HEAD COMPUTED TOMOGRAPHY SCAN PATTERNS OF PATIENTS SUSPECTED TO HAVE CEREBROVASCULAR DISEASE AT MOI TEACHING AND REFERRAL HOSPITAL –ELDORET, KENYA.

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SM/PGR/03/13

This Research thesis submitted in partial fulfillment of the requirements for the award of a Master of Medicine in Radiology and Imaging, Moi University.

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DECLARATION

Student’s Declaration

I declare that this is my original work and has not been presented in any other university or institution for an award of a degree or any academic credit. No part of this work may be reproduced or transmitted in any form without prior permission from the author and or Moi University.

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DEDICATION:

I would like to dedicate this research dissertation to God Almighty on Whose shoulders I stand. To my beloved wife Dr. Millicent Korir and my children; To my parents Mr. and Mrs. Edwin Chumo for their undying support in my pursuit for excellence. To my late brother Caesar Kibichii –may his soul rest in eternal peace; my siblings Caroline Chemutai and Pascal King; no one could have better moral support.

I am thus forever indebted to you all.
Background: Cerebrovascular disease (CVD) is a major cause of morbidity and mortality in both developed and developing countries due to aberrant and emerging trends in lifestyle. With rapid scan times and software aided ease of linear attenuation coefficients calculation, Computed tomography (CT) scanners are preferred for imaging CVDs. In spite of these, CVD patterns have not been established at Moi Teaching and Referral Hospital (MTRH), Kenya.

Objective: To describe the brain CT scan patterns of patients suspected to have CVD at MTRH.

Methods: This was a descriptive cross-sectional study done at Moi Teaching and Referral Hospital-Kenya from October 2014 to July 2015 where consecutive sampling technique was used on consenting patients with a clinical diagnosis of CVD who had CT scans. A structured questionnaire which had socio-demographic characteristics, signs and symptoms was administered to participants. Findings on CT scan and HU characteristic were noted and filled by interviewer into the questionnaire. Categorical variables were summarized as frequencies with corresponding percentages while continuous variables were summarized as median and the corresponding inter quartile range given. Data analysis and statistical computing was done with R Core Team software and results were presented in tables and graphs.

Results: A total of 225 participants were included in the study. The median age was 60.0 (IQR: 44.0, 77.0) years with a minimum and a maximum of 2.0 and 102 years respectively. One hundred and eighteen (52.4%) were male. More than a third of the participants, 36.9% had ischemic stroke, and 27 (15.1%) suffered hemorrhagic stroke. Cerebral atrophy was diagnosed in 41 (23.0%) of the participants. Forty two, 23.5%, of the total participants had normal CT, and 9 (5.0%) were diagnosed of tumors. Of those specifically with stroke, ischemic types were 66(71.1%) with an average HU of 18.0 +/- 8.6 and hemorrhagic stroke 27(28.9%) and average HU of 60.8 +/- 9.1. Majority, 26 (11.6%), of the CVD scan patterns were located in the parieto-temporal region.

Conclusion: The most common CT scan finding of patients suspected to have CVD was ischemic stroke and the average HU for Ischemic stroke was 18.0 +/- 8.6 and 60.8 +/- 9.1 for hemorrhagic stroke.

Recommendation: The use of Hounsfield Unit values in characterization of stroke is recommended.
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ACKNOWLEDGEMENT

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<thead>
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<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>ACA</td>
<td>Anterior cerebral artery</td>
</tr>
<tr>
<td>CVD</td>
<td>Cerebro-vascular Disease</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>GBM</td>
<td>Glioblastoma Multiforme</td>
</tr>
<tr>
<td>HU</td>
<td>Hounsfield unit</td>
</tr>
<tr>
<td>HS</td>
<td>Haemorhagic stroke</td>
</tr>
<tr>
<td>ICH</td>
<td>Intra-cerebral hemorrhage</td>
</tr>
<tr>
<td>IREC</td>
<td>Institutional Research and Ethics Committee</td>
</tr>
<tr>
<td>IS</td>
<td>Ischaemic stroke</td>
</tr>
<tr>
<td>IVB</td>
<td>Intra-ventricular bleed</td>
</tr>
<tr>
<td>MCA</td>
<td>Middle Cerebral Artery</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>MTRH</td>
<td>Moi Teaching and Referral Hospital</td>
</tr>
<tr>
<td>PCA</td>
<td>Posterior cerebral artery</td>
</tr>
<tr>
<td>SDH</td>
<td>Subdural hematoma</td>
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</table>
CHAPTER ONE: INTRODUCTION

1.1 BACKGROUND INFORMATION

Cerebrovascular disease (CVD) which can also be referred to as CVD, is the acute loss of brain function due to acute disturbance in it’s blood supply either due to ischemia or hemorrhage in whichever form (Sims & Muyderman, 2010). It is a major cause of morbidity and mortality in both developed and developing countries due to aberrant and emerging lifestyle trends (Romero et al., 2014; Walker et al., 2010). Western cohorts have been shown to have higher incidences than Africa and the other developing countries; though are fast joining the league of this burden due to adoption of emerging lifestyles, poor diagnostics and follow up that is wanting (Tibazarwa & Damasceno, 2014). CVDs have overbearing outcomes in both the afflicted and the society as it can in most times be debilitating (Garbusinski et al., 2005). CVD is categorized as haemorrhagic and ischaemic. Ischaemic CVDs form the bulk of the disease (Walker et al., 2010) as compared to haemorrhagic CVD. Studies in Hai, one of the rural districts of the Kilimanjaro region demonstrated overall crude yearly CVD incidence rates at 94.5 per 100,000 and 107.9 per 100,000 in Dar-es-Salaam which is an urban setting (Walker et al., 2010). When age-standardized to the WHO world population, yearly CVD incidence rates were 108.6 per 100 000 in Hai and 315.9 per 100,000 in Dar-es-Salaam (Walker et al., 2010). CT scanning machines were introduced into the Western region of Kenya in 1998 (Elias, 1999) and hitherto revolutionized diagnosis and subsequently management of CVDs. The advent of CT scanners meant a diagnosis of either haemorrhagic or ischaemic CVD could be made with a some degree of confidence (Health; 2013). With rapid scan times and ease for detecting intracranial hemorrhage, CT has been shown to be a preferred diagnostic
technology. MRI has been shown to easily detect ischemia with diffusion-weighted imaging and also detects hyperacute hemorrhage with proper sequences(Bushberg). It is however inferior to CT scanning in diagnosis of acute haemorrhagic bleeds in the brain which are either extra-dural or intra-dural. MRI is not as widely available as CT and is typically more costly(Health; 2013). This study would therefore seek to find out the CT scan patterns of patients presenting with suspected CVD and attempt to map out the sites and characteristics of the lesions.

1.2 PROBLEM STATEMENT
Cerebro-vascular disease is an important emerging non communicable disease whose burden is borne individually and by society owing to its debilitating characteristics which include, but not limited to handicaps, dementia, depression, fatigue, recurrent hospitalization and adverse consequences on socioeconomic standing(Jowi & Mativo, 2008). Death is an inevitable eventuality for the severe cases. It is a major cause of morbidity and mortality in both developed and developing countries due to aberrant and emerging trends in lifestyle (Romero et al., 2014);(Walker et al., 2010). While the available study data and evidence are limited, the burden of CVD in Africa appears to be increasing (Owolabi et al., 2015). Although primary prevention has contributed to a decrease in CVD incidence in high-income countries, it is a proven fact that epidemiological transition has led to an increase in incidence of CVD and its complications in middle-to-low-income countries as well(Bejot, Daubail, & Giroud, 2015). Between 1990 and 2010, the number of CVD survivors had almost doubled to reach 33million people and it is epidemiologically projected to hit the figure of 77 million by 2030(Bejot et al., 2015).
Proven risk factors such as arterial hypertension, cigarette smoking, diabetes mellitus, hyperlipidaemia, micro-vascular rupture, increasing expected life span and co-morbidities such as sickle cell disease, HIV/AIDS infection, are increasingly being encountered in the tropics (Jowi & Mativo, 2008). Despite the above stated statistics, the true patterns of cerebro-vascular disease have not been established at MTRH. In patients also proven to have stroke on CT scanning, there has never been established the Hounsfield unit characteristic of the dominant pattern and their temporal change over time.

1.3 JUSTIFICATION

CVD is a common diagnosis in the Radiology Department at MTRH and a major cause of morbidity and mortality in African countries (Tibazarwa & Damasceno, 2014; Walker et al., 2010). Its true prevalence in Kenya’s western region and in the MTRH’s Radiology department has not been documented. With the establishment of a Chronic Disease Center and establishment of a Cardiac Care Unit in the hospital, there will be a postulated increase in the at risk patients for CVDs. Elias (1999) defined general head CT scan diagnosis as on request and found out that intracranial haemorrhage was the leading disorder with 17.8%, followed by brain infarcts at 10.5%, hydrocephalus at 6.3% and brain tumours at 5.9% (Elias, 1999). Further attempt on subclassification of cerebrovascular disease was not done. For example what were the HU characteristics of the hemorrhagic and infarctive stroke?

This study will therefore try to define the CT scan patterns in CVD as presented at the department and with it aid in better prediction of disease pattern especially for clinicians. It will also increase Moi University’s pool of knowledge and quench the needs of consumers of knowledge as findings of this study shall form a benchmark for referencing on patterns of CVD as experienced at MTRH.
1.4 PURPOSE OF STUDY

The purpose of this study therefore was to determine the CT scan patterns of patients presenting with CVD and while at it, mapping of the sites and characteristics of the lesions.

1.5 RESEARCH QUESTION

What are the stroke patterns of patients suspected to have cerebro-vascular disease and their corresponding Hounsfield units at Moi Teaching and Referral Hospital?

1.6 OBJECTIVES:

1.6.1 Main Objective:

To describe the brain CT scan patterns of CVD patients suspected to have Cerebrovascular disease at Moi Teaching and Referral Hospital.

