be classified into 6 types (coup, contrecoup, intermediate coup, gliding, herniation, and fracture contusions). The distinction between coup and contrecoup is becoming less clear.

Contusions and Lacerations results from bruised neural parenchyma. Frontal and temporal contusions are the predominant ones regardless of the site of impact. Intermediate coup implies an injury within the neural parenchyma between the impact site and the opposite side of the brain. Gliding contusions occur at the vertex of the brain during de-acceleration injuries. Herniation contusions are produced by sudden forcing of a portion of the brain against a rigid opening, most often the incisura of the tentorium. These contusions are seen along the medial portion of the temporal lobes (uncus, hippocampus).

The majority of post traumatic Intraparenchymal Hemorrhages (ICH) s lies in frontal and temporal lobes. They can be multiple and are seen in about 10% of severe TBI often with coexisting cortical contusions and lacerations. Ethanol-induced coagulopathies probably help contribute to secondary ICH following trauma.

Direct Brainstem Trauma injuries occur most frequently in association with lesions elsewhere in the brain.

Direct Cranial Nerve Injuries occur commonly of the olfactory bulbs (I) and tracts. Traumatic lesions of the optic nerve (II) often involve hypothalamic injury. Avulsion of IIIrd nerve with mesencephalic pontine laceration. Injuries to IV, V, VI, VII, and VIII usually follow basal skull fractures

Direct vascular Trauma: Veins, arteries and dural sinuses may be lacerated, avulsed, or thrombosed leading Traumatic carotid-cavernous fistula, traumatic aneurysms or false aneurysms. Thrombosis of major intracranial vessels follows often trivial trauma direct lacerations of cerebral arteries and veins occurs and usually causes SDH.

Direct Hypothalamic Lesions; with severe TBI will demonstrate structural abnormalities in the hypothalamus and pituitary. Syndromes of Diabetes Insipidus or hypopituitarism do occur in closed TBI. Hypothalamic and pituitary dysfunction may follow secondary to brain shifts

3.6.2 Secondary TBI

"Secondary" injury is the result of physiological processes that are initiated following impact or soon thereafter. They process evolve and often lead to further damage of nervous tissue, leading to clinical deterioration. Conditions known to cause secondary brain injury can be divided into extra cranial (systemic) and intracranial insults.

Hypotension (Systolic < 90mmHg) and hypoxia (PaO2 < 60 mmHg) are the main and must potent cause of secondary brain damage. They are the avoidable secondary brain damage in TBI [2, 3, 12, 14] other systemic insults include hyper/hypoglycemia, electrolyte abnormality, coagulopathy, anaemia, hyperthermia, infection, seizures and intracranial surgical mass lesion. Focal or global brain swelling may lead to raised intracranial pressure and aggravate brain injury.

3.7 Management

3.7.1 Pre-Hospital Management

Emergency Medical Service (EMS) provider is the first health care provider at the scene of injury. They are the first critical designed to provide pre-hospital assessment and treatment. EMS is continues and completed once there is stability from cardiopulmonary standpoint.

There is no current treatment of primary TBI except prevention; the mainstay of management is on the management of secondary brain injury. The rationale for each treatment of TBI is based on the concept of the **Monro-Kellie doctrine**. (Appendix III)

Primary brain injuries sustained at the time of trauma cannot be reversed. The goal is to prevent secondary neuronal injury by preventing hypoxia and hypotension and recognize and treat surgical correctable extra cranial or intra cranial lesion. Other concomitant injuries should be attended.

Primary and secondary traumatic surveys of are done in all injured patient according to the advanced trauma life support (ATLS) guidelines. The first priority in any injured patient is cervical stabilization, establish an adequate A-airway, ensure adequate B-breathing (ventilation), and gain venous access to initiate volume resuscitation (C-circulation). Hypoxia and hypotension the most important source of secondary brain insult and should be cautiously but aggressively prevented. Patient with GCS of 8 or less should be intubated. Finally determine the level of consciousness and examination of pupils D-disability. An attempt should be made to classify severity of TBI, history of the mechanism preceding the trauma. In addition neurological examination should include: Brain stem examination (pupil examination size, light reaction ocular movement, corneal and gag reflex.). Reflexes and motor-sensory examination.

The severity of TBI according to the GCS score is as follows: Severe TBI = 8 or less, Moderate TBI = 9-12, Mild TBI = 13-15. (Appendix I)

Patients who are intubated are unable to speak. They are evaluated only with eye opening and motor scores, and the suffix **T** is added to their score to indicate that the patient is intubated. In intubated patients, the maximal GCS score is 10T and the minimum score is 2T.

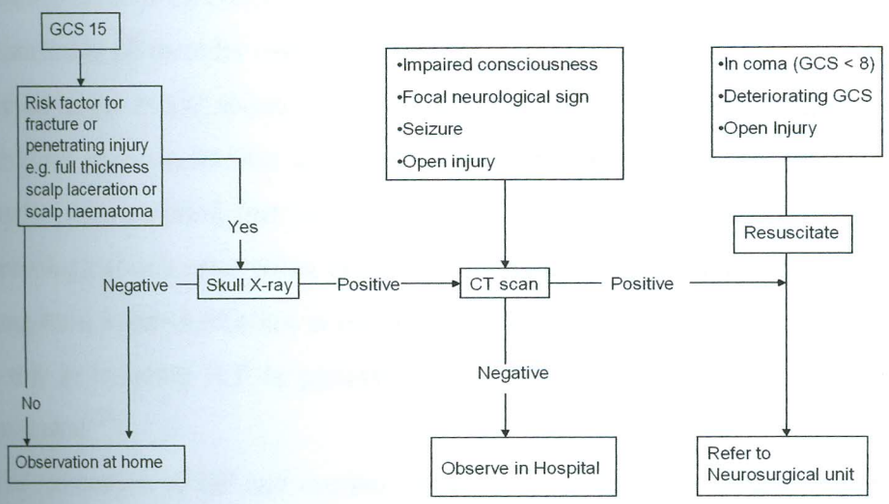
3.7.2 Early Hospital Management

The goals are to maintain, patient Airway, fluid resuscitation, and immobilize spinal injuries, especially cervical injuries; assessment of level of consciousness and attend and exclude extra cranial injuries followed by referral to a trauma center. (Appendix IV)

3.8 Diagnostic Studies

Skull radiography once an important part of TBI evaluation has been replaced by CT-scan. And it is a diagnostic study of choice in the evaluation of TBI, because of its rapid acquisition time. Unfortunately, this form of investigation is expensive and not universally available.

