IMPACT OF A SCREENING PROTOCOL ON DOOR TO ECG TIME IN THE ACCIDENT AND EMERGENCY DEPARTMENT OF KENYATTA NATIONAL HOSPITAL

DR. JULIA MBITHE NDAWA
MMED (Internal Medicine)
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A DISSERTATION WRITTEN IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTERS OF MEDICINE IN INTERNAL MEDICINE, UNIVERSITY OF NAIROBI
DECLARATION

I, Dr. J.M. NDAWA, declare that this is my original work and that to the best of my knowledge it has not been presented before for a degree at this or any other university.

Signed ........................... Date .................................
SUPERVISORS’ DECLARATION

This project proposal is submitted with our approval

1. Prof Mark Joshi
   Consultant Physician and Cardiologist,
   Associate Professor of Medicine,
   Department of Clinical Medicine and Therapeutics,
   University of Nairobi

   Signed ………………………..   Date ……………………

2. Dr. Loice Achieng
   Consultant Physician,
   Lecturer,
   Department of Clinical Medicine and Therapeutics,
   University of Nairobi

   Signed ………………………..   Date ……………………

3. Dr Benard Gitura
   Consultant Physician and Cardiologist,
   Kenyatta National Hospital
   Accidents and Emergency Department

   Signed ………………………..   Date ……………………
ACKNOWLEDGEMENTS

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I want to appreciate my three supervisors for their mentorship, commitment, support and advice as we worked through the development of this study protocol.

To all my colleagues, thank you for the moral support.

I want to express my heartfelt appreciations to my entire family and especially my husband Joseph Mwanga who has been my most ardent motivator. I wouldn’t have done this without him.
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<table>
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<th>Description</th>
</tr>
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<tbody>
<tr>
<td>ACS</td>
<td>Acute coronary syndrome</td>
</tr>
<tr>
<td>ACC</td>
<td>American College of cardiology</td>
</tr>
<tr>
<td>A&amp;E</td>
<td>Accident and Emergency</td>
</tr>
<tr>
<td>AHA</td>
<td>American Heart Association</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired Immunodeficiency Syndrome</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>ESC</td>
<td>European Society of Cardiologists</td>
</tr>
<tr>
<td>HIV</td>
<td>Human Immunodeficiency Virus</td>
</tr>
<tr>
<td>HDL</td>
<td>High Density Lipoprotein</td>
</tr>
<tr>
<td>KNH</td>
<td>Kenyatta National Hospital</td>
</tr>
<tr>
<td>LBBB</td>
<td>Left Bundle Branch Block</td>
</tr>
<tr>
<td>LDL</td>
<td>Low density Lipoprotein</td>
</tr>
<tr>
<td>SSA</td>
<td>Sub Saharan Africa</td>
</tr>
<tr>
<td>TG</td>
<td>Triglycerides</td>
</tr>
<tr>
<td>TEWS</td>
<td>Triage Early Warning Sign score</td>
</tr>
<tr>
<td>VLDL</td>
<td>Very Low Density Lipoprotein</td>
</tr>
</tbody>
</table>
ABSTRACT

Cardiovascular disease constitutes a major increasing disease burden in the developing world. Kenyatta National Hospital does not have a specific chest pain protocol despite Acute Coronary Syndrome (ACS) being a true medical emergency requiring prompt diagnosis and immediate management. The American College of cardiology/American Heart Association (ACC/AHA) recommends that patients presenting with suspected ACS should have an Electrocardiogram (ECG) within 10 minutes of first medical contact. This has a direct impact on the subsequent diagnosis and definitive management.

Objectives
To determine the current triage practice, implement a screening protocol and evaluate its impact on door to ECG time of patients with suspected Acute Coronary Syndrome presenting at the Accident and Emergency (A&E) Department in Kenyatta National Hospital (KNH).

Specific objectives
1. To determine the proportion of patients at high risk for acute coronary syndrome who obtain an ECG at the Accidents and Emergency department of KNH.
2. To determine the door to ECG time of patients at high risk for ACS at the A&E department.
3. To evaluate the impact of a screening protocol implemented at the triage station on the proportion and on the Door to ECG time of patients at high risk for ACS at the A&E department.

Methods
This was a quasi-experimental study in which assessment of door to ECG time before and after implementation of a triage screening protocol. The intervention involved provision of a simple ECG prioritization tool and free ECG service administered to non-surgical patients aged >30 years prior to doctors’ consultation.

Analysis
Door to ECG time was dichotomized to early <10 minutes verses delayed >10 minutes and presented as percentages with 95% confidence interval. Univariate analysis was done to compare the proportions of patients receiving ECG and of those receiving an early or delayed ECG before and after intervention using the McNemar test. Comparisons of the mean time to ECG before and after the intervention was done using Wilcoxon signed rank sum test. A p value of <0.05 was considered significant.

Results
It was found that 14.4% (95% CI 10.06 – 20.09) of patients with a high probability of Acute coronary syndrome had an ECG done with a mean door to ECG time of 10.2hrs (SD 7.18) median of 8.08hrs (IQR 4.65-13.1). None obtained an ECG within the recommended 10 minutes. Post intervention, 93.6% of patients had an ECG done with a mean door to ECG time of 2.5hrs (SD4.02), Median 69minutes IQR (20-163). 9.4% of patients obtained the ECG within 10 minutes and patients were 86.7 times more likely to receive an ECG post intervention p < 0.05.

Conclusion
The current screening and diagnosis of suspected ACS is sub-optimal. The institution of an ECG triage tool and free ECG service can significantly bridge this gap.
INTRODUCTION

Definitions

Acute coronary syndrome (ACS) is a composite term used to describe several clinical presentations that are a manifestation of underlying coronary artery disease. It includes ST segment elevation myocardial infarction, Non-ST elevation myocardial infarction, and Unstable Angina. It results from rupture of an atherosclerotic plaque within the cardiac arterial tree with secondary thrombosis of the affected vessel.

According to the 2015 ESC guidelines for the management of acute coronary syndromes, to make a diagnosis of acute myocardial infarction there needs to be an increase and or decrease of a cardiac biomarker with troponin being the gold standard biomarker combined with at least one other of the following:

1. Symptoms of ischaemia.
2. New or presumed new significant ST-T wave changes or left bundle branch block on a 12-lead ECG.
3. Development of pathological Q waves on ECG.
4. Imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality.
5. Intracoronary thrombus detected on angiography or autopsy (1)

2 Literature Review

2.1 Epidemiology

Cardiovascular disease is the leading cause of morbidity and mortality worldwide. Of all cardiovascular disease, ischaemic heart disease and cerebrovascular accidents are responsible for approximately 25% of all deaths worldwide. (2)

Historically cardiovascular disease was found to be quite rare in African black populations. As the developing world grapples with the unfinished agenda of infectious diseases, there is now an emergin epidemic of non-communicable diseases of which cardiovascular disease is the largest contributor. This is largely due to rapid urbanization with associated increased exposure to risk factors coupled with poor or absent primary prevention strategies. According to the global atlas on cardiovascular disease 2011, 80% of all deaths from cardiovascular disease occurs in low and middle income countries as well as 42% of premature deaths (3) In addition almost 90% of related morbidity and disability also occurs in the same setting worldwide,(4) Stroke is disproportionately high in Africa and the Caribbean but Ischaemic Heart disease is also rapidly increasing in incidence (5) There is also the impact of infectious diseases and their treatments modifying the cardiovascular risk factors on African populations. This is especially of note in HIV/AIDS which Africa has the lions share (6)

In Kenya, non-communicable disease accounted for 27% of all mortality with cardiovascular disease being responsible for 8% of all mortality (7)
FIGURE 1: World map of the distribution of CVD mortality rates in males (age standardized, per 100,000)

World map of the distribution of CVD mortality rates in females (age standardized, per 100,000)
2.1.1 Epidemiology of Ischaemic Heart disease

IHD is the number one cause of mortality in the developed world. In 2008, it was responsible for 12.7% of all deaths globally (8). The prevalence of coronary artery disease in the United states is 6.2% (9) in the overall population. According to the AHA/CDC Heart disease and stroke statistics updates 2017, USA has an annual incidence of 580,000 new and 210,000 recurrent myocardial infarctions and it was responsible for 1 in every 7 deaths in the year 2014 (10).

