CLINICAL PATTERN OF PEDIATRIC TRAUMATIC BRAIN INJURY AND EVALUATION OF THE PECARN HEAD TRAUMA RULE AT KENYATTA NATIONAL HOSPITAL.

A PROSPECTIVE STUDY

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DECLARATION

This research is my original work and has not been presented in any other forum, to the best of my knowledge. All references used will be duly noted and documented.

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LIST OF ABBREVIATIONS

A/E	Accident and Emergency
AEDH	Acute Extradural Hematoma
AHT	Abusive Head Trauma
ASDH	Acute Subdural Hematoma
ciTBI	Clinically Important Traumatic Brain Injury
CSF	Cerebrospinal Fluid
CT	Computerized Tomography
DAI	Diffuse Axonal Injury
DTICH	Delayed Traumatic Intracerebral Hemorrhage
IVH	Intraventricular Hemorrhage
KNH	Kenyatta National Hospital
MRI	Magnetic Resonance Imaging
SBS	Shaken Baby Syndrome
TBI	Traumatic Brain Injury
TICH	Traumatic Intracerebral Hemorrhage
tSAH	Traumatic Subarachnoid Hemorrhage
PECARN	Pediatric Emergency Care Applied Research Network

OPERATIONAL DEFINITIONS

TBI as seen on CT

This is definition entails:

- Diffuse axonal injury
- Intracranial hemorrhage or contusion
- Diastasis of the skull
- Cerebral edema
- Shearing injury
- Midline shift of intracranial contents or brain herniation features
- Traumatic infarction
- Pneumocephalus
- Sigmoid sinus thrombosis
- Depressed skull fracture. By at least the thickness of the diploe.

Clinically-important TBI

This definition entails:

- Admission to the hospital for ≥2 nights because of TBI with findings on CT
- Surgical intervention by the neurosurgery team for TBI
- TBI resulting in intubation for >24 hours
- TBI resulting in death

The PECARN head trauma rule

a) For patients who are less than 2 years of age:

With one of the signs below:

- Altered mental status
- Palpable skull fracture
- GCS ≤14

Acquire a non-contrast CT head because the possibility of ciTBI is 4.4%

With one or more of the symptoms and signs below:

- Severe injury mechanism
    - A fall more than one meter or three feet
    - Head hit by high-impact object
    - A pedestrian or bicyclist lacking a helmet hit by motor vehicle
- Loss of consciousness for 5 seconds or longer
- Abnormal activity reported by parents
- A scalp hematoma that is non-frontal

Observation or a non-contrast CT head may be considered. The possibility of ciTBI is 0.9%

b) For patients who are 2 years of age or more:

With one of the signs below:

- Altered mental status
- Basilar skull fracture signs
- GCS ≤14

Acquire a non-contrast CT head because the possibility of ciTBI is 4.3%

With one or more of the symptoms and signs below:

- Severe injury mechanism
    - A fall more than two meters or five feet
    - Head hit by high-impact object
    - A pedestrian or bicyclist lacking a helmet hit by motor vehicle
- Vomiting
- Loss of consciousness
- A headache that is severe

Observation or a non-contrast CT head may be considered. The possibility of ciTBI is 0.9%

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ABSTRACT

Introduction

Traumatic brain injury (TBI) is the most common cause of mortality and morbidity in children. Pediatric TBI may significantly differ from adult TBI in both pathophysiology and management. As a result of this variations, results derived from adult population studies cannot be applied to the pediatric population. Therefore, there is a definite need for more research studies to be designed for this unique population.

From my review of literature, there has been no research done within the last 10 years that specifically looks at the clinical pattern of pediatric TBI in Kenya. This study intends to investigate the pattern of pediatric TBI in patients admitted at Kenyatta National Hospital with an attempt at defining the demographics involved, their characteristics and management with the aim of generating information that will guide the development of strategies that will help in preventing and managing pediatric TBI in our setup. Using the data obtained, we will also evaluate the PECARN head trauma rule in the Kenyan pediatric population as seen at KNH.

Broad Objective

To determine the clinical pattern of pediatric traumatic brain injury and to evaluate the PECARN head trauma rule in mild TBI at Kenyatta National Hospital.

Methodology

A prospective descriptive cross sectional study was conducted on a total of 101 children who were seen in the Kenyatta National Hospital accident and emergency unit and diagnosed with TBI over a six-month period between August 2019 and January 2020. Cases of pediatric traumatic brain injury that met the inclusion criteria within the time frame specified were selected for study. SPSS statistical package was utilized for analysis. Frequencies, means and proportions were calculated. To compare variables student t-test and Chi-square test were undertaken. Statistical significance was taken at the level $p < 0.05$.

Results

There were 69 males (68.3%) and 32 females (31.7%). The mean age was 4.7 years. Falls (63.5%) and road traffic accidents (23.8%) were the leading causes of head injury. The distribution of head injury severity was mild in 74.3%, moderate in 19.8%, and severe in 5.9%. Good functional recovery was achieved by 93 (92%) of the patients in our series, whereas moderate and severe disability each accounted for 2% while mortality accounted for 4%. Younger age (p=0.029), a lower Glasgow coma scale score, anisocoria and admission into our Intensive Care Unit were all associated with poorer outcomes (p=0.000). The PECARN head trauma rule had a negative predictive value of 100.0% and a sensitivity of 100.0% for detecting clinically-important TBI.

Conclusion

Pediatric TBI is a significant health burden in our setup. We therefore recommend implementation of prevention strategies and establishment of a pediatric head trauma registry to help in the surveillance of pediatric TBI. The PECARN head trauma rule was shown to be effective in detecting clinically important traumatic brain injury in our setup. It should therefore be applied in mild pediatric TBI where it could limit CT use, protecting children from unnecessary radiation risks.

INTRODUCTION

Traumatic brain injury(TBI) is the most common cause of mortality and morbidity in children.[1,2,3,4] In the group of children who are more than one year of age, it accounts for more than 80% of deaths due to trauma.[1] Majority of pediatric TBI occurs following falls, assaults, road traffic accidents, activities for recreation, and abuse.[1,5,6,7] In Kenya, the leading cause of pediatric head injury is falls.[5]

Pediatric TBI may significantly differ from adult TBI in both pathophysiology and management. This difference may be ascribed to the injury mechanism and the child's physical ability, age-related structural change, and the difficulty in pediatric neurological evaluation.[2] As a result of this variations between adult and pediatric TBI, results derived from population studies based on adults may not be readily applied to a population of children. Therefore, this creates a need for additional research studies to be designed for this unique population which will form a foundation for the development of specific pediatric management guidelines.

To my knowledge, there has been no research that specifically looks at the clinical pattern of pediatric TBI in Kenya in the last 10 years although it still remains the most common source of pediatric morbidity and even mortality. This study intends to investigate the pattern of pediatric TBI in patients admitted at Kenyatta National Hospital with an attempt at defining the demographics involved and their clinical characteristics with the aim of generating information that will guide the development of strategies that will help in preventing and managing pediatric TBI in our setup.

The PECARN head trauma rule is a criterion that was developed to help recognize children who have a low possibility of having clinically important pediatric TBI for whom CT might be unnecessary. Avoiding unnecessary CT scans in children greatly reduces the risk of malignancies brought about by radiation. Nonetheless, this prediction rule has yet to be evaluated in the Kenyan pediatric population. Therefore, in addition to studying the pattern of pediatric TBI, this study will also use the data obtained to evaluate the PECARN head trauma rule.

LITERATURE REVIEW

Clinical pattern of pediatric traumatic brain injury and the PECARN head trauma rule

Epidemiology

The yearly incidence of pediatric TBI is around 200 per 100,000 in the United States. In a local study done 13 years ago at Kenyatta National Hospital, the hospital-based incidence was 20.2 patients per month which translated to about 242 admissions per year. [5] All through childhood, head trauma is relatively evenly distributed but there is an increase in incidence in 2 age groups. The first group is infants, where there is an increased incidence of head trauma ascribed to child abuse and falls.[8] The second group is at approximately 15 years, where there is a significant increase, largely in males. In Kenyatta National Hospital, the average age of pediatric TBI was 4.9 years. The 3-5 years age group had the highest risk. The risk of head injuries in males is twice that of females. In addition, the risk of fatal trauma is 4 times higher in males. [1] Mwangi et al in Nairobi, found a male to female ratio of 1.7:1in Kenyatta National Hospital. [5] The traumatic injury death rate in children less than four years is higher than in children between 5-14 years of age, at 5 per 100,000. This may be a reflection of the number of injuries caused by abuse in children less than four years of age. [9]

Majority of pediatric TBI occurs following falls, assaults, road traffic accidents, activities for recreation, and abuse. Vulnerability to pediatric TBI may be increased by factors such as attention deficit disorder, alcohol and drug abuse and seizures. Road traffic accidents contribute to 27-37% of all pediatric TBI. The child is usually a pedestrian or riding a bicycle in majority of the cases. Falls contribute to 24% of all pediatric TBI and are the commonest etiology in those younger than four years of age. In Kenya, falls were also responsible for majority of injuries at around 65%. [5] Recreational activities contribute to 21% of pediatric TBI and have a seasonal distribution. Assault accounts for 10% of all pediatric TBI. [1] Child abuse contributes to 24% of traumatic brain injury in those less than two years of age and was suspected in an additional 32%. Risk factors associated with child abuse include maternal single status, substance abuse, poor prenatal care, partner status and social concerns. The risk decreases with increasing gestational age at birth and increasing maternal age.[10]

Injury characteristics and anatomical considerations

Pediatric TBI has unique biomechanical properties because of a blend of higher plasticity and deformity. This change the way external forces are absorbed. In infants, the rigidity of the skull is less than that of adults and the presence of open sutures allow for some movement when responding to mechanical pressure. [11] A child has a bigger head in relation to body size when compared to adults. This means that there is an increased risk of the head of a child being injured in trauma. In addition, the pediatric head is heavier than the rest of the body with resultant altered dynamics of head acceleration when traumatic forces are applied. This increases vulnerability to pediatric TBI. [2]

In newborns, the cerebral white matter contains little myelin and its distribution is different from adults. The neonatal brain is watery but as the child grows older, the density increases because of increasing myelination and the water content reducing. The degree of myelination is significant in that it affects the pattern of absorption of forces that result from trauma, with unmyelinated regions being more susceptible to TBI. [12]

In TBI, the paranasal sinuses and the face limit brain damage because more energy is absorbed by the sinuses, reducing the amount of energy transmitted to the brain. Therefore children with poorly developed paranasal sinuses are more susceptible to brain damage because they lack the protection of the sinuses. Furthermore, young children have a protruding forehead which increases the chance of injury to the frontal area of the skull and the cerebral parenchyma that lies below it.

Children have a relatively heavy head and weaker neck muscles. In addition, they have flat facets and relatively flexible soft tissues and ligaments. This means that in severe trauma cases, they are at an increased risk of cranio-cervical junction lesions associated with cranio-cervical instability.

Sometimes forces applied on the fetal head as it passes through the birth canal may be associated with intra and extra cranial injuries. This may be aggravated by using instruments used in obstetrics such as delivery forceps. During this period, there is increased susceptibility to subgaleal hematomas and cephalohematomas. Violent shaking in children may also cause shear forces which usually lead to injuries of the brain parenchyma and cortical vessels. [13]

Clinical spectrum

1. Extra-parenchymal injury

a) Skull fractures

Skull fractures are linear, depressed, comminuted, and diastatic. Majority of the fractures in children are linear (90%) and the commonest location is the parietal bone. [1, 14] One of the clinical signs that may help diagnose skull fractures is extracranial subcutaneous swelling. With regards to depressed fractures in children, simple fractures are more prevalent. It is a consequence of a localized external force. Among childhood fractures, the compound type accounts for about 42%–66%. While in infants it accounts for 9% of all fractures. [2] Sometimes dural injury may be observed beneath fractures. Injuries to the dura that were associated with intracranial lesions accompanied around 11% of pediatric depressed fractures. [15, 16] Fractures that occur above dural sinuses have an increased risk of extradural haematomas and venous sinus thrombosis. In newborns, it is rare for ping-pong ball fractures to injure the dura. In depressed fractures in neonates, especially the simple type, reduction may occur spontaneously, but cranioplasty may be required in older children. Based on the current guidelines, surgical repair is recommended in the following cases: (1) where a foreign body is detected, (2) Cerebrospinal fluid leak identified, (3) infected wounds, (4) debridement is needed, (5) for cosmetic reasons, and (6) where evacuation of a hematoma is required. Some studies have found depressed skull fractures to be a risk factor for development of post-traumatic seizures. [2]

Basal skull fracture may be seen in 6-14% of pediatric head trauma patients. It may be associated with injuries to cranial nerves and vascular structures, more so in age groups where the facial bones are not fully developed. [18] In addition, it may also present as tardive meningitis. A study done by Alhelali et al. found basal skull fractures in 26% of their patients. Cerebrospinal fluid leaks were observed in 1/3rd of these cases and temporal bone fractures were seen in 2/3^{rds} of these cases. In conclusion, they found that basal skull fractures are suggestive of a linear blunt force and they are also predictors of mortality. [19]

Growing skull fractures can be found in young children, with majority occurring in those younger than 3 years of age. It is thought to occur due to skull fractures causing dural tears

allowing leptomeningeal cysts or cerebral parenchyma to herniate through. Cerebrospinal fluid pulsations erode the fracture margin, resulting in subsequent enlargement of the fracture line and non-union.

b) Acute epidural hematoma

It is not as common in early infancy. This is because the dura is firmly attached to the skull inner table, particularly around the sutures. In addition, the skull in neonates does not contain the middle meningeal artery and they have shallow arterial grooves from the external carotid artery which are less likely to be damage during TBI. [20, 21] Acute extradural hematoma (AEDH) in infants is mostly venous but the possibility of an arterial AEDH should not be discounted. It often occurs following rupture of emissary veins or after dural venous sinus injuries. For example, an occipital fracture overlying the transverse or the sigmoid sinus may result in a posterior fossa AEDH. Posterior fossa AEDH may also occur following head injury during birth in neonates. An AEDH of arterial origin usually peaks in size around 6-8 hours after the injury, unlike those of venous origin which may grow over 24 hours or more. [1] Usually, AEDH is limited by suture lines but if it is due to injury to a venous sinus it may cross suture lines. [22]

c) Acute subdural hematoma

Acute subdural hematoma (ASDH) develops between the dura mater and arachnoid membrane. It may result from bridging veins tearing or due to a laceration of the cortical arteries. It can be caused by a direct external force or because of a skull in motion colliding with an object that is stationary. Brain parenchymal injury and injury to the vasculature is caused by a force that is shearing in nature and a resultant rotational acceleration that results because of the abrupt deceleration. [23] ASDH is commonly associated with severe injury to the parenchyma, and a presentation that is characterized by a severe neurologic deterioration that is often progressive. [1] Of note, is that abusive TBI is a frequent cause of ASDH in the pediatric age group younger than 2 years of age. Suspect abusive TBI when there are multiple hematomas that occurred at different times (chronic and acute) when coagulopathy has been ruled out.

The bridging veins are at an increased susceptibility to traction and are at an increased risk of rupture when the subdural space is wide. [24] This can be seen in patients with benign external

hydrocephalus, cerebral atrophy, subdural effusion and hydrocephalus following shunt placement. [25] ASDH cases when compared to AEDH are associated with more frequent cerebral parenchymal contusions. Typical locations include within the interhemispheric fissure, calvarium, tentorium cerebelli and falx cerebri. Bilateral ASDHs are also common.

Abusive head trauma (AHT) is an important cause of severe pediatric TBI with an estimated incidence of between 14 and 40 in 100,000 in children aged less than 1 year. The injury may occur due to shaking, a direct blow or both. Hypotension and hypoxia may contribute as secondary causes of brain injury. The diagnosis of shaken baby syndrome is based on the behavior of abnormal shaking being observed as the principle cause of injury. [26] In children younger than 2 years of age, abusive head trauma is the most common cause of TBI therefore it is important for the clinician to have a high index of suspicion when accessing children with traumatic injuries. AHT may present with seizures, delayed developmental milestones, impaired consciousness and vomiting. Characteristic findings on imaging include ASDH (77%– 89% of cases), subarachnoid hemorrhage, cerebral contusion and intracerebral hemorrhage. [27, 28] On autopsy 83-90% of abusive head trauma cases have ASDH findings. [29, 30] With regards to physical examination, only "apnea" is specific to abusive head trauma, and it has a positive predictive value of 93%. [31] Other important findings include rib fractures (PPV of 73%), retinal hemorrhage (PPV of 71%) and seizures. Scalp injuries, skull fractures and long bone fractures did not show significant association with abusive head trauma. Retinal hemorrhages can be seen in 74% of AHT cases and are frequently bilateral and multilaminar. [32] Suspect abusive head trauma in patients who lack superficial trauma findings but have intracranial injury that is serious.