Indications for Imaging



Although MRI provides extraordinary anatomic detail, it is not commonly used to evaluate acute TBI because of its long acquisition times and the difficulty in obtaining MRIs in persons who are critically ill. MRI is superior to CT scan for helping identify diffuse axonal injury (DAI) and small intra-parenchyma contusions.

Patient blood is taken for Full Picture, Electolytes, glucose, Urea & creatinine, blood gases, alcohol and any drug levels.

3.9 Indication for Neurosurgical Consultation

- Abnormal CT scan
- Patient fulfils criteria for CT scan but this cannot be done within an appropriate period of time
- Persisting coma (GCS ≤ 8) after initial resuscitation
- Definite or suspected penetrating TBI
- Confusion persisting more than 4 hours
- Deterioration in level of consciousness after admission
- Focal neurological sign
- Seizure without full recovery
- Depressed skull fracture

3.10 ICU care

Patients are admitted in ICU to establish physiological monitoring and further management. Management of elevated ICP involves using a combination of treatment. Volume resuscitation is essential in all patients with acute TBI.

Initial target PCO2 should be 35 to 40 mm Hg. [75,76] hyperventilation reduces ICP by causing cerebral vasoconstriction and a subsequent reduction in CBF.Published randomized clinical study demonstrated that hyperventilation after TBI is associated with a significantly adverse neurological outcome when compared to patients kept normocapnic [77] Based on this data, long-term hyperventilation is no longer recommended.[76] Short-term hyperventilation does have a role in reducing ICP in patients who are rapidly deteriorating before other measures can be instituted.[78].

The restoration of BP and normal circulating volume is of utmost importance. According to the Brain Trauma Foundation guidelines, a mean arterial pressure of 90 mm Hg should be targeted; this was chosen based on attaining cerebral perfusion pressures > 70 mm Hg. [75].

If the ICP remains > 20 mm Hg, despite adequate sedation and elevation of the head of the bed (to 30°), Hyper-osmolar agents currently in clinical use for traumatic brain injury (TBI) are mannitol and hypertonic saline (HS). Its beneficial effects on ICP, CPP, CBF, and brain metabolism, and its short-term beneficial effect on neurological outcome are widely accepted as a result of many mechanistic studies performed in humans and in animal models. [79-81, 82].There is still controversy regarding the exact mechanisms by which it exerts its beneficial effect, [83]

Manitol may be an immediate plasma expanding effect, which reduces the hematocrit, increases the deformability of erythrocytes, and thereby reduces blood viscosity, increases CBF, and increases cerebral oxygen delivery [79-81,82]. The dose used is 0.25 to 0.5g/kg administered every

2 to 6 h to increase the serum osmolarity to 310 to 320 mOsm/kg H_2O. Thus it has been recommended that mannitol should be administered as repeated boluses rather than as a continuous infusion [84]. Arterial hypotension, sepsis, nephrotoxic drugs, or preexisting renal disease place patients at increased risk for renal failure with hyper-osmotic therapy.[85,86] Relatively little is known regarding Hypertonic Saline Mechanism of action. The principal effect on ICP is possibly due to osmotic mobilization of water across the intact blood–brain barrier (BBB) which reduces cerebral water content. [82, 87]

The only reliable way to determine CPP and cerebral hypo-perfusion is to continuously monitor ICP and blood pressure. As with any invasive monitoring device, ICP monitoring has direct costs, uses medical personnel resources for insertion, maintenance, troubleshooting, and treatment, [80-82]). When a ventricular catheter is being used for ICP monitoring, CSF drainage should be used for ICP if is pathologically (>20) elevated.

Seizure Prophylaxis: Post traumatic seizures (PTS)s are classified as early, occurring within 7 days of injury, or late, occurring after 7 days following injury.[88,89] It is desirable to prevent both early and late PTS. The incidence of seizures following penetrating injuries is about 50% in patients followed for 15 years.[90] Certain r factors increase risk for developing PTS.[9,14] These risk factors include the following:

Glasgow Coma Scale (GCS) Score<10, cortical contusion, depressed skull fracture, subdural hematoma, epidural hematoma, intra-cerebral hematoma, Penetrating head wound, Seizure within 24 h of injury. The, use of prophylactic anticonvulsants for the prevention of late PTS is debatable [91]. If late PTS occurs, patients should be managed in accordance with standard approaches to patients with seizure. The agent commonly recommended is phenytoin, with a loading dose of 18 mg/kg and usual maintenance dose of 5 mg/kg/d following serum drug levels to a goal of 10 to 20 mg/L.

Hyponatremia, hypomagnesaemia lowers the seizure threshold and the later can exacerbate cerebral edema.

Hyponatremia is relatively common after TBI. The etiology of the hyponatremia is complex, with both cerebral salt wasting and the syndrome of inappropriate antidiuretic hormone being implicated.