There is scarce accurate and objective data on prevalence, incidence and mortality from ischaemic heart disease in Africa as a whole. Most of the available data is from hospital based studies. One met-analysis found the prevalence ranging from 0.1% in a study in Senegal amongst patients admitted to a medical ward up to 10.4% amongst Sudanese diabetic patients who died in a hospital set up (11).

<table>
<thead>
<tr>
<th>TABLE 1: Prevalence of MI among study populations of African studies.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author/Year</td>
</tr>
<tr>
<td>------------</td>
</tr>
<tr>
<td>Kolo et al 2013</td>
</tr>
<tr>
<td>Shavadia et al 2012</td>
</tr>
<tr>
<td>Nguchu et al 2009</td>
</tr>
<tr>
<td>Seck et al 2007</td>
</tr>
<tr>
<td>Sani et al 2006</td>
</tr>
<tr>
<td>Ahmed et al</td>
</tr>
<tr>
<td>Joubert et al 2000</td>
</tr>
</tbody>
</table>
In a Tanzania multicenter population based study in 2013, there was documented higher prevalence of myocardial infarction among stroke patients than in age and sex matched controls from the population (12). The prevalence of coronary artery disease in Egypt is 8.3%(13).

According to WHO 2008, death from ischaemic heart disease in Kenya was 129/100000(8). Shavadia et al in 2012 documented ACS to account for 5.1% of all intensive care admissions in a prospective study found that 30% of patients with diabetes presenting to the casualty department in Kenyatta National Hospital had acute coronary syndrome based on Ischaemic changes on ECG and elevated troponin levels in 2009(15).

2.2 Shifting patterns and Acute Coronary Syndromes in Africa and Kenya

The epidemiologic pattern of acute coronary syndromes in Africa and particularly in black Africans mirrors that of Western industrialized countries more than five decades ago. The mean age of presentation of initial Acute myocardial infarction is 54.3 years, almost 4 years earlier than in the rest of the world according to the INTERHEART Africa study (16). This is in contrast to the average age of first presentation in the US which is 65 years in males and 71 years in females (9). The diagnosis is made more in black African individuals with tertiary education and higher incomes than Europeans (16). This has been attributed to the adoption of western lifestyles especially for Africans of higher income brackets.

Despite the seemingly low prevalence rates of ACS, there is a clear rising incidence of cardiovascular disease and this is explained in part by the explosion of multiple risk factors in African and specifically in our Kenyan population. In the INTERHEART-Africa study (16), it was revealed that the risk factors driving ischaemic heart disease in Africa are similar to those in western industrialized populations.

Hypertension is the single most prevalent and biggest contributor of cardiovascular disease in Africa. (6) There are twice as many Africans living with hypertension than there are people living with HIV/AIDS globally. Prevalence ranges from 15% to as high as 70%.(17). Unfortunately only less than 30% of African hypertensive patients are aware of their diagnosis, and just 18% are on treatment with only 7% having controlled disease (17) In Kenya, the WHO Non Communicable Disease report of 2014 documented a prevalence of Hypertension at 28% (7). Barely 20% of hypertensive patients in Kenya know their status and a mere 25% of these are on optimal medication(18–20)

Obesity and Physical inactivity are also on the rise. Overall prevalence of obesity in Kenya 4.2% (7). In a study of both private and private schools in Nairobi Kyallo F et al found 19% of children aged 9-14 years are either overweight or obese(21) In adults, obesity is more prevalent among women at almost 60% of females either obese or overweight according to Wanjiru et al
who studied cardiovascular risk factors in a Nairobi slum in 2013 (18). Level of physical activity are as low as 13% in some urban settings with an overall grade of C in the 2014 Report Card on the Physical Activity and Body Weight of Children and Youth (22,23).

Hyperlipidaemia especially a combination of increased very low density lipoproteins (VLDL), LDL with associated decreased HDL promotes atherogenesis. (24) Low HDL level is the most commonly found lipid abnormality found among Kenyan subjects. (25)

Diabetes Mellitus is an independent risk factor for cardiovascular disease and it modifies the presentation of ACS with silent disease occurring despite multivessel involvement. (26) In Kenya, the prevalence of Diabetes ranges from 2.2% to 12.4% in rural to urban populations respectively. (27)

The combination of three or more of the above mentioned risk factors in one individual constitutes the metabolic syndrome. The additive and multiplicative effects of several risk factors place such individuals at very high risk for multiple CVD and their complications. In a cross sectional household study in Nairobi in 2008, the prevalence of metabolic syndrome was found to be 34.6%, was much more frequent among females at 42% versus 29% and increased with socioeconomic status especially for men. (25)

Smoking prevalence in Kenya is 13% (7) It is more prevalent in men especially in western rural Kenya where as many as three times more men than women smoke (28). It is the commonest risk factor for ACS in an Egyptian cohort of patients (29), but was found at only 8.4% in a cohort of ACS patients in KNH (15).

Africa has the lion’s share of the HIV/AIDS pandemic and as the disease transforms to a chronic illness the cardiovascular effects of the disease are being seen. The various mechanisms responsible for this include immune activation with a chronic pro-inflammatory effect on the endothelium, a hypercoagulable state, direct toxicity of HIV antigens especially of GP-120 and Tat protein on the vessels, the impact of HAART especially Protease Inhibitors on glucose and lipid metabolism and accelerated aging among others.(30). The success of the treatment programs translate to longer life span and older age is associated with traditional risk factors such as hypertension and diabetes which tend to peak approximately a decade after the peak age of HIV infection in Africa. (6) The presentation of ischaemic heart disease in HIV infected populations is at much younger ages with a mean age of 48years. (30)
2.3 Diagnostic and management approach to patient with Suspected Acute Coronary Syndrome.

Definitive diagnosis of an ACS is normally made after reviewing a composite of clinical features, ECG abnormalities and cardiac biomarkers.

Other more advanced diagnostic tools in the diagnosis of acute coronary syndrome include Echocardiography, Cardiac CT/MRI, perfusion scans and Coronary angiograms. In the emergency departments, rapid and accurate diagnosis ensures timely treatment leading to better outcomes; according to the saying that time is muscle.

2.3.1 Clinical Features

The classic presentation of an acute coronary syndrome involves an at risk patient presenting with chest pain (1). The typical patient will have the aforementioned risk factors such as being middle to older age, male sex, obesity, with a hypertension and smoking history among others. They complain of chest pain which had distinct features described as constricting, heaviness or squeezing in the chest of gradual onset radiating to the jaw, neck, shoulder, upper limbs or epigastric area. It is worsened by exertion and may be relieved by rest.

The chest pain is usually associated with other symptoms such as dyspnoea, sweating, dizziness or lightheadedness and anxiety with sense of impending doom. At times patients present with complications such as arrhythmias, cardiogenic shock and cardiac arrest (31).
Despite the above mentioned features, history and its characteristics is inadequate to make a definitive diagnosis and has to be accompanied by other diagnostic tools to confirm a diagnosis of an ACS. This is because not all chest pain is of cardiac origin and important differential diagnoses to consider include Pericardial disease, vascular causes like aortic dissection, pulmonary embolism, pulmonary origin like pleuritis and or pneumonia, spontaneous pneumothorax, tracheobronchitis, GIT causes like esophageal reflux, peptic ulcers and pancreatitis. Studies have shown that the history is more valuable a tool for its negative predictive value for ACS especially pain described as sharp or stabbing with specific finger point location. However, patients with non-specific chest pain who have IHD tend to have worse outcomes. (31)

The other challenge in the evaluation of chest pain for ACS is up to a third of patients present in an atypical manner and do not have chest pain(32). These tend to be high risk patients including the elderly, women and diabetics. This means that they also tend to receive less evidence based therapy and end up with higher morbidity and mortality (33). These atypical presentations include dyspnea, nausea and/or vomiting, epigastric pain, palpitations, weakness, syncope, confusion or cardiac arrest. (34). Although women tend to experience more throat and jaw discomfort, studies have shown similar rates of chest pain and no statistically different association of symptoms and ischaemic heart disease between the sexes (35,36)