Accumulation of subdural fluid may also be associated with pediatric TBI. This accumulated fluid is mostly bilateral and can be a hematoma, concentrated protein or a combination of both. The presentation may be non-specific and includes decreased appetite, dysphoria, excitability, lethargy and an increased head circumference. Sometimes the diagnosis may be an incidental finding on imaging. With regards to management, subdural-peritoneal shunts may be considered. Y-shunt technique is often successful at reducing the subdural fluid that has collected over 2-3 months with a good prognosis. [33] Nonetheless, delayed enlargement of subdural space may

occur in AHT cases (especially in the bifrontal area), but it could also be subdural fluid that collects to compensate for areas of encephalomalacia.

d) Traumatic subarachnoid hemorrhage

Traumatic SAH (tSAH) results from rupture of small vessels located within the subarachnoid space or running on the surface of the cerebral cortex and from intraventricular hemorrhage or other intracranial hematomas being circulated and redistributed in the subarachnoid space. A retrospective study done by Hochstadter et al. reviewed 171 patients with a Glasgow Coma Scale (GCS) ≤ 8 before sedation who were subjected to CT head imaging within the first 24 hour period of admission into the hospital. Traumatic SAH was encountered in approximately 50% of severe traumatic brain injury patients of the pediatric age group. tSAH was indicative of pediatric TBI severity and a higher level of care needed at discharge. Traumatic SAH in pediatric TBI patients was not independently associated with an increased risk of mortality. [34]

e) Intraventricular hemorrhage

Intraventricular hematomas are often a result of intracerebral hematomas adjacent to ventricles extending into the ventricles. Furthermore, intraventricular hemorrhage (IVH) may be encountered when subependymal vessels rupture and when there is injury to the structures around the ventricle such as corpus callosum, septum and fornix. Sometimes traumatic SAH may flow backwards into the ventricle systems. Rotational and shear forces in diffuse axonal injuries may rupture ventral corpus callosum and subependymal blood vessels, causing intraventricular hemorrhage. Post-traumatic hydrocephalus is a complication that usually follows obstruction of the aqueduct or subarachnoid granular obstruction causing malabsorption of CSF. Moreover, degradation red blood cell products can lead to chemical ependymitis. Intensive care is necessary in order to monitor and detect signs of secondary hydrocephalus in pediatric IVH. [35]

2. Intra-parenchymal injury

a) Cerebral contusions

In pediatric TBI, cerebral contusions are relatively common. They usually occur under the region where the external force was exerted, also known as the coup injury. Cerebral contusions are frequently observed on the gray matter. The white matter is usually fairly well maintained. They are often found in both temporal and frontal lobes. This is due to the irregular cranial base and the anatomical contiguity of the petrous bone and crista galli to the cortex. [36] Contre-coup injuries are rarely seen in infants. A primary contusion may cause cerebral swelling which may progress into a hypoxic lesion which surrounds the initial lesions. As this focal hypoxic lesions progress, they can cause secondary injury to the brain and also negatively affect blood circulation. Consequently, the ischemia in the region of the initial cerebral lesion may worsen. An elevation in cerebral pressure can quickly advance to herniation of the cerebrum and can lead to death, especially in limited compartments like middle or posterior fossa.

b) Diffuse axonal injury

The classical presentation of diffuse axonal injury (DAI) includes coma which may be associated with a decorticate or decerebrate posture. MRI findings showing radiological evidence of DAI may allow a more definitive diagnosis. [37] There are multiple external forces acting on the brain from varied motions resulting in complex pathology. Nerve tissues and vasculature are often damaged by shear forces in DAI. In addition, changes may be seen in the frontal and parietal subcortical white matter axons, the basal ganglia, central corpus callosum, internal capsule, and the ampulla. [2]

c) Intracerebral hemorrhage

Traumatic intracerebral hemorrhage (TICH) is often seen in white matter of the temporal and frontal lobes but with injury to the perforators, it may be seen in the basal ganglia or cerebellum.

Delayed traumatic intracerebral hemorrhage (DTICH) characteristically occurs in geriatric TBI patients with multiple intraparenchymal contusions with likely coagulopathy.

Nevertheless, it should be included in the differential diagnosis of children who develop an abrupt neurological deterioration requiring urgent intervention. [38-40]

PECARN head trauma rule

The origin of this rule is from the multicenter PECARN network. It was developed to detect clinically important traumatic brain (ciTBI) injury in children to age 18yrs old after blunt head trauma. [41] It had been observed that less than 10% of CT scans in minor pediatric TBI show traumatic brain injuries. In addition, injuries requiring neurosurgery are uncommon in children with GCS scores of 14-15. [42-45] Therefore a large number of CT scans done in minor pediatric TBI are usually unnecessary.

Avoidance of unnecessary CT imaging is important in head-injured children because ionizing radiation from CT scans can cause lethal radiation-induced malignancy. [46-48] Children are much more sensitive to radiation-induced cancer than adults. This was observed from the data obtained from survivors of the atomic bombs dropped on Japan in 1945. The reason for this is twofold. The first reason is that children have more time to express a cancer than adults because of their longer life expectancy. Secondly, children are essentially more sensitive to radiation simply because they have more dividing cells and radiation basically acts on dividing cells. [46] In addition, the organ doses from a CT scan are clearly higher for children than for adults. The approximate rate of lethal malignancies from CT is between 1 in 1000 and 1 in 5000 pediatric cranial CT scans, with risk increasing as age decreases. [46, 47]

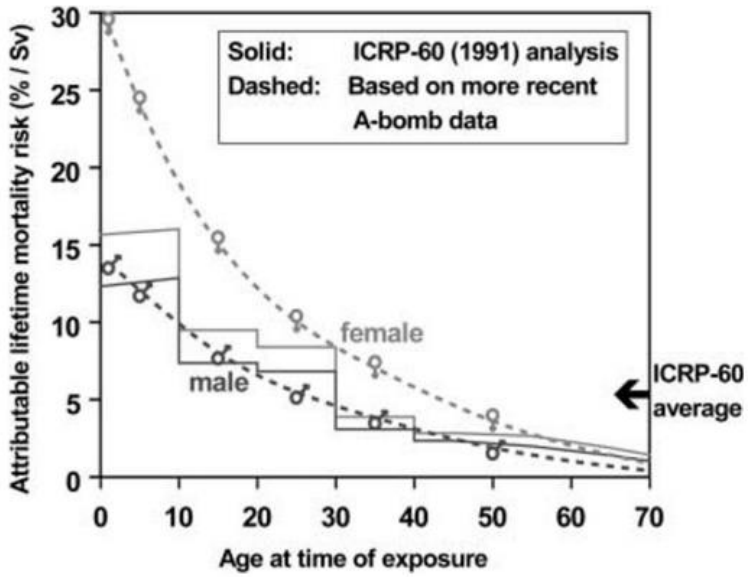


Figure 1. Estimated lifetime risks after radiation exposure based on atomic bomb data [46]

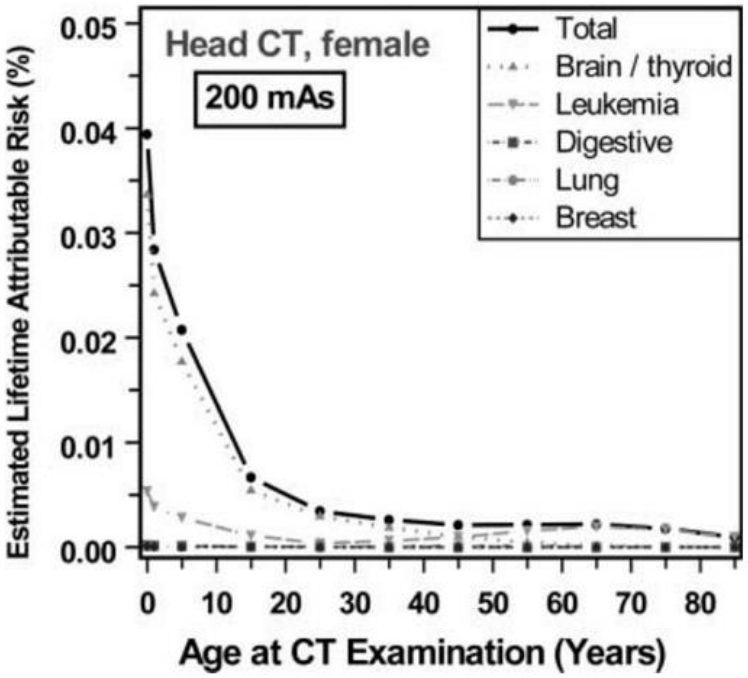


Figure 2. Head CT: estimated lifetime cancer mortality risk (%) [46]

The study that this rule was derived from was a prospective cohort study of patients who were younger than 18 years with head trauma in 25 emergency departments of a pediatric research network. [41, 49] Regarding the inclusion and exclusion criteria, children presenting within 24 hours of head trauma were eligible. Children with trivial injury mechanisms defined by falls that were at ground level or walking or running into objects that were stationary, and no signs or symptoms of head trauma other than scalp abrasions and lacerations were excluded. Patients with penetrating trauma, known brain tumours, pre-existing neurological disorders complicating assessment, or neuroimaging at an outside hospital before transfer were also excluded. Patients with ventricular shunts in situ, bleeding disorders, and GCS scores less than 14 were enrolled but were analyzed separately. [41]

The main outcome measure was ciTBI which was defined as death from traumatic brain injury, neurosurgery, intubation for more than 24 hours for traumatic brain injury, or hospital admission of two nights or more associated with TBI on CT. This outcome was defined to exclude brief intubations for imaging or overnight admission for minor CT findings. Outcomes were verified

by medical record review by site investigators who were unaware of emergency department data. CT scans were ordered and obtained at the discretion of the emergency department clinician. Helical CT scanners with radiographic slices separated by 10 mm or less were used. CT scans were viewed and interpreted by site faculty radiologists. A study pediatric radiologist, unaware of clinical data, made definitive interpretations of inconclusive CT scans. TBI on CT was defined as outlined in the definitions section.

The PECARN head trauma prediction rule characteristics include [41]:

1. For patients less than 2 years old with TBI on CT: Negative predictive value: 100.0%, (97.8–100·0), sensitivity:100.0% for TBI on CT (94.7–100.0)
2. For patients less than 2 years old with ciTBI: Sensitivity: 100.00% (86.3–100.00), Negative predictive: 100.00% (99.7–100·00)
3. For patients 2 years and older with TBI on CT: Negative Predictive Value for TBI on CT: 439/446 (98.4%, 96.8–99.4), Sensitivity of 94.0% 109/116 (88.0–97.5)
4. For patients 2 years and older with ciTBI: Sensitivity: 96.8% (89.0–99.6), Negative Predictive Value: 99.95% (99.81–99.99)

Management overview

Initial management of pediatric TBI is guided by the Advanced Pediatric Life Support (APLS) guidelines which involve: primary survey, secondary survey and definitive management. During the primary survey life-threatening injuries are identified and simultaneously resuscitation is begun. It entails assessment of the airway maintenance with cervical spine protection, assessment of breathing and ventilation, circulation, disability and exposure and environment control. Secondary survey begins once primary survey is completed and vital signs are normalizing. It involves a head-to-toe evaluation of the patient including a complete history and physical examination. Imaging indicated from the clinical findings is obtained. [50]

History taking

History taking entails a brief history of the event including: time of injury and mechanism of injury. Symptoms and their progression since the injury, including: Loss of consciousness, vomiting, headache, seizures, confusion, amnesia, irritability, lethargy, or agitation etc. Details of any significant medical history and history of alcohol or other drug use.

Physical Findings

Important physical findings to be obtained include vital signs, that is, respiratory rate, blood pressure, temperature and pulse. Head examination to look for scalp hematomas, depressed fractures, haemotympanum, rhinorrhea, otorrhea, head enlargement, bulging anterior fontanelle, racoon eyes and a battle sign. A complete neurological examination should also be obtained to identify any focal neurological deficits. A complete physical examination for other injuries on the rest of the body should also be carried out. The severity of TBI is then classified based on the pediatric Glasgow Coma Scale score: mild TBI – GCS 13-15, moderate TBI – GCS 9-12 and severe TBI – GCS 3-8.

Mild TBI

Mild TBI is seen in majority of the children, and it may be associated with brief or no loss of consciousness. Patients who fall under this group have heterogenous pathophysiology. [51]

This has caused the management mild TBI to remain controversial because reports of neurologically intact children with intracranial injuries have caused some to recommend cautious management while the infrequency of serious intracranial injuries have caused others to be less conservative. [51,52]

Duus et al [53] identified intracranial complications in 9 patients out of 1876 patients classified as minor head injuries. Factors that significantly predicted the risk of developing an intracranial complication included the presence of agitation, vomiting, amnesia >5 minutes, positive neurological signs and impaired consciousness. Iverson et al[54] observed abnormal CT scan head findings in 16-21 % of patients with mild TBI. They found a relationship between the presence of intracranial abnormalities with lower GCS scores, skull fractures and frequency of loss of consciousness. Batchelor et al [51] identified 5 predictive symptoms for an abnormal CT scan head, which included: severe headache, nausea, vomiting, dizziness and blurred vision. Borczuk et al [55] found an abnormal CT scan in 8.2% of patients with mild TBI after blunt head trauma. These cases were predicted significantly by the presence of a focal neurological deficit, cranial soft-tissue injury and signs of basilar skull fracture. The best predictor of subsequent deterioration or intracranial hemorrhage requiring emergent surgical intervention in patients has been shown to be the neurological examination.

Risk stratification

Patients may be stratified into one of three groups based on the likelihood of intracranial injury: low, moderate and high risk for intracranial injury.

1. Low risk for intracranial injury

In this group, there is an extremely low likelihood of intracranial injury (ICI) (incidence of ICI: ≤8.5 in 10,000 cases with 95% confidence level. [56,57] Patients in this group are characterized by:

- Asymptomatic
- Headache
- Dizziness
- Scalp hematoma, laceration, contusion, or abrasion
- No moderate nor high risk criteria

- No history of loss of consciousness

2. Moderate risk for intracranial injury

- History of change or loss of consciousness on or after injury
- Progressive headache
- Alcohol or drug intoxication
- Post-traumatic seizure
- Unreliable or inadequate history
- Age <2 years (unless trivial injury)
- Vomiting
- Post-traumatic amnesia
- Signs of basilar skull fracture
- Multiple trauma
- Serious facial injury
- Possible skull penetration or depressed fracture
- Suspected child abuse
- Significant subgaleal swelling

3. High risk for intracranial injury

- Depressed level of consciousness not clearly due to alcohol, drugs, metabolic abnormalities, postictal, etc.
- Focal neurological findings
- Decreasing level of consciousness
- Penetrating skull injury or depressed fracture

Management of mild TBI

1. Low risk mild TBI

This group includes patients with a mild TBI who meet the low risk criteria for intracranial injury. CT scans of the head are not usually indicated and plain skull radiographs are not recommended. Patients in this group who meet criteria below for observation at home may be managed with observation at home with written head-injury discharge instructions:

- Head CT scan not indicated, or CT scan normal if indicated
- Initial GCS≥ 14
- No high risk criteria
- No moderate risk criteria except loss of consciousness
- Patient is now neurologically intact (amnesia for the event is acceptable)
- There is a responsible, sober adult that can observe the patient
- Patient has reasonable access to return to the hospital E/R if needed
- No "complicating" circumstances (e.g. no suspicion of domestic violence, including child abuse)

Head injury discharge instructions include:

- Seek medical attention in case of:
    1. A change in level of consciousness
    2. Abnormal behavior
    3. Increased headache
    4. Slurred speech
    5. Weakness or loss of sensation
    6. Persistent vomiting
    7. Enlargement of one or both pupils that does not get smaller when a bright light is shined on it
    8. Seizures (convulsions or fits)
    9. Significant increase in swelling at injury site

- Do not take sedatives or pain medication stronger than acetaminophen (paracetamol in some countries) for 48 hours.
- Aspirin or other anti-inflammatory medications are contra-indicated because of interference with platelet function and theoretical increased risk of bleeding

In-hospital observation is indicated to rule-out neurological deterioration if patient does not meet the criteria for observation at home.

2. High/moderate risk mild TBI

In this group, clinical grounds alone may miss important lesions so a head CT scan is indicated. [58] 8–46% of patients with minor head injury have an intracranial lesion with the most frequent finding being hemorrhagic contusions. [59] Patients who are high risk for intracranial injury will require admission to the hospital. If there are focal findings on neurological examination, the operating room needs to be notified to be on standby.

Admitting orders for mild TBI

1. Bed rest. Elevate the head of bed 30–45°.

2. Neurological checks 2 hourly (Hourly if more concerned) to monitor for neurological deterioration.

3. Nil per oral until alert; then clear liquids, advance as tolerated.

4. Isotonic intravenous fluids (e.g. Normal saline and 20 mEq KCl/L) run at maintenance (2000 $cc/m^2/d$).

5. Mild analgesics: acetaminophen (PO, or PR if NPO)

6. Anti-emetic: give infrequently to avoid excessive sedation, avoid phenothiazine anti-emetics

(Which lower the seizure threshold)

7. CT scan head if indicated. (Especially if patient develops neurological symptoms, GCS decline or persistent vomiting >4 hours after admission)

Admitting orders for moderate TBI

1. Orders as for minor head injury above except patient is kept NPO in case surgical intervention is needed.

2. For GCS=9–12 admit to ICU.

3. CT scan head if indicated. (Especially if the patient has not improved in 2 hours)

Patients with normal or near-normal CT scans should improve within hours. Any patient who fails to reach a GCS of 14–15 within 12 hrs should have a repeat CT at that time [60]

Admitting orders for severe TBI

1. Admit to the intensive care unit

2. Monitor vital signs. Aim for: $Pa0_2$ > 80 mmHg, PaC02 35-40 mmHg, BP systolic> 80 mmHg (Or age appropriate equivalents).