Deep Vein Thrombosis Prophylaxis; Patients with severe TBI are at significant risk of developing venous thrombo-embolic events. However, no reliable data can support a

recommendation regarding when it is safe to begin pharmacological prophylaxis. Moreover, no recommendations can be made regarding medication choice or optimal dosing regimen for patients with severe TBI, based on the current evidence. [92]

To achieve full caloric replacement by 7 days, nutritional replacement is usually begun no later than 72 h after injury. Zinc is the only supplement studied in detail in a TBI population. One small pilot Class II study reported a better 24-h peak GCS motor score at two time points after injury (days 15 and 21) with zinc supplementation by the end of the first week. [91]

3.11 Outcome Assessment

There is a wide range of factors to account when making clinical decisions and assessing prognosis. In practice, relatively few features have been found to contain most of the prognostic information, These include the patient age, clinical indices indicating the severity of brain injury and the results of investigation (hypotension and hypoxia) and imaging studies, particularly intracranial pressure (ICP) and computed tomography (CT) scanning, which demonstrates the nature of brain injury and its effects on intracranial dynamics.[29, 30, 63]

Nevertheless there are still debates about the precise nature of their relationships and about exactly how the different features should be assessed, categorized, and most importantly utilized [42, 62,63].

Thornel et al made the following observation (below)

Table 3 :Outcome – Death & disability

	Outcome 1 year after head injury			
Initial Severity	**Dead or vegetative**	**Severe disability**	**Moderate disability**	**Good outcome**
Mild (GCS 13-15)	8 %	20 %	28 %	45 %
Moderate (GCS 9-12)	16 %	22 %	24 %	38 %
Severe (GCS 3-8)	38 %	29 %	19 %	14 %

Adapted from Thornhill et al, BMJ 2000; 320: 1631-1635.

4.0 RATIONALE OF THE STUDY

Traumatic TBI is a major cause of mortality and morbidity at the ED in MOI. The risk of traumatic brain injuries is on the rise due to the steady increase in urbanization, industrialization and motorization. There is lack of adequate information to reasonably address the problem. A hospital based study on TBI could contribute in the management of TBI in our setup and indeed in establishing a TBI program in the future.

5.0 METHODOLOGY

5.1 (a) Main Objectives

To study the demographic pattern and outcome of patients hospitalized with TBI at MOI, Neurosurgical Unit.

5.2 (b) Specific Objectives

To determine the demographic pattern and cause of TBI.

To determine the pattern of the severity of TBI, duration of hospital stay and outcome.

5.3 Patient and Methods

The study was a simple cross-sectional retrospective study of patient hospitalized due to traumatic TBI at the MOI from October 2003 to Sept. 2004.The study population included all patients admitted at MOI, Neurosurgical Unit with TBI.

Patients with co-morbid conditions of cardiovascular disease, birth brain traumas, congenital CNS abnormalities or metabolic disease were excluded.

The sample size was determined using a statistical formula derived from Epi info 6 software: Taking into account the finite population correction (i.e. N=1200), the minimum required sample size was 473

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

N = population size

n = sample size required

Z = percentage point corresponding to 5% level of significant

p = expected mortality rate in the population

d =margin of error

0n specifying the death rate p=4.8

Margin of error to be = 1.5%

A questionnaire was used and the following data was retrieved from the patient's files:

Hospital number, age, sex, date of admission, date of discharge. Level of consciousness (GCS), cause of injury, associated extra-cranial injuries, diagnostic studies and Outcome.

Registrars and residents in the department were involved in the management of patients. Data gathered was entered in a coded data sheet and analyzed using Statistical Package for Social Scientists (SPSS) version 10. Software.

The proposal was submitted to the hospital ethical management committee for consent/clearance and then forwarded to the college research and publications committee of MUHAS for authorization.

5.4 ETHICAL CONSIDERATIONS

The proposal was submitted to the hospital ethical management committee for consent/clearance and then forwarded to the college research and publications committee of MUCHS for authorization.

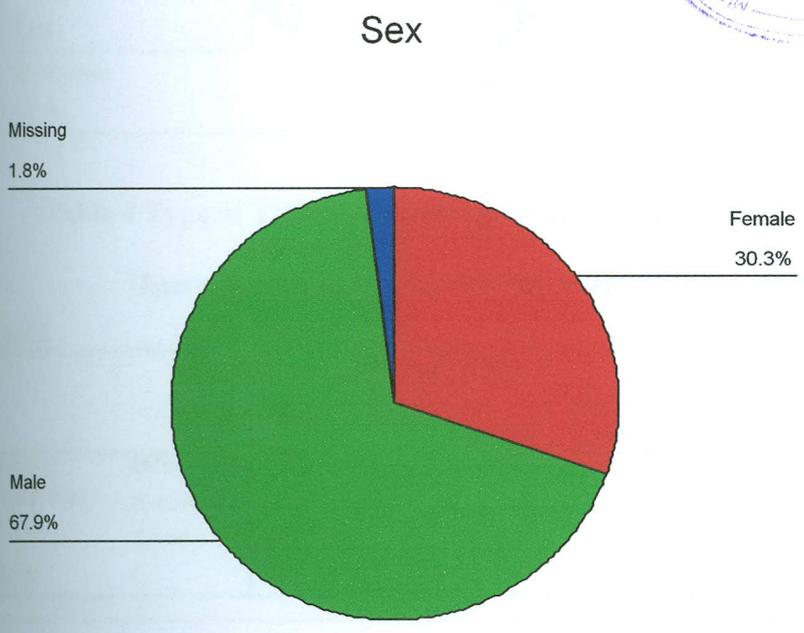
Relevant information was given to the patient, parent/guardian regarding the study for an informed consent. All relevant information gathered was treated with outmost confidentiality

5.5 RESULTS

5.5.1 Age and Sex

Fig. 2: Distribution of TBI by Sex

MUHIMBILI MEDICAL UNIVERSITY COLLEGE LIBRARY DSM.