<table>
<thead>
<tr>
<th>Pain Descriptor</th>
<th>Reference</th>
<th>No. of Patients</th>
<th>Positive Likelihood Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased likelihood of AMI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radiation to right arm or shoulder</td>
<td>29</td>
<td>770</td>
<td>4.7 (1.9-12)</td>
</tr>
<tr>
<td>Radiation to both arms or shoulders</td>
<td>14</td>
<td>803</td>
<td>4.1 (2.5-6.5)</td>
</tr>
<tr>
<td>Associated with exertion</td>
<td>14</td>
<td>893</td>
<td>2.4 (1.5-3.8)</td>
</tr>
<tr>
<td>Radiation to left arm</td>
<td>24</td>
<td>278</td>
<td>2.3 (1.7-3.1)</td>
</tr>
<tr>
<td>Associated with diaphoresis</td>
<td>24</td>
<td>8426</td>
<td>2.0 (1.9-2.2)</td>
</tr>
<tr>
<td>Associated with nausea or vomiting</td>
<td>24</td>
<td>970</td>
<td>1.9 (1.7-2.3)</td>
</tr>
<tr>
<td>Worse than previous angina or similar to previous MI</td>
<td>29</td>
<td>7734</td>
<td>1.8 (1.6-2.0)</td>
</tr>
<tr>
<td><strong>Described as pressure</strong></td>
<td>20</td>
<td>11504</td>
<td>1.3 (1.2-1.5)</td>
</tr>
<tr>
<td><strong>Decreased likelihood of AMI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Described as pleuritic</td>
<td>29</td>
<td>8822</td>
<td>0.2 (0.1-0.3)</td>
</tr>
<tr>
<td>Described as positional</td>
<td>29</td>
<td>8330</td>
<td>0.3 (0.2-0.5)</td>
</tr>
<tr>
<td>Described as sharp</td>
<td>20</td>
<td>1088</td>
<td>0.3 (0.2-0.5)</td>
</tr>
<tr>
<td>Reproducible with palpation</td>
<td>29</td>
<td>8822</td>
<td>0.3 (0.2-0.4)</td>
</tr>
<tr>
<td>Inframmary location</td>
<td>31</td>
<td>903</td>
<td>0.8 (0.7-0.9)</td>
</tr>
<tr>
<td>Not associated with exertion</td>
<td>14</td>
<td>803</td>
<td>0.8 (0.6-0.9)</td>
</tr>
</tbody>
</table>

Abbreviations: AMI, acute myocardial infarction; CI, confidence interval.
2.3.2 Electrocardiography (ECG)

ECG is the initial tool used for the screening of patients presenting with chest pain to the hospital settings. Soon after its inception >100 years ago, it was recognized as a tool in the diagnosis of cardiac pain though the full impact of its usefulness was not fully exploited until the 1940s-50s (37). It is used in the evaluation of multiple cardiac abnormalities including conduction abnormalities, arrhythmias, electrolyte abnormalities and ischaemic heart disease among others.

ECG diagnostic criteria of an ACS

May either be ST Elevation Myocardial infarction (STEMI) or or Non-ST Elevation Myocardial Infarction NSTEMI.

ST segment elevation at the J point of ≥2mV in V2-V3 or ≥1 mV in all other leads in two or more contiguous leads constitutes an STEMI. New or presumed new Left Bundle branch block (LBBB) is considered a STEMI equivalent and should be managed as such in the same way.

Non-STEMI ACS constitutes other ECG features suggestive of ischaemia such as ST segment elevation of lesser magnitude than the one mentioned above, ST segment depression of 0.05Mv in V2-V3 and 0.1 in all other leads measured at 0.08 seconds from the J point and this should not be associated with ventricular hypertrophy (38).

T wave changes such as hyper-acute tall T waves, flattening or inversion at least 1mm deep that is present in two continuous leads that have dominant R waves also suggest myocardial ischaemia.

The third type of ACS is unstable angina with dynamic ECG changes without troponin elevation.

ECG Utility

In a study evaluating the utility of pre-hospital ECG for the diagnosis of Acute Coronary Syndrome, it was found that the ECG is invaluable in the early screening of chest pain and more ST segment and Q wave abnormalities were found in patients with Acute Coronary Syndrome - than those without (39). ECG has the advantage of displaying features of myocardial ischaemia even in the absence of coronary vessel occlusion like what is seen in Prinzmetal’s angina or Takusubo cardiomyopathy.

ECG Sensitivity

Compared to troponin confirmed AMI, ECG has a sensitivity of 38% and a specificity of 98%. When compared to Coronary angiography, ECG has a sensitivity range of 36-87% (37) Doing serial ECG can also increase sensitivity (39). Addition of leads can increase the sensitivity from 33% to 45% if using 19 lead and to 49% with a 24 lead ECG compared to a standard 12 lead ECG at admission. However there would be a slight reduction in specificity (40). Increase in number of leads also improves the diagnosis of posteriorly located infarcts (37). The low
sensitivity of ECG means that it cannot be used in isolation but needs to be used in combination with other tools for the definitive diagnosis.

**ECG Timing**

The AHA/ACA guidelines recommend a 12 lead ECG to be done and interpreted within 10 minutes of first medical contact for patients presenting with chest pain or angina equivalent (41). This time target remains a challenge in the developed world. In the crusade initiative, it was shown that only 33% of patients with high risk STEMI had an ECG within the recommended 10 minute bracket and females were more likely to have delayed ECG’s(42) Timely ECGs have been documented to improve management outcomes such as door to needle or door to balloon times (43)

**Limitations of ECG**

ST Elevation can occur in other settings other than ACS giving rise to over-diagnosis.

<table>
<thead>
<tr>
<th>TABLE 3: Conditions with ST- segment elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-infarction, trans mural ischemia (Prinzmetal's angina pattern or acute takotsubo syndrome)</td>
</tr>
<tr>
<td>Post-MI (ventricular aneurysm pattern)</td>
</tr>
<tr>
<td>Myocarditis</td>
</tr>
<tr>
<td>Massive Pulmonary Embolism</td>
</tr>
<tr>
<td>Acute pericarditis</td>
</tr>
<tr>
<td>Myocardial tumour</td>
</tr>
<tr>
<td>Hyperkalaemia</td>
</tr>
<tr>
<td>Myocardial trauma</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>Brugada-type patterns</td>
</tr>
<tr>
<td>Hypothermia</td>
</tr>
<tr>
<td>Paced rhythms</td>
</tr>
<tr>
<td>Normal variants</td>
</tr>
<tr>
<td>Hypercalcaemia</td>
</tr>
<tr>
<td>Post DC Cardioversions</td>
</tr>
</tbody>
</table>
Other limitations with ECGs

ECG interpretation may impact its utility. Physician interpretation has a specificity of 45% while computerized interpretation sensitivity is just about 36% (37). The presence of arrhythmias also complicates ECG interpretation and so does pacemaker presence. These may require cardiologist consultation for correct interpretation.

ECG’s are also real time pictures and may be normal in transient ischaemia leading to a false reassurance. To avoid such scenarios, it is important to do serial ECGs or stress ECG in patients with high probability of an ACS and combine it with evaluation of cardiac biomarkers.

2.3.3 Cardiac biomarkers

Upon cardiac myocyte injury, various molecules are released from the cells into the extra cellular compartment. These include Troponin, Myoglobin, Creatinine Kinase, Lactate dehydrogenase and heart type fatty acid among others. These have been used in the diagnosis and monitoring of cardiac injury for decades and recent advances in the use of highly sensitive Troponin tests have made rapid and accurate diagnosis possible.

Historical biomarkers

Lactate Dehydrogenase

Consists of 5 isoenzymes, 1 to 5 with LD-1 being found mostly in the heart. LD1 is also found in other tissues such as the kidney, stomach, pancreases and Red blood cells, thus making it less sensitive to cardiac injury. Its use has been overtaken by the more sensitive and specific assays such as troponin.

Myoglobin

A heme protein rapidly released from damaged tissue. Has a short half-life and is non-specific for injury to the heart. Was initially used as an adjunct for troponin assays in the early diagnosis, however with the advent of highly sensitive troponin assays with lower cut off ranges, there is no clinical value of adding a myoglobin test (44)

CK-MB

This is an enzyme involved in the regeneration of phosphocreatine from creatine and ATP thus providing an energy source in the cells of various tissues such as skeletal muscle, brain, spermatozoa. It exists in three isoforms MM, BB and MB. Total creatine kinase lacks specificity and may be raised in conditions of skeletal muscle injury.

