ECG PATTERNS AND THREE MONTHS OUTCOMES IN PATIENTS ADMITTED WITH NEW ONSET STROKE AT MUHIMBILI NATIONAL HOSPITAL

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by Muhimbili University of Health and Allied Sciences a dissertation entitled "**ECG PATTERNS AND THREE MONTHS OUTCOMES IN PATIENTS ADMITTED WITH NEW ONSET OF STROKE AT MUHIMBILI NATIONAL HOSPITAL"** in fulfillment of requirements for the degree of Master of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences.

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DECLARATION
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I, Dr Khuzeima Khanbhai declare that this **dissertation** is my own original work and that it has not been presented and will not be presented to any other university for a similar or any other degree award.

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DEDICATION

This dissertation is dedicated to my grandfather Mr. Sheikh Jafferbhai Khanbhai, My parents Mr. Shabir and Mrs. Nafisa Khanbhai, my sister Lamiya ,my wife Zahabiyah and my two little kids Sakina and Abbas Khanbhai who have always been instrumental and inspirational in my life and my career as a doctor.

ABSTRACT

Background

Stroke is on the rise in Tanzania with great impact on morbidity and mortality. One of the factors associated with poor outcome among stroke patients are some abnormal electrocardiographic (ECG) changes resulting from massive autonomic discharge along the sympathetic outflow tracts of the nervous system that interferes with cardiac autonomic control. This study aimed at determining the ECG patterns among new onset stroke patients and their clinical outcomes in terms of morbidity and mortality at 3 months.

Objective

To determine ECG patterns and three months outcomes of patients admitted with new onset stroke at Muhimbili National Hospital.

Methodology

This was a descriptive follow up study conducted at the Muhimbili National Hospital in-patient neurology units. We enrolled 160 patients with new onset stroke regardless of its type. All eligible patients who consented for the study underwent 12 lead resting ECGs. These patients were then followed up at 1 month and 3 months from the day of enrollment for determination of their survival/morbidity and mortality outcomes.

Descriptive statistics were used to describe the socio-demographics of the study population in terms of frequencies. Chi-square test was used to compare proportions of different ECG patterns, type of stroke, and outcomes in terms of mortality or survival in 1 month and 3 months of follow up. Multivariate analysis was used to control for confounding factors on socio-demographic characteristics and ECG patterns as related to the outcomes.

Results

From August 2012 to mid December 2012 a total of 527 patients were admitted at MNH neurology unit. Out of these 160 patients met the inclusion criteria and consented for the study. Their mean age was 58 years with standard deviation of ± 14.695.

Of these 53.1% were Males. 87.5% had HTN, 41.3% were diabetic (DM), 39.4% had both HTN and DM. A total of 9 ECG patterns were identified out of which LVH, ischemic changes and QT Prolongation were dominant; (83.1%, 63.1% and 50.6% respectively). QT prolongation and Ischemic changes were found to be statistically significant in predicting outcomes at 1month and at 3 months with P-value <0.001 and 0.036 respectively .Those with QT prolongation and Ischemic changes had poor outcomes than other patterns.

Patients with more than three patterns had poor outcomes as compared to those with less than three patterns (p=0.018) with risk of three times likely hood of mortality at three month.

Patients with hemorrhagic stroke were more likely to present with multiple ECG patterns than were patients with ischemic stroke (p <0.001). Patients with Hemorrhagic stroke had poor outcomes compared to ischemic stroke (p<0.001) in terms of mortality at 3 months

.

Conclusion

- Bed side ECG was a significant tool in identifying certain ECG patterns associated with predicting poor outcomes.
- ECG patterns such as QT-prolongation and Ischemic changes were associated with poor outcomes at three months, irrespective of the co-existence of co-morbid conditions such as (HTN, DM).
- Although not definite some ECG patterns such as QT-prolongation was highly predictive of type of stroke (hemorrhagic vs. Ischemic stroke). But CT-scan still remains definitive tool.

Recommendation

- Every stroke patients should have a bedside ECG which is easily available, and which will guide in overall management of patients and prognosis.
- A new study should be done to see different ECG patterns in patients with HIV disease who presents with acute event of stroke.

ABBREVIATIONS

AAN	-	American Academy of Neurology
AF	-	Atrial Fibrillation
AHA	-	American Heart Association
CBF	-	Cerebral blood flow
CNS	-	Central Nervous System.
CT scan	-	Computed tomography
DSS	-	Demographic Surveillance Sites
ECG	-	Electrocardiogram.
GCS	-	Glasgow Coma Scale
HIV	-	Human Immunodeficiency Virus
ICH	-	Intracranial Hemorrhage
LVH	-	Left Ventricular Hypertrophy
MNH	-	Muhimbili National Hospital
MRI scan	-	Magnetic Resonance Imaging
MUHAS	-	Muhimbili University of Health Allied Science
NIHSS	-	National Institute of Health Stroke Scale
PVC	-	Premature Ventricular contraction
QTc	-	Corrected QT interval
QTd	-	QT dispersion

SAH	-	Subarachnoid Hemorrhage
SpO2	-	Pulse oximetry
SSA	-	Sub-Saharan Africa
SVT	-	Supraventricular Tachycardia
tPA	-	tissue Plasminogen Activator
TSIP	-	Tanzania Stroke Incidence Project
USA	-	United States of America
VT	-	Ventricular Tachycardia
WHO	-	World Health Organization

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CHAPTER ONE

1.0 INTRODUCTION AND LITERATURE REVIEW

1.1 EPIDEMIOLOGY

An electrocardiogram (ECG) is a device used for recording electrical activity of the myocardium by detecting transmission of cardiac impulse through the conductive tissues of the heart muscles. It allows diagnosis of specific cardiac rhythm abnormalities.[1]

CNS disorders, including stroke may produce ECG changes and arrhythmia. For the first time in 1944, Byre and colleagues described the ECG changes in sufferers of subarachnoid hemorrhage [2]. ECG changes have been reported in 60-90% of patients with intra-parenchymal or subarachnoid bleed and in about 5-20% of patients with acute ischemic stroke [3]. The underlying cause has been postulated to be disordered depolarization process[4]. There is also a relationship between these changes and sudden death in sufferers of stroke [5].

In a study by Kager A.G et al, abnormalities such as ischemic-like ECG changes and/or QT prolongation, were found in 76% (95% CI 73–90) of patients with subarachnoid hemorrhage, irrespective of whether they had preexisting heart disease or not.[6]

In the same study by Kager A.G et al it was found out that of 555 consecutive black patients with acute stroke done in South African population, 72% had cerebral infarction and 28% had intracranial hemorrhage (ICH). ECG changes compatible with myocardial ischemia were present in 81 patients (15%). Pathological Q waves compatible with previous myocardial infarction (MI) were present in 4 (1%) patients. Changes considered to indicate coronary artery disease were present in 17% of the patients. [7]

Stroke and related ECG changes

In patients with stroke the ECG abnormalities most frequently noted are ischemic changes (35%), prolongation of QT interval (45%) and disturbances in rate and rhythm (25%), which include atrial fibrillation, premature atrial and ventricular complexes, supra-ventricular tachycardia (SVT) and ventricular tachycardia (VT), torsades de pointes or polymorphic ventricular tachycardia's [8,9].

Stroke induced ECG changes are transient (temporary,) resolving over a period of days to months. The frequency and severity of ECG changes is highest within 48 hours of the onset of stroke which explains the importance of continuous ECG monitoring for these patients.[10]

The ECG abnormalities and part of mortality in stroke patients have been found to be attributable to cardiac autonomic imbalance generated by acute cerebral lesion.[9] Massive autonomic discharge along the sympathetic outflow tracts of the nervous system produces tachyarrhythmias.[11]

A study by Muhamed A.A *et al* reported repolarization abnormalities, manifesting as prolonged QTc to be one of the most frequent ECG abnormalities irrespective of the stroke type (63.63% vs. 68.29%).The two varieties of stroke (ischemic and hemorrhagic stroke) manifest more or less similar patterns of ECG changes.[12]

In another recent study by Ince B et al, the ECG of patients receiving emergency treatment for an ischemic stroke could help doctors predict the outcomes in this patient in the next three months.[5]

In some studies in patients with ischemic stroke, the prognostic importance of these ECG parameters, particularly ST-segment changes and prolonged QT interval has been demonstrated.[13.14]

A prolonged QT interval has been associated with increased risk for ventricular tachyarrhythmia's, like torsades de pointes and which is a risk factor for sudden death.[15]

QTc which is corrected for heart rate, has been found to be an independent indicator of arrhythmias and mortality in conditions such as long-QT syndrome, cardiomyopathy and chronic heart failure.[16,17]

A study by Dr. Latha G Stead et al reports that stroke patients with QTc interval longer than normal are at a higher risk within the next three months, being highest for women

with a QTc interval over 440 milliseconds and for men with a QTc interval over 438 milliseconds.[18]

Nazire Afsar, et al, provided further evidence for an interaction between the CNS and the cardiovascular system during acute cerebrovascular events.[19] The conclusion was that he noted ECG abnormalities inclusive QTc dispersion in the first 24 hours were as a result of incident stroke.

Prevalence of Stroke

Stroke is the second commonest cause of death worldwide,[20] with two-thirds of these deaths occurring in developing countries. The incidence of stroke in developing countries is also expected to rise in the future as the populations undergo what has been referred to as the health transition.[21]

Estimates suggest that 8% of all first-ever strokes occur in Africa and that 5% of the 30 million stroke survivors worldwide live in Africa.[22] Ischemic stroke is said to be equally common in the black race, [23,24] though some studies claimed that cerebral hemorrhage might be commoner.[25] When compared with subarachnoid hemorrhage, it occurs in older patients[26] and causes less mortality,[27] though it is associated with considerable disability and morbidity.

In the Tanzania Stroke Incidence Project (TSIP), patients with stroke were identified by community-based investigators and by liaison with local hospital and medical centre staff. Deaths from stroke were identified using verbal autopsy. A total of 201 strokes were identified by the TSIP and 435 strokes that had not been identified by TSIP were identified by verbal autopsy.[28]

Age-specific incidence rates in both the Hai demographic survey and the Dar-es-Salaam survey study (DSS)were similar or higher than reported from high-income countries.