Males were twice the number of females with a ratio 2:1 (fig.2). The ages ranged between 1 year and 85 years (mean=28.95 years). The average age of females [26.57] years being less than that of males (30.35 years) .From this data male have a more likelihood of sustaining TBI ($p<0.001$) (Appendix 5)

MUHIMBILI MEDICAL UNIVERSITY COLLEGE LIBRARY DSM.

Fig. 3: Distribution of TBI by age

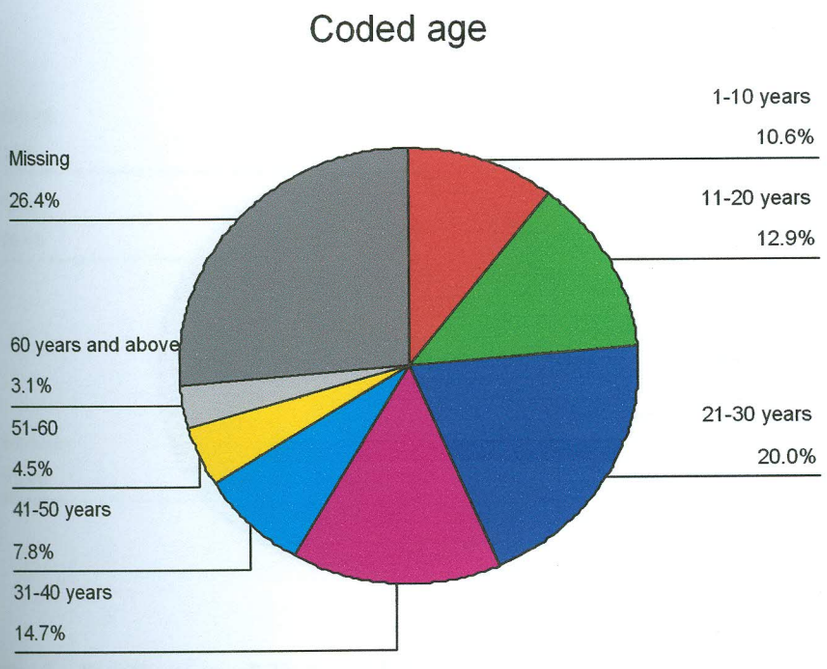


Table 4 Type of TBI by Age group cross tabulation

Type of head injury * Age groups Cross tabulation

Count

		Age groups			
		Below 20 years	20-40 years	Above 40 years	Total
Type of Head injury	Mild	80	109	45	234
	Moderate	15	18	16	49
	Severe	15	39	13	67
Total		110	166	74	350

The majority of the TBI (43.5%) occurred in young people (<40 years) with peak overall age group at 21-30(20%) years. There were significant fewer cases of TBI above the age of 50 years (table 4). There were 139(26.4%) cases whose age data was missing since p-value is greater than 0.05 and concluded that there is no association between Type of Head Injury and Age for this data.

5.5.2 Type of TBI

Fig. 4: Distribution by type TBI

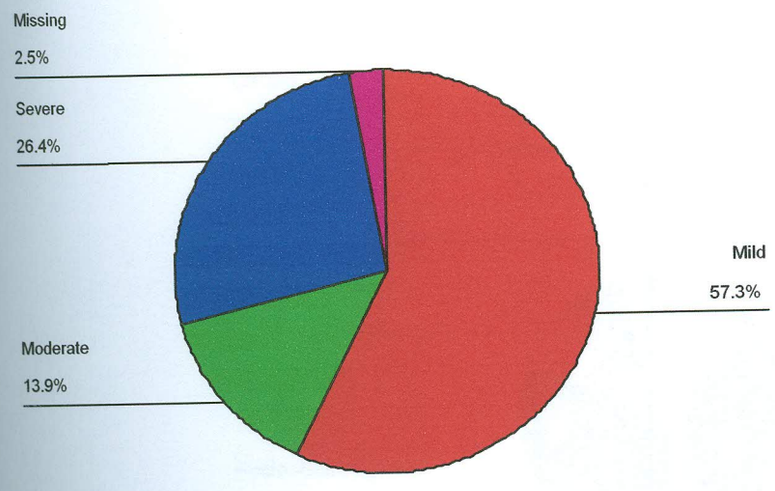


Table 5: distribution of Type of TBI by Sex Cross tabulation

Type of head injury * Sex Crosstabulation

Count

		Sex		
		Female	Male	Total
Type of head injury	Mild	104	175	279
	Moderate	12	56	68
	Severe	27	102	129
Total		143	333	476

Mild injuries (57.3%) occurred more frequently followed by severe TBI (26.4%) and moderate TBI. Aanalysis by Chi-Square test give a *P*-value of 0.001 which is clearly less than 0.05 indicating that there is a very significant relationship between sex and type of TBI. From our data, males are more likely to have one of the types of TBI than the females. No association could be established between type of TBI and age. ($p>0.05$) (Table 4)