CK-MB is more specific to the heart. It is rarely elevated in myositis except in very severe inflammation. It goes up 3-12 hours post infarction, peaks at 18-24 and returns to baseline in 36-
48hrs. This makes it useful in the assessment of re-infarction but it has no role in the late diagnosis of older infarction. Currently it is used in combination with a troponin assay in the emergency evaluation of acute coronary syndromes where standard troponin assays may fail to detect myocardial injury in the initial 6 hours.

**Troponin Assay**

Cardiac Troponins are regulatory proteins located at regular intervals along tropomyosin. They have three subunits (T, C and I) with different functions. Troponin T attaches to tropomyosin, Troponin C is the binding site for Calcium and Troponin I inhibits the binding of myosin to actin. They are coded for by specific genes giving rise to different amino acid sequences specific to cardiac and skeletal troponin. This makes troponin assay very specific to cardiac injury. There are two pools of troponin, the cytosolic or early release pool and the structural pool. Approximately 7% of cTn T and 3.5% of cTn I is cytosolic (45)

Troponin is released into the blood stream when cardiac myocytes are damaged. It is the most sensitive biomarker currently in use for the acute diagnosis of Myocardial infarction. It rises soon after myocyte injury in 2-3 hours and remains high for up to 10 days and this allows for late diagnosis of myocyte damage. In acute myocardial infarction, elevated troponin levels are those above the 99th percentile of the normal population. Use of the highly sensitive troponin enables early diagnosis of Acute MI without the need to use the early rising biomarkers like myoglobin (44). It can also be used to identify patients at low risk for myocardial infarction with a negative predictive value of up to 99.6% (46) and this may facilitate early discharge of chest pain patients due to non cardiac causes.

Troponin is also elevated in a variety of non ischaemic cardiac pathologies such as myocyte injury due to myocarditis, myocardial contusion, atrial fibrillation and congestive cardiac failure. It is also elevated in some non-cardiac conditions such as chronic renal failure, chronic obstructive pulmonary disease, and pulmonary embolism, critically ill patients in ICU, HDU and in Cerebrovascular illnesses especially subarachnoid hemorrhage. (47)

It is also a marker of prognosis. Elevated Troponin T and I are key indicators of risk of death, myocardial infarction and death from cardiovascular causes in patients with unstable angina pectoris and Non ST Elevation Myocardial Infarction(48,49) This has also been observed in diabetic patients with stable coronary disease where elevated troponin are a marker of worse prognosis despite intensive medical care or invasive revascularization therapy. (50)

**2.4 MANAGEMENT OF ACUTE CORONARY SYNDROME**

**2.4.1 DEFINITIVE MANAGEMENT**

Primary PCI is the gold standard of treatment. It is indicated for STEMI and NSTEMI in institutions where it is available to those with symptoms lasting 12 hours or less. It should be
done within 90 minutes of first medical contact. However if the patient is in cardiogenic shock, heart failure from the ACS it should be done despite the time delays. This is a class 1 recommendation. (51)

Thrombolytic therapy is the recommended reperfusion strategy if PCI is not available as long as there are no contraindications. (51) The STREAM trial demonstrated that fibrinolytic therapy can be a viable option in patients who present early and are unable to access PCI within 1 hour of medical contact (52). In a study reviewing acute coronary syndromes in a PCI enabled tertiary hospital in Kenya 55% of patients with ACS received fibrinolytic therapy as opposed to 18% who underwent PCI (14). This seems to be the best option for third world countries such as Kenya where there are only 7 PCI enabled hospitals with only one in the public sector. (53)

2.4.2 SUPPORTIVE MANAGEMENT

Dual antiplatelete administration with Aspirin and a PY2 inhibitor such as clopidogrel or prasugrel (51). Anticoagulation with either Unfractionated heparin, enoxaparin, bivalirudin and fondaparinux are indicated as adjunctive therapies to PCI and should be initiated as soon as contact is made. Nitrates are used for anginal pain relief but these are contraindicated if patient is hypotensive, has had prior use of 5’phosphodiesterase inhibitors within the last 72 hours especially for tadalafil, or have severe heart rate abnormalities(51). Oxygen therapy is indicated in hypoxic, dyspnoeic or has heart failure. (51) but routine indiscriminate use of oxygen may be harmful (54). Analgesia with morphine as the drug of choice in ACS due to its analgesic, anxiolytic, and relief of the work of breathing especially in pulmonary edema. NSAIDS and COX -2 inhibitors are harmful and should be avoided(51). RAAS Blockade Ace inhibitors should be given to all patients routinely unless in hypotension, renal failure or hyperkalemia. Beta blockers should be given to all patients routinely within 24 hours and especially if they have persistent hypertension unless there is a contraindication such as reactive airway disease, heart block, severe heart failure or cardiogenic shock (51) Lipid management with high dose statins should be administered rapidly in all patients despite their lipid profiles because this has been shown to provide clinical benefit and plaque stabilization even with low LDL cholesterol (55). High dose atorvastatin is preferred due to its clinical and mortality benefit (51)

2.5 PROBLEM STATEMENT

Despite the wealth of knowledge available of the prevalence, etiology, clinical features and management strategies for ACS, the overall care of ACS in Kenya is suboptimal especially in the public sector. Varied factors weigh in on these shortcomings and can be categorized as either patient factors, healthcare provider factors or health systems and delivery factors.

Patient factors

The Health seeking behavior of patients in SSA, especially regarding cardiovascular disease is quite discouraging. Despite increasing literacy, even in urban settings, most individuals do not
proactively participate in screening for non-communicable disease like high blood sugar or cholesterol levels. (56) Late presentation is an almost universal feature in studies evaluating ACS in the African setting. 20% of patients with an ACS presenting to A&E in a Kenyan tertiary hospital did not receive fibrinolytic therapy because they presented late. (14) In KNH, almost 80% of ACS patients presented more than 6 hours after symptoms onset, averaging 5.5 days duration before contact with medical personnel. (15) This is mostly due to lack of awareness of the gravity of symptoms.

**Health care workers and systems**

In a study in 5 emergency units in the United states, poor ECG interpretation and failure to adhere to evidence based guidelines led to 22% of eligible patients with ischaemia and ST elevation myocardial infarction failing to receive reperfusion therapy(57) In South Africa, the ACCESS study evaluated the practices and patterns of the care of patients with ACS and found that despite adherence to recommended evidenced based initial medical therapy such as Aspirin, statins, beta blockers and anticoagulants, the definitive interventional therapy (PCI) rates were much lower compared to European and North American at 59% versus 80% (58). In Kenya, a retrospective review of the acute management of ACS at the Aga Khan found adherence to recommended guidelines quite variable with excellent Door to ECG time, but less than 50% patients receiving fibrinolytic therapy in time and barely 30% undergoing PCI within 90 minutes. (59)

**2.6 Protocol Based ACS Care**

Protocols for clinical management have been shown to streamline care subsequently improving clinical outcomes and other variables such as reducing hospital costs and shortening length of stay(60,61). This has been very effective in infectious disease programmes in Sub-Saharan Africa particularly in the management of HIV/AIDS and Tuberculosis. These protocols contain clear guidelines with simple checklists and have made screening, diagnosis and treatment of these conditions very effective across different cadre of healthcare professionals.