1.6.2 Specific Objectives:

- To assess the head CT scan patterns of CVD patients suspected to have Cerebrovascular disease at MTRH
- To establish the subtypes of CVD found on CT scanning and their Hounsfield Unit (HU) characteristic.
CHAPTER TWO: LITERATURE REVIEW:

2.1 CT SCAN PATTERNS OF STROKE.

According to the World Health Organization (WHO), worldwide, 15 million people annually suffer a stroke. Of these, 5 million die and another 5 million are left permanently disabled placing a burden on the family and the community (http://www.who.int/cardiovascular_diseases/en/cvd_atlas_15_burden_stroke.pdf?ua=1).

WHO further elaborates that it is uncommon in people under 40 years and that if it occurs the main cause would be high blood pressure. It has also noted that it occurs in 8% of children with sickle cell disease (Feigin et al., 2014). In 2001 it was estimated that cerebrovascular diseases (stroke) accounted for 5.5 million deaths worldwide, equivalent to 9.6% of all deaths (WHO, 2002). Two-thirds of these deaths occurred in people living in developing countries and 40% of the subjects were aged less than 70 years and although age-standardized rates of stroke mortality have decreased worldwide in the past two decades, the absolute number of people who have a stroke every year, stroke survivors, related deaths, and the overall global burden of stroke (DALYs lost) are great and increasing (Feigin et al., 2014). In the UK for example the age adjusted annual death rates from strokes is about 200 per 100,000 (WHO Statistical Information System. Causes of death: mortality and health status. WHO data and statistics. [accessed September 20]). WHO data in 2004 reported that the age-standardized death rate per 100,000 for cerebrovascular disease in Thailand is 100 (Tangcharoen T, 2007). Although primary prevention has contributed to a decrease in stroke incidence in high-income countries, epidemiological transition has led to an increase in incidence in middle-to-low-income countries as well.
Between 1990 and 2010, the number of stroke survivors has doubled to reach 33 million people and is epidemiologically projected to hit the figure of 77 million by 2030 (Bejot et al., 2015). It has been shown that stroke was the third leading cause of hospital admission among cardiovascular diseases with an in-hospital mortality rate of acute stroke of 11.9% within the same population (Tangcharoen T, 2007). For the Eastern Mediterranean Region hospital-based studies have indicated that type of stroke pattern may be similar to that of Western countries (Yaqub BA & PJ., 1991). Hospital-based stroke register from Buenos Aires, Argentina, reported that intracerebral hemorrhage is more frequently occurring in natives than in Caucasians (Saposnik G, 2000), though it is hard to generalize it on the population with certainty.

Studies on stroke type from Africa are generally limited by small sample sizes and being hospital-based (J., 1991; Matenga J., 1986; Rosman, 1986). Nakibuuka et al (2013) demonstrated a 77.6% incidence of ischaemic stroke in a cross-sectional study done at Mulago-Uganda (Nakibuuka et al., 2013). Similar findings were also demonstrated by Jowi (2008). In this retrospective study, of the 80 patients sampled during the study period, 68 (85%) had ischemic stroke, 7 (8.8%) had haemorrhagic stroke while 5 patients (6.3%) had clinical symptoms but could not be subclassified on computed tomography (Jowi & Mativo, 2008).

The subtype of these types of strokes were however not demonstrated for example if found to be ischaemic, was it a lacunar or thromboembolic in nature?; if hemorrhagic..Was it intracerebral? And if it was, what region showed preponderance?; was it subarachnoid?, was the diagnosis different? Maybe a tumour? Cerebritis?, abscesses?, normal CT findings?

Introduction of the CT scan machine by Sir Geoffrey Hounsfield in 1971 and subsequent practical use in 1972 has revolutionized neuro-imaging in that accurate and non invasive
diagnosis of CVD can be made. Digital geometry processing is used to generate a three-dimensional image of the inside of an object from a large series of two-dimensional radiographic images taken around a single axis of rotation (Herman, 2009). It has thus made it possible to define intracranial anatomy by visualizing structures of different radio-densities and further detect and define the underlying pathologic processes more directly than it was possible with other diagnostic procedures and reducing the need for invasive contrast procedures (Weisberg, 1979). The advent of CT scanners meant a diagnosis of either haemorrhagic or ischaemic CVD could be made with some degree of confidence (Health, 2013). With rapid scan times and ease of detecting intracranial hemorrhage, CT has been shown to be a preferred diagnostic technology.

Hounsfield unit (HU) scale is a linear transformation of the original linear attenuation coefficient measurement into one in which the radiodensity of distilled water at standard pressure and temperature (STP) is defined as zero Hounsfield units (HU), while the radiodensity of air at STP is defined as -1000 HU (Hounsfield, 1980). For a material X with linear attenuation coefficient \( \mu_X \) the HU value is therefore given by:

\[
HU = \frac{\mu_X - \mu_{\text{water}}}{\mu_{\text{water}} - \mu_{\text{air}}} \times 1000
\]

Where \( \mu_{\text{water}} \) and \( \mu_{\text{air}} \) are linear attenuation coefficients of water and air respectively (Hounsfield, 1980).

The CT number of water and air is defined as 0 HU and -1000 HU respectively; this scale has no limit in the positive range of values. Medical scanners typically work in a range of –1024 HU to +3071 HU. As for windowing in the CT image, density values are represented
as gray scale values. The human eye however can discern only approximately 80 gray scale values and therefore not all possible density values can be displayed in discernible shades of gray. It is for this reason that, the density range of diagnostic relevance is assigned the whole range of discernible gray values in a process called windowing. To set the window, it is first defined which CT number the central gray scale value is to be assigned to. By setting the window width, it is then defined which CT numbers above and below the central gray value can still be discriminated by varying shades of gray, with black representing tissue of the lowest density and white representing tissue of the highest density (Berrington de Gonzalez et al., 2009).

Combined with other features of stroke on CT scan like loss of cortico-medulary differentiation, loss of insular ribbon, early features like dense MCA sign, (for ischaemic stroke) and hyperdense moieties due to blood spillage (for hemorrhagic stroke), importance of Hounsfield units use in characterization of cerebral lesion cannot be understated. Arun S.G. (2015) states that CT has the ability to quantify the beam attenuation, hence the measurements are expressed in Hounsfield units (HU), which indicates the HU values of various stages of infarct pathologies associated with the brain (Arun S Govind, 2015). Attenuation values of hemorrhagic strokes can also be graded as per stage in temporal evolution.

2.2 SUB-TYPES OF STROKE AS SEEN ON CT

Stroke can broadly be classified as either ischaemic or hemorrhagic (Feigin et al., 2014).
2.2.1 ISCHEMIC STROKE

Adams et.al (1993) came up with definitions for use in a multicenter clinical trial in classifications of subtypes of ischemic stroke in the TOAST trial (Adams et al., 1993). With it, are sub-classification of ischaemic strokes as shown below;

- Large artery atherosclerosis
- Cardio-embolism.
- Small artery occlusion (lacunae).
- Acute stroke of other determined etiology.
- Stroke of undetermined etiology.

Studies have shown that the bulk of all stroke cases are ischemic. In Caucasian populations for example approximately 80% of all strokes are ischemic, 10%-15% intracerebral hemorrhage (ICH), 5% subarachnoid hemorrhage (SAH), and the rest is due to other causes of stroke (Sudlow & Warlow, 1997).

Large artery atherosclerosis occurs when occlusion or stenosis of >50% of a major artery to the brain (Adams et al., 1993). Cardio-embolism would also be another cause of ischaemic brain stroke and would occur in at risk patients. CT scan imaging findings are similar in these two cases and a clinical examination would delineate the two (Adams et al., 1993; Health;, 2013 ).
Image 1: Patient’s axial non contrast enhanced CT with a 30 minute history of left sided weakness showing acute thrombosis of the MCA

In the acute period (6-24 h), the changes of ischemia become more apparent on the non-contrast CT scan. The loss of gray-white interface, sulcal effacement, hypodensity of basal ganglia, and hypodensity of the insular cortex become prominent (Truwit CL, 1990). The vascular distribution of the infarct becomes increasingly clear during this stage (Image2, Image3). In severe cases, edema and mass effect can appear at this stage. Chronic infarctions are characterized by marked hypodensity and lack of mass effect on CT scans; HU of the afflicted zone would then be similar to cerebrospinal fluid.
Image 2: Cerebral infarcts at 12 and 36 hrs as seen on axial CT

Image 3: Middle cerebral artery territory infarct..36 hrs
Small artery occlusion or lacunar infarcts or small subcortical infarcts result from occlusion of a single penetrating artery (Image4, Image5) and account for one quarter of cerebral infarctions (Arboix & Marti-Vilalta, 2009). Clinical examination in patients will demonstrate pure lacunar syndrome where symptoms of pure motor hemiparesis, pure sensory syndrome, sensorimotor stroke, ataxic hemiparesis or dysarthria-clumsy hand or with atypical lacunar syndrome (Arboix & Marti-Vilalta, 2009) predominate. Salgado et al (1996) showed that lacunar infarcts are associated with low stroke recurrence, mortality rates and overall has a better clinical outcome (Salgado, Ferro, & Gouveia-Oliveira, 1996).

Image 4: Lacunae infarct
2.2.2 HEMORHAGIC STROKE

2.2.2.1 Intracerebral hemorrhage

Spontaneous intra-cerebral hemorrhages as opposed to traumatic ones are mainly due to arteriolar hypertensive disease, and rarely due to blood dyscrasias, cerebral vascular malformation within the brain, and high alcohol consumption (Feigin et al., 2014). It forms between 10-15% of the classified strokes (Sudlow & Warlow, 1997). It involves acute accumulation of blood in the brain’s parenchyma. Albrecht (2014) in a study on stroke incidence following traumatic brain injury in older adults showed that there was a 6-fold increase in the rate of hemorrhagic stroke following trauma compared with the pre-traumatic period (Albrecht et al., 2014). CT scanning is quite sensitive in identifying various forms of acute intracranial hemorrhage and other gross lesions that would preclude the use of thrombolytic therapy (Adams et al., 1993). Acute haemorrhagic strokes appear hyper dense (Cohen W, 1992) (Image6, Image7, Image8) in comparison with the surrounding brain tissue.