5.5.3 Causes of TBI and associted factors

Fig.5: Causes of TBI

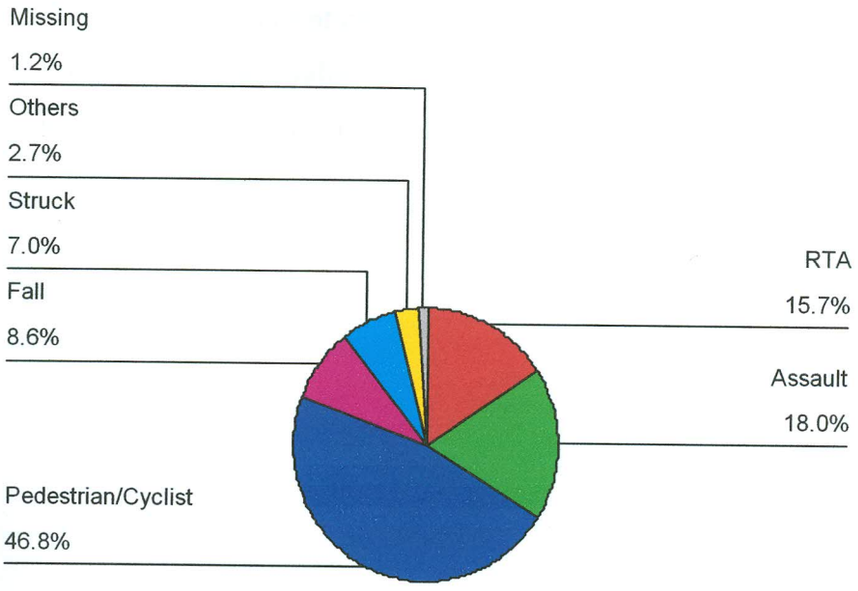
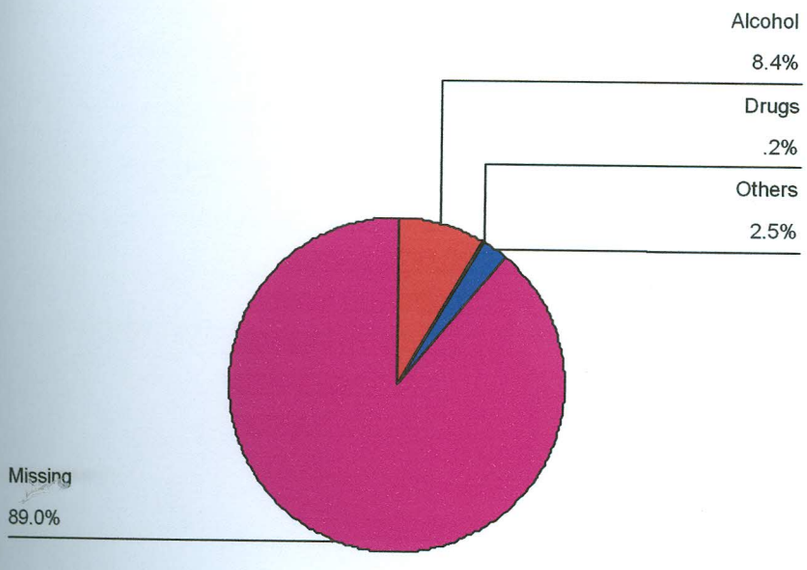


Figure 6; Associated factors of TBI



Traffic related accidents were predominantly the major cause at 62.5% and of these Pedestrian and cyclist related injuries (46.8%) presented a larger number of injuries compared to other causes of TBI. Other causes included assault (18%) and RTA (15.7%).Analysis give a *P*-value of 0.329 (which is greater than 0.05) and thus implies that the associated cause has no major influence on the type of TBI. The number of observations from the "Associated cause" had 435 missing cases and therefore this is not a valid variable of statistical analysis to use in reporting due to the large percentage of missing observations (fig. 6).

Figure 7: Outcome

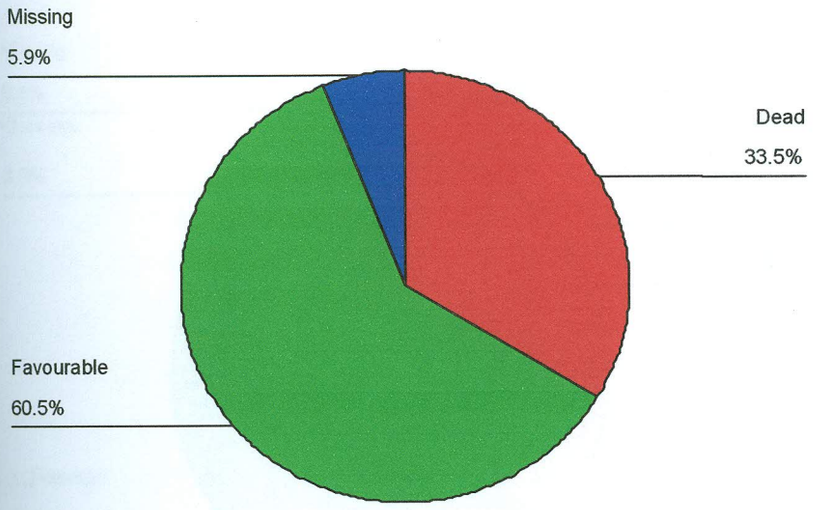


Table 6:Type of head injury * Outcome

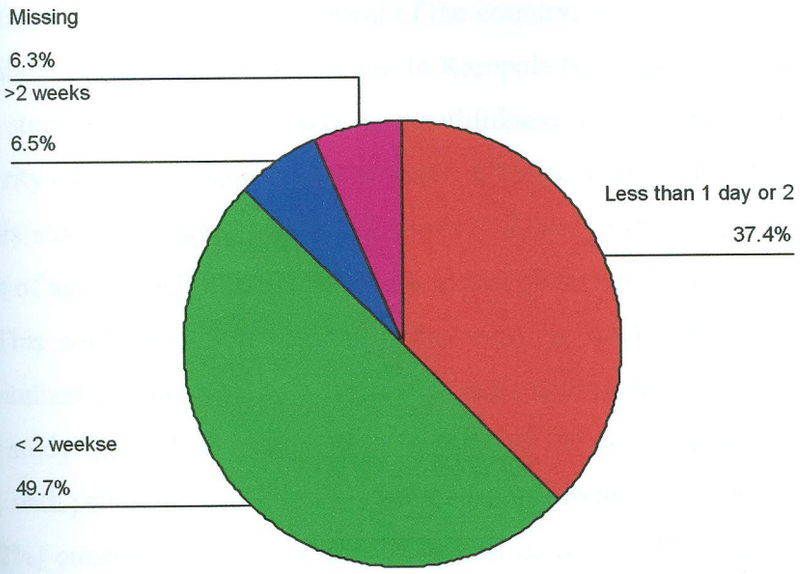
Count

		Outcome		
		Dead	Favorable	Total
Type of head injury	Mild	35	231	266
	Moderate	21	43	64
	Severe	106	18	124
Total		162	292	454