The ACC/AHA recommends that each institution needs to come up with customized standards of care and protocols for the implementation of the evidence based recommendations it provides for the management of ACS. Several institutions have developed strategies to prevent missed diagnoses, enable accurate and timely diagnosis of ACS so as to ensure patients receive specific and definitive therapy rapidly. The earliest point of intervention is at the first contact, during triage and screening of patients to determine the probability of an ACS and immediately start the process of confirming or ruling out the diagnosis. Door to ECG time has been identified as a predictor of meeting subsequent treatment goals like door to needle or door to balloon time. (62,63) Focus has been aimed at shortening this time to achieve the recommended 10 minutes from first medical contact. The strategies are varied and customized to the individual institutions.
Table 4: STRATEGIES TO SHORTEN DOOR TO ECG TIME

<table>
<thead>
<tr>
<th>YEAR</th>
<th>INTERVENTION</th>
<th>N</th>
<th>D2ECG BEFORE</th>
<th>D2ECG AFTER</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2007</td>
<td>Introduction of ER greeters to screen for likely candidates and obtain ECGs</td>
<td>126</td>
<td>29.6 minutes</td>
<td>8.8 minutes</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>Purim-Shem-Tov et al (64) Chicago university hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2009</td>
<td>Design patient prioritization process for cardiac triage Assigning specific staff to provide rapid ECGs Feedback and review of any case falling out of the 10-minute timeline Training and education of staff on identification of high risk patients</td>
<td></td>
<td>21.8 +/- 5.49 minutes</td>
<td>9.47 +/- 2.48 minutes</td>
<td>55% improvement</td>
</tr>
<tr>
<td></td>
<td>Phelan et al (43) Cleveland clinic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2009</td>
<td>Training registration staff on cardiac triage Designating one Technician to do ECGs all day Provision of a dedicated ECG phone to call the ECG Tech</td>
<td>718</td>
<td>16 min</td>
<td>9 min</td>
<td>0.0001</td>
</tr>
<tr>
<td></td>
<td>Thomas Jefferson University Hospital Takakuwa et al (63)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2010-2012</td>
<td>Creation of a cardiac triage designation in addition to emergency and urgent to fast track reviews Movement of ECG technician and station to the initial triage area</td>
<td>232</td>
<td>43 min</td>
<td>30 min</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Los Angeles County/ University of Southern California Coyne et al (62)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A pertinent question arises as to who should obtain a screening ECG at first medical contact. How is patient selection done for a fast tracked cardiac evaluation done? A study was done to derive and subsequently validate a simple tool for the prioritization of immediate ECG acquisition before physician review in North Carolina, USA. This was done from a cohort of more than 3.5 million Emergency department visits in 107 hospitals over a two year period from 2007-2008. It was stratified according to age due to increasing age being a risk factor for ACS and included clinical symptoms and traditional risk factors that can rapidly be elicited at a point of triage. It also included symptoms like syncope and epigastric pain that may be attributed to non cardiac etiologies especially in atypical ACS presentation without chest pain. It had a sensitivity of 91.7%, a specificity of 76.3%, a positive predictive value of 0.67% and a negative predictive value of 99.98% for diagnosis of acute myocardial infarction (32) Making it and excellent screening tool for initial evaluation and determining probability of acute presentation of ischaemic heart disease.

**FIGURE 2: ECG PRIORITIZATION RULE**
2.7 STUDY JUSTIFICATION

Acute coronary syndrome is increasing in our environment. Gaps have been identified in the acute care of ACS in varied set ups in the western countries, Africa and Kenya showing that there are many missed diagnosis and opportunities to improve care. There is need to audit our current practice and identify both our shortcomings and opportunities which will inform the implementation of clear specific customized protocols to improve care of acute coronary syndromes. It is possible to provide timely and evidence based management of ACS and achieve recommended international guidelines and targets.

2.8 RESEARCH QUESTION

What is the current ACS screening practice at Kenyatta National Hospital Accidents and Emergency Department and how would a protocol influence that?

NULL HYPOTHESIS

Implementing a diagnostic care package does not improve screening of ACS at Kenyatta National Hospital Accidents and Emergency Department.

2.9 OBJECTIVES

2.9.1 BROAD OBJECTIVES

To determine the current triage practice, implement a screening protocol and evaluate its impact on door to ECG time of patients at high risk for Acute Coronary Syndrome presenting at the Accident and Emergency Department in Kenyatta National Hospital.

2.9.2 SPECIFIC OBJECTIVES

1. To determine the proportion of patients at high risk for acute coronary syndrome who obtain an ECG at the Accidents and Emergency department of KNH.
2. To determine the door to ECG time of patients at high risk for acute coronary syndrome at the Accidents and Emergency department.
3. To implement a triage protocol and evaluate its impact on the proportion and on the Door to ECG time of patients at high risk for acute coronary syndrome at the Accident and Emergency department.
3.0 METHODOLOGY

3.1 STUDY DESIGN

Quasi experimental design with two phases before and after intervention.

3.1.2 STUDY SETTING

The Kenyatta national hospital accident and emergency department.

3.1.3 STUDY POPULATION

Non-surgical adult patients aged above 30 years presenting to the adult triage station in the A&E department of KNH.

3.1.4 CASES DEFINITION

The case definition was derived from the ECG prioritization rule derived and validated in North Carolina outlined previously (32). This defined a case as any medical patient aged above 30 years with a high probability of ACS. Medical patient was a triage designation assigned by the nursing staff at first contact depending on presenting complaints. High probability of ACS was defined by history or presence risk factors which were stratified according to age. This was categorised as age above 30 with of either of diabetes or hypertension and chest pain or age above 50 with either of dyspnoea, altered mental status, syncope, upper limb pain, and age above 80 with epigastric pain, nausea or vomiting.

Once and ECG was done, abnormal ECG features were defined according to the AHA/ACA recommendations for standardization of ECG (38) consistent with ACS and included, ST segment elevation, ST segment elevation/Depression, Pathological Q waves, T wave inversion/hyper-acute T waves, new or presumed new LBBB

INCLUSION CRITERIA

All the patients who fit the case definition and provided written informed consent were included in the study.

EXCLUSION CRITERIA

Patients with symptoms attributed to trauma

Psychotic patients (due to inability to do an ECG)
3.1.5 SAMPLE SIZE

SAMPLE SIZE DETERMINATION AND FORMULA USED

This was an interventional study with patients selected before and after the intervention hence a formula for comparing proportions was used to calculate sample size. It was estimated that 50% of patients have ECG’s done pre intervention. A sample size of at least 170 in each group was calculated to detect a minimum intervention effect size of 15% with 80% power with 95% confidence using the following formula:

\[
n = \frac{2(Z_{\alpha/2}+Z_{1-\beta})^2 P_{av} (1-P_{av})}{(P_0-P_1)^2}
\]


N is the sample size required in each group

\(Z_{\alpha/2}\) refers to the level of significance or confidence interval – 1.96 for 95% CI

\(Z_{1-\beta}\) refers to the power of obtaining difference between the two groups – 0.84 for 80% power

\(P_0\) – Proportion of patients who obtain an ECG in pre-intervention period – 50%

\(P_1\) – Proportion of patients who obtain an ECG in post-intervention period – 65%

\(P_{av}\) – Average outcome in the two groups – 57.5%

Substituting into the formula:

\[n = 170\] patients per group was studied to detect a minimum effect size of 15% after the intervention.

SAMPLING METHODS

Consecutive sampling
3.1.6 SCREENING AND RECRUITMENT

Pre Intervention Triage Practice and patient flow within KNH A&E.

All patients presenting at the A&E are first attended to at the triage a unit with two stations manned by a minimum of three nurses. The nurses evaluate patients and fill in a triage form with information on bio data, vital signs, chief presenting complaint, time of triage and a TEWS score (Triage Early Warning Sign). In emergencies (TEWS score more than 7) patient is immediately wheeled to resuscitation room B (RRB) for management. For non-emergent cases, the patients are designated with color codes for varying degrees of urgency. They proceed to registration desk to obtain an outpatient file number and a consultation card before paying a consultation fee at the cashier. Subsequently, they proceed to the waiting bay before being called into doctor’s room 1 to 4 for consultation by one of the medical officers on duty who after assessment of the patient and determines whether or not a 12 lead ECG is required. Patient have to pay for the procedure at the cashier before going to Resuscitation room B for the ECG which is administered by the nurse stationed there. They subsequently return to the medical doctor for interpretation.

Figure 3 ILLUSTRATION OF PATIENT FLOW AT KNH ACCIDENTS AND EMERGENCY DEPARTMENT

RRA: Resuscitation Room A, RR-B: Resuscitation Room B, 1-6 Steps between triage and acquisition of an ECG

Pre-intervention Screening and Recruitment
This was done at the triage station where two research assistants were stationed. During the regular process of triage outlined above, any non-surgical patient aged >30 was identified. A
A description of study intent was given to the patient about the desire to follow up on their progress during management at the accidents and emergency department. Consent was obtained and patient details and phone numbers were taken to facilitate follow up. Patients who did not give consent or patients presenting with psychosis were excluded. This was due to the technical difficulty of doing an ECG on a psychotic patient.