It is also known that among blacks, stroke is more common, more severe, and carries a higher mortality when compared with other races. This is attributed by the fact that blacks have an increased frequency of traditional risk factors for stroke such as diabetes mellitus, hypertension, and obesity.[28]

1.2 EFFECT OF STROKE ON CARDIAC ELECTROPHYSIOLOGY.

There is strong clinical evidence of cerebrogenic cardiac arrhythmias and myocardial changes during ischemic stroke, intracerebral hemorrhage, and subarachnoid hemorrhage. Alternative scenarios include cardioembolic stroke, multisystem diseases with stroke-like and cardiac features. Cardiac effects of stroke may be severe or even fatal and worsen the prognosis of stroke.[29] the stroke worsened prognosis may partly be due to stress response as well as cardiac changes following stroke. These changes may have role in future therapeutic strategies. Damage to the right insula (part of the brain) may hold a key role for the stress response and cardiac changes after stroke.[30]

Clinical and experimental studies suggest that cortical and subcortical structures such as the insular cortex and amygdala play a pathogenic role. The peripheral mechanisms involve abnormal sympathetic activity, altered parasympathetic activity, and raised levels of circulating catecholamine's, whereas the central mechanisms are largely unknown.[31]

The two major mechanisms causing brain damage in stroke are ischemia and hemorrhage. In ischemic stroke, which represents about 80% of all strokes, decreased or absent circulating blood deprives neurons of necessary substrates.[32]In hemorrhagic stroke, SAH have been associated more than 25% to 90% in disregulation of cardiac autonomy and is a poor prognostic predictor of the patient's outcome independently.[13]

During the acute episode of stroke, there is disruption of cardiac autonomy, which causes direct effect on cardiac electrical impulses. The mechanism by which these abnormalities occur is not well understood but may involve the insular cortex.[33] Insular damage may cause activation of the sympathico–adrenal system because of decreased inhibitory insular activity.[34]Intraoperative insular cortex stimulation studies indicate right sided dominance in mediation of sympathetic cardiovascular effects.[35]

SAH in the right sylvian fissure has been shown to have cardiac consequences.[36] these findings have been supported by animal models,[37] leading to the hypothesis that insults to the right insular in a direct or indirect manner affect cardiac function; a possible consequence may be sudden cardiac death after stroke.[33]

Recent experimental evidence in humans with stroke suggests that increased sympathetic nervous system activity accounts in part for the ECG abnormalities associated with stroke. There is association between ECG changes and elevated catecholamine levels in patients with subarachnoid hemorrhage.[34]

ECG findings may also have prognostic significance in stroke patients. Ventricular arrhythmias, concurrent MI, and a prolonged QT interval have all been associated with increased mortality in stroke patients.[38]

1.3 CLINICAL PRESENTATION

The symptoms and signs of stroke start suddenly and ranges from mild to severe depending on the area of brain insult. The more extensive the area of insult the more functions likely to be lost. This may be associated with localizing neurological signs.

During the event of stroke there is activation of sympathetic system, which accounts in part for cardiovascular symptoms, signs and ECG abnormalities associated with stroke. Increased sympathetic activity symptoms may include palpitations, sweating, extreme hypo- or hypertension, increased frequency of QT prolongation and new ischemic changes. Other ECG abnormalities may be seen such as high frequency of left ventricular ischemic changes, Q waves, arrhythmias, bundle branch blocks, left ventricular hypertrophy and myocardial necrosis.[13]

Additionally, symptoms of raised ICP can be seen due to compression of brain parenchyma because of intracerebral hemorrhage.

1.4 DIAGNOSIS

Stroke is diagnosed through several techniques: a thorough medical history and neurological examination (such as the NIHSS) gives an evaluation of the location and severity of a stroke. Ideally all stroke patients should also have an ECG and other diagnostic investigations such as CT scans or MRI scans, Doppler ultrasound, and cerebral-arteriography.

Different imaging techniques in stroke patients are of diagnostic importance. For ischemic stroke, CT scans (without contrast enhancements) has a sensitivity of 16% and specificity of 96% and MRI scan has a sensitivity of 83% and specificity of 98%. In the case of hemorrhagic stroke, CT scans (without contrast enhancements) has a sensitivity of 89% and specificity of 100% and MRI scan has a sensitivity of 81% and specificity of 100%.[1] For detecting chronic hemorrhages, MRI scan is more sensitive.[39]

Other methods to identify the underlying cause of stroke include duplex carotid studies. Additional studies include Holter monitoring studies to identify intermittent arrhythmia; angiogram of the cerebral vasculature if a bleed is thought to have originated from an aneurysm or arteriovenous malformation. Blood tests to establish presence of other risk factors such as hypercholesterolemia, Diabetes, and HIV should be done.

1.5 MANAGEMENT OF STROKE PATIENTS.

Stroke patients are best managed in an acute stroke unit during their initial presentation. A cardio cerebral approach is desirable with close cardiovascular and neurologic monitoring.

Prolonged and intensive cardiovascular monitoring is recommended in patients manifesting cerebrogenic cardiovascular disturbances after an insular stroke, especially in those with advancing age, coexisting hypertension or coronary artery disease.

Management strategy including physiotherapy is focused towards rehabilitation, improving symptoms and improving quality of life.

CHAPTER TWO

2.1 PROBLEM STATEMENT:

Stroke is one of the common conditions contributing to morbidity and mortality all around the world. In Sub-Saharan Africa the burden is high and with transition in life style, the burden of patients with strokes is on the rise. Following a stroke there is disregulation of cardiac autonomy which may cause different ECG changes, Some of these ECG changes have been associated with mortality out comes. [15]

In Tanzania studies have revealed that the burden of stroke is on the rise, and increase in morbidity and mortality among stroke patients is increasing. But there have been no studies in Tanzania to associate ECG changes in stroke patients with mortality and morbidity outcomes in these patients

2.2 STUDY RATIONALE:

A study by Dr. Latha G Stead et al reported that stroke patients with abnormal ECG patterns are at high risk of death during the next three months.[18]

In Tanzania there have been no studies to associate ECG changes in stroke patients with morbidity /mortality outcome. This study was aimed at identifying ECG patterns in patients presenting with acute stroke and determine their significance in relation to outcome at three months.

2.3 STUDY OBJECTIVES:

Broad Objective:

To determine ECG patterns and three months outcome in patients admitted with new onset stroke at Muhimbili National Hospital.

Specific Objectives:

1. To describe the ECG patterns at admission in patients with new onset stroke at MNH.

2. To determine the association of admission ECG patterns in patients with new onset stroke and their outcomes at 3 months.

CHAPTER THREE

3.0 METHODOLOGY:

3.1 Study design:

This was a descriptive follow up study. Patients with new onset stroke were seen at admission, investigated and followed up at one and three month's interval for their outcome in terms of mortality.

3.2 Study population:

Patients who were clinically diagnosed to have new onset stroke admitted at neurology unit at MHN.

3.3 Study site:

This study was conducted at Muhimbili National Hospital (MNH) medical wards, particularly at neurology units. The units have a total of 18 beds and at least 2 patients with acute stroke were admitted per day.

MNH is situated in Dar es Salaam, the largest commercial city in Tanzania. It is the largest tertiary referral hospital in the country receiving referred patients from all the regions in the country and is also teaching hospital for the Muhimbili University of Health and Allied Sciences (MUHAS). Dar es Salaam has an estimated population of 3.8 million people according to the 2012 census. The city is administratively divided into three districts, namely Kinondoni, Temeke and Ilala.

3.4 Inclusion criteria:

1) All patients presenting with symptoms and detectable neurological signs of stroke
2) Patients who are 18 years or older were included.

3.5 Exclusion criteria:

1) HIV infected patients.
2) Patients who had a history of previous stroke

3.6: Sampling technique

Patients were consecutively recruited into the study daily as they were admitted in the neurology units until the study sample size was reached.

3.7 Sample size calculation

Sample size was calculated from the following formula.

$$(z_1\text{-}\alpha/2 + z_1\text{-}\beta)^2 \left\{ \frac{\pi0\,(1 - \pi0) + \pi1\,(1 - \pi1)}{n0 \qquad\qquad n1} \right\} \div (\pi1 - \pi0)$$

$\pi0$ = Proportion of low risk group mortality

$\pi1$ = Proportion of high risk group mortality

Zx = Point of standard normal distribution with area to the left = 1.96

α = type I error rate 5%

β = type II error rate 10%

Study power 85%

n0 = 2n

n1 = 5n

n = Sample size

n = 94 + (10%) = 104

(Taking top 6 confounders Age, HTN ,DM, Alcohol, Smoking, LVH)

Then **n = 160**

The study by GOLDSTEIN et *al*[13] was used to calculate the sample size

3.8 Definitions of the Study

STROKE

- Stroke is defined as rapidly developing clinical signs of focal or global disturbance of cerebral function lasting >24 hours or leading to death with no apparent cause other than vascular origin.

HYPERTENSION

- SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg on two readings at physical examination, also those who are already on medications for hypertension.

DIABETES

- WHO Criteria for blood sugar levels : Fasting > 6.8mmols/L, Random >10mmols/L
- Patients on oral hypoglycemic/insulin

HYPERLIPEDEMIA

- Total Serum Cholesterol > 5.70 umol/l, or those who are already on lipid lowering drugs due to previous hypercholesterolemia.

OUTCOMES

- Mortality was the overall outcome in this study

3.9 Recruitment of study participants:

The investigator visited the neurology units daily to identify those admitted with new onset stroke. Patients with stroke (or their relatives on their behalf if the patients were moribund) were informed about the study and requested to give consent to the study.

Patients or their relative contact details were recorded for follow up purposes.

Follow up of the patients was done at proposed clinic dates one month and three month after discharge. Patients who failed to show up to the clinic were telephoned to see how they were fairing and encouraged to attend the subsequent clinic. The collected information also included date of death. A minimum of 3 clinics was scheduled for each patient.

The principal investigator and a research assistant examined these patients and collected all the data using a pre-tested structured questionnaire. A 12 lead ECG was done by the investigator to all the recruited patients using General Electronic Mac400 machine with the ECG paper set at 25mm/s,10mm/mv, 50~0.05-15Hz.The ECGs were interpreted by the investigator and the findings were confirmed by a senior cardiologist.

Physical Examination

- General and neurological examination was carried out by the investigator and confirmed by the attending physician in the neurology ward within 24 hrs following admission.

- Blood Pressure; Pulse rate, Heart rate, Pulse deficit, Respiratory rate, Spo2, Temperature were recorded.

1) With the patient lying in bed, two blood pressure readings were taken five minutes apart on the UN affected arm using standard mercury BP machine. The average of the two readings was recorded as the patient's blood pressure.

2) Oxygen saturation(Spo2)was recorded using Finger tip pulse oximetry

Serum biochemistry:

- Blood sugar: Random blood glucose was checked for each patient as a bedside test. A pricker was used to prick a middle finger of the unaffected limb. A drop of blood was put on GLUCOSTIX, tested by GLUCO-PLUS glucometer. The serum blood glucose was recorded in mmol/L. Readings above 33mmol/L were recorded as HIGH by the glucometer and these were also recorded as such in the questionnaire.
- Total serum cholesterol Urea and Creatinine: Veno-puncture was done on the veins in the cubital fossa of the unaffected limb after cleansing using methylated spirit in a cotton swab. 5mls of blood was drawn and put in an empty sterile bottle and sent to MNH laboratory for total cholesterol, urea and createnine analysis.