Image 5: Illustration of lacuna infarct occurrence
Image 6: Serial axial non contrast enhanced CT of intra-cerebral (thalamic) hemorrhage

Image 7: ICH with ventricular spillage
Image 8: Cerebellar hemorrhage

Acute hemorrhage will appear hyperdense on cranial CT. This is attributed to the fact that the haemoglobin molecule is denatured into Fe++ and globin chains. The Ferrous component is relatively dense, and hence effectively absorbs x-ray beams (Peron, 2007). Acute blood is typically in the range of 50-100 Hounsfield units. As the blood becomes older and the haemoglobin molecule breaks down further, it will lose this hyper-dense appearance, beginning at the periphery and working centrally. On CT scan blood will first become iso-dense with the brain at between 4 days to 2 weeks, depending on clot-size, and finally hypo-dense to brain at greater than 2-3 weeks (Peron, 2007).
CHAPTER THREE: RESEARCH DESIGN AND METHODOLOGY

3.1 STUDY SITE

The study was done at Moi Teaching and Referral Hospital, specifically at its Radiology and Imaging Department.

The Hospital is located in Eldoret town the headquarters of Uasin Gishu county and is 350 Kilometers Northwest of the Kenya’s capital Nairobi. It is the second National Hospital and serves as a teaching hospital for Moi University Schools of Medicine (MUSOM), Public Health (SPH), Nursing (SON) and Dentistry (SOD). Kenya Medical Training Center (KMTC), Eldoret and University of Eastern Africa Baraton School of Nursing also access its facilities in training. MTRH is an internship center for medical, clinical and nursing officers and is the referral hospital for the Western part of Kenya and North rift with a bed capacity of over 700. It has a catchment population of approximately 20 million people. The facility has several departments including Surgery, Pediatrics and Radiology and Imaging, Cardiac Care Unit Accident and Emergency Center (www.mtrh.or.ke/).

3.2 STUDY DESIGN

This was a cross-sectional descriptive study done from 13\textsuperscript{th} October 2014 to 30\textsuperscript{th} July 2015, where by all CT scans of patients with a clinical diagnosis of cerebrovascular disease were reviewed for determination and characterisation of patterns.
3.3 STUDY POPULATION

It included all patients with a clinical diagnosis of CVD who had CT scans done at the Radiology and Imaging Department during the period of study.

3.4 ELIGIBILITY CRITERIA

3.4.1 Inclusion criteria.

- Consent to participate in study.

- Patients who had been reviewed by a clinician within or without MTRH, suspected to have CVD and referred to the Radiology and Imaging department for imaging.

- Patients with a clinical suspicion of CVD and had head CT scans done.

3.4.2 Exclusion criteria

- Patients with head injuries secondary to trauma.

- Patients who were eligible but consent was not received from self or guardians.

3.5 SAMPLING TECHNIQUES

3.5.1 Sampling method

Consecutive sampling technique was used whereby all patients presenting to the Radiology Department with a clinical diagnosis of cerebrovascular accidents were included in the study.
after consent. For those who were incapacitated i.e. coma or confusion, their next of kins were approached to give an informed consent.

**3.5.2 Sample size determination**

The sample size required in order to be 95% sure that the proportion of patients with ischemic CVD among those who had head CT scan is within plus or minus 5% of the population proportion of 54.8% was estimated using the following formula (Cochran WG. Sampling Techniques. 2nd ed. New York: John Wiley and Sons)

\[
n = \left( \frac{Z_{1-\%}}{\delta} \right)^2 P(1-P)
\]

\[
= \left( \frac{1.96}{0.05} \right)^2 \times 0.548 \times (1-0.548)
\]

\[
= 381
\]

Where \( P \) is the population proportion of ischemic CVD among patients, who had head CT scan,

\( \delta \) is the margin of error equal to the 5% used in this case, and

\( Z_{1-\%} \) is the \((1 - \%\)\(\times 100\% \) quantile of the standard normal distribution.

Adjusting this sample size for finite population gave us a minimum sample size of

\[
\left( \frac{381}{1 + \frac{381}{500}} \right) = 217 \approx 220
\]

participants. The prevalence of ischemic CVD among the patients with head CT was obtained from Asefa and Meseret in Ethiopia (Asefa G). This sample size was sufficient to study the various types/patterns of celebrovascular accidents among the patients who had head CT examination.
The finite adjusted population size for was obtained from the Moi Teaching and Referral Hospital records as follows: There was an average of 80 patients suspected to have CVDs per month as found on CTs giving a total of 480, approximately 500, within a period of 6 months within which the study lasted.

3.6 STUDY PROCEDURE

Patients who were clinically diagnosed to have CVAs and who met all the inclusion criteria were identified mainly at the CT scan room wing of the Radiology and Imaging Department. They included all patients triaged and assessed at the Accident and Emergency Department and also those who were referred from peripheral facilities for CT scanning. The subjects underwent non contrast enhanced (NCECT) and contrast enhanced (CECT) CT scanning based on the protocols laid down by the department(Appendix II) and subsequently reporting was done by the principal investigator ; aided by two consultant radiologists. The bio-data and demographics were collected from both the subjects and hospital records. The questionnaire was subsequently filled after consent was given. During the period of study, the filled questionnaires were always under lock and key and accessed by the principal investigator and the supervisors.
Figure 1: Algorithm for study procedure

3.7 DATA COLLECTION AND MANAGEMENT

3.7.1 Data collection.

Data relevant to the study was collected from October 2014 to July 2015. They were then entered into questionnaires and later double entered into a computer database. The data was
kept confidential (and still is) only accessible to the principal investigator and supervisors. Identities of the patients were serialized. Data collection forms were reviewed by day end to ensure completion in filling and subsequently coded.

3.7.2 Quality control

All head CT scans were done at MTRH CT SCAN room that had internal quality controls. Patients who had been referred from peripheral facilities, accident and Emergency department or general wards were treated in the same manner, that is same CT scan protocols(Appendix II)applied. The scans were reported by the principal investigator in collaboration with at least two consultant radiologists.

3.7.3 Statistical Data Analysis

A software for data analysis and statistical computing known as R {Team, 2015 #57} was used. Categorical variables were summarized as frequencies with corresponding percentages while continuous variables were summarized as median and the corresponding inter quartile range given. Gaussian assumptions were assessed using Shapiro Wilk test. Analysed results were presented in tables and graphs.

3.8 ETHICAL CONSIDERATIONS:

It is paramount that research subjects' autonomy, dignity, and rights(Pina, 2012) are maintained. It was with this regard in mind that research approval was sought out from the Institutional Research and Ethics Committee and the Director of Moi Teaching and Referral Hospital. Explicit consent was obtained from the subjects or their next of kin after
explanation of the laid down CT scan protocol, the benefits of the procedure and possible side effects of contrast media.

All the subjects received care without bias whether they approved of consent or not. Furthermore no incentives or coercion were used to influence consent. After the procedure, reports about the scan were availed to the patient whilst confidentiality of the data collected enforced.

Confidentiality of all data was (and still is) maintained. The data collection forms used did not contain the names of the patients nor their personal identification numbers, rather random indexing was used. Data collecting material was kept in a locked cabinet in the office of the principal investigator during the study period and the soft copy versions saved and protected with passwords.

Analyzed results and conclusions of the study will be presented to the University’s Department of Radiology and Imaging and to the MTRH management for their consumption. It shall further be presented for publication in reputable journals for the furtherance of medical frontiers of knowledge for health care providers and references for researchers.
3.9 Study Limitations

1. Cost of CT scanning was borne by the patient.

2. Study was a hospital based design and thus was not immune to bias.

3. Power outages of the national grid on occasion disrupted the CT generators’ functionality.
CHAPTER FOUR: RESULTS

A total of 225 participants were included in the study. The median (IQR) age was 60.0 (IQR: 44.0, 77.0) years with a minimum and a maximum of 2.0 and 102 years respectively.

Figure 2: Age distribution
Figure 3: Gender distribution

Of the total number of participants were 118 (52.4%) male participants (Figure 2).

**County of residence**

<table>
<thead>
<tr>
<th>County</th>
<th>n</th>
<th>percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baringo</td>
<td>9</td>
<td>4.0</td>
</tr>
<tr>
<td>Bomet</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Bungoma</td>
<td>10</td>
<td>4.4</td>
</tr>
<tr>
<td>Busia</td>
<td>6</td>
<td>2.7</td>
</tr>
<tr>
<td>Elgeyo- marakwet</td>
<td>20</td>
<td>8.9</td>
</tr>
<tr>
<td>Kakamega</td>
<td>8</td>
<td>3.6</td>
</tr>
<tr>
<td>Kapenguria</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Kericho</td>
<td>5</td>
<td>2.2</td>
</tr>
<tr>
<td>Kisii</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Kisumu</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Lodwar</td>
<td>2</td>
<td>0.9</td>
</tr>
<tr>
<td>Meru</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Migori</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Muranga</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Nakuru</td>
<td>5</td>
<td>2.2</td>
</tr>
<tr>
<td>Nandi</td>
<td>46</td>
<td>20.4</td>
</tr>
<tr>
<td>Nyamira</td>
<td>2</td>
<td>0.9</td>
</tr>
<tr>
<td>Siaya</td>
<td>2</td>
<td>0.9</td>
</tr>
<tr>
<td>Trans-nzoia</td>
<td>7</td>
<td>3.1</td>
</tr>
<tr>
<td>Turkana</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>Uasin gishu</td>
<td>87</td>
<td>38.7</td>
</tr>
<tr>
<td>Vihiga</td>
<td>1</td>
<td>0.4</td>
</tr>
<tr>
<td>West pokot</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>225</td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
Figure 4: Distribution of participants by the level of education

More than one third of the participants had no formal education. The proportion of the participants who had primary and secondary level education was almost similar being 20% and 19.6% respectively. Slightly more than a quarter had tertiary level of education. Of those who had primary level of education, 17 (37.8%) attained classes 1-4 while the rest, 28 (62.2%) attained classes 5-8.
Figure 5: Distribution of participants by occupation