Results from this analysis give a *P*-value of 0.000 (which is less than 0.05) and thus indicating that there is a very significant relationship between the outcome and type of TBI. From the data, a favorable outcome is more likely under a mild type of TBI. Out of the 489 patients, 454 had outcomes recorded. 162 (33.5%) died while 292 (60.5%) had a favorable outcome.

5.5.4 Type of TBI versus period of hospital stay

Figure 8: hospital stay



Results from this analysis clearly indicate that there is a very strong relationship with a *P*-value of 0.000(which is less than 0.05) between the type of TBI and the period of stay in hospital. We therefore reject the null-hypothesis in favour of the alternative which implies the duration of stay in hospital is strongly associated the severity of TBI.

6.0 DISCUSSION

TBI data are difficult to compare internationally for various reasons of inconsistencies and complexities of diagnostic coding and inclusion criteria [1, 8, 11,93]. Nevertheless, the world's literature on traumatic brain injury (TBI) grows annually including new reports on epidemiologic findings from many regions with the wide variety of reports emphasizing various relevant factors on incidence, severity, external cause, gender, mortality, prevalence, cost and related factors.[94,64,93].

However the present result profile of this study is similar to that obtained in most African countries in that TBI is a public health problem. It is prevalent and on the rise [97, 12, 16, 18, 19, 23]. Males are said to be about twice as likely as females to sustain a TBI [44-45]

In this study 489 cases TBI, 332 (67.9%) comprised males and 148 (30.3) females. In China the male-female ratio was 1.7 to 1 in the six-city study (62.4%)male and 37.6% female), and 2.5 to 1 in the rural areas survey (71.3%) male and 28.7 female)[46]. This is a study done in Dar es Salaam, a major commercial capital of the country, young men were the largest consumers of the hospital emergency trauma services. In Kampala the largest occupational category of the injured were students which may reflect the youthfulness of the population. Whereas in South Africa the majority of injured persons were workers, with students a minority [95].

In this study the majority of the TBI (43.5%) were experienced by people who were below 30 years of age. Which is consistent with global pattern of TBI as shown in studies elsewhere [52,18-22]. This contrast with the USA in that Persons aged ≥75 years had the highest TBI-related hospitalization rate; persons aged 15--24 years had the next-highest rate. (47-49)

This study highlights traffic-related incidences in Dar es salaam as the major cause (62.5%) of TBI, with pedestrian and cyclist related injuries (46.8%) presenting the larger number and assault (18.2%) constituting the second commonest cause of TBI. Pedestrian risk correlates to similar studies done in Tanzania in traffic related accidents [19]. Pedestrian /pedal cyclists are more exposed to injury in a collision than the occupants of motor vehicles [95]. The fomer are vulnerable lacking physical protection afforded by cars and buses.

Similar studies report victims of assaults being beaten to death by angry citizens following robbery or caught stealing [96]. These findings describe the situation in Dar Es Salaam but may as well reflect problems faced by other large African cities and perhaps cities of low income countries elsewhere[31] Mob justice is a reflection. of inadequacy or ineffectiveness of law enforcement and/or lack of faith in the judiciary. Black males and American Indian/Alaska Native males had the highest rates of TBI attributable to assault approximately 4 times the rate for white males [46]. Fall constituted only 8.7% and only the third cause in this study. In USA as in most western countries [12, 15, 44-46], falls were the leading cause of TBI for both children and older age group

Mild injuries (57.3%) were more frequently reported followed by severe TBI (26.4%) and (13.9%) Moderate. From this study males are twice as likely to have one of the types of TBI as the females. There was a very significant relationship (p-value of (0.001) between sex and the type of TBI. This generally compares favorably with literatures elsewhere. [2, 43, 44, 46, 47]. In this study (p-value > 0.05) we found no association between Type of TBI and age. However, there

was a very strong relationship ($P< 0.000$) ($P< 0.05$) respectively between the type of TBI and the period of stay in hospital and outcome. The crude case fatality for mild HI and severe HI was 13.15% and 85.48% respectively. This phenomenon compare favorably with many studies done elsewhere. [1.10, 22.23,97]

The majority of patients therefore attending Emergency department and of those admitted, having mild head injuries which compares with many studies [1, 97, 10, 22-23]. The fact that patients with mild injuries without obvious brain damage make a considerable impact on the health care system mild TBI are crowding secondary and tertiary care services. This deprives the needed care for severe injuries.

In North America, the annual number of serious head and spinal injuries has steadily decreased over the past two decades, and of those patients who reach the hospital, the mortality and long-term morbidity have also declined [47,98].

7.0 CONCLUSION

There seem to be little awareness of the magnitude and the consequence and of contribution of TBI to the disease burden in developing countries and apparently and seemingly seriously neglected in research and policy [3, 14, 16,17-19, ,22,, , 97]. There are several facts that are known and others that need to be addressed

In comparison with the burden of disability, funding for injury is less than almost any other human misery [93, 99]. Head injured and impaired consciousness people present an important exception in clinical research because they are unable to give informed consent. This has posed an obstacle in enrolling in randomanized studies.