The following ECG indices and patterns were identified and analyzed

i. Corrected Qt (**QTc) INTERVAL**

1. Corrected QT was calculated for each ECG using Bazetts formula

    1. $$QTcB = {}^{QT}\!/\!_{\sqrt{RR}}$$

2. Where **QTc** is the QT interval corrected for heart rate, and RR is the interval from the onset of one QRS complex to the onset of the next QRS complex, *measured in seconds*, often derived from the heart rate (HR) as 60/HR (here QT is measured in milliseconds).
3. The potential significance of QT prolongation is that it leads to life threatening arrhythmias TORSADES-DE-POINTES, which is associated with high mortality rates.

ii. ISCHEMIC CHANGES

1. ST segment elevation or T wave invesion in two consecutive leads of > 2mm, and T wave inversion in corresponding leads

2. Presence of Q-waves in two consecutive leads , suggesting of old myocardial infarct

iii. **HEART RATE (HR)**

1. Tachycardia HR > 90 beats/min
2. Bradycardia HR< 60 beats/min

iv. ARRYTHMMIAS

a. SINUS ARRYTHMIAS

i. Rhythmic repetitive irregularity of the heart beat ,the heart being under normal control of its natural pacemaker the sino-atrial node.[41]

b. Atrial Fibrillation

i. The normal rhythmic contraction of the cardiac atria are replaced by rapid irregular twitching of muscular wall. The ventricles responds irregularity to the dysarrythmic bombardment from the atria. [41]

c. Premature Ventricular contraction

i. Compression within the lower cardiac chambers the ventricles. Such contractions may occur because of abnormal electrical activity of these chambers. [41]

d. **Left Ventricular hypertrophy**

Sokolow-Lyon criteria

i. Sum of S-wave in lead V1, and R-waves in lead V5 or V6 ≥

3) **CT scan of the brain**. Where possible patients had a CT (cost for the CT scan were paid by the relatives). The CT scans were interpreted by consultant radiologist at MNH. Those CT scans which didn't reveal information were interpreted by other consultant radiologist.

3.10 Data analysis:

- SPSS version 20 was used for data entry and analysis
- Descriptive statistics were calculated for both categorical and continuous variables.
- Chi square test was used to determine association between categorical variables.
- Comparison of the means was used to associate continuous and categorical variables at bivariate level.
- Multivariate logistic regression analysis was used to associate ECG patterns with 1 month and 3 months mortality
- P value of < 0.05 was considered statistically significant

Patient's demographic characteristics, risk factors and impact of the disease on daily life were summarized using frequency distribution tables.

3.11 Ethical Consideration

The proposed study possessed negligible risks to the study population. The aims of the study and expected outcomes and utility of knowledge derived from this study was explained to patients and guardians. Study participants were enrolled after the participant/guardian gave written informed consent which was administered by the principal investigator. Participants/guardians who declined to consent did receive the same quality of care as those who consented to participate in the study.

All data collected during the study were treated with strict confidence.The results were also availed to the attending physician at the stroke unit for appropriate treatment. Ethical clearance was given by the Research and Publications Committee of MUHAS and the director of clinical services of MNH.

CHAPTER FOUR

RESULTS

During the study period from August 2012 to mid December 2012, a total of 529 patients were admitted in the Neurology unit at MNH due to various neurological diseases. Out of those, 265 (50.1%) patients were admitted with a clinical diagnosis of stroke. We excluded 105 patients because they were either HIV infected or had an old stroke, thus 160 patients were recruited into the study. There were 2 females who declined to be part of the study of total females with stroke 124. Therefore total of 122 females were part of study.

Out of 160 patients recruited, 82 (51.25%) were males and 78 (48.75%) were females. (Figure 1)

At one month of follow-up a total of 8 (5%) patients were lost to follow up and at 3 months total of 27 (16.8%) patients were lost to follow up. Of 160 patients, 151 (95%) patients had CT-scan of the brain and the type of the stroke could therefore be identified. The rest 5% of patients could not afford CT- scan or had died before the procedure. (Figure 1)

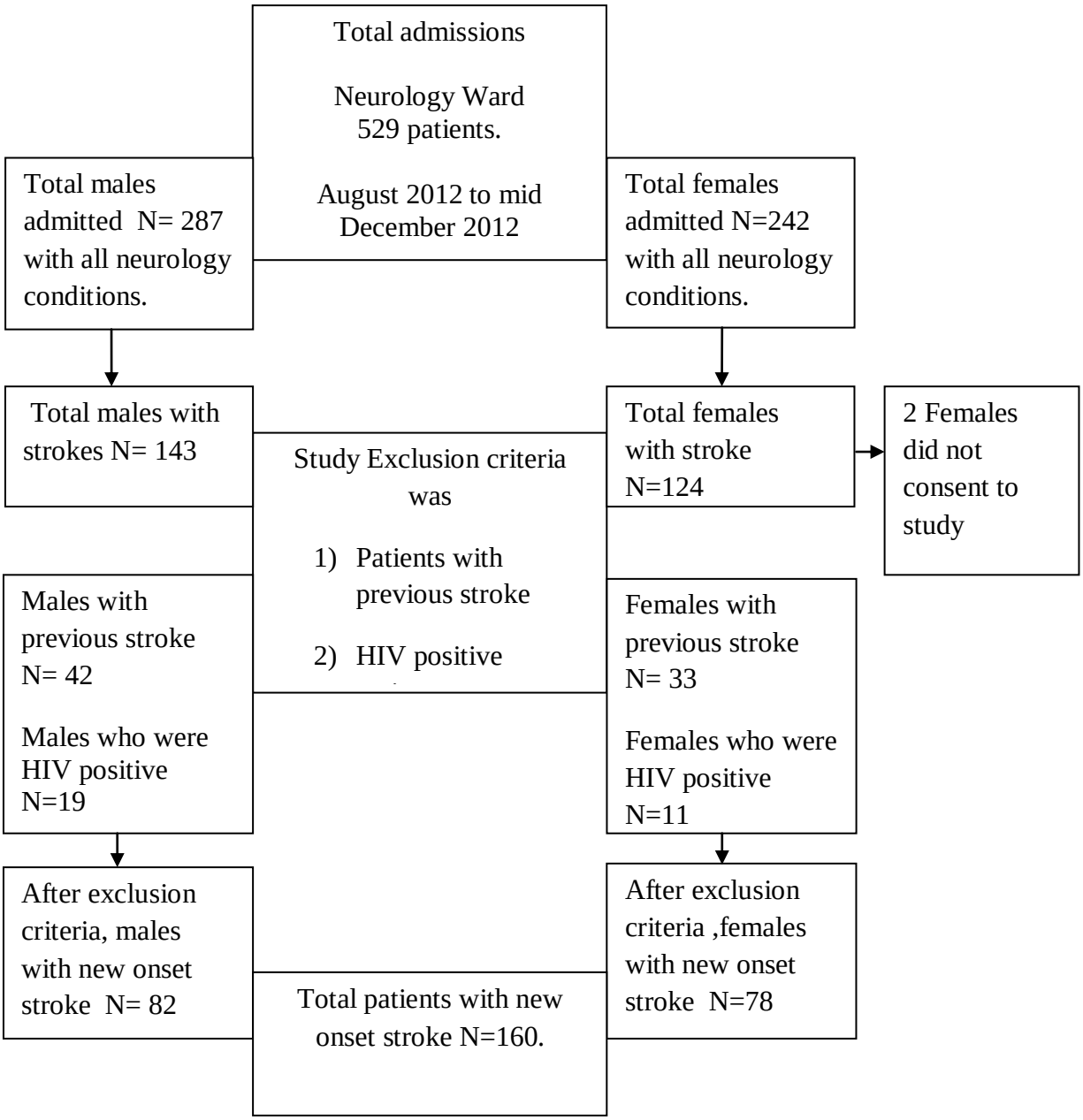


Figure 1: Flow chart of patient recruitment.

The baseline characteristics of the study population are as shown in table 1. Male patients comprised of 51.25% of the study population. Majority (45%) of the study population were in the age group of 50-65 years. The mean (± SD) age of the study population was 58.43 (± 14.695) years. Majority of the study population were married (93.1%), did unskilled work (30%), and had attained only primary education (59.4%).

Table 1: Baseline social demographics characteristics of patients admitted with new onset stroke at Muhimbili National Hospital (N=160)

Characteristic	Number	Percentage
SEX: Male	82	51.25
AGE (years)		
18-30	4	2.5
31-39	17	10.6
40-49	24	15.0
50-64	72	45.0
>65	43	26.9
MARITAL STATUS		
Married	146	91.3
Single	8	5.0
Divorced	3	1.9
Cohabiting	3	1.9
OCCUPATION		
Unemployed	43	26.9
Casual laborer	44	27.5
Unskilled work	48	30
Skilled work	13	8.1
Business owner	12	7.5
EDUCATION		
None	39	24.4
Primary	95	59.4
Secondary	17	10.6
College	9	5.6

Co-morbidities in new stroke patients.

As shown in table 2, majority of the patients (87.5%) were hypertensive. Diabetes mellitus was seen in 41.3% of the patients while co-existence of hypertension and diabetes mellitus was seen in 39.4% of the study population. Only 1.3% of the patients had dyslipidemia at baseline. Females and males were similarly affected by these co-morbidities as all the p-values were not statistically significant.

Smokers comprised of 21 (13.1%) of the total new onset stroke patients, of which 19 (90.5%) were males. Of the smokers, 12/21 (57.1%) were past smokers while 9/21 (42.9%) were current smokers. Males predominated the ever smoked group (91.7%) and the current smoker group (88.9%), p values 0.04 and 0.02 respectively. Taking alcohol was also found to be statistically significant in males who had ever taken alcohol and who were still taking alcohol with p-values 0.04 and 0.03 respectively compared to females. More males were also found to use other forms of tobacco compared to females, however this was not statistically significant. Statistical difference was found with those who were still using tobacco (p 0.048).