3. Endotracheal intubation for airway protection and facilitation of ventilation

4. Head of bed elevation 30-45 degrees

5. Isotonic intravenous fluids (e.g. Normal saline and 20 mEq KCl/L) run at maintenance (2000 $cc/m^2/d$).

6. Prophylactic anticonvulsants e.g. phenytoin

7. Proton pump inhibitors e.g omeprazole

8. Anti-emetic

9. Analgesics

10. Urinary catheterization and monitoring of input and output.

11. Nasogastric tube insertion.

12. Thromboembolism prophylaxis e.g. Thromboembolism deterrent stockings

13. CT scan head immediately after resuscitation

14. Consider intracranial pressure monitoring.

15. If evidence of raised intracranial pressure or risk of intracranial herniation, options include:

- Sedation and paralysis
- Draining 3-5ml cerebrospinal fluid if an intraventricular catheter present
- Hyperventilation
- Mannitol 0.25–1 gm/kg, then 0.25 mg/kg q6 hrs, Furosemide
- Hypertonic saline
- High dose barbiturate therapy
- Hypothermia
- Decompressive surgery

16. Neurosurgery if surgical indications are met.

Outcomes

Evaluation of both physical and intellectual disability outcomes requires long-term follow-up. A comprehensive, multidisciplinary rehabilitation protocol was suggested by Kuihara et al to help promote recovery and facilitate a smooth transition back to school and home activities. [61]

Fulkerson et al studied long-term (median 10.5 years) outcomes in a cohort of TBI children who presented with traumatic brain injury and an initial Glasgow Coma Scale score of 3 or 4. 56.6% of these patients died within 1 year, but approximately 15% had good outcomes at 10 or more years. Pupillary response at admission, mechanism of injury and occurrence of hypothermia were all associated with survival and outcomes. [62]

Severe pediatric TBI patients as expected, have worse outcomes. Mwang'ombe et al studied the outcome of severe injuries managed at Kenyatta National Hospital. [62] In the pediatric age group, that is patients aged below 13 years, they reported an age specific mortality of 35.7%. Sousa et al who also studied severe TBI observed 25% functional outcome and 75% poor outcome (death and severe disability). [63]

Mild pediatric TBI has a lower mortality rate, with overall mortality rates of 1-3% being reported. [64,65] Majority of mild pediatric TBI cases do not result any adverse effects, but long-term problems in psychosocial function occur in more severe cases, particularly when this event occurs during the preschool years. [66] Functional and cognitive recovery is variable and may be delayed or impaired.

Outcome assessment

The Glasgow outcome scale (GOS)

Score	Meaning
5	Good recovery - resumption of normal life despite minor deficits
4	Moderate disability - disabled but independent
3	Severe disability - conscious but disabled - dependent
2	Persistent vegetative state - unresponsive and speechless
1	Death

Prevention of head injuries.

Close supervision by an adult caregiver is crucial in pediatric TBI prevention. Infants and toddlers should not be left unattended, especially on a baby cot without barriers or on an adult bed. Care should be taken with baby walkers because they can topple over. Violent shaking of babies should be discouraged. Children should be secured in a child safety seat or seat belt while in the car. Children should also wear properly fitted helmets when participating in activities like riding horses, bicycles and using skates. Children should also be educated on road safety rules.

Options for home safety include, installation of window guards which can protect young children from falling out when the windows are open. Use of safety gates placed at the top and bottom of stairs. Removal of tripping hazards which include small area rugs and electrical cords that are loose. Non-slip mats may be utilized in the bathtub and on shower floors. The playgrounds also need to be well designed and maintained. For instance, the surface of the playground should be made of shock-absorbing material such as mulch, pea gravel or rubber.

Conclusion

Traumatic brain injury(TBI) is the most common cause of mortality and morbidity in children. After the initial evaluation, prompt diagnosis, multimodal monitoring, and titrated management of intracranial hypertension are necessary to minimize pathophysiological damage to the developing brain. Because pediatric TBI may significantly differ from adult TBI in pathophysiology, this creates challenges in management. A study on the patterns of pediatric TBI in our setting will therefore help set the foundation for the development of strategies that will help in preventing and managing pediatric TBI in our setup. In addition, evaluation of the PECARN head trauma rule in the Kenyan pediatric population will help encourage its use and therefore reduce unnecessary CT imaging.

PROBLEM STATEMENT/RESEARCH PROBLEM

There has been no research done at Kenyatta National Hospital or any other center in Kenya within the last 10 years that focusses specifically and solely on the clinical pattern of pediatric traumatic brain injury. Given the relatively common occurrence of this condition, its significant contribution to childhood morbidity and mortality, and the specialist care that its successful management often demands, there is an urgent need to investigate it fully and possibly identify factors that could contribute to prevention and better management. Lack of current research in this unique population means that no statistics currently exist that would be useful in establishing standardized management protocols for our Kenyan pediatric population. Thus, this knowledge gap can only be filled by a research study such as this one.

Traumatic brain injury (TBI) is the number one cause of morbidity and mortality in children. Pediatric TBI is associated with several distinctive characteristics that differ from adults and are attributable to age-related anatomical and physiological differences, pattern of injuries based on the physical ability of the child, and difficulty in neurological evaluation in children. Evidence suggests that children exhibit a specific pathological response to TBI with distinct accompanying neurological presentation. This research hopes to elucidate this pattern of pediatric TBI as seen in our Kenyan pediatric population, sensitize clinicians on the differences between adult and pediatric TBI, ensure early referral for specialist care and, via identification of modifiable risk factors/causes, reduce recurrence and incidence of this condition.

There is also paucity of data in Kenya on the incidence and prevalence of both confirmed and suspected child abuse resulting in pediatric TBI. This I suspect may be due to poor reporting. Cohen et al, who studied current patterns of inflicted head injury in children, found that child abuse is a frequent cause of pediatric TBI. [67] He also found that child abuse cases correlated strongly with low socioeconomic status. With majority of the Kenyan population being of low socioeconomic status, I suspect that the incidence of child abuse leading to pediatric TBI may be significant. I hope that my study will be able to describe the incidence and pattern of pediatric TBI secondary to child abuse.

It has also been observed that CT scan use has more than doubled with many of the traumatic brain injuries identified on CT not requiring acute intervention, and some being false positives or non-traumatic findings. The use of the PECARN head trauma rule has been shown to be sensitive in detecting clinically important traumatic brain injury in international studies and thus reducing unnecessary CT imaging. [41] This rule has yet to be evaluated in the Kenyan pediatric population.

STUDY/RESEARCH JUSTIFICATION.

Researching into pediatric traumatic head injury and defining the demographics involved and their clinical characteristics will help in generating information that will guide the development of strategies that will help in preventing and managing pediatric TBI in our setup. In addition, evaluation of the PECARN head trauma rule in the Kenyan pediatric population will help encourage its use and reduce unnecessary CT imaging.

BROAD OBJECTIVE

To determine the clinical pattern of pediatric traumatic brain injury and to evaluate the PECARN head trauma rule in mild pediatric TBI at Kenyatta National Hospital.

SPECIFIC OBJECTIVES

1. To describe the epidemiology of pediatric traumatic brain injury.
2. To describe the spectrum of clinical presentation of patients diagnosed with pediatric traumatic brain injury.
3. To determine the radiological pattern of pediatric traumatic brain injury.
4. To determine the immediate outcome at discharge.
5. To evaluate the PECARN head trauma rule in mild pediatric TBI

RESEARCH METHODOLOGY

Study Design

This is a prospective descriptive cross-sectional study that will analyze a sample of cases of pediatric traumatic brain injury in Kenyatta National Hospital between the months of June 2019 and November 2019.

Actual patients meeting the inclusion criteria who present at the study site during this period will be recruited after an informed consent has been obtained. Data will be obtained from interviewing the guardians, examining the patient, recording their radiologic imaging findings if any and passively observing their management and outcome until the point of discharge from the hospital. The investigator will only observe and will not interfere with the management of the patient.

After we have obtained the data, we will then analyze the data collected. We will look back at each patient's clinical presentation data and check whether it meets the PECARN head trauma rule criteria which uses clinical presentation to detect clinically important TBI. Then we will confirm whether the patient truly developed clinically important TBI by reviewing the data we obtained while following the patient until the point of discharge. This will allow us to ascertain the number of true and false positives and true and false negatives with regards to detection of clinically important TBI by the PECARN rule. We will then be able to evaluate the PECARN head trauma rule by measuring its sensitivity, specificity, positive predictive value and negative predictive value from the data obtained.

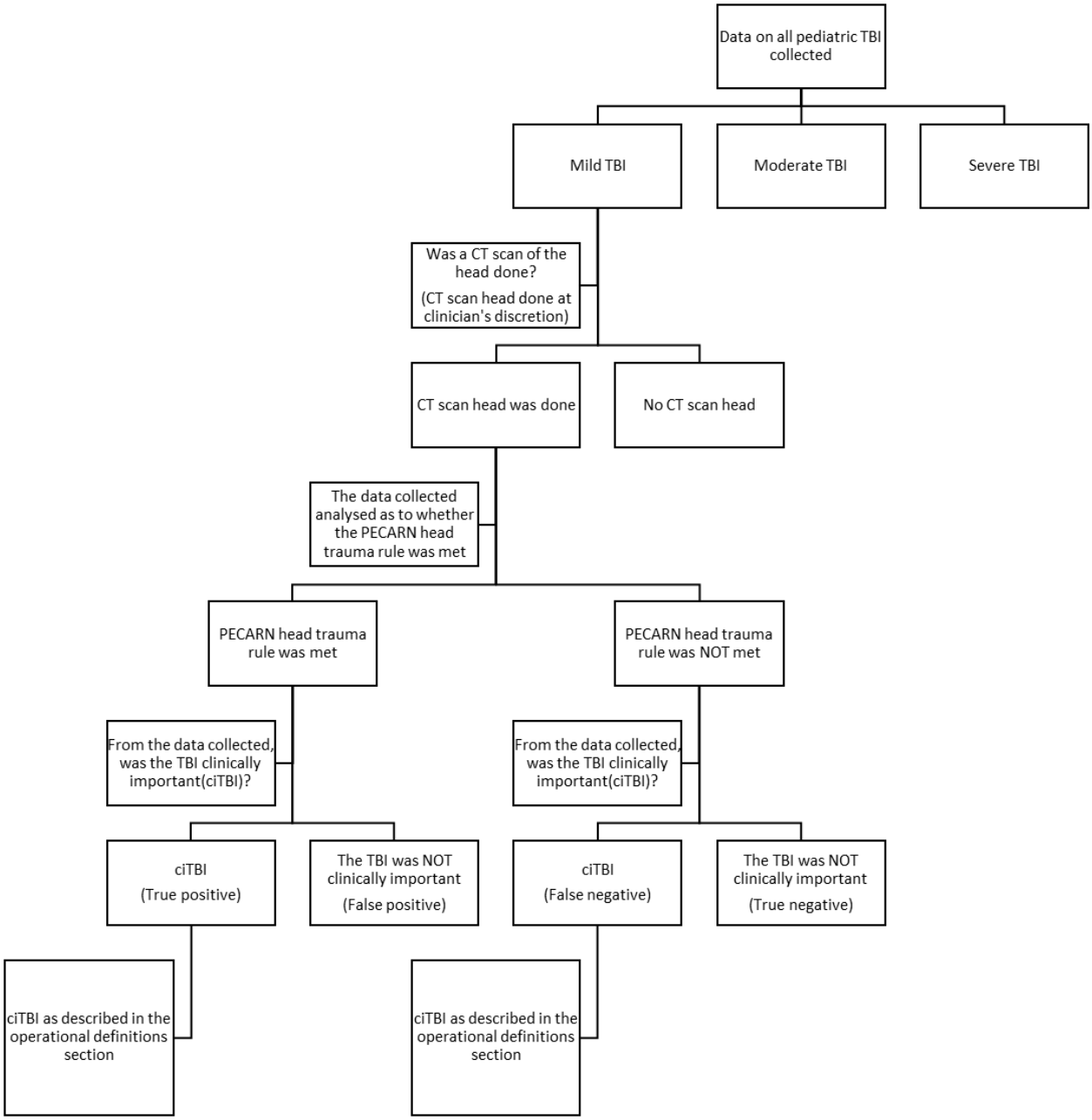


Figure 3. Data analysis with regards to PECARN CT head rule

Study Site

The study will be conducted at Kenyatta National Hospital, a level 6 Teaching and referral hospital based in the capital city of Nairobi. The specific study area within Kenyatta National Hospital will include the accident and emergency unit (casualty), neurosurgical ward (4c), pediatric neurotrauma ward (4a) and intensive care unit. Kenyatta National Hospital receives a high volume of pediatric TBI, about 4-5 cases per day on average.

Study Population

The study will include all patients with diagnoses of pediatric traumatic brain injury within the time period specified, and who meet the inclusion criteria outlined below. Actual patients will be recruited (not health records).

SELECTION AND ENROLMENT OF PATIENTS

Inclusion Criteria

- All patients with confirmed diagnoses of pediatric traumatic brain injury, aged 13 years and below, regardless of sex, ethnicity or socio-demographic variables, within the time period specified.
- All patients meeting the above criteria whose guardians give informed consent for their children to participate in the research study. If guardians have not accompanied the patient at presentation, they will be contacted via telephone and informed consent will be taken once they arrive at the hospital. Assent from minors will not be possible to obtain post traumatic brain injury because of altered level of consciousness and confusion that frequently accompanies pediatric traumatic brain injury.

Exclusion Criteria

- All patients with confirmed diagnoses of pediatric traumatic brain injury, aged 14 years and above.
- Patients whose guardians do not consent to participate in the study.
- Patients with no guardians to give consent to participate in the study.

Sample Size Determination

Sample size calculated as per Fisher's formula for calculating one sample size using precision around a proportion will provide minimum sample required.

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

N= minimal sample size required for the study.

z= 1.96 (normal deviate corresponding to 95% confidence level)

d= 0.1 (degree of precision around the mean)

P= 29% (mortality rate of head trauma in children in the United States: National Center for Health Statistics, 2017)

Thus $N = \frac{1.96^2 \times 0.29 \times 0.71}{0.01}$ The minimum sample size N= **79**

Convenience sampling will be used to recruit the 79 cases as they present at the accident and emergency unit at Kenyatta National Hospital.

Study procedures

Sampling, recruitment and consenting

Convenience sampling will be used to recruit the 79 cases who meet the inclusion criteria as they initially present at the accident and emergency unit at Kenyatta National Hospital.
The patient will only be recruited once the guardian has given informed consent for the child to participate in the research study.
The investigator will provide all the information necessary for an informed consent.
If guardians have not accompanied the patient at presentation, they will be contacted via telephone and informed consent will be taken once they arrive at the hospital. Assent from minors will not be required as this will not be possible post traumatic brain injury.

At point of presentation

At the point of presentation, the investigator will then interview the guardian to obtain the history of clinical presentation, physically examine the child and record the radiological imaging findings using the data collection sheet provided in the appendices.

Follow up

At the point of discharge from the hospital, the investigator will record the clinical management administered to the patient and the Glasgow outcome scale score at discharge using the data collection sheet provided in the appendices.

QUALITY ASSURANCE PROTOCOL

There will only be one principal investigator who is a neurosurgical resident and 2 supervisors who are consultant neurosurgeons. The principal investigator shall be responsible for obtaining the clinical history and initial examination of the patient and any equivocal signs elicited will be confirmed with the consultant neurosurgeon on call on that day.

All patients' clinical histories and examination findings will then be reviewed by all the consultant neurosurgeons in the neurosurgical unit during the major ward rounds and the findings obtained by the principal investigator will be confirmed. Radiological imaging findings recorded will be confirmed by an independent radiologist.

Clinical management including investigations and interventions will be left to the discretion of the consultant neurosurgeon on call on each day. The investigator will not interfere with patient management.

At the point of discharge, the principal investigator will be responsible for recording the clinical management that was administered to the patient. The supervisors will randomly review 20% of the data collected and confirm the findings. Data entry will be done by the principal investigator and the supervisors will randomly review 20% of the data entered to ensure accurate data entry.

The scientific concept and general aims of the study have been presented to the neurosurgical unit at a meeting organized by the principal investigator and has been approved by the neurosurgery department which will offer its full support.

ETHICAL CONSIDERATIONS

A permission to carry out this study will be sought from the Kenyatta National Hospital Ethical Review Committee.