In comparison to developed countries, the annual number of serious head and spinal injuries has steadily decreased over the past two decades, and of those patients who reach the hospital, the mortality and long-term morbidity have also declined [44,45,47-49], The burden and pattern of injuries in Africa and other developing areas however, are poorly known and not well studied.

Road traffic injuries are the leading cause of death by injury and accounts for most of the severe TBI. With increasing motorization the global burden of TBI can only be expected to increase. It is estimated that by 2020 RTA will be the third leading cause of disability-adjusted life years lost worldwide and second leading cause in developing countries [16, 93, 99].

There are two divergent trends evident in international road safety report [58]. In Europe in the past 30 years, road deaths have fallen by 35%, despite the fact that traffic growth has tripled. This trend is also seen in the USA, Canada, and Australia. However, in many industrializing countries, especially in Africa, Latin America and Asia except Japan, deaths have increased alarmingly. Industrializing countries have 85% of death toll, but only 32% of the global motor vehicle fleet [-100]

7.1 Limitation of the Study

The present study analysis is based on retrospective coding of routinely collected information obtained from patient admission case notes. Therefore there is concern of potential under-reporting.

Generally, the analysis provides the table of statistics for the various variables whose descriptive variables will be captured in the database that are not sufficient for further analysis beyond the descriptive stage.

Follow-up data on disability and other long-term outcomes was problematic. Subjects were lost from follow-up.

8.0 RECOMMENDATIONS

TBI is a prevalent public health problem in our community probably reflecting the level of socio-economic and infrastructural development. Dar es Salaam, the capital city of Tanzania unfortunately, needs institutionalized pre-hospital neuro-trauma emergency care system. The absence of pre-hospital care and transport might and probably in the coming future, may worsen the status of these patients on admission.

The fact that patients with mild injuries without obvious brain damage make a considerable impact on the health care system becomes of concern on definitional issues and hospital admission policies. Evaluation of head injured patient is has increasingly come to rely on CT scanning. Theoretically an establishment of a good pre-hospital care, and an improvement in diagnostic facility at the emergency department, may shift the treatment of mild head injury from hospital inpatient service to outpatient service. However this effect is limited by the fact that many hospitals do not have CT scanner. There is therefore a need to provide guidelines for the

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11.0 APPENDICES

Appendix 1: Glasgow Coma Scale (GCS)

Eye Opening		
Score	**>1 Year**	**0-1 Year**
4	Spontaneously	Spontaneously
3	To verbal command	To shout
2	To pain	To pain
1	No response	No response
Best Motor Response		
Score	**>1 Year**	**0-1 Year**
6	Obeys command	
5	Localizes pain	Localizes pain
4	Flexion withdrawal	Flexion withdrawal
3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)
2	Extension (decerebrate)	Extension (decerebrate)
1	No response	No response

Best Verbal Response			
Score	**>5 Years**	**2-5 Years**	**0-2 Years**
5	Oriented and converses	Appropriate words	Cries appropriately
4	Disoriented and converses	Inappropriate words	Cries
3	Inappropriate words; cries	Screams	Inappropriate crying/screaming
2	Incomprehensible sounds	Grunts	Grunts
1	No response	No response	No response

Appendix 2: Glasgow Outcome Scale (GOS)

State	**Description**
Dead (D)	
Persistent vegetative state (PVS)	Wakefulness without responsiveness
Severe disability (SD)	Conscious but dependent
Moderate disability (MD)	Independent but disabled
Good recovery (GR)	Capacity to reintegrate

Appendix: 3

A: Monro-Kellie doctrine

This concept is defined

V (i/c) = V (brain) + V (cerebrospinal fluid) + V (blood)

This states as follows:

- Total volume of intracranial contents must remain the same(Vi/c)
- Venous volume + Arterial volume + Brain + CSF = Constant
- Intracranial compensation for expanding mass
- Volume pressure curve shows point of decomposition with rapid rise in ICP

In adult,

- Intracranial volume is approximately 1500 mL
- Brain accounts for 85-90%,
- Intravascular cerebral blood volume accounts for 10%
- Cerebrospinal fluid (CSF) accounts for the remainder (<3%).

B: Intracranial compliance

Defined as the change in pressure due to changes in volume

Compliance = Change in volume / change in pressure

States

- The intracranial volume is fixed,
- The pressure within this compartment rises unless some compensatory action occurs, such as a decrease in the volume of one of the other intracranial components.
- Compliance is based on the pressure volume index (PVI) within the intracranial compartment. The PVI describes the change in intracranial pressure (ICP) that occurs when a small amount of fluid is added to or withdrawn from the intracranial compartment.

The brain has very limited compliance and cannot tolerate significant increases in volume that can result from diffuse cerebral edema or from significant mass lesions such as a hematoma.

C: Cerebral perfusion pressure (CPP)

CPP is the net pressure of blood delivery to the brain. It is the difference between the mean arterial pressure (MAP) and the ICP.

CPP = MAP – ICP

- Normal Cerebral Blood Flow (CBF) 50ml / 100g brain / minute

- Normal Mean Arterial Pressure (MAP) 50-160mmHg
- Normal ICP is considered 0-15 mm Hg.(In young children may be considered 10 mm Hg)

Cerebral blood flow (CBF) is maintained at a constant range by auto regulation of the arterioles, which constrict or dilate within to maintain a constant amount of blood flow to the brain.

When the MAP is less than 50 mm Hg or greater than 150 mm Hg, the arterioles are unable to auto regulate and blood flow becomes entirely dependent on the blood pressure, a situation defined as **pressure-passive flow**. The CBF is no longer constant but is dependent on and proportional to the CPP.