Table 2: Co-morbidities in new stroke patients admitted at Muhimbili National Hospital. (N=160)

Co-morbidity	Total	Males N=82	Females N=78	M VS F P-Value
	Number (%)	Number (%)	Number (%)	
Hypertension	140 (87.5)	71 (86.6)	69 (88.5)	0.720
Diabetes mellitus	67 (41.3)	35 (42.7)	32 (39.7)	0.706
Dyslipidemia	2 (1.3)	2 (2.4)	0 (0.0)	0.169
Hypertension and Diabetes mellitus	63 (39.4)	33 (40.2)	30 (38.5)	0.659
Smokers N=21	21 (100)	19 (90.5)	2 (8.5)	0.01
Ever smoked	12 (100)	11 (91.7)	1 (8.3)	0.04
Current smoker	9 (100)	8 (88.9)	1 (11.1)	0.02
Alcohol use N=109	109 (100)	71 (65.1)	38(34.9)	0.03
Ever used	59 (100)	39 (66.1)	20 (33.9)	0.04
Current user	50 (100)	32 (64.0)	18 (36.0)	0.03
Use of other forms of tobacco N=9	9 (100)	8 (88.8)	1 (11.1)	0.68
Ever used	5 (100)	4 (80)	1 (20)	0.19
Current use	4 (100)	4 (100)	0 (0.0)	0.05

Clinical characteristic of study patients

Clinical characteristics of the study are as shown in Table 3. About 45% of the patients were in coma (Glasgow coma score 3-8) at the time of admission. The mean (±SD) systolic blood pressure was above normal: systolic pressure, being 156.5 ± 20.22 mmHg while diastolic pressure it was 88 ± 13.16 mmHg. The mean random blood glucose (RBG) was within normal range being 8 ± 2.48 mmol/L. Total cholesterol was 4.95mmol.l, triglyceride was 1.51mmol/l, LDL was 3.19 mmol/l, and HDL was 1.75 mmol/l. The mean BUN was 6.04 and creatinine was 91.76 mmol/l

Table 3. Clinical characteristic in new stroke patients admitted at Muhimbili National Hospital (N=160)

Variable	N=160 (%)
COMA (GCS 3-8)	71 (44.4%)
BloodPressure	
Systolic (Mean ±SD)	156.5±20.22
Diastolic (Mean ±SD)	88±13.16
RBG (Mean ±SD)	8±2.48
Total cholesterol (Mean ±SD)	4.95±0.98
Triglyceride (Mean ±SD)	1.51±0.71
LDL (Mean ±SD)	3.91±0.88
HDL (Mean ±SD)	1.75±0.27
Urea (Mean ±SD)	6.04±4.04
Creatinine (Mean ±SD)	91.76±47.6

Figure 2 shows the different ECG patterns found in patient with acute stroke. The majority were found to have left ventricular hypertrophy (83.1%), followed by ischemic changes (63.1%) and QT prolongation (50.6%). Other patterns which were found were tachycardia (35.6%), U-waves (35.6%), Arrhythmias (15%), and Q-waves (6.3%). It was also found that (31.9%) of study participants had strain pattern on their ECGs.

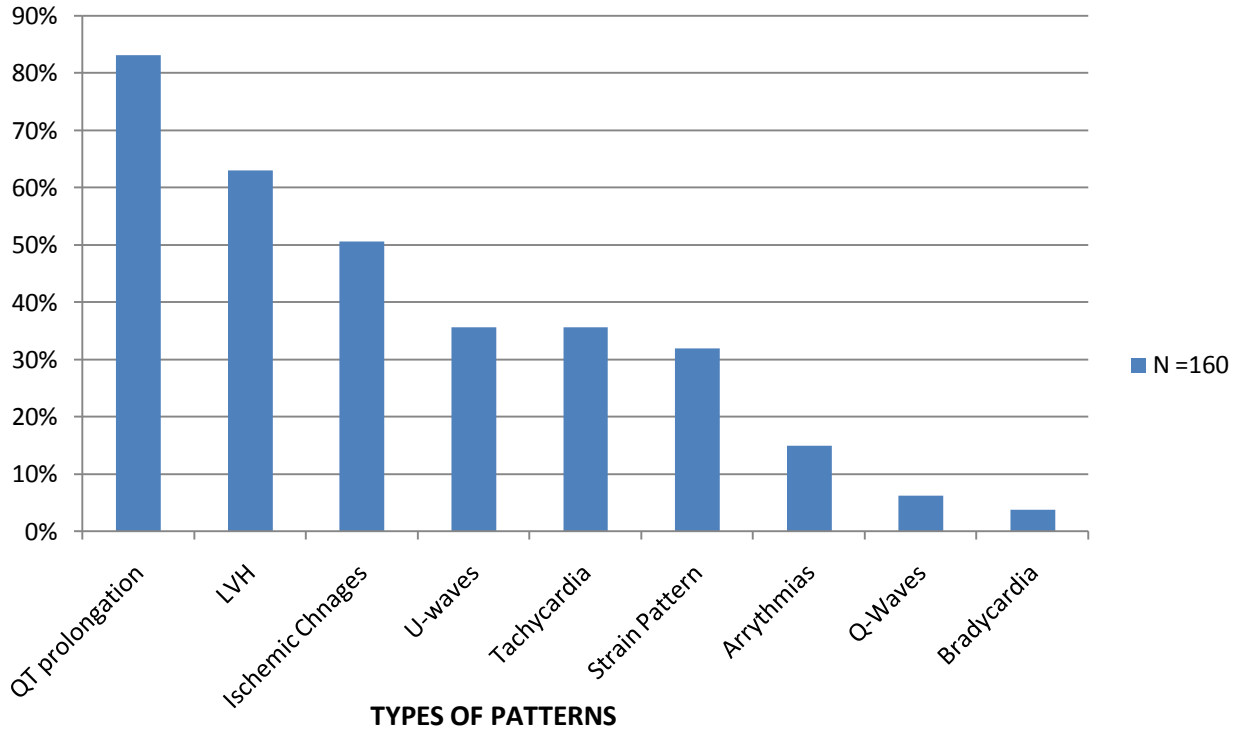


Figure 2 : ECG patterns in patients with new stroke at Muhimbili National Hospital (N=160)

Figure 3 is showing type of stroke and different ECG patterns. Out of 160 participants, 151 Ct scans films were available, 9 study participants were unable to get CT- scans. On univariate analysis, QT prolongation was seen more in patients with hemorrhagic stroke compared to ischemic stroke (87.2% vs. 12.3%) with statistically significant p <0.001. Similarly patients with hemorrhagic stroke exhibited Ischemic changes more than patients with ischemic stroke (70.5% vs. 57.5%) with statistically significant p=0.096. Other patterns were statistically insignificant.

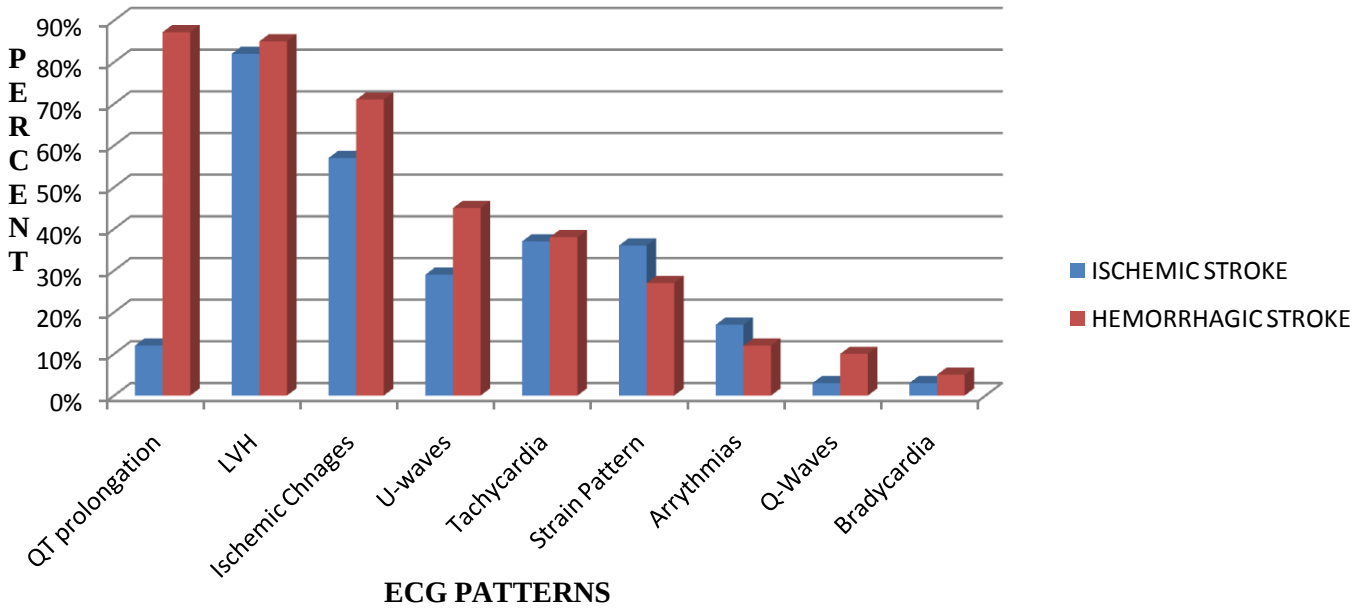


Figure 3: ECG patterns in patients and type of stroke at Muhimbili National Hospital (N=151)

Table 4 shows multivariate logistic analysis on ECG patterns and type of stroke. Ischemic changes, U-waves and Q-waves were not associated with type of stroke. Patients with QT-prolongation had 92.8 times likely hood to have hemorrhagic stroke then ischemic stroke (OR 92.8, CI 26.16-329.6), p-value of <0.001 which is highly statistically significant. However, the 95% CI is very wide which might be due to smaller sample size.

Table 4: Multivariate logistic regression analysis of ECG patterns with type of stroke in patients admitted at Muhimbili National Hospital (N=151)

VARIABLE	cOR (95% CI)	P-VALUE	aOR(95% CI)	P-VALUE
1) QT-Prolongation N=30				
Ischemic stroke	Reference		Reference	
Hemorrhagic stroke	48.435 (18.46-126.9)	< 0.001	92.8 (26.16-329.6)	<0.001
2) ISCHEMIC changes N=36				
Ischemic stroke	Reference		Reference	
Hemorrhagic stroke	1.866 (0.954-3.65)	0.068	1.237 (0.064-24.1)	0.888
3) **U-waves N=5**				
Ischemic stroke	Reference		Reference	
Hemorrhagic stroke	2.16 (1.092-4.262)	0.027	0.722 (0.212-2.815)	0.695
4) Q-waves N=6				
Ischemic stroke	Reference		Reference	
Hemorrhagic stroke	4.06(0.83- 19.78)	0.083	3.21 (0.26-40.196)	0.366

Table 5 shows one month mortality in patients with different ECG patterns. Univariate analysis shows that those patients with QT prolongation and ischemic changes as a pattern had highest mortality with p-values of < 0.001 and 0.039 respectively. Other patterns were statistically not significant.