The information will be kept confidential and within limits of research objectives. Data recording and storing will be done by the investigator and carefully stored.

Every single case we use for this study will have the signed informed consent of the participant(s) concerned.

The information we collect from these records in our questionnaire will be destroyed after the completion of the study to maintain the privacy of participants.

DATA COLLECTION, MANAGEMENT AND ANALYSIS

Data management

All cases that meet the inclusion criteria will be studied individually, and the relevant data from each case will be captured. The data collection tool will be a structured questionnaire which will be filled by the investigator.

Data analysis

Variables

Variable Name	Brief description	Classification; nature of measurements (quantitative – continuous or discrete; qualitative – nominal or ordinal)
Demographic variables		
Study_num	Study number : Patient unique identifier	Quantitative- discrete
County	Current county of residence of the patient	Qualitative- categorical nominal
Dob	Date of birth	Quantitative- discrete
Age		Quantitative- discrete
Gender	Male/Female	Qualitative-categorical nominal
Clinical presentation variables		
Date	Date of presentation	Quantitative- discrete

moi	Description of mechanism of injury	Qualitative-categorical nominal
Predisposing factor	Predisposing factor	Qualitative-categorical nominal
Pt_amnesia	Post-traumatic amnesia	Qualitative-categorical nominal
Loc	History of loss of consciousness	Qualitative-categorical nominal
Pt_seizure	Post-traumatic seizure	Qualitative-categorical nominal
Headache		Qualitative-categorical nominal
Vomiting		Qualitative-categorical nominal
Dizziness		Qualitative-categorical nominal
Behaviour	Parental report: Is child acting normally?	Qualitative-categorical nominal
Irritability		Qualitative-categorical nominal
Negative_feeding	Negative change in feeding habit	Qualitative-categorical nominal
Confusion		Qualitative-categorical nominal
Otorrhea		Qualitative-categorical nominal
Rhinorrhea		Qualitative-categorical nominal
Scalp_swelling	Scalp swelling	Qualitative-categorical nominal
Scalp_lacerations	Scalp lacerations	Qualitative-categorical nominal
Facial_lacerations	Facial lacerations	Qualitative-categorical nominal
hr	Heart rate	Quantitative- discrete
rr	Respiratory rate	Quantitative- discrete
Sp02	Oxygen saturation	Quantitative- discrete
bp	Blood pressure	Quantitative- discrete
Temperature		Quantitative-continuous

Weight	Weight (in kgs)	Quantitative- continuous
GCS	Glasgow coma scale score	Quantitative- discrete
orientation	Oriented to person/place/time	Qualitative-categorical nominal
Cn_palsy	Cranial nerve palsies	Qualitative-categorical nominal
Papilledema		Qualitative-categorical nominal
Anisocoria		Qualitative-categorical nominal
Motor_weakness	Motor weakness	Qualitative-categorical nominal
Sensory_loss	Sensory loss	Qualitative-categorical nominal
Abn_reflexes	Abnormal reflexes	Qualitative-categorical nominal
Prim_reflexes	Primitive reflexes	Qualitative-categorical nominal
Abn_tone	Abnormal muscle tone	Qualitative-categorical nominal
Abn_bulk	Abnormal muscle bulk	Qualitative-categorical nominal
Abn_coordination	Coordination abnormalities	Qualitative-categorical nominal
Abn_gait	Gait abnormalities	Qualitative-categorical nominal
intoxication	Suspected alcohol or drug intoxication	Qualitative-categorical nominal
Head_circum	Head circumference	Quantitative- continuous
Bulging_AF	Bulging anterior fontanelle	Qualitative-categorical nominal
Rhinorrhea		Qualitative-categorical nominal
Otorrhea		Qualitative-categorical nominal
Racoon_eyes	Racoon eyes	Qualitative-categorical nominal
Battle_sign	Battle sign	Qualitative-categorical nominal

Hemotympanum		Qualitative-categorical nominal
Scalp_lacerations	Scalp lacerations	Qualitative-categorical nominal
Facial_lacerations	Facial lacerations	Qualitative-categorical nominal
Periorb_edema	Periorbital edema	Qualitative-categorical nominal
Palp_skull_abn	Palpable skull deformities/fractures	Qualitative-categorical nominal
Scalp_hematoma	Scalp hematoma	Qualitative-categorical nominal
Trauma_above_clav	Signs of trauma above the clavicles	Qualitative-categorical nominal
Non_cranial_trauma	Other significant non-cranial trauma	Qualitative-categorical nominal
Ct_obtained	Ct scan obtained	Qualitative-categorical nominal
Ct_observation_period	Was the child observed in the accident and emergency unit after the first evaluation to determine if an CT scan should be done?	Qualitative-categorical nominal
Ct_main_finding	Ct scan main findings	Qualitative-categorical nominal
MRI_obtained	MRI obtained	Qualitative-categorical nominal
MRI_observation_period	Was the child observed in the accident and emergency unit after the first evaluation to determine if an MRI should be done?	Qualitative-categorical nominal
MRI_main_finding		Qualitative-categorical nominal

Management and outcome variables		
Interventions	Neurosurgical interventions performed	Qualitative-categorical nominal
Duration_intub	Duration of intubation for intubated patients	Quantitative- continuous
Adm_indication	Reason for hospital admission	Qualitative-categorical nominal
Adm_duration	Hospital admission duration	Quantitative- continuous
Disposition	Home, general ward, ICU, operating room, death	Qualitative-categorical nominal
Gos_outcome	Glasgow outcome scale score at discharge (1-5)	Quantitative- discrete

Analysis

The data will be sourced from interviewing the patients' guardians, physical examination of the patient done by the principle investigator, radiological imaging findings and observed management and outcome recorded by the principle investigator. The data collected by the questionaire will be entered into Epinfo 6 data sheet and analyzed or exported to SPSS version 17.0 statistical package for analysis. Descriptive statistics such as frequencies, proportions, measures of central location and variation (mean, mode, ranges and standard deviation) will be used for most variables (Age, Gender, among others). To compare means student t-test will be used or its non-parametric equivalent if data will not be normally distributed. To compare proportions the chi-square test will be undertaken. Statistical significance will be taken at the level $p < 0.05$. Results will be presented and displayed in form of frequency tables, bar graphs, linear graphs or charts as appropriate.

The positive predictive value, negative predictive value, sensitivity and specificity of the PECARN CT head rule in detecting clinically important TBI will be calculated from the true positive, false positive, true negative and false negative rates obtained from analysis of the data collected as shown in figure 3 on the following page. Discussions will be made by comparing findings of this study with other similar studies.

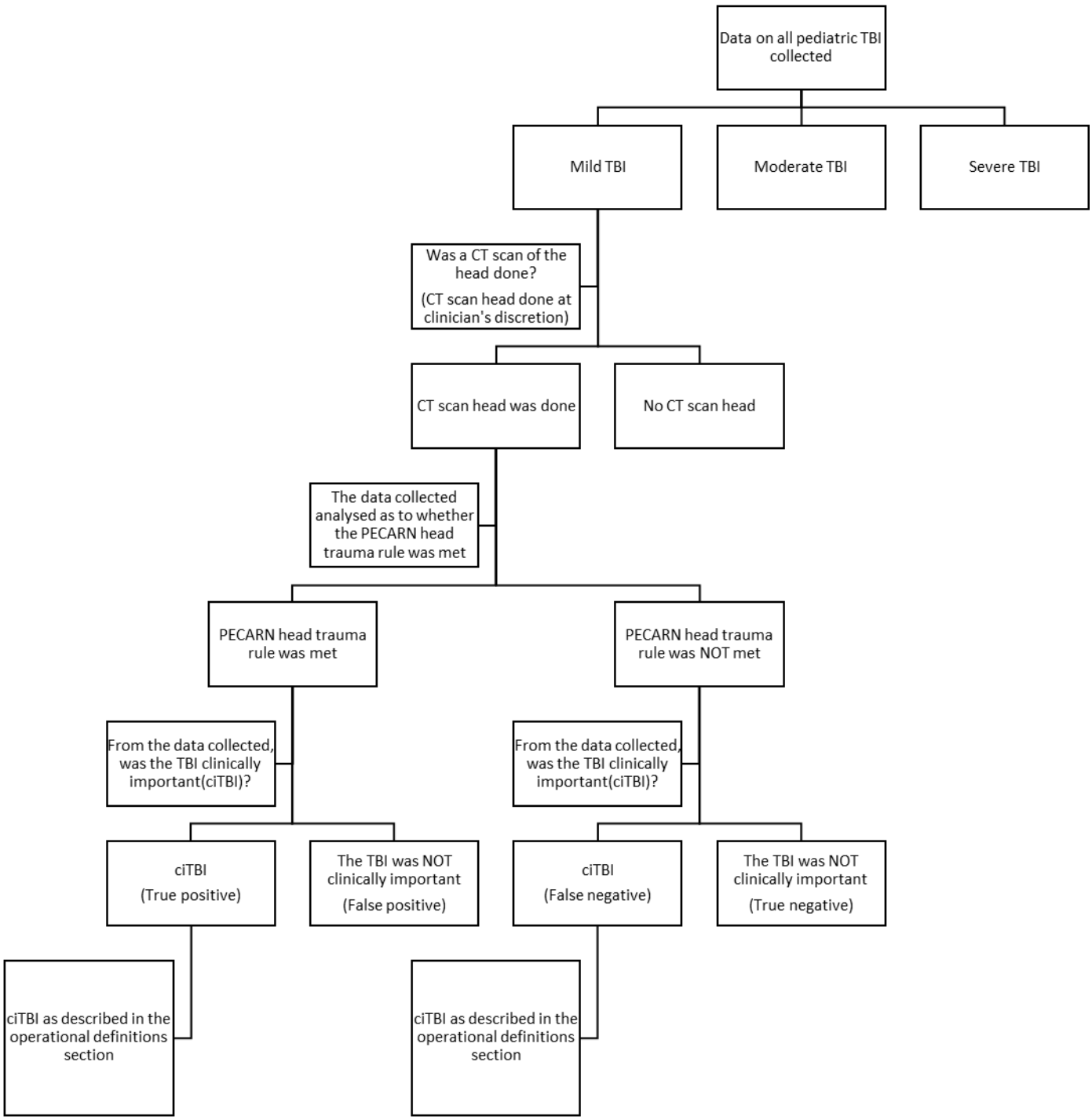


Figure 4. Data analysis with regards to PECARN CT head rule

DISSEMINATION OF RESULTS

The results of the study will be presented to Kenyatta National Hospital, University of Nairobi and to surgical scientific journals for publication. The results will also be presented in scientific conferences.

STUDY LIMITATIONS

- The ideal time would be to cover a much longer period (e.g. 5 years), but due to budgetary and time constraints, the study will be limited to 6 months. However, we are confident that results will achieve objectives of the study and will provide useful, actionable information to guide prevention, diagnosis and improved management of this condition in our setup.
- Patients who die at the scene of injury will not be included in the sample whereas they represent a vital part of the group.
- The study is limited to Kenyatta National Hospital hence the results may not represent what happens in the whole country.

RESULTS

One hundred and one children with head injury were seen in the accident and emergency unit over a six-month period between August 2019 and January 2020.

1. Demographic characteristics

There were 69 males (68.3%) and 32 females (31.7%). Male to female ratio, 2.2:1.

The age range recorded was between 6 months and 13 years (mean age 4.7 years). Most of the patients were between 3 and 5 years (42.6%). 71.3% were below 6 years.

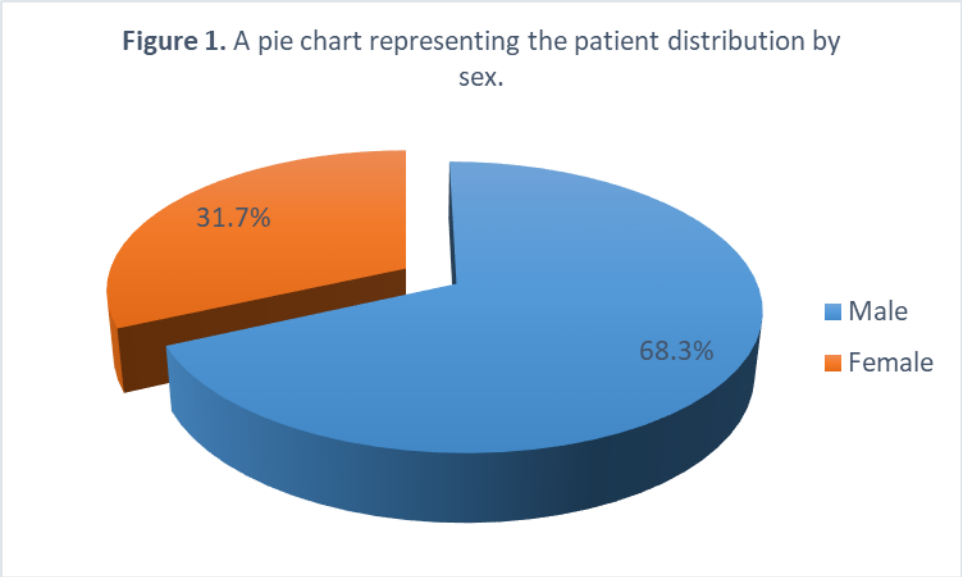
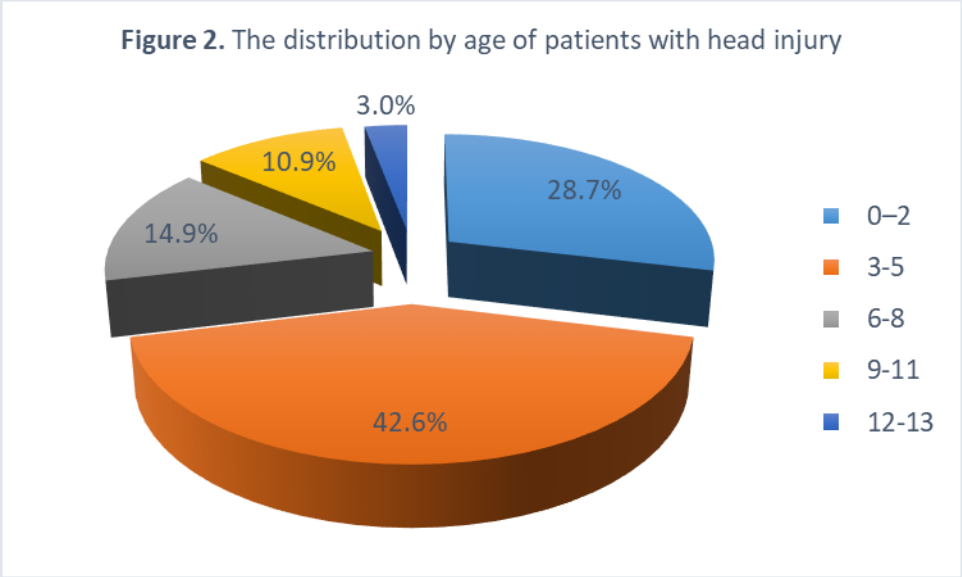


Figure 1. A pie chart representing the patient distribution by sex.



2. Injury pattern

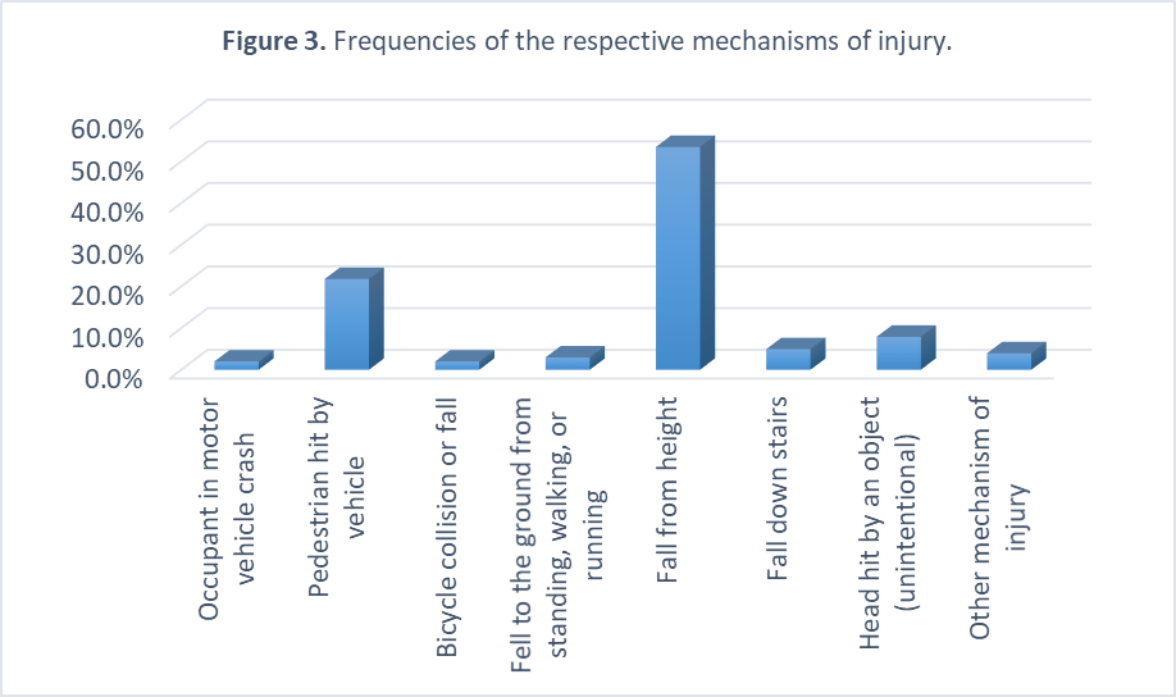
Majority of the cases (70.3%) were from Nairobi county followed by neighbouring counties of Muranga (6.9%) and Kajiado (4%).