Once a patient was identified and consent obtained the patient continued with subsequent management as usual in the A&E department. The ECG prioritization rule which also served as our case definition was applied retrospectively to all the patients identified using information recorded in the triage book and casualty cards evaluated by the end of the work day (24 hours after triage). This further classified the patients into either high or low probability of ACS. If patients had a high probability of ACS as per the ECG rule, they were recruited into the study. Follow-up phone calls were made to the patient or the next of kin mobile phones to confirm ECG request, acquisition and if possible retrieve a copy of the ECG.

**FIGURE 4: SCREENING AND RECRUITMENT PRE INTERVENTION**
The following information was derived for the patient

1. Bio data (Age, sex)
2. Door time (Indicated in the triage form of all patients) and date
3. Presenting symptoms and duration of symptoms
4. Risk Factors (DM, Hypertension, Smoking etc)
5. Whether a 12 lead ECG was done on the patient.
6. If done, the time from arrival to ECG time.
7. Whether a troponin tests was ordered or not and the values obtained
8. Whether a diagnosis of ACS was made or not

**The intervention and post intervention recruitment**
The intervention was a composite of 2 actions

1. The introduction of an ECG prioritization screening tool.
2. Administration of a free ECGs on eligible patients immediately after identification before doctors’ consultation for free.

The ECG prioritization tool which was used to determine which patient was to be fast tracked in the obtaining of an immediate ECG was provided to the triage staff and research assistant as an enlarged chart and smaller A-4 copies to facilitate the selection of patients who required an immediate ECG.

**FIGURE 2 ECG Prioritization Rule**

```
AGE >30 WITH CHEST PAIN, DIABETES OR HYPERTENSION

AGE >50 WITH DYSPNOEA ALTERED MENTAL STATUS UPPER LIMB PAIN SYNCOPE WEAKNESS

AGE >80 WITH ABDOMINAL PAIN NAUSEA VOMMITING
```
When an ECG was indicated according to the rule above, the research assistant then immediately directed the patient to the ECG room adjacent to registration desk which is in close proximity to the triage station and administered a standard 12-Lead ECG to the patient. This was at no cost to the patient. When features consistent with ischaemia were found, blood samples were also drawn for troponin level evaluation. Therefore the prioritization rule was applied prospectively.

**FIGURE 5 POST INTERVENTION MOVEMENT OF PATIENT FOR ECG IN A&E**

![Diagram](image)

RR-A Resuscitation room A, RR-B Resuscitation room B, 1-6 Steps in the normal care

### 3.2 CLINICAL METHODS

**History Taking**

This was as quick cardiac focused history to elicit any symptoms of chest pain or angina equivalent (epigastric pain, jaw/upper extremity pain, dyspnoea, altered mental status) and associated risk factors such as history of diabetes, hypertension, dyslipidaemia and smoking. It was done by the research assistant stationed at the triage station as part of the triage.

**ECG administration**

A standard resting 12 lead ECG was done by the research assistant using a GE Marquette MAC 5500 EKG Machine / ECG System machine.

**ECG Interpretation**

Features consistent with ACS included

- ST segment elevation
• ST segment elevation/Depression
• Pathological Q waves
• T wave inversion/hyper-acute T waves
• New or presumed new LBBB

ECGs were presented to the casualty medical officers in room 1-4 for interpretation and their findings documented. The medical officers were not sensitized on the details of the research prior. A second analysis and interpretation was done by the Principle Investigator (PI). If PI was not at the Accidents & Emergency department, a picture of the ECG was taken by the Research Assistant and sent via WhatsApp phone application to the PI for interpretation. This too was documented.

3.3 STUDY VARIABLES –

Dependent
1. ECG done Yes or no
2. Door to ECG time

Independent
a. Age
b. Sex
c. Risk factors (DM, HPT, Smoking, Obesity)
d. Time of arrival (AM versus PM)
e. Day of the week (weekday versus weekend)

3.4 QUALITY ASSURANCE PROCEDURES

Screening was done by trained Research Assistants who were trained in triage and ECG administration. ECG reading and interpretation was done twice by casualty Medical officer and by the PI. In the event of lack of clarity, further clarification was sought with cardiologists on call.
3.5 DATA MANAGEMENT AND STATISTICAL METHODS

3.5.1 Data Collection and Handling

Data was keyed into two tools
- A hard copy Study Proforma.
- Keyed into a MS access database.

Data was coded and serialized without using patient identifiers to maintain confidentiality. Double data entry technique was used to enter data into a password protected MS Access database managed by the statistician. Data verification, cleaning and validation was done in MS Excel. Data capture forms were stored and are available for scrutiny and a soft copy of all data was archived. Data analysis was done in SPSS version 21.0.

3.5.2 Statistical Analysis

Categorical data such as sex and risk factors was summarized using frequencies and proportions. Continuous data such as age and door-to-ECG time was summarized using means (with standard deviations) and medians (with inter-quartile ranges).

Door to ECG time was further dichotomized to early <10 minutes verses delayed >10 minutes and presented as percentages with 95% confidence interval. Univariate analysis was done to compare the proportions of patients receiving ECG and of those receiving an early or delayed ECG before and after intervention. This comparison was done using the McNemar test. Comparisons of the mean time to ECG before and after the intervention was done using Wilcoxon signed rank sum test. A p value of <0.05 was considered significant.

Data was organized into tables, pie charts, histograms, bar charts and line graphs where necessary.

3.6 ETHICAL CONSIDERATIONS

This research was approved by the University of Nairobi department of Clinical Medicine and Therapeutics and the KNH Ethics committee. The objectives and procedures intended were explained to the patient in a language that he/she understood. Next of kin or guardians were used to aid in the translation for illiterate patients. Patients were free to decline participation. In the pre-intervention phase, the consent was obtained at first contact so as to facilitate follow up by way of phone calls during care.
In the post second phase, the intervention represented recommended standard of care, we did not withhold care and administered ECGs to all eligible patients. Obtaining ECGs and samples for troponin assays was done in the routine manner and did not present any additional risks to the patients. Informed consent was obtained after ECG administration for the use of patients’ data into the study. Any suspected diagnosis of an ACS or other findings relevant for management was documented and communicated to the management team at the KNH casualty to facilitate prompt management. We provided dual antiplatelete therapy Aspirin and Clopidogrel for any patients who required it as the A&E team took over the subsequent care. All data collected was stored confidentially.
4.0 RESULTS

A total of 4,061 patients were seen at the KNH triage between 14th September and October 24th 2016. Out of these 1,599 were medical patients aged above 30 years. This represented 34% of all patients presenting to the A&E.

The pre-intervention phase ran between 14th and 30th September during which the ECG prioritization rule was applied retrospectively on 877 medical patient charts aged above 30 years. 259 of these met the criteria for high probability of ACS and after excluding 59 cases in whom medical records were incomplete could not be traced and 10 who did not provide consent, 190 were and included in the final analysis.

**Figure 6. SCREENING AND RECRUITMENT PRE INTERVENTION**

The post intervention phase ran between 6th and 24th October 2016. The ECG prioritization rule was applied prospectively on 722 medical patients aged above 30 years and 232 met the criteria for high probability of ACS. 6 of these did not give consent and 54 could not be traced and were missed. 172 patients were recruited and included in the final analysis. In total, the medical patients who fit the criteria for high probability of ACS were 30% of all the medical patients.
Baseline Characteristics of the study participants Pre-Intervention

The characteristics of the patients were evaluated and found to be similar in both pre and post intervention patient groups. The mean age was 56(SD16) median 56 years in the pre intervention group and 55(SD17) median 53 in the post intervention group. Majority of the patients were in the 45-65 age group in both pre and post intervention phase. Females accounted for 57% of the respondents in pre intervention group and 55% in post intervention group. About half of the respondents were peri-urban residents and at least 70% of the patients had attained a secondary school level of education in both groups. Most of the patients were attended and recruited on weekdays with less than 15% being seen on weekends. This distribution of the respondents in terms of age, sex, residence and education level is further illustrated in Table 1.
Table 5 Baseline Demographic Features