Table 5. ECG Patterns and 1month mortality in patients admitted with stroke at Muhimbili National Hospital (N=152)

ECG PATTERNS	MORTALITY 1 MONTH N=152		P- VALUE
	DEAD (57)	ALIVE(95)	
QT PROLONGATION	44 (77.2)	34(35.8)	< 0.001
ISCHAEMIC CHANGES	43(75.4)	56(58.9)	0.039
TACHYCARDIA	25(43.9)	36(37.9)	0.468
BRADYCARDIA	3(5.3)	3(3.2)	0.519
U-WAVES	22(38.6)	34(35.8)	0.728
ARRYTHMIAS	10(17.5)	12(12.6)	0.405
Q-WAVES	5(8.8)	5(5.3)	0.398
LVH	50(87.7)	77(81.1)	0.168
STRAIN PATTERN	13(22.8)	33(34.7)	0.121

Table 6 shows multivariate logistic analysis of ECG patterns predicting mortality at 1 month. It shows that patients with QT prolongation had 3 times likelihood of dying of stroke in the 1st month of the diagnosis of new onset stroke, aOR (95%CI) = 3.122 (1.16-5.306); p-value of <0.001. It was also noted that patients above age of 65 years had 13.96 times likelihood of dying of stroke in the 1st month of the diagnosis of stroke, aOR (95%CI) = 13.96 (2.94-16.3), p< 0.001 .

Table 6: Multivariate Logistic regression analysis of ECG patterns predicting mortality at 1 month among patients with new onset stroke at MNH. (N=152)

Variable	cOR (95% CI)	P-VALUE	aOR(95% CI)	P-VALUE
1) QT-Prolongation				
Alive	Reference		Reference	
Dead	6.072 (2.876-12.823)	< 0.001	3.122(1.16--5.306)	< 0.001
2) Ischemic changes				
Alive	Reference		Reference	
Dead	2.139(1.032-4.43)	0.041	1.598(0.53-4.817)	0.405
3) U-waves				
Alive	Reference		Reference	
Dead	1.128(0.572-2.223)	0.728	0.579(0.180-1.149)	0.224

Continuation of table 6…….

4) Q-waves				
Alive	Reference	0.403	Reference	0.459
Dead	1.173(0.478-6.261)		1.937(0.357-11.17)	
5) LVH				
Alive	Reference	0.174	Reference	0.190
Dead	1.987(0.74-5.34)		2.392(0.65-8.82)	
6) HTN				
Alive	Reference		Reference	
Dead	1.339(0.511-3.513)	0.971	1.024(0.213-3.85)	0.971
7) DM				
Alive	Reference		Reference	
Dead	0.78(0.393-1.533)	0.465	0.974(0.358-2.65)	0.959
8) Smoking				
a) Ever				
Alive	Reference		Reference	
Dead	2.52(0.76-8.36)	0.131	1.285(0.078-21.04)	0.860

Continuation of table 6…….

b) Current				
Alive	Reference		Reference	
Dead	2.19(0.56-8.50)	0.26	1.380(0.055-34.6)	0.845
9) Alcohol				
a) Ever				
Alive	Reference		Reference	
Dead	1.732(0.88-3.40)	0.111	0.962(0.173-5.351)	0.965
b) Current				
Alive	Reference		Reference	
Dead	1.894(0.94-3.81)	0.073	2.22(0.39-12.8)	0.365
10) AGE				
≤18	Reference		Reference	
19-30	2.79(0.98-7.91)	0.055	2.63(0.76-9.10)	0.13
31-49	1.133(0.41-3.1)	0.806	1.80(0.44-7.40)	0.41
50-64	0.857(0.3-2.6)	0.774	1.39(0.34-5.33)	0.68
≥ 65	6.5(1.82-9.6)	0.004	13.96(2.94-16.3)	0.001

Univariate analysis in table 7 associates different ECG patterns with 3 months mortality. It shows that QT prolongation and ischemic changes associated with mortality at 3 months have statistically significant p-values of <0.001 and 0.01 respectively. Other patterns showed no statistical significance.

Table 7: ECG Patterns and 3 month mortality in patients admitted with new onset stroke at Muhimbili National Hospital N=133

ECG PATTERNS	MORTALITY 3 MONTH N=133		P- VALUE
	DEAD (79)	ALIVE(54)	
QT PROLONGATION	59 (74.7)	14(25.9)	<0.001
ISCHAEMIC CHANGES	60(75.9)	26(48.1)	0.01
TACHYCARDIA	32(40.5)	24(44.4)	0.651
BRADYCARDIA	5(6.9)	1(1.9)	0.222
U-WAVES	37(46.8)	15(27.8)	0.027
ARRYTHMIAS	12(15.2)	8 (14.8)	0.953
Q-WAVES	7(8.9)	2(3.7)	0.245
LVH	68(86.1)	42(77.8)	0.214
STRAIN PATTERN	18(22.8)	24(44.4)	0.008

On multivariate logistic analysis as shown in table 8, taking into account other ECG patterns and the confounders, we found that patients with QT prolongation had 17 times likelihood of having mortality at 3 months with aOR of 17.12 and a statistically significant p-value of <0.001, 95% CI (0.527-52.17). Patients with Ischemic changes had 3 times likelihood of mortality at 3 months with aOR of 3.22 and a statistically significant p-value of 0.047, 95% CI(1.014-10.602). This was after adjusting for the confounders. We also found that on those age 19-30 , cOR revealed that they have 2.8 times likely hood of getting a stroke . After adjusting we found that young age was protective against getting a stroke with aOR of 0.232.

Table 8: Multivariate logistic regression analysis of ECG patterns predicting mortality at 3 month among patients with new onset stroke at MNH.(N=133)

Variable	cOR (95% CI)	P-VALUE	aOR(95% CI)	P-VALUE
1) QT-Prolongation				
Alive	Reference		Reference	
Dead	8.07 (3.79-17.20)	<0.001	17.277(0.572-52.17)	<0.001
2) Ischemic changes				
Alive	Reference	0.003	Reference	0.047
Dead	3.010(1.47-6.2)		3.280(1.014-10.602)	
3) U-waves				
Alive	Reference		Reference	
Dead	2.103(1.042-4.247)	0.038	0.678(0.222-2.070)	0.495

Continuation of table 8…….

4) Q-waves				
Alive	Reference		Reference	
Dead	1.886(0.467-7.612)	0.37	1.228(0.163-9.237)	0.842
5) LVH				
Alive	Reference	0.371	Reference	0.217
Dead	1.505(0.74-5.34)		2.262(0.619-8.276)	
6) HTN				
Alive	Reference	0.854	Reference	0.050
Dead	1.090(0.437-2.717)		3.943(1.00-15.5)	
7) DM				
Alive	Reference	0.515	Reference	0.066
Dead	1.253(0.635-2.475)		2.819(0.935-8.94)	
8) Smoking				
a) Ever				
Alive	Reference		Reference	
Dead	1.390(0.388-4.980)	0.613	0.062(0.003-1.161)	0.063
b) Current				
Alive	Reference		Reference	

Continuation of table 8…….

Dead	1.311(0.301-5.571)	0.718	17.05(0.561-51.79)	0.103
9) Alcohol				
a) Ever				
Alive	Reference	0.370	Reference	0.561
Dead	1.371(0.688-2.730)		1.673(0.295-9.495)	
b) Current				
Alive	Reference		Reference	
Dead	1.635(0.94-3.81)	0.186	1.052(0.175-6.324)	0.956
10) AGE				
≤18	Reference		Reference	
19-30	2.865(1.001-8.199)	0.050	0.232(0.057-0.947)	0.042
31-49	1.173(0.387-3.550)	0.778	0.246(0.05-1.211)	0.085
50-64	0.821(0.252-2.670)	0.743	3.459(0.808-14.81)	0.094
≥ 65	3.263(1.089-9.776)	0.635	0.346(0.093-1.281)	0.112

Figure 4a shows the number of patterns and their outcome in terms of mortality at one month. It reveals that the higher the no of patterns the poorer the outcome in term of mortality. However, this table does not indicate which specific patterns were contributing to the poor outcome in terms of mortality.

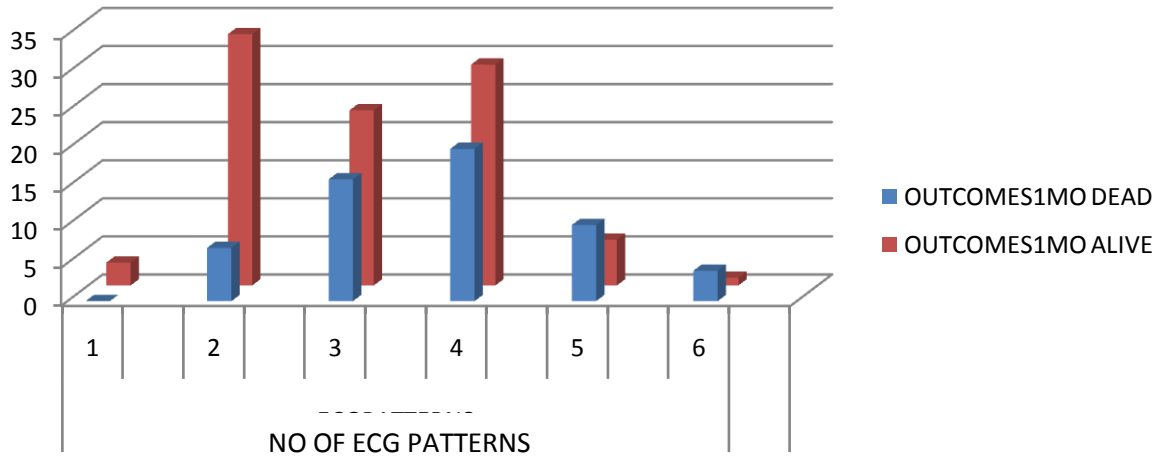


Figure 4a: Association between number of ECG patterns and mortality at 1month in patients with new onset stroke at Muhimbili National Hospital. (N=152)

As shown in Figure 4b the higher the combination of different numbers of patterns the poorer the outcome at three months. Those who had more than 3 patterns had poor outcomes at three months; however, again this figure doesn't represent the type of combination patterns being associated with increased mortality.