The commonest mechanism of injuries was falls (63.5%). 53.5% of the cases seen resulted from falls from heights. 5% were from falls down stairs. 3% fell to the ground from standing, walking or running. 2% were from bicycle collisions or falls. Road traffic accidents accounted for 23.8% cases. 7.9% of cases had their head being hit by an object unintentionally. 1% of children were injured by walking or running into stationary objects. 4% of children had other mechanisms of injury which included jumping from moving vehicles and being shot by a stray bullet.

Most of the children who had history of falls were in the younger age groups (<=5 years). Falls from residential building balconies and buildings under construction while playing were the commonest causes of falls.

Road traffic accidents accounted for 24 of the cases. 21.8% were pedestrians hit by vehicles while 2% were vehicle occupants in a motor vehicle crash. Majority of the pedestrians hit by vehicles were hit by motorcycles.

8 children had their heads being hit by objects unintentionally. This included being hit by bricks at a construction site, having a temporary kiosk structure collapse on them, having a building collapse on them, and one had a pen penetrate through his pharynx when he fell from a bicycle while holding that pen in his mouth. Only one child was shot by a stray bullet.



3. Clinical presentation

The clinical presentation comprised of the following symptoms and signs:

Table 1. Frequency of clinical variables in patients in our series

Clinical variable	Frequency	Percentage
Post-traumatic amnesia	6	5.9%
History of loss of consciousness	74	73.3%

Post-traumatic seizure	22	22.2%
Headache	25	24.8%
Vomiting	52	51.5%
Parental report: child is not acting normally	94	93.1%
Irritability	78	77.2%
Negative change in feeding habit	83	82.2%
Confusion	17	16.8%
Otorrhea	9	8.9%
Rhinorrhea	2	2%
Scalp swelling	26	25.7%
Scalp lacerations	11	10.9%
Facial lacerations	12	11.9%

73.3% of the children had history of loss of consciousness with majority (60.4%) having a duration of greater than 5 minutes.

Post-traumatic seizures occurred in 22.2% of patients lasting for 2 to 30 minutes in duration. With a mean duration of 9.2 minutes. 78.9% of post-traumatic seizures occurred within 1 hour of injury.

24.8% of patients complained of headache which varied in severity from mild (2%), moderate (15.8%) and severe (5%).

Vomiting occurred in 51.5% of patients, with vomiting episodes ranging from 1 to 7. Timing of onset of vomiting after injury ranged from immediate to 24 hours after injury.

Table 2. Frequency of clinical signs in patients in our series

Clinical variable	Frequency	Percentage
Disoriented to person/place/time	41	40.6%
Focal signs	6	5.9%
Papilledema	3	3%
Anisocoria	6	5.9%
Rhinorrhea	2	2%
Otorrhea	9	8.9%
Racoon eyes	14	13.9%
Scalp lacerations	11	10.9%
Facial lacerations	11	10.9%
Periorbital edema	35	34.7%
Palpable skull deformities/fractures	13	12.9%
Scalp hematoma	15	14.9%
Other significant non-cranial trauma	9	8.9%

99 patients (98%) had blunt trauma and 2 patients (2%) had penetrating head injury.

Scalp lacerations were present in 8.9% of patients, while scalp hematomas were present in 14.9% of patients. 60% of the scalp hematomas were 3cm or less in size, while 40% were more than 3cm. They were located in the temporo-parietal region (7.9%), frontal region (23.1%) and occipital region (15.4%) and were boggy in character.

A number of base of skull fracture signs were found, including rhinorrhea (2%), otorrhea (8.9%) and raccoon eyes (13.9%).

Other significant non-cranial trauma mainly included injuries to the extremities such as radio-ulna fractures (4%), femur fractures (3%), tibia-fibula fractures (1%) and humerus fractures (1%).

4. Injury severity grades

Based on the Glasgow coma scale score obtained at presentation, mild head injuries accounted for 75 (74.3%) patients, which was the majority of patients. Followed by moderate head injury which accounted for 20 (19.8%) patients and severe head injury which accounted for 6 (5.9%) patients.

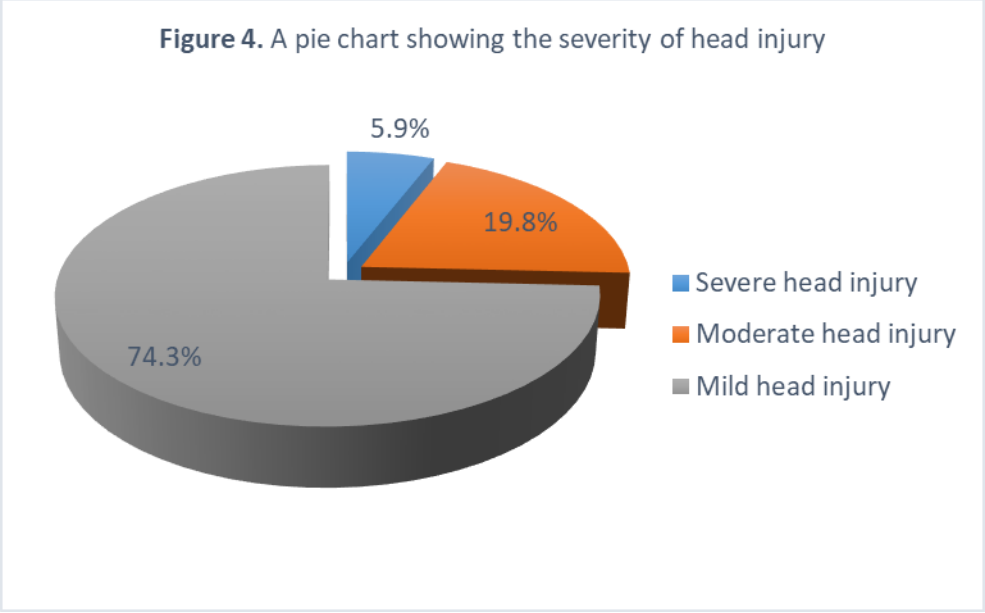


Figure 4. A pie chart showing the severity of head injury

Of the 6 patients who were severely injured, 4 had fallen from heights, 1 was a pedestrian hit by a vehicle and 1 had jumped from a moving vehicle.

Table 3. Frequency of clinical variables in different grades of head injury severity

Clinical variable	Mild head injury N=75	Moderate head injury N=20	Severe head injury N=6	Total
Post-traumatic seizure	13 (17.3%)	6 (30%)	3 (50%)	22
Headache	24 (32%)	1 (5%)	0	25
Vomiting	43 (57.3%)	7 (35%)	2 (33.3%)	52
Anisocoria	2 (2.6%)	0	4 (66.7%)	6
Focal signs	3 (4%)	1 (5%)	2 (33.3%)	6

Severe head injury was noted to be associated with anisocoria, focal signs, post-traumatic seizures and loss of consciousness. Headache and vomiting were more frequent in moderate and mild head injury.

5. CT scan

CT scans were done for 97 patients (96%). Indications for CT scanning sited included altered level of consciousness, pupillary changes, palpable depressed skull fractures, focal neurological deficits, penetrating head injury and closed head injury. None of the children who had a CT head done was observed in the accident and emergency unit after the first evaluation to determine if a CT should be done.

Table 4. Frequency of CT scan findings in patients in our series

CT scan finding	Frequency	Percentage
Skull fracture	62	61.4%
Intracranial hematoma	25	24.8%
Subarachnoid hemorrhage	5	5%
Cerebral contusions	20	19.8%
Traumatic infarction	1	1%
Pneumocephalus	7	6.9%
Herniations	4	4%
Diffuse axonal injury	2	2%
Scalp hematoma on CT scan	14	13.9%

Skull fractures were seen in 61.4% of patients. Majority (51.5%) were simple fractures, while 9.9% were compound fractures. 36.6% were linear fractures, 11.9% were comminuted fractures, 17.8% were depressed fractures and 6.9% were base of skull fractures.

Intracranial hematomas were seen in 24.8% of patients. Majority were extradural hematomas (12.9%), followed by subdural hematomas (8.9%), intracerebral hematomas (2%) and intraventricular hematomas (1%).

6. PECARN head trauma rule evaluation

Mild head injuries accounted for 75 patients but CT scans of the head were done for 70 of these patients. For each of these 70 patients, each patient's clinical presentation data was analyzed and checked to see whether it met the PECARN head trauma rule criteria. In addition, each patient's

CT scan findings were also analyzed to determine if they had clinically-important TBI as described in the operational definitions section. These patients were further subdivided into 2 groups as per the PECARN head trauma rule: those who are less than 2 years of age and those who are 2 years of age or older.

Those who were less than 2 years of age comprised 9 patients, only 8 of whom met the PECARN head trauma rule criteria. Of these patients, only 6 had clinically-important TBI. Hence a negative predictive value of 100.0%, a positive predictive value of 75.0%, a sensitivity of 100.0% and a specificity of 33.3% for patients less than 2 years old with clinically-important TBI.

Those who were 2 years of age or older comprised 61 patients, only 58 of whom met the PECARN head trauma rule criteria. Of these patients, only 30 had clinically-important TBI. Hence a negative predictive value of 100.0%, a positive predictive value of 51.7%, a sensitivity of 100.0% and a specificity of 9.7% for patients 2 years of age or older with clinically-important TBI.

Table 5. Evaluation of the PECARN head trauma rule

Age group	CiTBI present	PECARN positive	PECARN negative	Total
<2 years of age	Yes	6	0	6
	No	2	1	3
	Total	8	1	9
Age group	**CiTBI present**	**PECARN positive**	**PECARN negative**	**Total**
≥2 years of age	Yes	30	0	30
	No	28	3	31
	Total	58	3	61

7. Management

There were 94 (93.1%) hospital admissions while 7 (6.9%) patients were reviewed in the accident and emergency unit and discharged home. All the patients who were reviewed and discharged home had mild head injury. 5 of the injuries were from falls, while 1 was from a bicycle collision and 1 was from running into a stationary object.

The hospital admission duration ranged from 1 to 60 days with a mean of 4.47 days. Factors that were associated with prolonged hospital stay were high injury grade, anisocoria, focal signs, intracranial hemorrhage and ICU admission. Majority of the patients were admitted to the general ward (87.1%) followed by intensive care unit (5.9%). The indications for admission were mainly for neuro-observation (71.3%) and surgical intervention (28.7%).

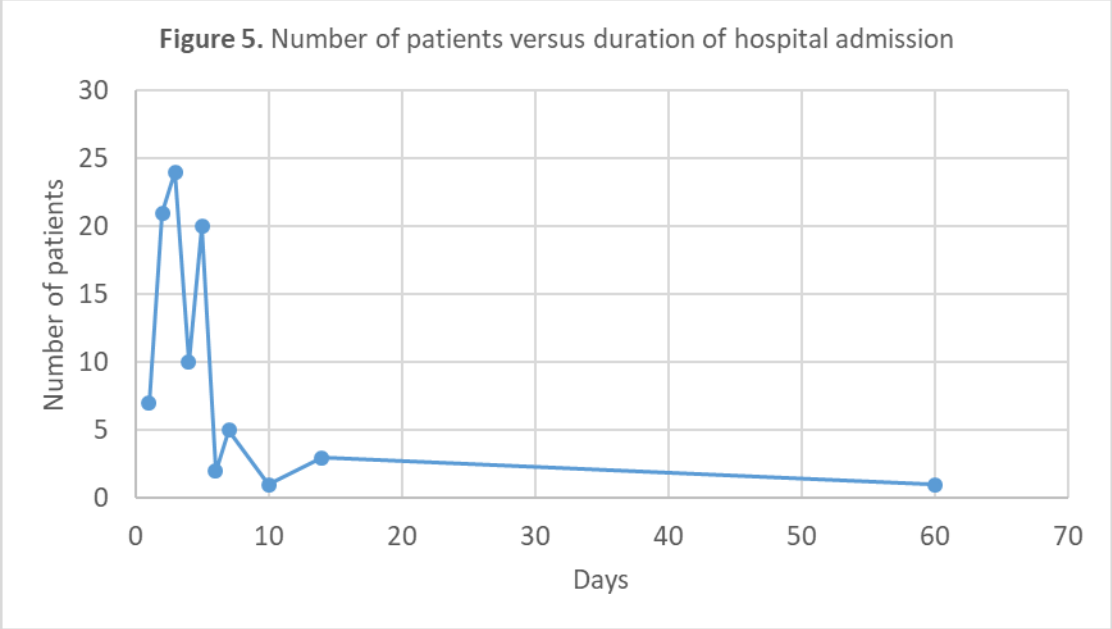


Figure 5. Number of patients versus duration of hospital admission

5 of the 6 children with severe head injuries were admitted to the intensive care unit, 4 had fallen from heights and 1 had jumped from a moving vehicle. 1 patient with mild head injury was admitted to ICU after surgery to remove a pen that had penetrated through his pharynx into the posterior fossa when he fell from a bicycle while holding that pen in his mouth.

Majority of the patients admitted to the general ward had mild head injury (76.2%), while 22.7% had moderate head injury and 1.1% had severe head injury.

22 (21.8%) children had neurosurgical intervention.

Table 6. Frequency of neurosurgical interventions in patients in our series

Operation	Frequency	Percentage
Elevation of a depressed skull fracture	10	9.9%
Evacuation of a hematoma	8	7.9%
Debridement	10	9.9%
Burr hole for tension pneumocephalus	1	1%
Transoral removal of a foreign object	1	1%

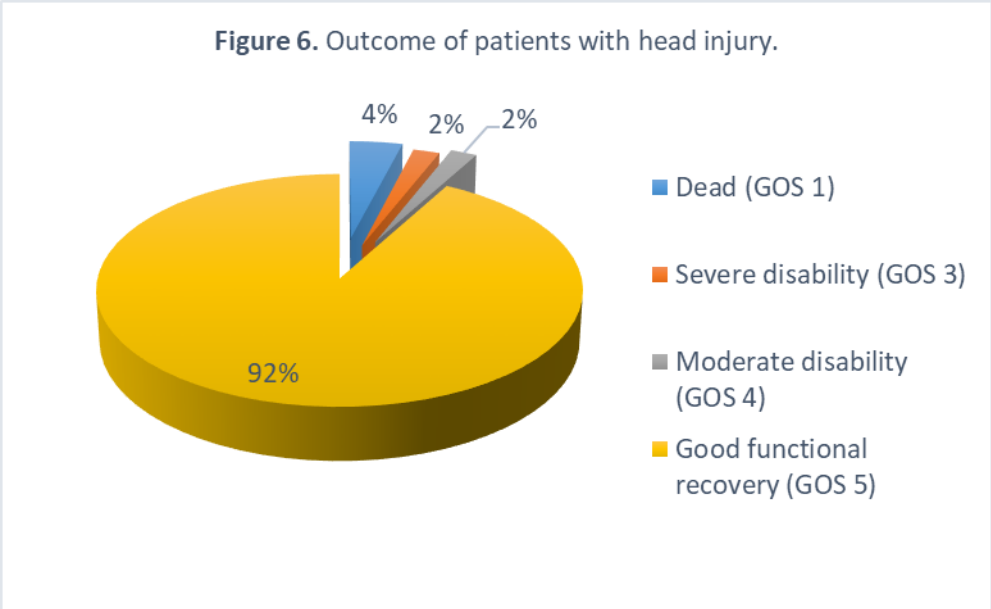
4 patients underwent intubation. The duration of intubation ranged from 2 to 30 days, with a mean of 9.5 days. All 4 intubated patients had severe head injury.

8. Outcome

At the time of discharge, outcome was assessed using the Glasgow outcome scale score. 93 (92%) patients had a good functional recovery, 2 (2%) patients had moderate disability, 2 (2%) patients had severe disability and 4 (4%) patients died.

Table 7. Frequency of the various outcomes met by patients in our series

Outcome	Frequency	Percentage
Dead	4	4%
Severe disability	2	2%
Moderate disability	2	2%
Good functional recovery	93	92%
Total	101	100%



Females had a better functional recovery rate (100%) than males (88.4%) but this was not statistically significant (P=0.258). Furthermore, 0% of female patients had severe disability as compared to 2.9% of male patients. Males had a higher mortality (5.8%) as compared to females (0%). Patients who were 6 years and older had a slightly better outcome than patients younger than 6 years. 7.1% of patients who were 6 years and older did not achieve good functional

recovery compared to 8.3% of patients younger than 6 years (P=0.029). Furthermore, mortality was also higher in those younger than 6 years at 5.8% as compared to the other age sets.