When auto regulation is impaired as in the injured brain, pressure-passive flow occurs within and around injured areas and, perhaps, globally in the injured brain.

The brain is at risk of ischemia if MAP falls below 50 mm Hg while MAP greater than 160 mm Hg causes excess CBF that result in increased ICP.

D: Intracranial Pressure (ICP)

Normal ICP is considered 0-15 mm Hg.(In young children may be considered 10 mm Hg)

- Increase ICP is defined as pressure >20mHg (81, 82).
  Elevations in ICP result in decreased CPP and decreased CBF,
- < 20-25ml - Diminishing EEG activity
- ~ 5ml = Irreversible damage / cell death
- Uncontrolled ICP may cause herniation resulting in irreversible and often fatal cerebral injury

Appendix 4: Indications for admission to Hospital

A: Impaired consciousness (GCS < 15)

GCS 15 but any of the following risk factors exist:

- History of loss of consciousness
- Posttraumatic amnesia

- Severe or persistent headache or vomiting
- Irritability or abnormal behaviour
- Seizure at any time after injury
- Significant associated injuries
- Significant alcohol or drug intoxication (unable to assess reliably)
- Suspicion of non-accidental injury in children
- Focal neurological signs
- Clinical or radiological evidence of skull fracture or penetrating injury
- An abnormal CT scan
- Significant medical problems
- (e.g. anticoagulant use)
- No reliable companion at home / unable to return promptly

B: Mild TBI

GCS 14 – 15

- Admission to hospital or CT scan if indicated.
- If patient does not meet criteria for admission or CT scan → discharge with HI instructions + review in 1 week

C: Moderate TBI

- GCS 9 – 13 (can follow simple commands)
- All should undergo CT scan.
- If no neurosurgical intervention necessary → admit + frequent neurological observations.
- If patient improves (90%) → discharge with HI instructions + review in 1 week
- If patient deteriorates (10%) → repeat CT scan and treat according to severe HI protocol

Appendix 5: Statistical Report for TBI

The database contains data for 489 patients on several variables some of which have large numbers of missing observation that are not sufficient for further analysis beyond the descriptive stage.

Generally, the analysis starts by providing the table of statistics for the various variables whose descriptive variables are captured in the database. The row "Valid" means the number of subjects

whose observations were available while "Missing" gives the number of subjects whose observations were unavailable.

The descriptive tables for the various variables are provided showing the amount of available and missing data. For example, the table for "Associated cause" shows that there are 435 cases of missing observations hence not an good dataset to use for making clinical decisions. However, this could be a good dataset showing the challenges of recording important variables within the clinical set-up.

Type of Head injury versus Outcome

Type of head injury * Coded hospital stay Crosstabulation

			Coded hospital stay			
			Less than 1 day or 24 hours	Not more than two weeks	Greater than two weeks	Total
Type of head injury	Mild	Count	107	143	13	263
		Expected Count	106.0	138.2	18.7	263.0
	Moderate	Count	12	47	5	64
		Expected Count	25.8	33.6	4.6	64.0
	Severe	Count	62	46	14	122
		Expected Count	49.2	64.1	8.7	122.0
Total		Count	181	236	32	449
		Expected Count	181.0	236.0	32.0	449.0

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	26.365[a]	4	.000
Likelihood Ratio	27.631	4	.000
Linear-by-Linear Association	.005	1	.943
N of Valid Cases	449		

a. 1 cells (11.1%) have expected count less than 5. The minimum expected count is 4.56.

Ho: No association

H1: There is an association

Since p-value is less than 0.05, we reject the null hypothesis and conclude that there is a strong association between Type of Head Injury and Duration of hospital stay.

Type of Head injury versus Age Groups

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Type of head injury * Age groups	350	71.6%	139	28.4%	489	100.0%

Type of head injury * Age groups Crosstabulation

Count

		Age groups			
		Below 20 years	20-40 years	Above 40 years	Total
Type of head injury	Mild	80	109	45	234
	Moderate	15	18	16	49
	Severe	15	39	13	67
Total		110	166	74	350

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.749[a]	4	.068
Likelihood Ratio	8.492	4	.075
Linear-by-Linear Association	2.190	1	.139
N of Valid Cases	350		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 10.36.

Ho: No association

H1: There is an association

Since p-value is greater than 0.05, we fail to reject the null hypothesis and conclude that there is no association between Type of Head Injury and Age for this data.

Type of Head injury versus Sex

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Type of head injury * Sex	476	97.3%	13	2.7%	489	100.0%

Type of head injury * Sex Crosstabulation

Count

		Sex		
		Female	Male	Total
Type of head injury	Mild	104	175	279
	Moderate	12	56	68
	Severe	27	102	129
Total		143	333	476

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	17.014[a]	2	.000
Likelihood Ratio	17.636	2	.000
Linear-by-Linear Association	13.425	1	.000
N of Valid Cases	476		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 20.43.

Ho: No association

H1: There is an association

Since p-value is less than 0.05, we reject the null hypothesis and conclude that there is a strong association between Type of Head Injury and Sex.

Type of Head injury versus Outcome

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Type of head injury * Outcome	454	92.8%	35	7.2%	489	100.0%

Type of head injury * Outcome Crosstabulation

Count

		Outcome		Total
		Dead	Favourable	
Type of head injury	Mild	35	231	266
	Moderate	21	43	64
	Severe	106	18	124
Total		162	292	454

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	193.038[a]	2	.000
Likelihood Ratio	200.747	2	.000
Linear-by-Linear Association	186.236	1	.000
N of Valid Cases	454		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 22.84.

Ho: No association

H1: There is an association

Since p-value is less than 0.05, we reject the null hypothesis and conclude that there is a strong association between Type of Head Injury and Outcome.

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