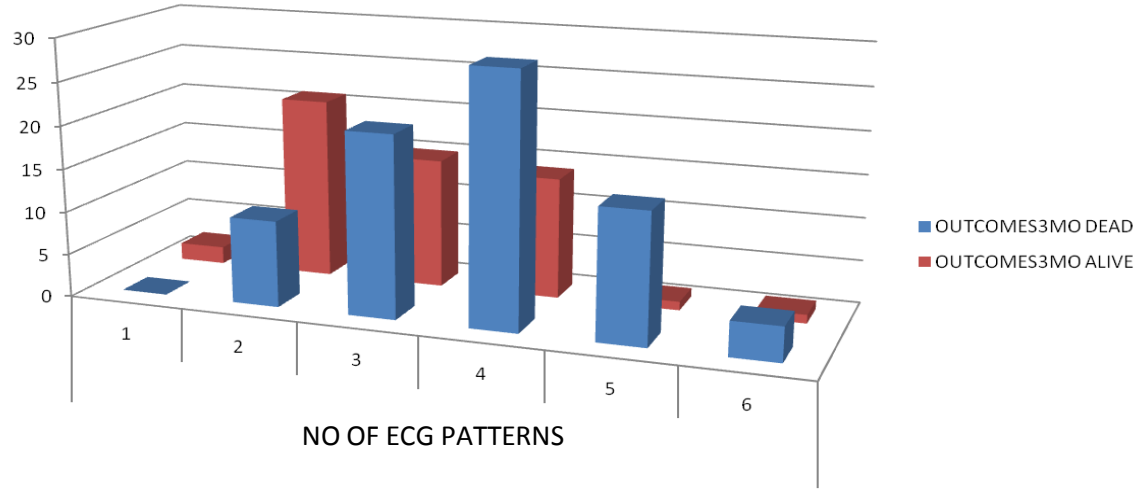
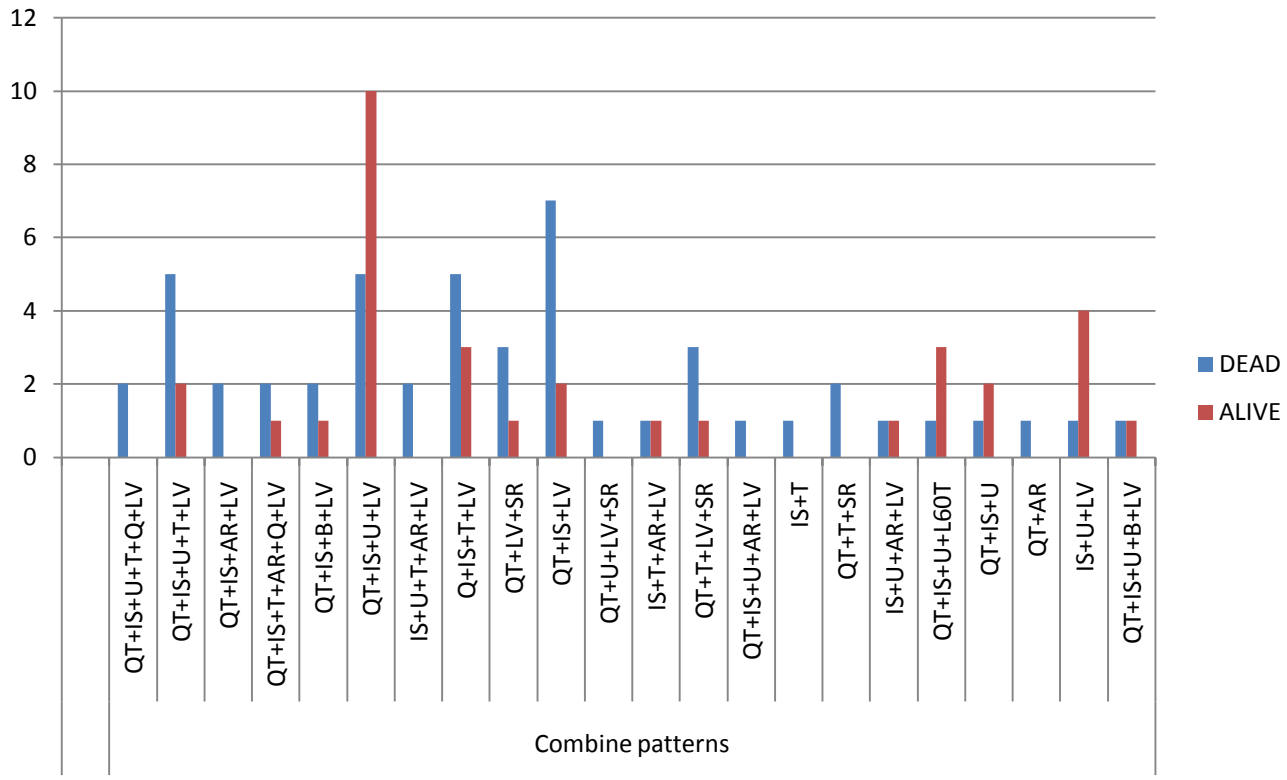


Figure 4b: Association between number of ECG patterns and mortality at 3 months in patients with new onset stroke admitted at Muhimbili National Hospital (N=133)

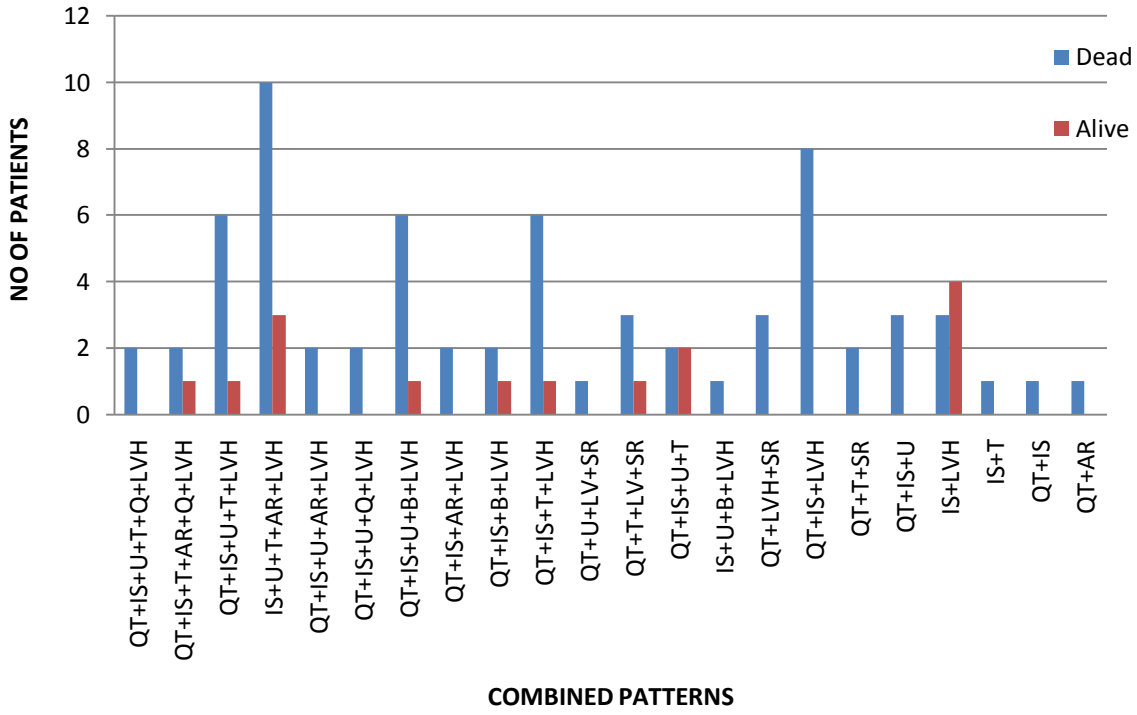
A Total of 47 different combinations of patterns were identified. Figure 5a shows combined ECG patterns and 1 month mortality, all those patterns with inclusive of QT prolongation and Ischemic changes were statistically significant in causing mortality with p value <0.001



KEY: **QT**=Corrected QT**, IS**=Ischemic changes, **AR**=Arrythmias**, Q**= Q-waves, **U**=U waves, **T**=Tachycardia, **B**=Bradycardia, **SR**=Strain pattern.

Figure 5a. Association of combined Patterns which cause most mortality at 1 month in patients with new onset stroke admitted at Muhimbili National Hospital N=152

Figure 5b also shows that the combination patterns which was associated with increased mortality at 3 months in patients with acute stroke were those inclusive of QT prolongation < 0.001



KEY: **QT**=Corrected QT**, IS**=Ischemic changes, **AR**=Arrythmias**, Q**= Q-waves, **U**=U waves, **T**=Tachycardia, **B**=Bradycardia, **SR**=Strain pattern.

Figure 5b: Association of combined Patterns which cause most mortality at 3 months in patient with new onset stroke admitted at Muhimbili National Hospital (N=133)

Table 9 shows mortality at one month and three months in relation to type of stroke. At one month we had 151 patients whose diagnosis was confirmed by CT scan. Of those who died, 57.5% had hemorrhagic stroke compared to 15.5% with ischemic stroke (p <0.001). Similarly at three months there was higher mortality in patients who had suffered hemorrhagic stroke (87.3%) compared to those with ischemic stroke (25.8%) with p<0.001. This signifies that patients with hemorrhagic stroke have poor outcomes in terms of mortality at one and three months compared to ischemic stroke.

Table 9. Mortality by type of stroke in patients admitted with stroke at Muhimbili National Hospital at one month (N=151) and three months (N=133)

TYPE OF STROKE	**OUTCOME ONE MONTH N=151 (%)**		**P- VALUE**
	DEAD	**ALIVE**	
ISCHEMIC	11(15.1)	62(84.9)	**<0.001**
HEMORRHAGIC	45(57.5)	33(42.3)	

TYPE OF STROKE	**OUTCOME 3 MONTH N= 133 (%)**		**P VALUE**
	DEAD	**ALIVE**	
ISCHEMIC	16 (25.8)	46(74.2)	**<0.001**
HEMORRHAGIC	62(87.3)	9(12.7)	

CHAPTER FIVE

5.1 DISCUSSION

Cardiac autonomic imbalance generated by acute cerebral insults plays an important role, not only in producing electrocardiographic abnormalities, but also in predisposing the patients towards early mortality.

1) ECG PATTERNS AND CO-MORBIDITIES

In this study it was found that patients with new onset of stroke had at least one ECG abnormalities at the time of ECG recording. A part from the ECG changes associated with acute stroke event, some of these ECG abnormalities could be due to underlying co-morbidities. Majority of the patients at baseline had underlying co-morbidity such as 87.5 % were hypertensive, 63% had diabetes mellitus. Around 44.4% of patients had poor Glasgow coma score on arrival they were in coma (GCS 3-8) and of these 58.3% were with hemorrhagic stroke compared to 35.6% with ischemic stroke.

In the majority of patients the hypertension was not controlled. The mean systolic blood pressure among the study patients was 156 mmhg and mean diastolic being 88 mmhg.

2) ECG PATTERNS AND MORTALITY

Among the 160 study patients , 47 different ECG patterns were identified. Individually the range of patterns noted in each study patient from study patients was from one single pattern to six patterns together. The study result shows that those who had more than three patterns had poorer outcomes within the next 3 months in terms of mortality. The results also indicate that those patients who had Qt prolongation and Ischemic changes as part of their ECG post stroke event had poorer outcomes at three months in terms of mortality. Thus not all ECG patter combination is associated with increased mortality at one and three months.