Majority of the participants were either farmers or retired farmers. Housewives and business men or women followed in that order. The “other” occupations include: Athlete, Carpenter, Matatu tout, Mechanic, Tailor, and Watchman.
Figure 6: Distribution by marital status

More than two thirds of the participants, 159 (70.7%), were married. One seventeenth of the participants were either widows or widowers.
Table 1: Next of kin

<table>
<thead>
<tr>
<th>Next of kin</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father</td>
<td>6 (2.7%)</td>
</tr>
<tr>
<td>Mother</td>
<td>5 (2.2%)</td>
</tr>
<tr>
<td>Brother</td>
<td>23 (10.3%)</td>
</tr>
<tr>
<td>Sister</td>
<td>7 (3.1%)</td>
</tr>
<tr>
<td>Husband</td>
<td>34 (15.2%)</td>
</tr>
<tr>
<td>Wife</td>
<td>19 (8.5%)</td>
</tr>
<tr>
<td>Daughter</td>
<td>13 (5.8%)</td>
</tr>
<tr>
<td>Son</td>
<td>75 (33.6%)</td>
</tr>
<tr>
<td>Daughter in-law</td>
<td>24 (10.8%)</td>
</tr>
<tr>
<td>Good Samaritan</td>
<td>10 (4.5%)</td>
</tr>
<tr>
<td>Others**</td>
<td>7 (3.1%)</td>
</tr>
</tbody>
</table>

**- Cousin, colleague, Grandson, Granddaughter, Nephew

One third of the next of kin were sons of the participants. Brothers, husbands, wives, daughter’s in-law.
Figure 7: Diagnosis at the referral point

Over 90% of the participants were diagnosed of CVA at the referral point (Figure 6). Five participants were diagnosed of CVD. The rest of the diagnoses (confusion, dementia, hypertension, intracerebral bleeding seizures and unconsciousness) were each diagnosed in one individual.
### Table 2: Symptoms and signs (S/S)

<table>
<thead>
<tr>
<th>S/S</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paralysis of both lower limbs</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Weakness of both lower limbs</td>
<td>9 (4.0%)</td>
</tr>
<tr>
<td>Weakness of one side of the body</td>
<td>118 (52.4%)</td>
</tr>
<tr>
<td>Left side</td>
<td>37 (31.4%)</td>
</tr>
<tr>
<td>Right side</td>
<td>55 (46.6%)</td>
</tr>
<tr>
<td>Missing response</td>
<td>26 (22.0%)</td>
</tr>
<tr>
<td>Loss of vision on one eye</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Left side</td>
<td>1 (100.0%)</td>
</tr>
<tr>
<td>Right side</td>
<td>-</td>
</tr>
<tr>
<td>Loss of vision on both eyes</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Headache</td>
<td>47 (20.9%)</td>
</tr>
<tr>
<td>Confusion</td>
<td>45 (20.0%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>18 (8.0%)</td>
</tr>
<tr>
<td>Memory impairment</td>
<td>10 (4.4%)</td>
</tr>
<tr>
<td>Inability to talk</td>
<td>42 (18.7%)</td>
</tr>
<tr>
<td>Drooling saliva</td>
<td>7 (3.1%)</td>
</tr>
<tr>
<td>Loss of coordination</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td><strong>Other S/S</strong></td>
<td></td>
</tr>
<tr>
<td>Mutism</td>
<td>11 (14.5%)</td>
</tr>
<tr>
<td>Facial palsy</td>
<td>27 (35.5%)</td>
</tr>
<tr>
<td>Unconsciousness</td>
<td>20 (26.3%)</td>
</tr>
<tr>
<td>Myalgia</td>
<td>8 (10.5%)</td>
</tr>
<tr>
<td>Convulsion</td>
<td>8 (10.5%)</td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>1 (1.3%)</td>
</tr>
<tr>
<td>Neuralgia</td>
<td>1 (1.3%)</td>
</tr>
</tbody>
</table>

†n=76
Assessment of the symptoms revealed one participant with paralysis of both limbs, 9 (4.0%) with weaknesses in both limbs and 52.4% with weakness of one side of the body. Only one participant had loss of vision on one eye. One fifth had a headache, 45 (20.0%) developed confusion, and 18 (8.0%) were vomiting.

Memory impairment, inability to walk, drooling saliva, and loss of coordination were seen in 10 (4.4%), 42 (18.7%), 7 (3.1%), and 1 (0.4%) respectively.

Other symptoms include autism, facial palsy, unconsciousness, myalgia, convulsion and urinary incontinence that were reported in 11 (14.5%), 27 (35.5%), 20 (26.3%), 8 (10.5%), 8 (10.5%), and 1 (1.3%) respectively.
Figure 8: Symptoms duration

Majority of the participants, 120 (53.3%), have had the symptoms between 1-3 days. Fifty participants, 12.2%, had their symptoms classified as subacute, and 5.8% had hyper acute symptoms. A significant proportion of the participants had chronic symptoms.

None of the participants had recently been involved in an accident.
Table 3: Scan findings

<table>
<thead>
<tr>
<th>Scan findings (n=225)</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic CVD</td>
<td>60 (26.7%)</td>
</tr>
<tr>
<td>Normal CT</td>
<td>41 (18.2%)</td>
</tr>
<tr>
<td>Cerebral atrophy</td>
<td>36 (16.0%)</td>
</tr>
<tr>
<td>Hemorrhagic CVD</td>
<td>27 (12.0%)</td>
</tr>
<tr>
<td>Subarachnoid bleed</td>
<td>23 (10.2%)</td>
</tr>
<tr>
<td>Tumors</td>
<td>9 (4.0%)</td>
</tr>
<tr>
<td>Cerebral atrophy &amp; Ischemic CVD</td>
<td>5 (2.2%)</td>
</tr>
<tr>
<td>Encephalitis</td>
<td>4 (1.8%)</td>
</tr>
<tr>
<td>Edema</td>
<td>3 (1.3%)</td>
</tr>
<tr>
<td>Abscess</td>
<td>2 (0.9%)</td>
</tr>
<tr>
<td>Bilateral Frontal CSF hygroma</td>
<td>2 (0.9%)</td>
</tr>
<tr>
<td>Ischemic CVD and tumors</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Bilateral Basal ganglia calcification</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Bilateral thalamic calcification/brain atrophy</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>CSF hygroma</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Left frontal cerebritis</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Left lateral ventricular dilation</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Left sided toxoplasmosis</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Porencephalic cyst</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Raptured anterior cerebral aneurysm</td>
<td>1 (0.4%)</td>
</tr>
<tr>
<td>Superior sagittal sinus thrombosis</td>
<td>1 (0.4%)</td>
</tr>
</tbody>
</table>

†Other associated scan findings

<table>
<thead>
<tr>
<th>Scan findings</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compensatory hydrocephalus</td>
<td>18 (62.1%)</td>
</tr>
<tr>
<td>Bilateral CSF hygroma</td>
<td>2 (6.9%)</td>
</tr>
<tr>
<td>Intraventricular bleed</td>
<td>2 (6.9%)</td>
</tr>
<tr>
<td>Ventricular seepage</td>
<td>2 (6.9%)</td>
</tr>
<tr>
<td>Bilateral basal ganglia calcification</td>
<td>1 (3.4%)</td>
</tr>
<tr>
<td>Compensatory left ventricular dilation</td>
<td>1 (3.4%)</td>
</tr>
<tr>
<td>Frontotemporal atrophy</td>
<td>1 (3.4%)</td>
</tr>
<tr>
<td>Multiple brain aneurysm</td>
<td>1 (3.4%)</td>
</tr>
<tr>
<td>Right sided subdural haematoma</td>
<td>1 (3.4%)</td>
</tr>
</tbody>
</table>

†n = 29

More than a quarter of the participants (29.3%) had ischemic CVD, and 27 (12.0%) suffered hemorrhagic CVD. Cerebral atrophy was diagnosed in 41 (18.2%) of the participants. Forty one (18.2%) of the total participants had normal CT, and 10 (4.4%) were diagnosed of
tumors. Encephalitis, edema, and abscess was reported in 4 (1.8%), 3 (1.3%), and 2 (0.9%) respectively.

Among those who had other scan findings included compensatory hydrocephalus that was found in 18 (62.1%). Of this number, 12 (66.7%) had cerebral atrophy, 2 (11.1%) had cerebral atrophy as well as CVD, 1 (5.6%) had hemorrhagic CVD, and 3 (16.7%) had ischemic CVD.