Patients who fell from a height and pedestrians who were hit by a vehicle were less likely to have good functional recovery (90.7% and 95.5% respectively) than those who were occupants in motor vehicle crash, involved in a bicycle collision, those who fell to the ground from standing, walking, or running, those who fell down stairs and those whose head was hit by an object (100%) but this was not statistically significant (P=0.258). Severe disability was more prevalent in pedestrians who were hit by vehicles (4.5%) while mortality was higher in children who fell from heights (7.4%). Children with skull fractures had a high proportion of good functional recovery (93.5%) when the type of head injury was analyzed, while children with intracerebral hematomas, subdural hematomas, subarachnoid hemorrhage, contusions and extradural hematomas had functional recovery rates of 50%, 55.6%, 80%, 85% and 100% respectively. Traumatic infarction had the lowest functional recovery rate of 0% and the greatest mortality (100%) but there was only 1 case recorded in the study. Intracerebral hematomas had a percentage mortality of 50%, followed by subdural hematomas (33.3%) and subarachnoid hemorrhage (20%) whereas contusions and extradural hematomas had a percentage mortality of 5% and 0% respectively.

Glasgow Coma Scale (GCS) score on admission was recorded for 101 patients, among whom 75(74.3%), 20 (19.8%), and 6 (5.9%) patients had mild, moderate, and severe head injury respectively. When outcome was cross-tabulated against admission GCS, it was observed that the proportion of patients with good functional recovery increased gradually from 16.7% in patients with GCS score 3-8, to 90% and 98.7% for patients with GCS scores 9–12 and 13-15, respectively (P=0.000). Moreover, the mortality rate significantly increased with decreasing GCS score. Mild head injury patients (GCS score 13-15) and moderate head injury patients (GCS score 9-12) had a percentage mortality of 0% as compared to 66.7% in those who had severe head injury (GCS score 3-8).

Anisocoria was recorded in 6 patients and was associated with poor outcomes (P=0.000). With regard to these patients, only 1 achieved good functional recovery, 1 had moderate disability and 3 (50%) died. Details regarding loss of consciousness following trauma was available for 101 patients, with 74 (73.3%) having a positive history. Patients who did not have traumatic loss of

consciousness were more likely to have good functional outcome (100%) whereas those who had such a history were more likely to have a lower proportion achieving good functional outcome (89.2%) and a higher proportion having severe disability (5.4%) (P=0.366).

22 (21.8%) of the patients in our series had surgical intervention. Patients who had surgical intervention were less likely to achieve functional outcome (86.4%) as compared to 93.7% in those managed conservatively. In addition, severe disability was also more common among surgically (4.5%) than conservatively managed patients (3.8%). Nonetheless, this finding was not statistically significant (P=0.574).

The mean duration of hospital stay was 4.47 days (±6.3) days. Patients who stayed for a shorter duration had a better outcome (P=0.003). Those who stayed for 1 day or less, those who stayed for 2 to 5 days and those who stayed for 6 to 10 days had a good functional recovery of 100%, 94.7% and 87.5% respectively. In contrast, those who stayed greater than 10 days had a functional recovery of 25%. 5.9% of the patients in our series were admitted to our Intensive Care Unit (ICU). These patients had a poorer outcome with only 16.7% achieving good functional recovery, as compared to 96.8% functional recovery achieved by patients not admitted in ICU (P=0.000).

Table 8. Clinical variables and their influence on patient outcome

Clinical Variables	No. of Patients	Mortality	Severe disability	Moderate disability	Good Recovery	P
Totals	101	4	2	2	93	
Sex						0.258
Male	69	4	2	2	61	
Female	32	0	0	0	32	
Age (years)						0.029
0–2	29	1	0	1	27	
3-5	43	3	2	0	39	
6-8	15	0	0	0	15	
9-11	11	0	1	0	10	
12-13	3	0	0	1	2	
Mechanism of injury						0.258
Occupant in motor vehicle crash	2	0	0	0	2	
Pedestrian hit by vehicle	22	0	1	0	21	
Bicycle collision or fall	2	0	0	0	2	
Fell to the ground from standing,	3	0	0	0	3	

walking, or running						
Fall from height	54	4	0	1	49	
Fall down stairs	5	0	0	0	5	
Head hit by an object	8	0	0	8	8	
Other mechanism of injury	4	0	1	1	2	
Glasgow Coma Scale score						0.000
≤8	6	4	0	1	1	
9–12	20	0	1	1	18	
≥13	75	0	1	0	74	
Pupillary abnormalities						0.000
Anisocoria	6	3	1	1	1	
Focal signs	6	1	1	1	3	0.001
History of loss of consciousness						0.366
Yes	74	4	2	2	66	
No	27	0	0	0	27	
History of convulsions						0.031
Yes	22	3	1	1	17	
No	77	1	1	1	74	

Duration of hospital admission (Days)						0.003
≤1	7	0	0	0	7	
2-5	75	3	0	1	71	
6-10	8	0	1	0	7	
>10	4	1	1	1	1	
ICU stay						0.000
Yes	6	4	0	1	1	
No	95	0	2	1	92	
Surgery done						0.574
Yes	22	1	1	1	19	
No	79	3	1	1	74	
Type of surgery						
Elevation of a skull fracture	10	0	0	0	10	0.812
Evacuation of a hematoma	8	1	0	1	6	0.075
Debridement	10	0	1	0	9	0.234
Burr hole for tension pneumocephalus	1	0	0	0	1	0.981
Transoral removal of a foreign object	1	0	0	0	1	0.710

DISCUSSION

In this study, we set out to look at the clinical pattern of pediatric traumatic brain injury and to evaluate the PECARN head trauma rule at Kenyatta National Hospital. With regards to the clinical pattern, we studied one hundred and one children who presented with head injuries in the accident and emergency unit over a six-month period between August 2019 and January 2020. The age pattern observed was an age range of between 6 months and 13 years and a mean age of 4.7 years, with a peak incidence at 42.6% between 3 and 5 years. Mwangi et also found similar findings with a mean age of 4.65 years and a peak incidence also between 3 and 5 years. Although he recorded a lower male:female ratio of 1.12:1, as compared to 2.2:1 recorded in this study, head injury in males was still more common than in females.[5] Onyemkpa et al also found a male:female ratio of 1.5:1.[4] This may reflect the fact that boys may be involved in more forceful and risky recreational activities than girls.

Our findings showed that majority of the head injuries occurred in children younger than 6 years with a peak in the 3-5 years age group at 42.6%. This was consistent with another Kenyan study which also reported that the 3-5 year age group has the highest risk and suggested that they should be targeted in prevention strategies.[5] Data from the U.S. Centers for Disease Prevention and Control regarding pediatric TBI was similar but only differed slightly, revealing that emergency consultations were most common among children aged 0–4 years.[68]

The commonest mechanism of injuries was falls (63.5%), which is consistent with other studies which have been done in our region. 53.5% of the cases seen resulted from falls from heights which affected mainly children residing in high-rise estates. In his study, Mwangi et al found that children residing in low and medium socio-economic high-rise estates have an increased risk of falls which may explain these findings.[5] Environmental factors such as poorly designed housing with unsafe balconies may also compound the risk. Younger children are also at a greater risk due to immature supporting and grasping movements in addition to increased level of activity and play.[5] Data from developed countries was also similar, showing that falls are the leading cause of TBI in children who are less than 14 years of age.[68]

Road traffic accidents accounted for 23.8% cases and were therefore the second commonest cause of pediatric TBI. Contrary to our findings, a Nigerian study found that road traffic

accidents were the most common cause of pediatric TBI in their setup.[4] According to the authors, factors contributing to this were the use of walking as a means of transportation common among school children coupled with the large number of poorly licensed motorists in their setup. In Kenya, the incidence of road traffic accidents may be due to underlying interrelated factors such as: rapid growth in motorisation and human population; increased traffic volume and movement; deficiencies and problems in road user behaviour; and poor public transport system with special reference to buses, matatus and motorcycles; the declining economic conditions in Kenya; deficiencies in road network development and maintenance; and deficiencies in road safety planning, management and interventions.[69] Lack of use of personal protective equipment at time of injury is an additional contributory factor. Seat belts and airbags for motor vehicle occupants[70, 71] and helmets for both motorcyclists[72,73] and bicyclists[74] have been demonstrated to substantially reduce the risk of head injury.

Mild head injuries accounted for 75 (74.3%) patients, which was the majority of patients. This was a similar finding to a prior local study where mild head injuries accounted for 83.2%.[5] A study by Trefan et al that was based on the UK population also found a significant burden of mild head injury at 86.2%.[75] This suggests that most injuries in young children are of low energy. In addition, children have unique biomechanical properties that may make their heads more resilient to external forces such as softer bones and open sutures that allow for some movement when responding to mechanical pressure.

Severe head injuries accounted for only 6 (5.9%) patients. Mwangi et al found that severe head injuries accounted for 8.9% of pediatric head injuries.[5] Kiboi et al found that children accounted for only 10% of severe head injuries at Kenyatta National Hospital.[62] The percentage of children with severe head injuries was lower in UK, where Trefan et al found that it accounted for 1.9%.[75] This was a multi-center study as opposed to our single center hospital based study. Severe head injury was noted to be associated with anisocoria(66.7%), focal signs(33.3%), post-traumatic seizures(50%) and loss of consciousness(33.3%). This was consistent with local studies. For instance Mwangi et al found that post-traumatic seizures and anisocoria accounted for 55.5% and 44.4% of severely injured patients respectively.[5]

CT scans were done for 97 patients (96%). This is a substantial increase from a CT scanning rate of 23.8% recorded in 2005 in the same facility, Kenyatta National Hospital.[5] The reason for the

low CT scanning rate cited by the authors was the unavailability of computed tomography facilities at that time, which resulted in more plain radiographs of the skull being routinely taken for almost all patients. In contrast, no skull radiograph was obtained in our study population. This may be explained by the significant increase in CT scan facilities countrywide, making it an easily accessible and efficient modality to evaluate pediatric TBI. Indications for CT scanning were found to be the same as previous local studies, this included: altered level of consciousness, pupillary changes, palpable depressed skull fractures, focal neurological deficits, penetrating head injury and closed head injury.[5, 64]

In our study, 75 patients had mild head injury and 70 of this patients underwent CT scanning. 45% of this patient did not end up having clinically-important TBI, therefore had underwent unnecessary CT scan imaging. International studies have observed that less than 10% of CT scans in minor pediatric TBI show traumatic brain injuries. In addition, injuries requiring neurosurgery are uncommon in children with GCS scores of 14-15.[42-45] Therefore a large number of CT scans done in minor pediatric TBI are usually unnecessary as observed in our findings. Avoidance of unnecessary CT imaging is important in head-injured children because ionizing radiation from CT scans can cause lethal radiation-induced malignancy.[46-48] Children are much more sensitive to radiation-induced cancer than adults. This was observed from the data obtained from survivors of the atomic bombs dropped on Japan in 1945. The reason for this is twofold. The first reason is that children have more time to express a cancer than adults because of their longer life expectancy. Secondly, children are essentially more sensitive to radiation simply because they have more dividing cells and radiation basically acts on dividing cells.[46] In addition, the organ doses from a CT scan are clearly higher for children than for adults. The approximate rate of lethal malignancies from CT is between 1 in 1000 and 1 in 5000 pediatric cranial CT scans, with risk increasing as age decreases.[46, 47]

The performance of the PECARN head trauma rule in its utility in detecting clinically important traumatic brain (ciTBI) injury was impressive when it was evaluated using the data we obtained. For patients who were less than 2 years of age, we found a negative predictive value of 100.0% and a sensitivity of 100.0% for detecting clinically-important TBI. These findings were comparable to those found by Kupperman et al, where they found a negative predictive of 100.0% and a sensitivity of 100.0% in this age group.[41] For patients who were 2 years of age or

older, we found a negative predictive value of 100.0% and a sensitivity of 100.0% for detecting clinically-important TBI. These findings were also comparable to those found by Kupperman et al, where they found a negative predictive of 99.95% and a sensitivity of 96.8% in this age group.[41] Based on these findings, application of the PECARN head trauma rule in mild pediatric TBI in our setup could limit CT use, protecting children from unnecessary radiation risks. It will be a valuable tool to assist clinicians in CT decision making after pediatric head trauma.

There were 94 (93.1%) hospital admissions while 7 (6.9%) patients were reviewed in the accident and emergency unit and discharged home under parental supervision. Although patients who were reviewed and discharged home had low risk mild TBI, this decision requires careful patient selection. Distance from the hospital, transport access and the availability of responsible care are important to ensure instructed monitoring and timely consultation should deterioration occur.[76] If the guardian is incompetent, unavailable, intoxicated, or otherwise incapacitated, provisions for admission must be made to ensure adequate observation of the child.

Of the 94 (93.1%) hospital admissions, the most common indication for admission was for neuro-observation (71.3%). This consisted of regular evaluation for development or progression of symptoms, evaluation of vital signs and frequent neurological examinations for early identification and intervention in deteriorating patients. Supportive management was also instituted including analgesia, anticonvulsants, antibiotics, antiemetics and intravenous fluids where indicated. The average duration of stay for patients admitted for neuro-observation in our setup was 3.22 days. The American Academy of Pediatrics recommends a period of neuro-observation that extends at least 24 hours. The reasoning behind this practice was based on the fact that most life-threatening complications occur within 24 hours after head injury.[50]

At time of discharge, 92% of the children in this study had a good functional recovery, 2% had moderate disabilities, 2% had severe disabilities and 4% died. Our mortality rate was slightly higher than that recorded by prior studies in this facility. The most recent study that was done by Mwangi et al recorded an overall mortality rate of only 1%.[5] Our mortality rate was also slightly higher than the 2% mortality rate recorded by Onyemkpa et al in a Nigerian tertiary center.[4] Luerssen et al[77] stated that age is a good indicator of mortality in cases of traumatic brain injury. There is an increased chance of survival in children, they tolerate periods of coma better than adults, and have fewer life-threatening complications. Those in the extreme age

groups have a higher mortality. Our current study showed a highly significant relation between age and outcome (p = 0.029). The age group that recorded the highest percentage mortality was that of those younger than 6 years, they had a percentage mortality of 5.8%. GCS score at admission was also found to significantly influence outcome. Mild head injury patients (GCS score 13-15) and moderate head injury patients (GCS score 9-12) had a percentage mortality of 0% as compared to 66.7% in those who had severe head injury (GCS score 3-8). Anisocoria was also associated with poor outcomes (P=0.000) with 50% of patients who presented with anisocoria dying. This findings were echoed by Luerssen et al[77] and Mwangi et al[5] who also illustrated a good correlation between the GCS score and anisocoria on neurological outcome.

This study had a number of limitations including the fact that this was a single center hospital based study because of the lack of a well-established trauma registry and head injury surveillance systems in Kenya. Therefore, our findings may not represent the pattern of pediatric TBI in the rest of the population. In addition, the center of study is a level 6 tertiary facility which is well equipped with facilities and specialists but it also receives a large proportion of pediatric TBI patients being referred from other centers which may have poor or no facilities, which may result in a bias when assessing the outcome of patients with pediatric TBI. Also, this study only looked at short term outcomes at discharge by using GOS which is an early global measure, therefore more detailed follow-up studies are needed to identify the specific long-term outcomes of pediatric TBI.

The pattern of pediatric TBI in Kenyatta National Hospital is characterized by a huge burden of mild head injury and a high incidence of fall-related head injuries which comprise a higher proportion of the more severe injuries. A follow-up study to assess the utilization and the role played by rehabilitation services, the socio-economic impact and long term outcome of pediatric TBI is recommended. There is currently no national pediatric head trauma registry and any system of surveillance to quantitatively characterize and accurately assess the scope of pediatric TBI. In developed countries such as USA, such systems have been shown to reduce mortality as illustrated by Nathens et al.[78] Majority of pediatric head injury is preventable, therefore prevention measures targeting groups at risk should be instituted. This should include improving public awareness of this menace. Child-proofing balcony rails and installation of window guards should be a priority especially for residents of highrise buildings. Prevention measures such as

road safety rules should be incorporated into the education curriculum to prevent children putting themselves at risk. Nonetheless, the road traffic safety legislation should also be strictly enforced.

CONCLUSION

Pediatric TBI is a significant health burden in our setup. This study has shown that males and children aged 3-5 years were affected more often. Falls were the leading cause of pediatric TBI at Kenyatta National Hospital with majority of this children falling from high-rise residential estates. Majority of the children who had history of falls were in the younger age groups (<=5 years). Prevention strategies should therefore be geared to prevention of falls and should involve the caregivers because most of this falls happen at home. Road traffic accidents were the second commonest cause of pediatric TBI with a child pedestrian being hit by a vehicle being the commonest mechanism. This shows that a deliberate effort has to be made to improve child pedestrian safety knowledge and road-crossing behavior. This may be achieved by developing a child pedestrian safety curriculum that can be taught in schools. Majority of the children had mild head injuries (74.3%) with the rest of the patients having moderate (19.8%) and severe head injuries (5.9%).