The most common ECG patterns found among the study population were LVH (83.1%), followed by Ischemic changes (63.1%) and QT prolongation (50.6%).

The findings of this study are consistent with some other studies done elsewhere, where Qt prolongation and Ischemic changes found in patients with new onset stroke were associated with poor outcomes in terms of mortality at 3 months.[13]

With regards to prevalence of QT prolongation in stroke patients : a study by Goldstein et al found that 45% had QT prolongation as compared to 50.6% noted in our study. The slight differences can be due to the difference in study design. Goldstein et al had done a retrospective study in which they compared two population groups through computer generated charts; while this study was a descriptive follow up of new onset stroke patients who had ECGs at admission and followed up for three months. In the same study, LVH was detected more frequently in sufferers of hemorrhagic stroke (69%) than cerebral infarction (15.5%). But in our study we found out that suffers from hemorrhagic stroke and ischemic stroke had almost equal but high prevalence of LVH on ECG (84.6% and 82.2% respectively). This difference can be attributed to underlying high prevalence (87.1%) of hypertension. We also found that 31.9% of our patients had strain pattern on ECG. Which had not been noted previous studies? This could be in association to severe LVH and endocardial wall ischemia due to uncontrolled hypertension. This could have been ascertained or excluded at echocardiography if this was performed on the study patients. However this pattern had no statistical significance on overall outcomes.

3) ECG PATTERNS AND STROKE

In this study the three month mortality in patients with ischemic stroke and ECG changes was 25.8%. in a study done by Ince B et al, [4] six months mortality in patients with ischemic stroke with ECG changes was 38.9%. The study duration is different by three months. However in the same study Ince et al found that ECG changes following acute event of stroke were associated with high mortality.[4] Similarly acute stroke patients had various ECG changes and overall mortality was similarly high.

In terms of outcomes at 1 month this study revealed that there was a statistical significance in mortality in patients who had more than 3 ECG patterns, especially if inclusive of Qt prolongation and ischemic changes as shown on univariate analysis. However, when multivariate analysis was done only Qt prolongation was found to be statistically significant on overall outcome with $p<0001$.

When this study looked at 3 months outcomes it was found that patients who had QT-prolongation and Ischemic changes , irrespective of underlying co-morbidities present, this patterns were found to predict poor prognosis in this group of patients.($p<0.001$ and $p=0.047$ respectively)

Of the patients with GCS of <8, 58.3% suffered hemorrhagic stroke while the rest suffered Ischemic stroke. This shows that patients with hemorrhagic stroke had poor GCS as well as outcomes in terms of mortality. There were no studies which had looked into patient's neurological status in terms of GCS.

4) STROKE AND AGE

This study shows that it is less likely for young patient to get a stroke, this was seen by after adjusting for all the confounders with the aOR of 0.232 and with statistical significant p-value of 0.0405. Those aged between 50-65 years were the most effected group to have suffered the acute event of stroke.

5.2 Conclusion.

- Bed side ECG was a significant tool in identifying certain ECG patterns associated with predicting poor outcomes.
- ECG patterns such as QT-prolongation and Ischemic changes were associated with poor outcomes at three months, irrespective of the co-existence of co-morbid conditions such as (HTN, DM).
- Although not very definite some ECG patterns such as QT-prolongation was highly predictive of type of stroke (hemorrhagic vs. ischemic stroke). But CT-scan still remains definitive tool.

5.3 Study Limitation

The study results cannot be extrapolated to the general population. This is to the fact that MNH is a tertiary referral hospital the majority of the cases would have had primary interventions before being treated at MNH.

There was also difficulty in follow up of the patients, some could not attend clinics on the proposed dates, some could not be available through phone calls, and some of the patients were not captured within the time frame which contributed to the loss of follow up in the study. 27 patients were lost to follow up during the study period and which might have given extra information if they would have been captured.

One of the limitations was also that we lacked ECGs from before onset of stroke, and there for we cannot conclude that observed abnormalities were due to acute event of stroke.

There are also medications which can cause QT prolongation, however this was difficult to ascertain in our settings as patients don't remember their medications or have records with them.

Majority of our patients had co-morbidities such as HTN which might be one of the confounding factors. Others factors include DM, AF and medications used which might prolong QT inte

5.4 Recommendation.

- Every stroke patients should have a bedside ECG which is easily available, and which will guide in overall management of patients and prognosis.
- A new study should be done to see different ECG patterns in patients with HIV disease who presents with acute event of stroke.

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APPENDIX I

QUESTIONAIRRE (ENGLISH)

DEMOGRAPHIC DATA AND BACKGROUND INFORMATION:

1. Serial number: _____
2. Name: _____
3. Telephone contact _____
4. Date of birth (dd/mm/yy): _____
5. AGE ☐
    1. 18-29
    2. 30-40
    3. 40-50
    4. 50-65
    5. > 65
6. Sex: 1. M ☐ 2. F ☐
7. Marital status

    1. Single ☐ 2. Married ☐ 3. Divorce ☐ 4. Co-habiting ☐
8. Relationship of caregiver to the patient
    1. ☐ Husband
    2. ☐ Wife
    3. ☐ Mother
    4. ☐ Father
    5. ☐ SON

6. ☐ Daughter

7. ☐ Other – specify _____

SOCIOECONOMIC ASSESSMENT:

9. Type of housing: what kind of roofing material does your house have?

1 .Grass/leaves/mud ☐ 2. Iron sheets ☐ 3. Tiles/Concrete/ ☐ Asbestos

10. Ownership of assets: does your household have: (tick those applicable)

1. Electricity ☐ 2. Television ☐ 3.Refrigerator ☐

11. Occupation

1.Unemployed ☐ 2.Casual labourer ☐ 3.Formal employment ☐ (unskilled work)

4.Formal employment (skilled job) ☐ 5.Business owner with ☐ employees

12. What is your usual mode of transport

1.On foot ☐ 2.Bicycle ☐ 3.Motorbil ☐ 4.Public ☐ transport

5.Private car ☐

13. Highest level of education completed

1.None ☐ 2.Primary ☐ 3. Secondary ☐

4. College / University ☐

14. Have you ever been diagnosed to have any of these conditions

1. Hypertension 1. Yes ☐ 2. NO ☐

2. Diabetes 1.YES ☐ 2. NO ☐

3. Hypercholesteremia 1. YES ☐ 2. NO ☐

4.Cardiac disorders

i. STROKE 1. YES ☐ 2. NO ☐

ii. EPILEPTIC SEIZURE 1. YES ☐ 2. NO ☐

5. Syphillis 1. ☐ 2. ☐

15. Have you /or do you still use any of the following

a. Smoking

i. YES ☐ 2. NO ☐ 3. If yes specify ……

b. Alcohol

i. YES ☐ 2. NO ☐ 3 .If yes specify ……..

c. Tobacco

i. YES ☐ 2. NO ☐ 3.If yes specify……

The following data will be filled by the principal investigator

1: Clinical Data of the patient

BP: _____ PR:_____

HR:_____ PD:_____

RR: _____ SpO2:_____ TEMPRETURE:_____

2: Signs and Symptoms

a) Level of Consciousness (GCS) ACTUAL SCORE ☐

1) Severe ≤ 8 ☐

2) Moderate 9 -12 ☐

3) Mild ≥ 13 ☐

a) Hemiplegia ; 1 YES ☐ 2. NO ☐

1.Right ☐ 2. Left ☐

b) Hemiparesis : 1 YES ☐ 2. NO ☐

1. Right ☐ 2. Left ☐

c) Quadriplegia: 1. YES ☐ 2. NO ☐

d) Tetraplegia 1. YES ☐ 2. NO ☐

e) Paraplegia : 1. YES ☐ 2. NO ☐

f) Headache 1. YES ☐ 2. NO ☐

g) Facial Deviation 1. YES ☐ 2. NO ☐

1. Right ☐ 2. Left ☐

h) Parasthesia 1. YES ☐ 2. NO ☐

3: ECG Patterns

a) QT prolongation 1) YES ☐ 2) NO ☐

a. Corrected QTc interval ……. (ECG FINDING)

b) ISCHAEMIC CHANGES 1) YES ☐ 2) NO ☐

c) U-WAVE 1) YES ☐ 2) NO ☐

d) TACHYCARDIA (HR > 90 b/min) 1) YES ☐ 2) NO ☐

e) BRADYCARDIA (HR < 60 b/min) 1) YES ☐ 2) NO ☐

f) ARRYTHMIAS 1) YES ☐ 2) NO ☐

a. SINUS ARRYTHMIAS ☐ 2) AF ☐ 3) PVC ☐

g) Q WAVES 1) YES ☐ 2) NO ☐

h) LVH 1) YES ☐ 2) NO ☐

4) CT SCAN/ MRI SCAN REPORT

1) ISCHAEMIC STROKE

_____

_____

2) HAEMORRHAGIC STROKE

_____

_____

5) OUTCOMES

1) AT 1 MONTH

1) DEAD 2) ALIVE

2) AT 3 MONTHS

1) DEAD 2) ALIVE

6)LIPID PROFILE : 1. Cholesterol …….. 2. Triglyceride …….. 3. LDL ……
4. HDL……

5. VLDL ……..

7) RENAL PROFILE: 1. Creatinine …… 2. UREA …….

8) RBG: …….