Image 9: Left fronto temporal hemorrhagic CVD in a 2 year old male patient (Ruptured AVM?..child died same day ICU)
Image 10: Axial NCECT showing left thalamic hemorrhagic CVD with associated cerebral atrophy in 47 year old man

Image 11: Axial NCECT showing left basal ganglia acute infarct in a 54 year old man
Image 12: Axial NECT showing right basal ganglia infarction with accompanying compensatory hydrocephalus (largely right anterior horn) in a 72 year old man

Image 13: Axial NECT showing a left fronto-parietal subacute infarct with mass effect in a 56 year old man
Image 14: Axial NECT showing a right high parietal subacute infarct in a 51 year old woman

Image 15: Bilateral white matter disease (likely encephalitis) in a 17 yr old teenager
Image 16: Axial NECT demonstrating left occipital lobe infarct in a 12 year old girl
Image 17a: Axial occipital parafalcine meningioma with hemorrhagic CVD in a 59-year-old woman

Image 17b: Left thalamic hemorrhagic bleed with menigioma(Image 17a..same patient)
Image 18:  CECT in a 30 year old female with leptomeningeal enhancement (meningitis), right basal ganglia infarct, and left caudate infarct

Image 19:  Bilateral subdural haematoma in a 54 year old male
Image 20: Axial NECT in a 86year old female showing a lacunar infarcts in the basal ganglia bilaterally

Image 21: CECT in a 63yr old woman showing high grade astrocytoma( likely GBM)
Image 22: Axial CT of 70 year old man demonstrating frontopolar atrophy

Image 23: Axial contrast enhanced CT of a 34 year old male demonstrating left frontal abscess
Table 4: Location of the scan findings

<table>
<thead>
<tr>
<th>Findings</th>
<th>Location</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral lobes</td>
<td>Frontal</td>
<td>8 (3.6%)</td>
</tr>
<tr>
<td>(Penetrating cortical branches of ACA,MCA,PCA)</td>
<td>Frontoparietal</td>
<td>11 (4.9%)</td>
</tr>
<tr>
<td></td>
<td>Parietal</td>
<td>13 (5.8%)</td>
</tr>
<tr>
<td></td>
<td>Parieto-temporal</td>
<td>26 (11.6%)</td>
</tr>
<tr>
<td></td>
<td>Parietal-occipital</td>
<td>7 (3.1%)</td>
</tr>
<tr>
<td></td>
<td>Occipital</td>
<td>18 (8.0%)</td>
</tr>
<tr>
<td></td>
<td>Whole Cerebral lobe</td>
<td>4 (1.8%)</td>
</tr>
<tr>
<td>Basal ganglia (Lenticulostriate branches MCA)</td>
<td>Caudate</td>
<td>4 (1.8%)</td>
</tr>
<tr>
<td></td>
<td>Globus pallidus</td>
<td>9 (4.0%)</td>
</tr>
<tr>
<td></td>
<td>Putamen</td>
<td>4 (1.8%)</td>
</tr>
<tr>
<td></td>
<td>Internal Capsule</td>
<td>4 (1.8%)</td>
</tr>
<tr>
<td>Thalamus: (Thalamogeniculate branches PCA)</td>
<td></td>
<td>6 (2.7%)</td>
</tr>
<tr>
<td>Pons (Basilar branches)</td>
<td></td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Cerebellum: Penetrating branches PICA, AICA, SCA</td>
<td></td>
<td>4 (1.8%)</td>
</tr>
</tbody>
</table>

Majority of the scan patterns, 26 (11.6%), were located in the parieto-temporal. There were 18 (8.0%) that were located in the occipital region. Other findings are as shown in Table 4.
Table 5: Specific side of the brain where the patterns were found

<table>
<thead>
<tr>
<th>Location</th>
<th>Right n (%)</th>
<th>Left n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>7 (87.5%)</td>
<td>1 (12.5%)</td>
</tr>
<tr>
<td>Frontoparietal</td>
<td>7 (63.6%)</td>
<td>4 (36.4%)</td>
</tr>
<tr>
<td>Parietal</td>
<td>6 (46.2%)</td>
<td>7 (53.8%)</td>
</tr>
<tr>
<td>Parieto-temporal</td>
<td>15 (57.7%)</td>
<td>11 (42.3%)</td>
</tr>
<tr>
<td>Parietal-occipital</td>
<td>4 (57.1%)</td>
<td>3 (42.9%)</td>
</tr>
<tr>
<td>Occipital</td>
<td>9 (50.0%)</td>
<td>9 (50.0%)</td>
</tr>
<tr>
<td>Whole Cerebral lobe</td>
<td>3 (75.0%)</td>
<td>1 (25.0%)</td>
</tr>
<tr>
<td>Caudate</td>
<td>2 (50.0%)</td>
<td>2 (50.0%)</td>
</tr>
<tr>
<td>Globus pallidus</td>
<td>6 (66.7%)</td>
<td>2 (22.2%)</td>
</tr>
<tr>
<td>Putamen</td>
<td>2 (50.0%)</td>
<td>2 (50.0%)</td>
</tr>
<tr>
<td>Internal Capsule</td>
<td>1 (25.0%)</td>
<td>3 (75.0%)</td>
</tr>
<tr>
<td><strong>Thalamus</strong>: (Thalamogeniculate branches PCA)</td>
<td>3 (50.0%)</td>
<td>3 (50.0%)</td>
</tr>
<tr>
<td><strong>Cerebellum</strong>: Penetrating branches PICA, AICA, SCA</td>
<td>2 (50.0%)</td>
<td>2 (50.0%)</td>
</tr>
</tbody>
</table>

A higher proportion of the patients with scan findings located in the frontal and frontoparietal areas were found to be in the right, 7 (87.5%), 7 (63.6%) respectively. More than half, 7 (53.7%), of those found in the parietal were located in the left. Similarly, more than half, 15 (57.7%), of those located in the parietal-temporal were in the right. Three quarters of those in the whole cerebral lobe were in the right, and two thirds of those in the globus pallidus were also to the right. Of those found in the internal capsule were 75.0% located to in the left. Half of those located to the occipital, caudate, putamen, thalamus, and cerebellum were located either in the right or in the left.
Table 6: Summary of the Hounsfield units stratified by the side of the brain

<table>
<thead>
<tr>
<th>Location</th>
<th>Median (IQR) Hounsfield units</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
</tr>
<tr>
<td>Frontal</td>
<td>21.5 (13.8, 57.5)</td>
</tr>
<tr>
<td>Frontoparietal</td>
<td>20.5 (18.0, 21.0)</td>
</tr>
<tr>
<td>Parietal</td>
<td>32.7 (22.3, 43.3)</td>
</tr>
<tr>
<td>Parieto-temporal</td>
<td>19.0 (16.7, 26.7)</td>
</tr>
<tr>
<td>Parietal-occipital</td>
<td>20.6 (18.8, 21.3)</td>
</tr>
<tr>
<td>Occipital</td>
<td>17.9 (14.1, 28.4)</td>
</tr>
<tr>
<td>Whole Cerebral lobe</td>
<td>18.6 (16.3, 20.8)</td>
</tr>
<tr>
<td>Caudate</td>
<td>24.1 (22.0, 26.3)</td>
</tr>
<tr>
<td>Globus pallidus</td>
<td>43.0 (19.1, 67.5)</td>
</tr>
<tr>
<td>Putamen</td>
<td>43.0 (30.5, 55.5)</td>
</tr>
<tr>
<td>Internal Capsule</td>
<td>40.4 (40.4, 40.4)</td>
</tr>
<tr>
<td><strong>Thalamus</strong>: (Thalamogeniculate branches PCA)</td>
<td>62.1 (61.5, 65.0)</td>
</tr>
<tr>
<td><strong>Cerebellum</strong>: Penetrating branches PICA, AICA, SCA</td>
<td>34.6 (22.5, 46.7)</td>
</tr>
</tbody>
</table>

The median Hounsfield units for the scan patterns found to the left of the frontal area was high, 37.2 (IQR: 37.2, 37.2) compared to those in the right 21.5 (IQR: 13.8, 57.5). Similarly, the median Hounsfield units for the scan patterns found to the left of the frontalparietal area was high, 44.5 (IQR: 21.3, 68.3) compared to those in the right 20.5 (IQR: 18.0, 21.0).

The Hounsfield units to the right of the parietal, parieto-temporal, and parietal-occipital were high 32.7 (IQR: 22.3, 43.3), 19.0 (IQR: 16.7, 26.7), and 20.6 (IQR: 18.8, 21.3) respectively compared to those to the left 19.0 (IQR: 14.8, 19.9), 18.4 (IQR: 15.9, 42.7), and 18.0 (IQR: 14.1, 19.7) respectively.
Median Hounsfield units to the right of occipital was lower 17.9 (IQR: 14.1, 28.4) compared to that to the left 19.6 (IQR: 11.6, 59.2). Similar observation was made regarding patterns located to the caudate, globus pallidus, putamen, internal capsule, thalamus, and cerebellum.

The mean Hounsfield units for the participants who had hemorrhagic CVD was 60.8 ± 9.1. When classified based on severity the following results were obtained.

![Pie chart showing severity based on Hounsfield units](image)

**Figure 9: Severity based on Hounsfield units**

More than three quarter of the participants (77.8%) had hyper-acute hemorrhagic CVD (HU: \( \geq 55 \)), 3 (11.1%) had sub-acute (HU: 35-45), and another 3 (11.1%) had acute hemorrhagic CVD (HU: 45-55).
Table 7: Relationship between symptoms duration and Hounsfield units among those who had hemorrhagic CVD

<table>
<thead>
<tr>
<th>Time to symptoms occurrence</th>
<th>N</th>
<th>Hemorrhagic CVD, Median (IQR)</th>
<th>Kruskal Wallis test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 24 hours</td>
<td>3</td>
<td>68.0 (63.2, 68.4)</td>
<td></td>
</tr>
<tr>
<td>1-3 days</td>
<td>19</td>
<td>61.0 (58.5, 66.9)</td>
<td></td>
</tr>
<tr>
<td>4-7 days</td>
<td>3</td>
<td>61.0 (52.8, 66.4)</td>
<td>0.845</td>
</tr>
<tr>
<td>8-14 days</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>&gt;14 days</td>
<td>2</td>
<td>55.6 (48.8, 62.5)</td>
<td></td>
</tr>
</tbody>
</table>

Participants with longer duration of symptoms seem to have lower Hounsfield units. However, the drop across time was not statistically significant, p=0.845.

Similarly, the average Hounsfield units for the participants who had ischemic CVD was 18.0 ± 8.6. Classifications of the severity based on Hounsfield units revealed the following findings.

Figure 10: Severity of ischemic CVD based on Hounsfield units
Half of the participants with ischemic CVD had sub-acute ischemic CVD (HU: 9.55-19.13), and 26 (40.6%) had acute ischemic CVD (HU: >=19.13). The remaining 9.4% had chronic ischemic CVD (HU: <9.55).