<table>
<thead>
<tr>
<th></th>
<th>Pre Intervention (n=190)</th>
<th>Post Intervention (n=172)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;35</td>
<td>9% (17)</td>
<td>15.2% (26)</td>
<td>0.299</td>
</tr>
<tr>
<td>35-45</td>
<td>19.7% (37)</td>
<td>19.9% (34)</td>
<td></td>
</tr>
<tr>
<td>45-55</td>
<td>21.3% (40)</td>
<td>21.1% (36)</td>
<td></td>
</tr>
<tr>
<td>55-65</td>
<td>19.1% (36)</td>
<td>16.4% (28)</td>
<td></td>
</tr>
<tr>
<td>65-75</td>
<td>20.7% (39)</td>
<td>13.5% (23)</td>
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<tr>
<td>75-85</td>
<td>6.4% (12)</td>
<td>8.2% (14)</td>
<td></td>
</tr>
<tr>
<td>&gt;85</td>
<td>3.7% (7)</td>
<td>5.8% (10)</td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>57% (106)</td>
<td>55% (94)</td>
<td>0.70</td>
</tr>
<tr>
<td><strong>Residence</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>16% (29)</td>
<td>22.6% (38)</td>
<td>0.093</td>
</tr>
<tr>
<td>Peri-Urban</td>
<td>50.3% (91)</td>
<td>45.8% (77)</td>
<td></td>
</tr>
<tr>
<td>Rural</td>
<td>33.7% (61)</td>
<td>31.5% (53)</td>
<td></td>
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<tr>
<td><strong>Education</strong></td>
<td></td>
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</tr>
<tr>
<td>Primary</td>
<td>42.1% (53)</td>
<td>44.4% (68)</td>
<td>0.63</td>
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<tr>
<td>Secondary</td>
<td>33.2% (42)</td>
<td>28.1% (43)</td>
<td></td>
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<tr>
<td>Tertiary</td>
<td>24.6% (31)</td>
<td>27.5% (42)</td>
<td></td>
</tr>
<tr>
<td><strong>Day of the week</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weekday</td>
<td>93.1% (175)</td>
<td>88.3% (151)</td>
<td>0.117</td>
</tr>
</tbody>
</table>

**ECG prioritization Features**

As per the ECG prioritization rule, we screened for patients that had presented with chest pain, dyspnoea, syncope, upper limb pain and or abdominal symptoms in both pre and post intervention patients. The chest pain was the commonest feature fitting this rule at 52.6%. Dyspnoea was the next commonest followed closely by general body weakness. Abdominal symptoms specifically epigastric pain, nausea and vomiting were noted in 42% of patients. Upper limb pain and altered mental status were seen in 13.8% and 12.9% in pre and post intervention groups respectively. The median time to presentation from symptom onset was 2 days (IQR 1-30 days). The risk factors derived from a quick focused history at the triage station revealed hypertension as the commonest risk factor in both groups at 76% and 77% for pre and post intervention groups respectively. Smoking and self reported dyslipidaemia or use of lipid lowering agents were noted in a much smaller proportion.
TABLE 6 ECG Prioritization features

<table>
<thead>
<tr>
<th></th>
<th>Pre Intervention (n=190)</th>
<th>Post Intervention (n=172)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest Pain</td>
<td>53.7% (101)</td>
<td>53.2% (91)</td>
<td>0.93</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>47.3% (89)</td>
<td>53.8% (92)</td>
<td>0.221</td>
</tr>
<tr>
<td>Upper Limb Pain</td>
<td>13.8% (26)</td>
<td>12.9% (22)</td>
<td>0.789</td>
</tr>
<tr>
<td>Syncope</td>
<td>23.9% (45)</td>
<td>9.4% (16)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Epigastric Pain</td>
<td>25% (47)</td>
<td>9.4% (16)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>21.3% (40)</td>
<td>19.9% (34)</td>
<td>0.744</td>
</tr>
<tr>
<td>Hypertension</td>
<td>76.6% (144)</td>
<td>77.8% (133)</td>
<td>0.790</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>2.7% (5)</td>
<td>2.9% (5)</td>
<td>0.879</td>
</tr>
<tr>
<td>Smoking</td>
<td>13.8% (26)</td>
<td>14.0% (24)</td>
<td>0.9</td>
</tr>
</tbody>
</table>

A medical exam at the triage station revealed that almost half of the patients selected by the ECG prioritization criteria at the triage had elevated blood pressure in both groups and at least 60% were overweight or obese based on their BMI

Table 7 Physical Examination Findings

<table>
<thead>
<tr>
<th></th>
<th>Pre Intervention (n=190)</th>
<th>Post Intervention (n=172)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Systolic Blood Pressure</td>
<td>144 (31)</td>
<td>143 (31)</td>
</tr>
<tr>
<td>Pulse Rate Grouped</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low (&lt;50)</td>
<td>3.72% (7)</td>
<td>4.73% (8)</td>
</tr>
<tr>
<td>Normal (50-100)</td>
<td>65.95% (124)</td>
<td>66.83% (114)</td>
</tr>
<tr>
<td>High (&gt;100)</td>
<td>30.31% (57)</td>
<td>28.24 (48)</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>1.5% (3)</td>
<td>4.76% (8)</td>
</tr>
<tr>
<td>Normal</td>
<td>29.25% (55)</td>
<td>35.71% (60)</td>
</tr>
<tr>
<td>Overweight</td>
<td>17.55% (33)</td>
<td>24.4% (41)</td>
</tr>
<tr>
<td>Obese</td>
<td>51.59% (97)</td>
<td>35.11% (59)</td>
</tr>
</tbody>
</table>
Main Results

In the pre intervention phase, the ECG prioritization rule was applied retrospectively by tracking the patients’ cards or files for documentation of the processes done at A&E. Where details were not clear, patients were called via mobile phones and inquiry was made as to whether an ECG was done or not, when and where they were done. It was found that 14.4% (95% CI 10.06 – 20.09) of patients with a high probability of acute coronary syndrome had an ECG done. The mean door to ECG time was 10.2hrs (SD 7.18) with a median of 8.08hrs (IQR 4.65-13.1hrs). None of these patients had an ECG within the recommended 10 minutes. The patients who had ECGs had to have them done at cardiology unit 27 and in external centers outside the hospital facility.

After putting up the ECG prioritization tool and providing the ECG, 93.6% (95% CI (88.85 – 96.37) of patients with a high probability of ACS had an ECG done. A logistic regression was performed to determine the likelihood to have an ECG in the two phases. Patients were 86.7 times (95% CI 41-180) $p < 0.05$ more likely to receive an ECG during the post-intervention period than during the pre-intervention period. The mean door to ECG time post intervention was 2.4hrs (SD4.02), Median 69minutes IQR (20-163minutes ). Only 9.4% of these patients had an ECG done within the recommended 10 minutes. However, this time period was 7.8 hours faster than the average door to ECG time for the pre intervention period and this was statistically significant $p <0.05$.

FIGURE 8 DOOR TO ECG TIME PRE AND POST INTERVENTION
Other findings
On further evaluation of the 161 patients who did not obtain an ECG in the pre-intervention phase, it was found that in 74.3% there was no documented request in the patients’ casualty cards or files as part of the ER physician’s planned investigations. A further 15.0% had an ECG request documented but on inquiry sited financial constraints as the reason for not having an ECG done. 3.75% of the patients declined to have the test done. In the post intervention group, a faulty machine was the main reason for not having an ECG done and this occurred in 6.4% of all the post intervention patients.

Figure 9 REASON ECG NOT DONE PRE-INTERVENTION

ECG Findings
Most of the ECGs acquired were done in the post intervention group and only 14% were done in the pre-intervention group. All of them were analysed together for features consistent with ischaemia according to the AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram (38). Of all the ECG’s acquired only 12.4% (n=187) were found to be normal. Most of the abnormalities were non ischaemic 62.9% including conduction abnormalities and chamber enlargement. Ischaemic features were further described as ST segment elevation seen in 4.3% and other non ST segment changes (20.4% n=187).
In the ECGs that were considered suggestive of ischaemia, the commonest finding noted was T wave inversion not associated with ventricular enlargement seen in 15 patients and it was mostly in the lateral leads V5 and V6. ST segment elevation was noted in 8 patients, 4 in the anteroseptal leads, 3 in the lateral leads and 1 inferior leads. ST segment depression was seen in 13 patients. Presumed new LBBB was noted on 5 patients and pathological Q waves were noted in 4 patients.