APPENDIX II

DODOSO (KISWAHILI)

IDADI YA WATU WA TAKWIMU NA TAARIFA ZA MSINGI:

1. Serial namba: _____
2. Jina: _____
3. Simu: _____
4. Tarehe ya kuzaliwa (dd/mm/yy): _____
5. Sex: mke ☐ mme ☐
6. Hali ya ndoa:_____
7. UMRI _____
8. Uhusiano wa mtoaji huduma kwa mgonjwa:

   Mume ☐

   Mke ☐

   Mama ☐

   Baba ☐

   Ndugu ☐

   Wengine - Mtaje _____

KIJAMII NA KIUCHUMI TATHMINI:

9. Aina ya makazi ya nyumba: Nyumba yako ina aina gani ya vifaa vya kuezekea?

   Nyasi / majani / tope ☐

   Chuma ☐

   Tiles / Saruji / Asbestos ☐

10. Umiliki wa mali: Je, nyumba yako ina: (jibu inayohusika

i. Umeme ☐

ii. Televisheni ☐

iii. Jokofu ☐

11. Kazi

i. Hajaajiriwa ☐

ii. Mfanyakazi kawaida ☐

iii. Ajira ya rasmi (wahudumu kazi) ☐

iv. Ajira ya rasmi (wenye kazi ujuzi) ☐

v. Biashara mmiliki na wafanyakazi ☐

12. Njia ya kawaida ya usafiri

i. Kwa mguu

ii. Baiskeli ☐

iii. Pikipiki ☐

iv. Usafiri wa umma ☐

v. Gari binafsi ☐

13. Kiwango cha juu cha elimu kukamilika

i. Hajapata elimu ☐

ii. Shule ya Msing ☐

iii. Sekondari ☐

iv. Chuo / chuo kikuu ☐

14. Je, umeshawahi kutambuliwa na magonjwa mojawapo

a. Shinikizo la damu

b. Kisukari

c. Ugonjwa ya mafuta (high cholesterol)

d. Ugonjwa wa Moyo

i. AF ☐

ii. DCM ☐

iii. Kiharusi

iv. Kifafa

e. Kaswende ☐

15. Je, umeshawahi kutumia mmojawapo yafuatayo:

a. Sigara 1) Ndiyo 2) Siyo

i. kama ndiyo taja wakati wa matumizi…….

b. Pombe 1) Ndiyo 2) Siyo

i. kama ndiyo taja wakati wa matumizi…….

c. Tumbaku 1) Ndiyo 2) Siyo

i. kama ndiyo taja Wakati wa matumizi……

Data zifuatazo kujazwa na mpelelezi mkuu

1: Vipimo vya mgonjwa

BP: _____ PR:_____ HR:_____

PD:_____RR: _____

SpO2:_____

TEMPERATURE:_____

2: Dalili za mgonjwa

a) Level of Consciousness (GCS)

Severe ≤ 8 ☐

Moderate 9 -12 ☐

Mild ≥ 13 ☐

a) Hemiplegia Right ☐ Left ☐

b) Hemiparesis Right ☐ Left ☐

c) Quadriplegia ☐

d) Paraplegia ☐

e) Headache ☐

f) Facial Deviation ☐ Right ☐ Left

g) Parasthesia ☐

3: ECG Patterns

a. QT prolongation ☐

b. ISCHAEMIC CHANGES ☐

c. U-WAVES ☐

d. TACHYCARDIA ☐ HR > 90 b/min

e. BRADYCARDIA ☐ HR< 60 b/min

f. ARRYTHMIAS ☐

i. SINUS ARRYTHMIA ☐

ii. AF ☐

iii. PVC ☐

g. Q WAVES ☐

h. LVH ☐

4) Reporti ya scan ya Kichwa

1) ISCHAEMIC STROKE

_____

_____

2) HAEMORRHAGIC STROKE

_____

_____

5) OUTCOMES

1) AT 1 MONTH

1) DEAD 2) ALIVE2) AT 3 MONTHS

1) DEAD 2) ALIVE

6) LIPID PROFILE : 1. Cholesterol …….. 2. Triglyceride …….. 3. LDL …… 4. HDL…… 5. VLDL ……..

7) RENAL PROFILE: 1. Creatinine …… 2. UREA…….

8) RBG: ……

APPENDIX III

CONSENT FORM: ENGLISH

ECG PATTERNS AND THREE MONTHS OUTCOMES IN PATIENTS ADMITTED WITH STROKE AT MUHIMBILI NATIONAL HOSPITAL.

My name is Dr. Khuzeima Khanbhai 2nd year resident in the department of Internal Medicine at Muhimbili University of Health and Allied Sciences (MUHAS).

I am conducting a study with the above title as part of my study program.

Aims of the study:

The aim is to identify different patterns of cardiac abnormalities in patients who come with stroke , also this study will look at the outcomes of this patients in terms of morbidity and mortality within 3 months.

Participation in this study:

A diagnosis of of stroke will be made on clinical grounds and presenting history and clinical features, patients will be requested in the enrolment into the study. Patient him/herself or the next of kin if the patient has altered mental status/coma can give a written consent as to be enrolled into the study. The study involves performing ECG of the patient on admission and this will be interpreted and if immediate management is needed will be provided to the patient .

In addition, during this study other basic investigations will be conducted routinely as per patients clinical condition and requirements

If you choose not to participate in this study, you/ your patient will continue to receive the normal care at the wards and will not be compromised in any way.

Risks:

This study poses no risk to any participant in view that ECG is a non invasive procedure. However a minor risk is there during the veno-pucture for the basic blood investigations which were done , but they are negligible to cause any potential life threatening events.

Benefits:

If you agree to participate in this study, you or your patient will benefit in the treatment of underlying any cardiac condition which might be unidentified/undiagnosed previously .The patient will not incur in any cost of medical service for investigation performed . The results from this study will be used to see the different patterns and also so establish the risk score.

Confidentiality:

All data collected will be treated with strict confidence and stored in locked cabinets and on encrypted computers and will not be revealed to anybody outside the research team.

Cost:

You will not be required to make any payments to participate in this study and no payment will be made to you.

For further information, questions or queries, you can contact:

1. The Principal Investigator,

   Dr. Khuzeima Khanbhai

   Department of Internal Medicine,

   MUHAS

   P. O. Box 65001,

   Dar es Salaam.

   Tel: +255 713 460653

   Email: drkhuz@gmail.com

2. Dr Johnson Lwakatare

   Consultant Cardiologist and Senior Lecturer

   Department of Internal Medicine,

   MUHAS

   P. O. Box 65001,

   Dar es Salaam.

   Tel: +255 715 262648

   Email: medi@raha.com

3. Prof. M.M.Aboud

   Director of Research and Publications

   P.O. Box 65001

   Tel: 022-2150302-6

   MUHAS

I, _____, have read/been told of the contents of this form and have understood its meaning. I agree to participate in this study.

OR

I,_____, husband/wife/father/mother/_____ have read/been told of the contents of this form and have understood its meaning. I agree to enroll _____ (patient's name in full) in this study

Signature of patient/Next of kin_____

Signature of Researcher _____

Date _____

APPENDIX IV

CONSENT FORM: KISWAHILI
FOMU YA RIDHAA YA KUSHIRIKI KATIKA UTAFITI

Jina langu ni Dr.Khuzeima khanbhai, mwanafunzi wa udaktari bingwa (Internal Medicine) katika chuo kikuu cha afya na sayansi shirikishi Muhimbili (MUHAS). Ninafanya utafiti kuangalia na kutambua ya mifumo ya ECG ya wagonjwa na Stroke, kwa mgonjwa anayekuja na atayelazwa wodini Mwaisela katika hospitali ya Taifa, Muhimbili, Dar es Salaam.

Madhumuni ya utafiti:

Utafiti huu kuona ruwaza kwa tofauti ya ECG katika wagonjwa ambao endelevu kiharusi (stroke), na hii pia kuona matokeo ya wagonjwa hao ndani ya miezi mitatu ijayo. utafiti huu itatoa taarifa juu ya jinsi kiharusi disrupts uhuru wa moyo wakati wa matukio.

Ushiriki katika utafiti:

Uchunguzi wa wa kiharusi hufanywa kwa misingi ya kliniki na kuwasilisha makala historia na kliniki ya wagonjwa wataombwa katika uandikishaji wa wanafunzi katika masomo. Mgonjwa huyo / mwenyewe au ya pili ya jamaa kama mgonjwa wa akili hali ilibadilika / kukosa fahamu inaweza kutoa kibali cha maandishi kama kujiandikisha kwenye somo. Utafiti inahusisha kufanya ECG /EKGS /ya mgonjwa juu ya uandikishaji na hii itakuwa kufasiriwa na kama usimamizi wa haraka inahitajika zitatolewa kwa mgonjwa. Aidha, wakati wa utafiti huu mengine ya msingi ya uchunguzi itafanyika mara kwa mara kama sharti kwa kila wagonjwa kliniki na mahitaji ya
Kama kuchagua si kushiriki katika utafiti huu, wewe / mgonjwa yako itaendelea kupata huduma ya kawaida katika wodi ya mwaisela na wala kuathirika kwa njia yoyote.

Hatari:

Utafiti huu unaleta hakuna hatari kwa mshiriki yeyote katika mtazamo kwamba ECG / EKG ni zisizo vamizi utaratibu.

Faida za utafiti:

Kama unakubali kushiriki katika utafiti huu, wewe au mgonjwa wako watafaidika katika matibabu ya msingi na hali ya ugonjwa wa moyo ambayo inaweza kuwa wasiojulikana / undiagnosed awali mgonjwa si incur katika gharama yoyote ya huduma ya matibabu kwa ajili ya uchunguzi akifanya.. Matokeo ya utafiti huu utatumika ili kuona aina tofauti tofauti na pia ili kuanzisha alama ya hatari.

Usiri:

Taarifa zote zitakazokusanywa katika utafiti huu zitakuwa siri, hivyo ushiriki wako hautajulikana na mtu. Taarifa hizi zitajulikana kwenye timu ya watafiti tu.

Malipo:

Kwa kushiriki kwenye utafiti huu, hautalipwa wala hautalipa chochote.

Ukiwa na swali au tatizo lolote, unaweza kuwasiliana na wafuatao:

1. Mtafiti Mkuu,

   Dr.Khuzeima Khanbhai,

   Idara ya Internal Medicine

   MUHAS

   S.L.P 65001,

   Dar es Salaam.

   Simu: +255 713 460653

   Barua pepe: drkhuz@gmail.com

2. Dr Johnson Lwakatare

   Consultant Cardiologist and Senior Lecturer

   Department of Internal Medicine,

   MUHAS

   P. O. Box 65001,

   Dar es Salaam.

   Tel: +255 715 262648

   Email: medi@raha.com

Mimi, _____, nimesoma/nimesomewa maelezo yote yaliyomo kwenye fomu hii na nimeelewa.

AU

Mimi, _____, mume/mke/baba/mama/_____ nimesoma/nimesomewa maelezo yote yaliyomo kwenye fomu hii na nimeelewa. Nakubali mgonjwa wangu ashiriki katika utafiti huu.

Sahihi ya mzazi/mbadala _____

Sahihi ya Mtafiti _____

Tarehe _____

SUMMARY OF MEDICATIONS WHICH CAN CAUSE QT PROLONGATION.

Most Potent Group

1) Amiadarone
2) Azythromycin
3) Chloroquine
4) Clarithromycin
5) Domeperodine
6) Erythromycin
7) Flecanide
8) Ondesterone
9) Procanamide
10) Sotalol

POSSIBLE CAUSING QT PRONLONGATION

1) Atanzanavir
2) Fosphenytoin
3) Levefloxacin
4) Nicardipine
5) Ofloxacin
6) Roxithromycin
7) Tamoxifen

CONDITIONAL DRUGS CAUSING QT PROLONGATION

1) Amitriptilline	4) Fluoxetin	7) Ketoconazole	10) Ritonavir
2) Ciprofloxacin	5) Imipramine	8) Quinine Sulphate	
3) Fluconazole	6) Itraconazole	9) Septrin	