Table 8: Relationship between symptoms duration and Hounsfield units among those who had ischemic CVD

<table>
<thead>
<tr>
<th>Time to symptoms occurrence</th>
<th>n</th>
<th>Ischemic CVD, Median (IQR)</th>
<th>Kruskal Wallis test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 24 hours</td>
<td>1</td>
<td>19.0 (19.0,19.0)</td>
<td></td>
</tr>
<tr>
<td>1-3 days</td>
<td>39</td>
<td>18.0 (15.9, 20.7)</td>
<td>0.345</td>
</tr>
<tr>
<td>4-7 days</td>
<td>10</td>
<td>19.7 (16.7, 21.2)</td>
<td></td>
</tr>
<tr>
<td>8-14 days</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>&gt;14 days</td>
<td>14</td>
<td>15.4 (10.9, 18.9)</td>
<td></td>
</tr>
</tbody>
</table>

There was no evidence of any trend in the change in Hounsfield units across the duration of the symptoms among the participants who had ischemic CVD.
CHAPTER FIVE: DISCUSSION

5.1 Introduction

Cerebrovascular disease is a comprehensive term that explains a clinical event characterized by sudden onset of neurological deficits. Arterial infarction and ischemia is the most common cause of CVD, responsible for 80% of all cases and the remaining 20% of strokes are mostly hemorrhagic (Arun S Govind, 2015; Sims & Muyderman, 2010; Walker et al., 2010). Estimates from the Global Burden of Diseases, Injuries, and Risk Factors Study (Lozano et al., 2012) ranked stroke as the second most common cause of death and the third most common cause of disability-adjusted life-years (DALYs) worldwide in 2010 (Lozano et al., 2012).

5.2 Socio-demographic characteristics

Socio-demographic characteristics have been known to play a role in risk and occurrence in cerebrovascular disease. In a study by Jowi et al. on pathological sub-types, risk factors and outcome of stroke at the Nairobi Hospital, Kenya, it was found out that the mean age was 61.3 years, mode: 63 years, range 34-95 years. Males were 43 (53.8%), M to F ratio 1.2:1; in a study where 80 patients had been recruited (Jowi & Mativo, 2008). The age range and percentages of afflicted gender groups compares relatively well with our study in which of a total of 225 participants who were included; the median (IQR) age was 60.0 (IQR: 44.0, 77.0) years with a minimum and a maximum of 2.0 and 102 years respectively. Of the total number of participants, 118 (52.4%) were male. There is a marked difference in number of recruited subject which could be explained by the private setting of Jowi et al’s area of study in contrast to MTRH, a public facility which serves a significantly higher populace as shown.
earlier. The proportions though of male to female ratios had similarities and could be postulated as that all participants were from Kenya. In a slight contrast, Nakibuuka et al in a study done in Uganda found out that of 127 patients, 68 (53.5 %) were female. The age range was 19–99 years, with median 60 (IQR 49–75). It also be noted that Nakibuuka et al’s study was done as one month follow up of patients. There were however similar numbers of patients (in proportions) of less than 51 years (29.9 %) and greater than 71 years (29.1 %)(Nakibuuka et al., 2015). The age ranges are however concur in the three studies.

In this study, it was found that one third of the participants had no formal education while proportion of the participants who had primary and secondary level education were almost similar, each being 20% and 19.6% respectively. Slightly more than a quarter had tertiary level of education. Of those who had primary level of education, 17 (37.8%) attained classes 1-4 while the rest, 28 (62.2%) attained classes 5-8. A study in Mulago hospital in Uganda demonstrated more than half of the patients being unemployed and having either never attended school or attained a primary level of education (Nakibuuka et al., 2015). Having greater than one third in the current study and more than one half in another study of a neighboring country by Nakibuuka et al begs the question of significance of educational and intelligence status of an individual in getting chronic diseases CVD for example. In attempting to answer this, does educational status necessarily equate to intelligence? Probably so or not. Wraw et al(2015) in a study on Intelligence in youth and health at age 50 concluded that pre-morbid intelligence is significantly associated with a large number of health outcomes at age 50. Those who had a higher IQ in youth tended to have better overall physical health and were less likely to have a number of chronic health conditions (Beaver et al., 2016; Calvin et al., 2011; Vagero, 2011) in several ways go on to echo what Wraw et al
demonstrated. The link of education and or intelligence and morbidity should form a basis of a Kenyan study which currently is beyond the scope of this study.

Majority of the participants were either farmers 57(25.3%) or retired farmers 55(24.4%). House wives and business men/ women followed in that order. These findings need further in depth reviews because a question could arise...does someone say he is a farmer since he is not employed? Because one would argue that farming indeed is employment, and by being a retired farmer does it mean one is living off his/her children’s earnings or well wishers. This is a challenge also brought out in this study that would need the participation of human resource experts, sociologist and epidemiologists .The “other” occupations included: Athlete, Carpenter, Matatu tout, Mechanic, Tailor, and Watchman (Table 1.)

As relates marital status, majority of the participants 159(70.7%) were married. Widows and widowers were 38(16.9%), those who were single 16(7.1%), separated 3(1.3%) and the young classified as pupils were 9(4%). Thompson et al in a study on risk factors for CVD and myocardial infarction in women in the United Kingdom as assessed in general practice noted that single women had a lower risk of stroke and myocardial infarction than married women (Thompson, Greenberg, & Meade, 1989). Categorization of men was however not picked. An explanation of having a high number of stroke incidences among the married could be that of age. Chances of getting married grow as on ages and age is a known risk factor for stroke (Feigin et al., 2014; Jowi & Mativo, 2008; Lozano et al., 2012; Thompson et al., 1989).
5.3 Symptoms and signs

It was noted that majority of the participants, 120 (53.3%), had had the symptoms for between 1-3 days at presentation. Fifty participants (12.2%), had had symptoms for 4 to 14 days, and 13 (5.8%) presented within 24 hours of symptom onset. Forty two (18.7%) of the participants presented more than 14 days after event. In a study on interpretation of symptoms and delay in seeking treatment by patients who have had a stroke, it was noted that patients' median delay time from the onset of symptoms to admission for stroke in the ED was 16 hours with only 12 patients (31.6%) accessed the emergency department in less than 2 hours (Zerwic, Hwang, & Tucco, 2007). Kothari et al (1997) showed that almost 40% of patients in their study of 166, admitted with a possible stroke did not know the signs, symptoms, or risk factor of a stroke (Kothari et al., 1997). The two studies could explain the varied timings of patients presentation to hospital and subsequent management. Some could perceive the symptoms and seek care while others could not.

Assessment of the symptoms some picked from patients in outpatient files revealed one participant with paralysis of both limbs, 9 (4.0%) with weaknesses in both limbs and 52.4% with weakness of one side of the body. This in contrast with the study of Kothari et al (1997) who found unilateral weakness (26%) and numbness (22%) were the most frequently noted symptoms. This can be explained by the fact that in the stated study, the authors went out to ask patients if they knew of the symptom. In our study there was active involvement of clerkship notes of the clinician. Only one participant had loss of vision on one eye. One fifth had a headache, 45 (20.0%) developed confusion, and 18 (8.0%) were vomiting. Memory impairment, inability to walk, drooling saliva, and loss of coordination
were seen in 10 (4.4%), 42 (18.7%), 7 (3.1%), and 1 (0.4%) respectively. 25 patients presented with facial nerve palsy with or without other symptoms

5.4 CT Scan Findings.

Majority of the participants 66(29.3) who presented with a clinical diagnosis of cerebro-vascular disease, had ischemic stroke, while 27 (12.0%) suffered hemorrhagic stroke. This finding is in keeping with some studies, for example: In a study on pathological sub-types, risk factors and outcome of stroke at the Nairobi Hospital, Kenya, it was shown that ischaemic stroke form 85% of findings, whilst haemorrhagic stroke was 8.8%(Jowi & Mativo, 2008). Six point three percent showed no evidence of stroke sub-type. Nakibuuka et al also demonstrated the same finding where 69.3% of participants had ischemic stroke and 30.7% had hemorrhagic stroke. The above findings can be collaborated by Bejot et al (Bejot et al., 2015) and in the study on global and regional burden of stroke(Feigin et al., 2014; Lozano et al., 2012). Our finding are however in slight contrast with one which was done on stroke injury, cognitive impairment and vascular dementia (Kalaria, Akinyem, & Ihara, 2016) who concluded that ischaemic strokes is almost 4-fold greater than haemorrhagic strokes. We could put an argument forth that our study included all the possible CT scan findings in computing the percentages unlike Kalaria et al who dealt with only confirmed patients on imaging of stroke in their attempt to link it with cognitive impairment and vascular dementia. Ischaemic strokes in other similar studies were also found to be higher almost 10 fold(Sacco, Wolf, Kannel, & McNamara, 1982). Similar studies done in West Africa also corroborate our study findings; Ischaemic stroke was found the most common 66.2% among study participants, haemorrhagc stroke 32.3% and
combined ischaemic and haemorrhagic, 1.5% (Bello, Aremu, Mustapha, & Olugbenga-Bello, 2010). In a 2 year audit of cranial computerised tomography in a general medical unit; completed stroke, transient ischaemic attacks (TIA), epilepsy and headache were the most common indications for cranial CT while infarct, atrophy, haemorrhage and tumour were the most common abnormalities (Curley, Lynch, Walker, & Doyle, 1990).