There has been a very substantial increase in CT scanning rate (96%), mainly because of increased availability of computed tomography facilities as compared to the past. This has led to better and more accurate evaluation of head injury patients. Unfortunately, this high CT scanning rate has led to a number of children undergoing unnecessary CT scan imaging. Up to 45% of children did not end up having clinically-important TBI after imaging in our study.

The PECARN head trauma rule was shown to be effective in its utility in detecting clinically important traumatic brain injury when it was evaluated using the data we obtained from our setup. It had a negative predictive value of 100.0% and a sensitivity of 100.0% for detecting clinically-important TBI. Based on these findings, application of the PECARN head trauma rule in mild pediatric TBI in our setup could limit CT use, protecting children from unnecessary radiation risks. The PECARN head trauma rule is inexpensive and uses clinical information that is readily available to the attending clinician. It will be a valuable tool to assist clinicians in CT decision making after pediatric head trauma. Clinicians handling pediatric TBI should therefore be educated on the PECARN head trauma rule and its application. If possible, it should be incorporated in the standard of care of this patients.

At time of discharge, 92% of the children in this study had a good functional recovery, 2% had moderate disabilities, 2% had severe disabilities and 4% died. The high rate of good functional recovery seen in Kenyatta National Hospital may be because it has a specialized neurosurgical unit with more neurosurgeons and post-graduate neurosurgical residents as compared other peripheral facilities. Predictors for poor outcomes included younger age, a lower Glasgow coma scale score, anisocoria and admission into our Intensive Care Unit. Another study with a larger sample size and a longer follow-up period is recommended to further evaluate outcomes after pediatric TBI.

RECOMMENDATIONS

1. Establishment of prevention measures.

   The first step in prevention should be improving public awareness of traumatic brain injury as a common cause of mortality and morbidity in children. The Ministry of Health should launch educative campaigns through both traditional and social media educate the public. Caregivers should be targeted and encouraged to adopt home safety measures. Measures that can be advocated include child-proofing balcony rails and installation of window guards to curb falls especially for residents of high-rise buildings. Safety gates may also be placed at the top and bottom of stairs. Caregivers should remove all tripping hazards which may include small area rugs and electrical cords that are loose. Non-slip mats may be utilized in the bathtub and on shower floors.

   To prevent pediatric TBI arising from road traffic accidents, child pedestrian safety knowledge and road-crossing behavior needs to be improved. This may be achieved by developing a child pedestrian safety curriculum that can be taught in schools. Road traffic safety regulations also need to be strictly enforced. The penalties for drunk driving, over speeding and driving while distracted by use of cellphones should be high enough to dissuade such practices.

2. Utilization of the PECARN head trauma rule.

   Kenyatta National Hospital clinicians involved in the care of pediatric TBI patients should be educated on the PECARN head trauma rule and its application. The PECARN head trauma rule should then incorporated in CT decision making in all pediatric patients with mild TBI. This will help to reduce unnecessary CT scan imaging.

3. Development of a head trauma registry

   The Ministry of Health should develop a head trauma registry to constantly surveil and assess pediatric TBI. This would proof useful in policy making, establishing prevention strategies and also assessing the effect of those strategies.

4. A follow-up study to assess the utilization and the role played by rehabilitation services, the socio-economic impact and long term outcome of pediatric TBI.

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APPENDICES

Consent form

Patient's Study Number:_____ **Date:_____**

Study Title:

Clinical pattern of pediatric traumatic brain injury and evaluation of the PECARN head trauma rule at Kenyatta National Hospital.

Investigators/Researchers :

Dr. Hudson Nganga Kamau

Supervisors:

Dr. Kiboi Julius Githinji (MB ChB), M.Med (Neurosurgery), Neurosurgeon. Department of Surgery, University of Nairobi

Dr. Michael Magoha (MB ChB), M.Med (Neurosurgery), Neurosurgeon. Department of Surgery, University of Nairobi

Introduction:

The purpose of this consent form is to provide you with the information you will need to assist you in deciding whether you want to participate in the study. This process is called 'Informed Consent'. Please read this consent information carefully and ask any questions or seek clarification on any matter concerning the study with which you are uncertain.

What is the purpose of the study?

Pediatric traumatic brain injury is a debilitating, incapacitating neurological condition causing significant morbidity. This study aims at providing information that will guide the development of strategies that will help in preventing and managing pediatric traumatic brain injury in our setup with a view of improving management and outcomes.

What will happen if you decide you want your child to be in this research study?

If you agree for your child to participate in this study, the following things will happen:

You will be interviewed by the investigator in a private area where you feel comfortable answering questions. The interview will last approximately 15 minutes. The interview will cover the child's clinical presentation. Then the principal investigator will examine the child and record his/her radiological imaging findings. At the point of

discharge from the hospital, the investigator will also record the clinical management administered and the outcome of the child.

Are there any risks, harms, discomforts associated with this study?

The study carries no extra risk to the patient. There will be no invasive procedures carried out in the study that may harm the patient. Refusing to take part in this study will not jeopardize your treatment in any way.

The information obtained about you will be kept in strict confidence. No specific information regarding you will be released to any person without your written permission. We will, however, discuss general overall findings of the study regarding all patients assessed but nothing specific will be discussed regarding you.

Are there any benefits being in this study?

The results obtained from the study will be used as a basis to improve the quality of care offered to patients diagnosed with traumatic brain injury treated at Kenyatta National Hospital. The information will be shared among treating clinicians and the hospital.

Will being in this study cost you anything?

Being in this study will cost you nothing.

Is there reimbursement for participating in this study?

There is no reimbursement for participating in this study.

What if you have questions in future?

If you ever have any questions about the study or about the use of the results you can contact the principal investigator, **Dr. NGANGA,** Tel.**0726-990679,** or his supervisors, Dr. **KIBOI**, Tel.**0720-498015**, and Dr. **MAGOHA**, Tel.**0710388279**. If any queries arise regarding your rights as a research participant you can contact the **Kenyatta National Hospital Ethics and Research Committee (KNH- ESRC)** by calling **2726300 Ext. 44355.**

What are your other choices?

Your decision to have your child participate in this research is voluntary. You are free to decline or withdraw participation of your child in the study at any time without injustice or loss of benefits. Just inform the investigator and the participation of your child in the study will be stopped. You do not have to give reasons for withdrawing your child if you do not wish to do so. Withdrawal of your child from the study will not affect the services your child is otherwise entitled to in this health facility or other health facilities.

CONSENT FORM (STATEMENT OF CONSENT)

The person being considered for this study is unable to consent for him/herself because he or she is a minor (a person less than 18 years of age). You are being asked to give your permission to include your child in this study.

Parent/guardian statement

I have read this consent form or had the information read to me. I have had the chance to discuss this research study with a study investigator. I have had my questions answered by him or her in a language that I understand. The risks and benefits have been explained to me. I understand that I will be given a copy of this consent form after signing it. I understand that my participation and that of my child in this study is voluntary and that I may choose to withdraw it any time. I understand that all efforts will be made to keep information regarding me and my child's personal identity confidential.

By signing this consent form, I have not given up my child's legal rights as a participant in this research study.

I voluntarily agree to my child's participation in this research study:

Yes No

Parent/Guardian signature /Thumb stamp: _____ Date _____

Parent/Guardian printed name: _____

Researcher's statement

I, the undersigned, have fully explained the relevant details of this research study to the participant named above and believe that the participant has understood and has knowingly given his/her consent.

Name: _____ Date: _____

Signature: _____

Role in the study: _____

IDHINI YA KUJIHUSISHA NA UTAFITI

Namba ya utafiti:_____ **Tarehe:_____**

Jina la utafiti:

Ruwaza kliniki ya kuumia kwa ubongo kwa watoto na uthibitishaji wa sheria ya PECARN inayoshughulikia maumivu ya kichwa katika Hospitali ya Taifa ya Kenyatta

Kanuni mpelelezi:

Dr. Hudson Nganga Kamau

Wasimamizi:
Dr. Kiboi Julius Githinji, Dr. Michael Magoha

Utangulizi

Madhumuni ya fomu hii ya idhini ni kukupa maelezo unayohitaji ili kukusaidia kuamua kama utashiriki katika utafiti huu.. Utaratibu huu unaitwa 'Mtaalam wa Kibali'. Tafadhali soma habari hii ya ridhaa kwa uangalifu na uulize maswali yoyote au utafute ufafanuzi juu ya suala lolote linalohusu kujifunza ambayo huyajui.

Kusudi la utafiti ni nini?
Kujeruhiwa kwa ubongo kwa watoto ni ugonjwa wa kudhoofisha na unaosababisha ulemavu mkubwa. Utafiti huu unalenga kutoa taarifa ambayo itasaidia maendeleo ya mikakati ambayo itasaidia kuzuia na kutibu kuumia kwa ubongo kwa watoto katika kanda letu kwa mtazamo wa kuboresha matibabu na matokeo.

Nini kitatokea ikiwa utaamua unataka mtoto wako awe katika utafiti huu wa utafiti?

Ikiwa unakubaliana na mtoto wako kushiriki katika utafiti huu, mambo yafuatayo yatatokea:

Utashughulikiwa na uchunguzi katika eneo la kibinafsi ambako unasikia vizuri kujibu maswali. Mahojiano itaendelea dakika 15. Mahojiano itakuwa inahusu mambo yaliofanyika mpaka mtoto akaletwa hospitalini. Kisha mchunguzi mkuu atamtazama mtoto na kurekodi matokeo yake ya picha ya radiology. Wakati wa kutolewa kutoka hospitali, matibabu aliyoyapata mtoto pamoja na hali yake yatarekodiwa.

Je, kuna hatari yoyote, madhara, kutokuwepo na uhusiano na utafiti huu?

Utafiti huu hauna hatari zaidi kwa mgonjwa. Hakutakuwepo na taratibu za uvamizi zitazofanywa katika utafiti ambazo zinazoweza kumdhuru mgonjwa. Kukataa kushiriki hakutahatarisha matibabu yako kwa njia yoyote.

Taarifa itakayopatikana kuhusu wewe itahifadhiwa kwa siri. Hakuna taarifa maalum kuhusu wewe itatolewa kwa mtu yeyote bila idhini yako iliyoandikwa. Hata hivyo, tutajadili matokeo ya jumla ya utafiti kuhusu wagonjwa wote watakayopimwa lakini hakuna kitu kitajadiliwa kuhusu wewe.

Je, kuna faida yoyote kuwa katika utafiti huu?

Matokeo ya utafiti huu yatatumika kama msingi wa kuboresha ubora wa huduma zinazotolewa kwa wagonjwa wanaojeruhiwa na kuumia kwa ubongo wanaotibiwa katika Hospitali ya Taifa ya Kenyatta. Maelezo yatashirikiwa kwa madaktari na hospitali.

Je, kuwa katika utafiti huu unadai gharama yoyote?

Kuwa katika utafiti huu hakutakupa gharama yoyote.

Je, Kuna malipo kwa kushiriki katika utafiti huu?

Hakuna malipo ya kushiriki katika utafiti huu.

Ikiwa una maswali baadaye?

Ikiwa una maswali yoyote kuhusu utafiti au juu ya matumizi ya matokeo unaweza kuwasiliana na mpelelezi mkuu, **Dr NGANGA**, Tel.**0726-990679**, au wasimamizi wake, **Dr KIBOI**, Tel.**0720-498015**, na Dr. **MAGOHA**, Tel.**0710388279**. Ikiwa una maswali yoyote kuhusu haki zako kama mshiriki wa utafiti unaweza kuwasiliana na **Kenyatta National Hospital Ethics and Research Committee (KNH- ESRC)** kwa kupiga **2726300 Ext. 44355.**

Je, una chaguo zingine?

Uamuzi wa mtoto wako kushiriki katika utafiti huu ni kwa hiari. Wewe uko huru kukataa au kumondoa mtoto wako kutoka kwa utafiti saa yoyote bila udhalimu au kupoteza faida. Mjulishe tu mpelelezi na ushiriki wa mtoto wako katika utafiti utasimamishwa. Si lazima kutoa sababu za kumtoa mtoto wako ikiwa hutaki kufanya hivyo. Kuondolewa kwa mtoto wako kutoka kwenye utafiti hautaathiri huduma ambazo mtoto wako ana haki kupata katika kituo hiki cha afya au vituo vingine vya afya.

<u>FOMU YA IDHINI</u>

Mtu anayezingatiwa kwa ajili ya utafiti huu hawezi kutoa idhini kwa sababu yeye ni mdogo (mtu chini ya umri wa miaka 18). Unatakiwa kutoa ruhusa yako kuingiza mtoto wako katika utafiti huu.

Taarifa ya mzazi / mlezi

Nimesoma fomu hii ya idhini au nilisomewai. Nimekuwa na fursa ya kujadili utafiti huu na mpelelezi mkuu. Maswali yangu yamejibiwa na yeye katika lugha ambayo ninaelewa. Hatari na faida zimeelezwa kwangu. Ninaelewa kwamba mimi nitapewa nakala ya fomu hii ya idhini baada ya kutia sahihi. Ninaelewa kwamba ushiriki wangu na wa mtoto wangu katika utafiti huu ni kwa hiari na kwamba nikonachaguo la kuondoka kutoka utafiti wakati wowote. Ninaelewa kwamba jitihada zote zitafanywa kuweka taarifa kuhusu mimi na mtoto wangu siri.

Kwa kuweka sahihi kwa fomu hii ya kibali, sijaacha haki za kisheria za mtoto wangu kama mshiriki katika utafiti huu.

Mimi kwa hiari ninakubali ushiriki wa mtoto wangu katika utafiti huu:

Ndio la

Sahihi ya Mzazi / Mlinzi / Thumb stamp: _____ Tarehe _____

Mzazi / Mlinzi jina la kuchapishwa: _____

Taarifa ya Mtafiti

Mimi, jina langu hapo chini, nimeeleza kikamilifu maelezo muhimu ya utafiti huu kwa mshiriki jina lake hapo juu na kuamini kuwa mshiriki ameelewa na amepeana idhini yake.

Jina: _____ Tarehe: _____

Sahihi: _____

Jukumu katika utafiti: _____

Data Collection Sheet

PATTERN OF PEDIATRIC TRAUMATIC BRAIN INJURY AND EVALUATION OF THE PECARN HEAD TRAUMA RULE AT KENYATTA NATIONAL HOSPITAL.

STUDY NUMBER	
DATE	
COUNTY OF RESIDENCE	

1. Biodata:

1.1. Age(yrs) (Must be age 13 years and below)	0-2….3-5…6-8.…9-11….12-13….
1.2. Date of birth (dd/mm/yyyy)	
1.3. Gender (M=1/F=2)	

2. History

Mechanism of injury	**YES=1**	**NO=2**	**SPECIFY**
2.1. Occupant in motor vehicle crash			Ejection….. Rollover…… Death of other passenger…… Speed……. Restraint use……
2.2. Pedestrian hit by vehicle			
2.3. Bicycle rider hit by a vehicle			Helmet use Yes….. No…..
2.4. Bicycle collision or fall			Helmet use Yes….. No…..
2.5. Other wheeled transport crash			Motorised Yes….. No…..
2.6. Fell to the ground from standing, walking, or running			

2.7. Walked or ran into stationary object			
2.8. Fall from height			Estimated height……
2.9. Fall down stairs			Number of stairs…….
2.10. Sport-related			Sport type…… Helmet use Yes….. No…..
2.11. Assault			
2.12. Head hit by an object (unintentional)			
2.13. Other mechanism of injury			

	YES=1	**NO=2**	**SPECIFY**
2.14. Post-traumatic amnesia			
2.15. History of loss of consciousness			Duration: <5s…. 5-60s….. 1-5min….. >5min……
2.16. Post-traumatic seizure			Duration…… How long after injury……..
2.17. Headache			Currently present: Yes…..No….. Severity: mild…. moderate….. severe….. Location of headache…….. Timing of onset……
2.18. Vomiting			Number of episodes……

			When vomiting started…….
2.19. Dizziness			
2.20. Parental report: Is child acting normally?			
2.21. Irritability			
2.22. Negative change in feeding habit			
2.23. Confusion			
2.24. Otorrhea			
2.25. Rhinorrhea			
2.26. Scalp swelling			
2.27. Scalp lacerations			
2.28. Facial lacerations			

3. Predisposing factors

	YES=1	**NO=2**	**SPECIFY**
3.1. Convulsive disorder or seizures prior to injury			
3.2. Alcohol abuse			
3.3. Drug abuse			
3.4. Single parent status			

3.5. Patient not living with biological parents			
3.6. Poor prenatal care			
3.7. Parents involved in substance abuse			
3.8. Gestational age at birth (In weeks)			
3.9. Maternal age (In years)			

4. Physical examination

4.1. Vital signs at initial presentation

4.1.1. Heart rate	
4.1.2. Respiratory rate	
4.1.3. Oxygen saturation	
4.1.4. Blood pressure	
4.1.5. Temperature	
4.1.6. Weight (in kgs)	

4.2. CNS examination

4.2.1. GCS	E=	V=	M=	Total=

	YES=1	**NO=2**	**SPECIFY**
4.2.2. Oriented to person/place/time			

4.2.3. Cranial nerve palsies			
4.2.4. Papilledema			
4.2.5. Anisocoria			
4.2.6. Motor weakness			
4.2.7. Sensory loss			
4.2.8. Abnormal reflexes			
4.2.9. Primitive reflexes			
4.2.10. Abnormal muscle tone			
4.2.11. Abnormal muscle bulk			
4.2.12. Coordination abnormalities			
4.2.13. Gait abnormalities			
4.2.14. Suspected alcohol or drug intoxication			

4.3. Head examination

	YES=1	**NO=2**	**SPECIFY**
4.3.1. Head enlargement (specify circumference)			
4.3.2. Bulging anterior			

fontanelle			
4.3.3. Rhinorrhea			
4.3.4. Otorrhea			
4.3.5. Racoon eyes			
4.3.6. Battle sign			
4.3.7. Hemotympanum			
4.3.8. Scalp lacerations			
4.3.9. Facial lacerations			
4.3.10. Periorbital edema			
4.3.11. Palpable skull deformities/fractures			
4.3.12. Scalp hematoma			Size: <1 cm…… 1–3 cm…… >3 cm…… Location: frontal….. temporal–parietal…… occipital…… Character: boggy…….firm…….