After the age of about 50 years the normal brain loses mass and volume at a variable rate, perhaps averaging about 0.1–0.3% per year (Allison, 2008). Cerebral atrophy is largely asymptomatic owing to it being a natural course in an aging brain. For clinicians to send patients for imaging with a clinical diagnosis, focal neurological lesions might have been demonstrated to warrant suspicion of cerebrovascular disease. Cerebral atrophy was diagnosed in 41 (23.0%) of the participants while another important group whose proportions were lumped up with ischemic stroke was the group 5(2.8%) which had both ischaemic stroke and cerebral atrophy. One can draw comparatives with a study on association of cerebral atrophy and in ischemic stroke which found that cerebral atrophy was present in 771 of 1360 patients with acute stroke (56.7%), of whom 203 (26.3%) had isolated cortical atrophy, 144 (18.7%) had isolated sub-cortical atrophy, and 424 (55.0%) had both atrophy (Baker et al., 2010). In the same study on differential associations of cortical and sub-cortical cerebral atrophy, Baker et al. sub classified stroke into three; cortical, sub-cortical and a combination of both (Baker et al., 2010). Small vessel disease in the brain has been increasingly recognized to be one of the most prevalent neurological disorders and is also associated with aging and cerebral atrophy (Thompson CS, 2009; ). Image 22, for example, is a demonstration of fronto-polar atrophy in a 70 yr old man diagnosed clinically to have stroke; but these findings would indicate the contrary where a diagnosis of fronto-polar dementia was made.
A total of 42 (23.5%) participants had normal CT with clinical diagnosis of stroke. This drew the question that could likely causes of symptoms be conversion syndromes? Hypochondriasis? Malingering? Other imaging modalities brought in i.e. MRI’s Diffusion weighted imaging to rule out the acute ischaemic stroke, but this wasn’t part of the study. These findings were however not unique to our study. It was found out that five patients (6.3%) out of a total of 80 sampled showed no evidence of stroke (Jowi & Mativo, 2008). In another study of a sampled group of 86 participants, 65 (75.6%) had stroke. Normal study was noted in 7 (8.12%) patients and 14 (16.3%) patients had other intracranial conditions wrongly diagnosed clinically as stroke (Bello et al., 2010). In a similar study on the comparison of two hospital stroke scores with computerized tomography in ascertaining stroke type among Nigerians, it was found out that the prevalence of hemorrhagic stroke diagnosed by gold standard (CT) was 29.5% while the prevalence of ischemic stroke diagnosed by CT was 54.7%. The CT brain was normal in 15.8% (Salawu, Umar, & Danburam, 2009). It is therefore imperative that this “special” group of normal findings on CT patients to be managed interdepartmentally since as a medical fraternity we would need to address the root cause of symptoms.

Our study demonstrated 9 (5.0%) participants as having tumors either primary or metastatic. These included meningiomas, high grade astrocytomas likely Glioblastoma multiforme, choroid plexus carcinoma and metastatic tumours. Some tumours for example Image 17a showed an occipital parafalcine meningioma coexisting with a thalamic hemorrhagic stroke Image 17b. In a retrospective study between January 1984 and December 1993 on brain tumours at the Kenyatta National Hospital, Nairobi, demonstrable clinical signs were mainly headache, spontaneous vomiting, nausea, ocular palsies, altered level of consciousness, back pain and papilledema (Mwang’ombe & Ombachi, 2000). Other scan findings included
hematoma, and hydrocephalus diagnosed in 24 (28.6%) and 20 (23.8%) participants respectively. The study (Table 4.4) demonstrated 11(45.8%) left sided subdural haematomas, 8(33.8%) being right sided and 5 (20.8%) being bilateral i.e. Image 19. The remaining 40 participants had a varied number of symptoms listed above in insignificant proportions.

5.5 Location of stroke patterns.

Not putting into consideration the type of stroke (either ischaemic or haemorhagic) the study found out that the majority of cases were found in the cerebral cortex supplied by the perforating cortical branches of ACA,PCA and MCA, followed by the basal ganglia (lenticulostriate branches of MCA), the thalamus(thalamogeniculate branches of PCA) and finally the cerebellum(penetrating branches of PICA,AICA,SCA). No pontine ischaemic or haemorrhagic lesion was recorded during the time of study.

Further sub-classification of the cerebral lobe affliction showed that the majority of lesions were in the parietotemporal lobe 26(11.6%), followed by occipital lesions 18(8%), parietal 13(5.8%), frontoparietal 11(4.9%), frontal8(3.6), parietal occipital 7(3.1%), and finally whole cerebral lobe lesions 4(1.8%). Table 4.5 .Our findings are in stark contrast with a study done by Arun et al on grading of cerebral infarction using CT Hounsfied unit to report the Hounsfield in acute, subacute and chronic(Arun S Govind, 2015). Their study demonstrated that of the ischaemic strokes found, 9( 45%) were frontal, 7(35%)were frontoparietal and a combination of both frontoparietal and parietaltemporal 4(20%)(Arun S Govind, 2015). An explanation to this could be the small sample size and the bias of excluding patients with any other type of stroke or cerebral lesion. Some comparison however can be made with a West African study which sought to define cranial
computerized tomographic assessment of cerebrovascular disease. It could be concluded that most 60.5% of the ischaemic stroke lesions were localized to the region of the middle and anterior cerebral arteries (Bello et al., 2010). Our percentages of 83.03% in the cerebral lobes could be due to the fact that we included sites of lesions of haemorrhagic CVD in the numerator.

**5.6 Specific side of the brain where the patterns were found**

Save for the internal capsule 1(25%) and the parietal lobe 6(46.2%) cerebrovascular disease patterns were found to be predominantly in the right and more so for the frontal and parietotemporal. One would be drawn to conclude that the above mentioned areas predominantly demonstrating lesions are likely large artery arteriosclerosis. In the globally acclaimed Trial of Org 10172(Adams et al., 1993) in Acute CVD Treatment subclassification of CVD was done as : 1) large-artery atherosclerosis, 2) cardioembolism, 3) small-vessel occlusion, 4) CVD of other determined etiology, and 5) CVD of undetermined etiology for ischaemic CVD(Adams et al., 1993). Our study would seem to be in agreement with the aforementioned study. The risk of occurrence is also in this order. The pattern of brain lesions as shown by brain MR imaging can also be classified according to a modified Oxfordshire method, based on the anatomic distribution of the infarcts into six groups: (1) total anterior circulation infarcts, (2) partial anterior circulation infarcts, (3) posterior circulation infarcts, (4) watershed infarcts, (5) centrum ovale infarcts, and (6) lacunar infarcts(Rovira, Grive, Rovira, & Alvarez-Sabin, 2005). The same sequence also demonstrates risk of occurrence. One would then ask …why the use of ischemic CVD solely as a rating of risk occurrence in these two studies? But they would be drawn to note previous studies quoting the occurrence of CVD being 4 fold upto ten fold high(Bejot et al., 2015; Feigin et al., 2014; Jowi & Mativo, 2008; Nakibuuka et al., 2015; Sacco et al., 1982). So
yes.. hemorrhagic CVD would be included in the sub classification stated above. Of course further studies to demonstrate this small proportion but high mortality causing hemorrhagic CVDs are needed and advocated for. Other studies however differ on the categorization of TOAST grading on frequency of occurrence. Puig et al for example found out that CVD etiologic subtypes were 33.3% for large artery arteriosclerosis, cardioembolic in 46.7%, and indeterminate in 20%(Puig et al., 2012).

5.7 Hounsfield unit count variations of CVD type

Acute bleeding is typically in the range of 50-100 Hounsfield units(Peron, 2007). As the blood becomes older and the haemoglobin molecule breaks down, it will lose this hyper dense appearance, beginning at the periphery and working centrally. On CT blood will progress to be isodense with the brain (4 days to 2 weeks, depending on clot size), and finally darker than brain (>2-3 weeks)(Dolinskas, Bilaniuk, Zimmerman, & Kuhl, 1977; J. Kornbluth, 2015; Peron, 2007). Compared with unclotted whole blood (HU count of 56 with hematocrit level of 45%), when whole blood coagulates in vitro, the clot retracts and HU count rises to, for example, 75 with hematocrit level elevation to 80%(J. Kornbluth, 2015). The reduction in size and attenuation values in intracerebral hemorrhage have been estimated at rates of 0.65 mm and 1.4 Hounsfield units per day, respectively(Dolinskas et al., 1977).

The above mentioned findings were in concurrence with our study i.e. the mean Hounsfield unit count for the participants who had hemorrhagic CVD was 60.8 ± 9.1. More than three quarter of the participants 21(77.8%) had hyper-acute hemorrhagic CVD (HU: >=55), 3 (11.1%) had sub-acute (HU: 35-45), and another 3 (11.1%) had acute hemorrhagic CVD (HU: 45-55). The findings of finding a high number of hyperacute cases can be explained by
the rapid progression of symptoms in haemorrhagic CVD hence health seeking behaviours are very rapid (Nakibuuka et al., 2015). We however did not factor in haematocrit levels in our study to look at the correlations. We did not also follow up the evolution of the hemorrhagic CVDs.

On the other hand, 32(50%) of the participants with ischemic CVD had sub-acute ischemic CVD (HU: 9.55-19.13), and 26 (40.6%) had acute ischemic CVD (HU: >=19.13). The remaining 9.4% had chronic ischemic CVD (HU: <9.55). Explanation of the subacute patients being more can be explained by the fact that some CVD types i.e lacunae type have an indolent kind of progression. One of the participants for example, (Image 20) had right hand bothering weakness that had been present for over 3 months. In the TOAST classification of ischaemia large arteries have to be stenosed for over 50% for ischaemic symptoms to occur (Adams et al., 1993). That is however not to say that the ischaemic CVD have no acute symptoms. Thromboembolic and large artery atherosclerosis have acute progressions that could lead to death.

In contrast to our study findings Phan et al (2010) observed the range value of gray scale levels for main brain structures and for CVD tissues got the HU range as 20 to 30 acutely (Phan et al., 2010). It was encouraging however to note that this study results on ischaemic CVD had similarities with a study by Arun et al who found that 23 HU with standard deviation of +/- 1.369 HU was the average value of acute infarct. Then 16.95 HU was the average value of sub acute infarct with standard deviation of +/- 2.493. In chronic infarct average value was 6.35 HU with standard deviation of +/- 1.622 HU.
CHAPTER SIX: CONCLUSION AND RECOMMENDATION

6.1 CONCLUSION

1. The most common CT scan pattern of patients suspected to have CVD was ischemic stroke.

2. The average HU for ischemic and hemorrhagic stroke subtype was 18.0 +/- 8.6 and 60.8 +/- 9.1 respectively.

6.2 RECOMMENDATIONS.

1. The use of Hounsfield Unit values in characterization of suspected cerebro-vascular disease is recommended.

2. Further studies ought to include MRI series especially in patients found to have normal CT scans and have been suspected to have CVD.
REFERENCES:


APPENDICES

Appendix I: Consent form

English version

My name is Dr. Tarus Felix Kiplimo, duly registered by the Kenya Medical and Dentists Board to practice as a Medical officer. I am a registrar pursuing a Masters degree in Radiology and Imaging at Moi University’s Medical school. I would like to include you/your next of kin into my research trying to find out the patterns of cerebrovascular disease on CT scanning in patients at Moi Teaching and Referral Hospital.