4.4. Additional injuries on the rest of the body

	YES=1	**NO=2**	**SPECIFY**
4.4.1. Signs of trauma above the clavicles			Specify…….. Location…….
4.4.2. Other significant non-			Specify……..

cranial trauma			Location…….

5. CT scan head imaging

	YES=1	**NO=2**	**SPECIFY**
5.1. Was CT head obtained			If Yes, indications for CT: ……. ……..
5.2. Was the child observed in the accident and emergency unit after the first evaluation to determine if an CT should be done?			

CT findings if CT head was obtained:

	YES=1	**NO=2**
5.3. Skull fracture		

If skull fracture present:	
5.3.1. Location	
5.3.2. Type	Simple……..Compound……. Linear…….Comminuted…….Depressed (specify degree of displacement)..…… Diastatic…… Base of skull fracture……

	YES=1	**NO=2**
5.4. Intracranial hematoma		

If intracranial hematoma present:	
5.4.1. Type	EDH=1…..SDH=2…..ICH=3……IVH=4…….
5.4.2. Location	
5.4.3. Volume (in cc)	
5.4.4. Midline shift (mm)	
5.4.5. Thickness (mm)	
5.4.6. Intraventricular extension for ICH(specify)	

	YES=1	**NO=2**
5.5. Subarachnoid hemorrhage		

If subarachnoid hemorrhage present:	
5.5.1. Location	
5.5.2. Modified Fisher grade	

	YES=1	NO=2	SPECIFY
5.6. Cerebral contusions			
5.7. Traumatic infarction			
5.8. Pneumocephalus			

	YES=1	NO=2	SPECIFY
5.9. Herniations			

	YES=1	NO=2
5.10. Diffuse axonal injury		
5.11. Sigmoid sinus thrombosis		

	YES=1	NO=2
5.12. Scalp hematoma		

5.13. Marshall grade	
5.14. Other findings	

6. MRI Brain

	YES=1	**NO=2**	**SPECIFY**
6.1. Was MRI brain obtained			If Yes, indications for CT: ……. ……..
6.2. Was the child observed in the accident and emergency unit after the first evaluation to determine if an MRI should be done?			

	YES=1	**NO=2**
6.3. Intracranial hematoma		

If intracranial hematoma present:	
6.3.1. Type	EDH=1…..SDH=2…..ICH=3……IVH=4…….
6.3.2. Location	
6.3.3. Volume (in cc)	
6.3.4. Midline shift (mm)	
6.3.5. Thickness (mm)	
6.3.6. Intraventricular extension for ICH(specify)	

	YES=1	**NO=2**
6.4. Subarachnoid hemorrhage		

If subarachnoid hemorrhage present:	
6.4.1. Location	
6.4.2. Modified Fisher grade	

	YES=1	**NO=2**	**SPECIFY**
6.5. Cerebral contusions			
6.6. Traumatic infarction			
6.7. Pneumocephalus			

	YES=1	**NO=2**	**SPECIFY**
6.8. Herniations			

	YES=1	**NO=2**
6.9. Diffuse axonal injury		
6.10. Sigmoid sinus thrombosis		

	YES=1	NO=2
6.11. Scalp hematoma		

6.12. Marshall grade	
6.13. Other findings	

7. Interventions

Neurosurgical intervention

	YES=1	NO=2	SPECIFY
7.1. Elevation of a depressed skull fracture			
7.2. Ventriculostomy			
7.3. Evacuation of a hematoma			
7.4. Lobectomy			
7.5. Debridement			
7.6. Duroplasty			
7.7. Intracranial pressure monitoring			
7.8. Other			

Other interventions

	YES=1	**NO=2**	**SPECIFY**
7.9. Intubation			Duration of intubation……

8. Admission

	YES=1	**NO=2**	**SPECIFY**
8.1. Hospital admission			Duration …… Indication……..

9. Disposition

Home=1	
General ward=2	
Intensive care unit=3	
Operating room=4	
Death=5	

10. Glasgow outcome scale score at discharge

Death=1	
Persistent vegetative state=2	
Severe disability=3	
Moderate disability=4	
Good functional recovery=5	

Data collection sheet with the interview section in kiswahili (Section 1-3)

RUWAZA KLINIKI YA KUUMIA KWA UBONGO KWA WATOTO NA UTHIBITISHAJI WA SHERIA YA PECARN INAYOSHUGHULIKIA MAUMIVU YA KICHWA KATIKA HOSPITALI YA TAIFA YA KENYATTA.

NAMBARI YA UTAFITI	
TAREHE	
KITIKA YA KUTUMA	

1. Habari kuhusu mgonjwa:

1.1. Umri (miaka) (Lazima uwe na umri wa miaka 13 na chini)	0-2….3-5…6-8.…9-11….12-13….
1.2. Tarehe ya kuzaliwa (dd/mm/yyyy)	
1.3. Jinsia (M=1/F=2)	

2. Historia

Mechanism of injury	**NDIO=1**	**LA=2**	**FAFANUA**
2.1. Alibebwa na gari lililohusika na ajali			Alitupwa nje ya gari….. Gari liligeuka…… Kifo cha abiria wengine…… kasi……. Matumizi ya vifaa vya kuzuia abiria…….
2.2. Mwenda kwa miguu aliyegongwa na gari			
2.3. Mwenda kwa baiskeli aliyegongwa na gari			Alitumia helmeti Ndio….. La…..
2.4. Mgongano wa baiskeli au kuanguka			Alitumia helmeti Ndio….. La…..

2.5. ajali wa usafiri mwingine wa magurudumu			Ambao unatumia mtambo Ndio….. La…..
2.6. Kuanguka chini kutoka kwa kusimama, kutembea, au kukimbia			
2.7. Alikimbia au alitembea na kugonga kitu kilichoimara			
2.8. Kuanguka kutoka kwa urefu			Kiwango cha urefu ……
2.9. Kuanguka kwa ngazi			Idadi ya ngazi …….
2.10. Kuhusiana na michezo			Aina ya michezo …… Alitumia helmeti Ndio….. La…..
2.11. Kushambuliwa			
2.12. Kichwa kugongwa na kitu(ajali)			
2.13. Mfumo mwingine wa kuumia			

	NDIO=1	**LA=2**	**FAFANUA**
2.14. Usahaulifu baada ya kuumia			
2.15. Historia ya kupoteza fahamu			Muda: <5s…. 5-60s….. 1-5 dakika….. >5 dakika……
2.16. Pindupindu au mshtuko baada ya kuumia			Muda …… Muda gani baada ya kuumia ……..
2.17. maumivu ya kichwa			Hivi sasa iko: Ndiyo ..... La.....

			Ukali: kidogo.... wastani ..... kali .... Mahali ya maumivu ya kichwa …….. Muda wa kuanza ……
2.18. kutapika			Mara ngapi…… Kutapika ulianza lini…….
2.19. Kizunguzungu			
2.20. Ripoti ya wazazi: Je! Mtoto anaendelea kawaida?			
2.21. Hasira na kulialia			
2.22. Mabadiliko mabaya katika tabia ya kula			
2.23. Kuchanganyikiwa			
2.24. Maji kutoka kwa masikio			
2.25. Maji kutoka kwa mapua			
2.26. Kufura kwa ngozi ya fuvu la kichwa			
2.27. kukatika kwa ngozi ya fuvu la kichwa			
2.28. kukatika kwa ngozi ya uso			

3. Sababu ambazo zilizochangia kuumia

	NDIO=1	**LA=2**	**FAFANUA**
3.1. Pindupindu au mshtuko kabla ya kuumia			
3.2. Utumizi wa pombe			
3.3. Utumizi wa madawa ya kulevya			
3.4. Hali ya mzazi mmoja			
3.5. Mgonjwa haishi na wazazi wa kibiolojia			
3.6. Huduma mbaya kabla ya kujifungua			
3.7. Wazazi wanaohusika katika matumizi ya madawa ya kulevya			
3.8. Alibebwa kwa wiki ngapi kabla ya kuzaliwa			
3.9. Umri wa mama(kwa miaka)			

4. Physical examination

4.1. Vital signs at initial presentation

4.1.1. Heart rate	
4.1.2. Respiratory rate	
4.1.3. Oxygen saturation	
4.1.4. Blood pressure	

4.1.5. Temperature	
4.1.6. Weight (in kgs)	

4.2. CNS examination

4.2.1. GCS	E=	V=	M=	Total=

	YES=1	**NO=2**	**SPECIFY**
4.2.2. Oriented to person/place/time			
4.2.3. Cranial nerve palsies			
4.2.4. Papilledema			
4.2.5. Anisocoria			
4.2.6. Motor weakness			
4.2.7. Sensory loss			
4.2.8. Abnormal reflexes			
4.2.9. Primitive reflexes			
4.2.10. Abnormal muscle tone			
4.2.11. Abnormal muscle bulk			
4.2.12. Coordination			

abnormalities			
4.2.13. Gait abnormalities			
4.2.14. Suspected alcohol or drug intoxication			

4.3. Head examination

	YES=1	**NO=2**	**SPECIFY**
4.3.1. Head enlargement (specify circumference)			
4.3.2. Bulging anterior fontanelle			
4.3.3. Rhinorrhea			
4.3.4. Otorrhea			
4.3.5. Racoon eyes			
4.3.6. Battle sign			
4.3.7. Hemotympanum			
4.3.8. Scalp lacerations			
4.3.9. Facial lacerations			
4.3.10. Periorbital edema			
4.3.11. Palpable skull deformities/fractures			

4.3.12. Scalp hematoma			Size: <1 cm…… 1–3 cm…… >3 cm…… Location: frontal….. temporal–parietal…… occipital…… Character: boggy…….firm…….

4.4. Additional injuries on the rest of the body

	YES=1	**NO=2**	**SPECIFY**
4.4.1. Signs of trauma above the clavicles			Specify…….. Location…….
4.4.2. Other significant non-cranial trauma			Specify…….. Location…….

5. CT scan head imaging

	YES=1	**NO=2**	**SPECIFY**
5.1. Was CT head obtained			If Yes, indications for CT: ……. ……..
5.2. Was the child observed in the accident and emergency unit after the first evaluation to determine if an CT should be done?			

CT findings if CT head was obtained:

	YES=1	**NO=2**
5.3. Skull fracture		

If skull fracture present:	
5.3.1. Location	
5.3.2. Type	Simple……..Compound……. Linear…….Comminuted…….Depressed (specify degree of displacement)..…… Diastatic…… Base of skull fracture……

	YES=1	**NO=2**
5.4. Intracranial hematoma		

If intracranial hematoma present:	
5.4.1. Type	EDH=1…..SDH=2…..ICH=3……IVH=4…….
5.4.2. Location	
5.4.3. Volume (in cc)	
5.4.4. Midline shift (mm)	
5.4.5. Thickness (mm)	
5.4.6. Intraventricular extension for ICH(specify)	

	YES=1	**NO=2**
5.5. Subarachnoid hemorrhage		

If subarachnoid hemorrhage present:	
5.5.1. Location	
5.5.2. Modified Fisher grade	

	YES=1	**NO=2**	**SPECIFY**
5.6. Cerebral contusions			
5.7. Traumatic infarction			
5.8. Pneumocephalus			

	YES=1	**NO=2**	**SPECIFY**
5.9. Herniations			

	YES=1	**NO=2**
5.10. Diffuse axonal injury		
5.11. Sigmoid sinus thrombosis		

	YES=1	NO=2
5.12. Scalp hematoma		

5.13. Marshall grade	
5.14. Other findings	

6. MRI Brain

	YES=1	NO=2	SPECIFY
6.1. Was MRI brain obtained			If Yes, indications for CT: ……. ……..
6.2. Was the child observed in the accident and emergency unit after the first evaluation to determine if an MRI should be done?			

	YES=1	NO=2
6.3. Intracranial hematoma		

If intracranial hematoma present:	
6.3.1. Type	EDH=1…..SDH=2…..ICH=3……IVH=4…….
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6.3.3. Volume (in cc)	
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6.3.5. Thickness (mm)	
6.3.6. Intraventricular extension for ICH(specify)	

	YES=1	**NO=2**
6.4. Subarachnoid hemorrhage		

If subarachnoid hemorrhage present:	
6.4.1. Location	
6.4.2. Modified Fisher grade	

	YES=1	**NO=2**	**SPECIFY**
6.5. Cerebral contusions			
6.6. Traumatic infarction			
6.7. Pneumocephalus			

	YES=1	**NO=2**	**SPECIFY**

6.8. Herniations			

	YES=1	**NO=2**
6.9. Diffuse axonal injury		
6.10. Sigmoid sinus thrombosis		

	YES=1	**NO=2**
6.11. Scalp hematoma		

6.12. Marshall grade	
6.13. Other findings	

7. Interventions

Neurosurgical intervention

	YES=1	**NO=2**	**SPECIFY**
7.1. Elevation of a depressed skull fracture			
7.2. Ventriculostomy			
7.3. Evacuation of a hematoma			
7.4. Lobectomy			

7.5. Debridement			
7.6. Duroplasty			
7.7. Intracranial pressure monitoring			
7.8. Other			

Other interventions

	YES=1	**NO=2**	**SPECIFY**
7.9. Intubation			Duration of intubation……

8. Admission

	YES=1	**NO=2**	**SPECIFY**
8.1. Hospital admission			Duration …… Indication……..

9. Disposition

Home=1	
General ward=2	
Intensive care unit=3	
Operating room=4	

Death=5	

10. Glasgow outcome scale score at discharge

Death=1	
Persistent vegetative state=2	
Severe disability=3	
Moderate disability=4	
Good functional recovery=5	

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21st May, 2019

Dr. Hudson Kamau Ng'ang'a
Reg. No.H58/75291/2014
Dept. of Surgery
School of Medicine
College of Health Sciences
University of Nairobi

Dear Dr. Kamau

RESEARCH PROPOSAL: CLINICAL PATTERN OF PAEDIATRIC TRAUMATIC BRAIN INJURY AND EVALUATION OF THE PECARN HEAD TRAUMA RULE AT KENYATTA NATIONAL HOSPITAL – A PROSPECTIVE STUDY (P60/02/2019)

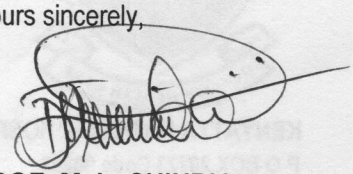
This is to inform you that the KNH- UoN Ethics & Research Committee (KNH- UoN ERC) has reviewed and **approved** your above research proposal. The approval period is 21st May 2019 – 20th May 2020.

This approval is subject to compliance with the following requirements:

a. Only approved documents (informed consents, study instruments, advertising materials etc) will be used.
b. All changes (amendments, deviations, violations etc.) are submitted for review and approval by KNH-UoN ERC before implementation.
c. Death and life threatening problems and serious adverse events (SAEs) or unexpected adverse events whether related or unrelated to the study must be reported to the KNH-UoN ERC within 72 hours of notification.
d. Any changes, anticipated or otherwise that may increase the risks or affect safety or welfare of study participants and others or affect the integrity of the research must be reported to KNH- UoN ERC within 72 hours.
e. Clearance for export of biological specimens must be obtained from KNH- UoN ERC for each batch of shipment.
f. Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. (*Attach a comprehensive progress report to support the renewal*).
g. Submission of an *executive summary* report within 90 days upon completion of the study. This information will form part of the data base that will be consulted in future when processing related research studies so as to minimize chances of study duplication and/ or plagiarism.

For more details consult the KNH- UoN ERC website **http://www.erc.uonbi.ac.ke**

Yours sincerely,



PROF. M. L. CHINDIA
SECRETARY, KNH-UoN ERC

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