Purpose. This study will try to benchmark CT scan patterns of patients presenting with cerebrovascular disease at MTRH.

Procedure; All patients who have a clinical diagnosis of CVD and from whom consent has been obtained shall have a CT scan head done and subsequently reported. The subject shall receive the report. Findings shall kept confidential throughout the research period and subjects’ identities shall never be revealed.

Benefits: There will be no direct benefits of participating in this study. Study subjects will be accorded same quality of management as non-study subjects.

Risks. There are no anticipated risks while undergoing CT scanning. However contrast is not 100% without side effects which could include flushing and nausea.

Confidentiality: All information obtained in this study shall be treated with utmost confidentiality and shall not be divulged to any unauthorized persons.

Rights to Refuse: Participation in this study is voluntary, there is freedom to refuse to take part and it shall have no consequence on quality of management. This study has been
approved by the Institutional Research and Ethics Committee (IREC) of Moi University/Moi Teaching and Referral Hospital

Sign or make a mark if you agree to take part in the study.

Patient/Next of kin…………………… Investigator……………………

Date……………………

Tafsiri kwa Kiswahili.


Lengo; Lengo kuu ya utafiti huu ni kupeleleza aina za kiharusi kulingan na picha za CT zipigwazo katika Hospitali Kuu ya Mafunzo na Rufaa ya Moi.

Utaratibu; Wagonjwa wote walioafikiwa na kiharusi na waliotoa kibali watafanyiwa picha ya CT ambapo pia watalamu wataidurusu na kutoa ripori yake. Mgonjwa atapewa ripoti ile na kisha data itakayopatikana kutoonyeshwa kwa mtu yeyote asiywe na idhini.

Faida; Hakuna faida moja kwa moja atakoyopata ye ye mgonjwa kwa kushiriki katika upelelezi huu

Madhara. Hakuna madhara yoyote katika kupigwa picha ya kichwa ya CT ingawa matumiza ya dawa ya contrast yanaweza kuleta kisunzi na kuhisi kutapika.
Haki ya kukataa: Ushiriki wako katika utafiti huu ni kwa hiari yako, kuna uhuru wa kukataa kujumlishwa. Utafiti huu imepitishwa na Utafiti wa Taasisi na Kamati ya Maadili (IREC) ya Chuo Kikuu cha kufundisha cha Moi na Hospitali kuu ya Rufaa.

Weka sahihi au kufanya alama ikiwa unakubali kushiriki katika utafiti Mgonjwa/Mlezi:.............................Mpelelezi:.............................Tarehe:.............................

......
Appendix II: Protocol for a Head CT scan.

Head CT scan was done using PHILIPS MX 4000 Dual Slice CT scan Machine.

Once the request form was received and indication for the CT scan confirmed, recording into the register along with the In/Out-Patient Number was done.

An intravenous canulla was put in place and for later use in case contrast was to be administered.

The patient was then positioned in a supine head first secured in position with pads on both sides and strapped in place to be firm.

Laser light was then centered at midline and about 2.5 cm from the vertex so as to include the entire head. (This was because the movement of the patient would be couch in such that they move inwards as the scan is done).

The settings were done as follows, Kilo-voltage 120Kv, 150MAs, 80 Window Width and 35 Window Level, Collimation of 5mm was used.

The scan was started with a scanogram for planning then necessary windows were set where brain and bone windows showed brain and bones respectively. Scanning was then done from the C2 and C1 spines to the vertex.

The images were shown on the computer screen and any intracranial lesion demonstrated. The images were then loaded onto the film format in the computer after which the film was then printed and interpretation done.
Appendix III: Data collection form

Instructions

1. To be filled by investigator once the client consents to the study.

### 1. DEMOGRAPHICS

<table>
<thead>
<tr>
<th>Date</th>
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</thead>
<tbody>
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</tr>
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</tr>
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<tr>
<td>Gender</td>
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<td></td>
</tr>
<tr>
<td>County of Residence</td>
<td></td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
</tr>
</tbody>
</table>
2. ATTENDANT

What is the guardian/attendant’s relationship with the patient?

Mother……………………………………

Father……………………………………

Brother……………………………………

Sister……………………………………

Good Samaritan…………………………

Other(Specify)…………………………

3. Clinical diagnosis at referral point

.............................................................................................................
4. **CLINICAL FINDINGS**

Tick the presence or absence of these clinical features

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paralysis of both lower limbs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness of both lower limbs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakness of one side of the body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of vision on one eye</td>
<td>Side:</td>
<td></td>
</tr>
<tr>
<td>Loss of vision on both eyes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Memory impairment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inability to talk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drooling saliva</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
5. When did the symptoms occur?

- Less than 24 hours (Hyperacute) 
- 1-3 days (Acute) 
- 4-7 days (Early subacute) 
- 8-14 days (Late subacute) 
- >2 weeks (Chronic)

6. Has patient been involved in an accident recently?

YES ☐ NO ☐
7. SCAN FINDINGS

<table>
<thead>
<tr>
<th>Findings (tick where it applies)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal CT</td>
</tr>
<tr>
<td>Ischaemic CVD</td>
</tr>
<tr>
<td>Haemorrhagic CVD</td>
</tr>
<tr>
<td>Tumours</td>
</tr>
<tr>
<td>Others:</td>
</tr>
</tbody>
</table>
Location of the finding

<table>
<thead>
<tr>
<th>Cerebral lobes (Penetrating cortical branches of ACA,MCA,PCA)</th>
<th>Location</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontoparietal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parieto-temporal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietal-occipital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occipital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole Cerebral lobe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal ganglia(Lenticulostriate br’s MCA)</td>
<td>Caudate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Globeus pallidus</td>
<td>Putamen</td>
<td></td>
<td></td>
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<tr>
<td>Internal capsule</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thalamus: (Thalamogeniculate br’s PCA)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pons (Basilar br’s)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebellum: Penetrating br.’s PICA, AICA, SCA</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hounsfield’s units at site of CVD: ....................................................
Appendix IV; Image formation and acquisition on CT

Figure 11: Computed tomographic (CT) images are produced from a large number of x-ray transmission measurements called rays.

A group of rays acquired in a certain geometry is called a projection or view. Two different geometries have been used in CT, parallel beam projection and fan beam projection (Bushberg)
Figure 12: Data acquisition in CT involves making transmission measurements through the object at numerous angles around the object (left).

The process of computing the CT image from the acquisition data essentially reverses the acquisition geometry mathematically (right). Each transmission measurement is back projected onto a digital matrix. After back projection, areas of high attenuation are positively reinforced through the back projection process whereas other areas are not, and thus the image is built up from the large collection of rays passing through it (Bushberg).
Appendix V: Approval to conduct research at MTRH

MOI TEACHING AND REFERRAL HOSPITAL

Telephone: 2033471/2/3/4
Fax: 61749
Email: director@mtbh.or.ke
Ref: ELD/MTRH/R.6/VOL.II/2008

Dr. Tarus Felix Kiplimo,  
Moi University,  
School of Medicine,  
P.O. Box 4606-30100,  
ELDORET-KENYA.

RE: APPROVAL TO CONDUCT RESEARCH AT MTRH

Upon obtaining approval from the Institutional Research and Ethics Committee (IREC) to conduct your research proposal titled:-

"Computed Tomography Scan Patterns of Patients Presenting with Cerebrovascular Disease at Moi Teaching and Referral Hospital - Eldoret".

You are hereby permitted to commence your investigation at Moi Teaching and Referral Hospital.

DR. JOHN KIBOSIA  
DIRECTOR  
MOI TEACHING AND REFERRAL HOSPITAL

CC - Deputy Director (CS)  
 - Chief Nurse  
 - HOD, HRISM
Appendix V: IREC Formal Approval

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 23
ELDoret
Tel: 254/0203
Reference: IREC/2014/122
Approval Number: 0001247

Dr. Tarus Felix Kipimo,
MoI University,
School of Medicine,
P.O. Box 4609-30100,
ELDoret-KENYA,

Ms. Dr. Tarus,

RE: FORMAL APPROVAL

The Institutional Research and Ethics Committee has reviewed your research proposal titled:

“Computed Tomography Scan Patterns of Patients Presenting with Cerebrovascular Disease at MoI Teaching and Referral Hospital - Eldoret.”

Your proposal has been granted a Formal Approval Number: FAIN: IREC 1247 on 25th August, 2014. You are therefore permitted to begin your investigations.

Note that this approval is for 1 year; it will thus expire on 24th August, 2015. If it is necessary to continue with this research beyond the expiry date, a request for continuation should be made in writing to IREC Secretariat two months prior to the expiry date.

You are required to submit progress report(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change(s) or amendment(s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc: Director - MTRH Dean - SOP Dean - SCM
    Principal - CHS Dean - SON Dean - SCD
Appendix VI: IREC Amendment letter

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

Reference: IREC/2014/122
Approval Number: 0001247

Dr. Tarus Felix Kiplimo,
Moi University,
School of Medicine,
P.O. Box 4609-30100,
ELDORET-KENYA.

Dear Dr. Tarus,

RE: APPROVAL OF AMENDMENT

The Institutional Research and Ethics Committee has reviewed the amendment made to your proposal titled:

“Head Computed Tomography Scan Patterns of Stroke Patients Suspected to have Cerebrovascular Disease at Moi Teaching and Referral Hospital – Eldoret, Kenya”.

We note that you are seeking to make an amendment as follows:

1. To change the title as above from: “Computed Tomography Scan Patterns of Patients Presenting with Cerebrovascular Disease at Moi Teaching and Referral Hospital - Eldoret”.

The amendment has been approved on 26th July, 2016 according to SOP’s of IREC. You are therefore permitted to continue with your research.

You are required to submit progress(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change(s) or amendment(s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc: CEO - MTRH Dean - SPH Dean - SOM
    Principal - CHS Dean - SOD Dean - SON