**Figure 10 ECG Findings**

**Figure 11 ECG Features consistent with Ischaemic**
5.0 DISCUSSION

This study set out to evaluate the current screening practice for patients with high probability of acute coronary syndromes, implement a screening protocol and evaluate its impact on door to ECG time at Kenyatta National Hospital. More than 30% of all non-surgical patients presenting to the Kenyatta National Hospital fulfilled the criteria of high probability of acute coronary syndrome based on the ECG prioritization rule. These patients should ideally be screened for the possibility of an Acute Coronary Syndrome with a baseline ECG.

Pre-intervention phase

Our research findings demonstrated that only 14.4% of these patients had an ECG done and the door to ECG time was dismally long at a mean of 10.2hrs. None of the patients achieved the prespecified AHA/ACC standard of 10 minutes from first medical contact. This compares poorly to another audit in Kenya at a private tertiary hospital by Wachira et al in 2013 where almost 90% of patients with STEMI acquired an ECG within <10minutes (59) although this audit was a retrospective review of patients confirmed to have MI and not of all high risk A&E patients. In another retrospective audit evaluating the acute care of STEMI patients at 3 public hospitals in Cape town, South Africa done in 2012, Maharaj et al found a median door to ECG time of 13minutes (IQR 1-402)(65).

The etiology of this failure to obtain ECGs and the delays at the Kenyatta National Hospital is multifactorial: the absence of a specific chest pain cardiac triage pathway to accelerate evaluation of potential ACS patients, secondly, the physician directed system where tests are requested for by doctors. Thirdly, the malfunctioning ECG at the A&E department forcing patients to go to other units such as the KNH cardiology unit which is quite a distance from the ER and only operates from 8:00AM to 5:00PM on weekdays and Saturdays. The WHO describes ECG machines as part of the essential package for provision of care for non communicable diseases and ideally there should always be a functional one at the KNH A&E department.(66) Patients presenting at night had to wait until the following day and some opted to have ECG’s done at peripheral centers. In the aforementioned audit by Wachira et al evaluating the emergency care of ACS patients in a tertiary Hospital in Nairobi where the target 10 minutes was achieved in nearly 90% of patients, it was noted that there was a clear chest pain pathway which outlined the responsibility of the triaging officer to screen and do initial ECGs and present these to the ER physicians for any patient likely to have ACS and this resulted in a very rapid evaluation (59).

On further interrogation of the patients who did not have an ECG done, 74.3%, had no documented plan or request for an ECG in the ER physician’s management plan on their casualty cards or files. The entire patient cohort was selected based on a criteria that picked out clear risk factors for ACS such as diabetes and hypertension and presentation features that were consistent with though not specific to acute myocardial ischaemia such as chest pain and dyspnoea.
lack of ECG requests may be due to a very low index of suspicion amongst health care workers for acute coronary syndromes informing a lack of investigation. It has been demonstrated that physicians with a low threshold for screening for ACS have a higher likelihood of missing the diagnosis (67). In a review of malpractice lawsuits in which there was delayed or missed diagnosis in the emergency department, failure to order appropriate tests was identified as the single largest point of failure. (68). An ECG was an appropriate test for this patient cohort. Further analysis of the clinician’s knowledge, attitude and practice toward ACS may be indicated to interrogate this further in order to draw accurate conclusions from this observation. In a further 15% there was a documented plan for an ECG in their management plan but on inquiry none was done. Financial constraint was cited as the cause of not having an ECG done. Laboratory and radiological tests are paid for before execution and majority of the patients seeking care at public, government-run facilities especially Kenyatta National Hospital do not have medical insurance despite the rollout of the national hospital insurance fund which is currently offering outpatient services. However, NHIF outpatient services are only accessed by civil servants at the KNH hence majority of the patients meet their medical costs out of pocket and the cost of healthcare has been quite high (69,70).

Post Intervention phase

The implementation of our study intervention enabled 93.6% of patients with a high probability of ACS to be screened with a baseline ECG. However, we did not achieve the desired 100% coverage. The mean door to ECG time was shortened from 10.2hrs to 2.4hrs and this reduction was statistically significant (p <0.05). Our median time to ECG was just over 1 hour at 69 minutes and 25% of our patients had an ECG done within 20 minutes of arrival. However, we only managed to attain the AHA recommended <10 minute for 9.4% of our patients. Achieving the recommended time frames has been elusive for many institutions even in the developed world especially if there are no clear cut, pre-specified pathways and protocols. However, once an institution identifies their specific bottlenecks and institutes customized clinical pathways, these targets become achievable with eventual benefit to patients as reperfusion therapy is delivered in a timely manner. For this study, the intervention involved introduction of the ECG prioritization rule that was applied to all medical patients as they were triaged and the immediate administration of ECGs prior to consultation with the ER doctors. This empowerment of the triage officers to administer ECG’s was central to effective and rapid screening leading to increased coverage and shorter door to ECG time. That is the consistent practice at all institutions that achieve this important time bound target including The Aga Khan University Hospital in Kenya where physicians were engaged at the point of ECG interpretation rather than requesting of the test.

The impact of similar protocols has also been demonstrated in prior studies where reduction of door to ECG time was achieved after implementation of varied strategies all of which involve ER physicians at the point of ECG interpretation. In one study in 2007 at the Rush Medical
Center, a private teaching hospital in Chicago Illinois with approximately 200 ED visits per day, Purim-Shem-Tov et al employed a strategy of addition of a greeter staff whose sole function was to identify potential ACS patients through focused questions and administer ECGs as indicated. This reduced the mean door to ECG time significantly from 29.6 minutes to 8.8 minutes (64). For the Kenyatta National Hospital, introduction of hospital greeters at triage with the sole function of screening for ACS and administer ECGs may not be feasible as it involves additional staff who would then have to be trained. This new staff may be underutilized in the department which also has to cater for multiple other emergencies such as trauma and acute gynaecological patients. The triage staff are adequate in number and are already trained in ECG administration. A second study was done in 2009 at The Thomas Jefferson University Hospital Pennsylvania USA, a private university hospital with approximately 330 ED visits per day in which Takakuwa et al utilized registration staff. These were trained to screen for possible ACS patients using focused questions then page or call an ECG technician to administer the ECG after a quick name and age registration. They were able to increase the proportion of patients who got a timely ECG in less than 10 minutes from 16% to 64% achieved a mean door to ECG time of 9 minutes (IQR8-12) post intervention. (63) However such an option of utilizing the registration clerks may also not be a feasible strategy due to the high patient volumes of up to 600 KNH encounters daily in addition to the unique challenges of literacy and finance that patient registration at our A&E already involves. In addition, it would also require training of non clinical staff to screen for ACS.

A third study done in 2012 by Coyne et al at a large public teaching hospital in Los Angeles USA with a daily ED visit of nearly 500 patients employed a two fold strategy of creating a new cardiac triage designation defined by features quite similar to the ECG prioritization rule used in our study and in addition moved their ECG machine to the triage station. With that intervention, they were able to significantly reduce the door-to-ECG time from 43 minutes to 30 minutes, with a median of 14 minutes (62). This environment closely resembled our KNH A&E department in terms of patient numbers. However, employing a similar strategy would not be practical due to lack of space for a couch for the patients to lie on. It would also create a bottle neck as delays would be encountered in the triage of other patients. In addition, the new KNH ECG room is in close proximity to the triage station and has a couch for ambulant patients as well as space to place a stretcher for immobile patients making such a move unnecessary.

The intervention used in our study would be most feasible for our institution as it would involve increasing the capacity of the triage staff who are nurses and are clinical staff. The elimination of the long waiting process from first medical contact and the involvement of the doctors at the point of ECG administration. It is important to note that due to the composite nature of our intervention, it may not be possible to clearly determine the individual effect of each step. The huge difference may have purely been due to the presence of a free ECG service post intervention.
The challenge of implementation of protocols is their uptake by the staff and sustainability post intervention. In this study, the screening and administration of ECGs was an experimental study directed activity carried out by the research assistants rather than standard hospital practice. The triage staff only served a supportive role rather than actively applying the ECG rule to all eligible patients. This resulted in several patients slipping through the triage station when the research assistant was not there and who were later identified at the registration desks or at the doctors consulting rooms resulting in the prolonged door to ECG time in the post intervention phase. The ECG’s were also administered for free therefore overcoming the hurdle of affordability and saving on time spent at queuing at the cashier for payment. All this raises the question of post intervention sustainability. It has been noted in previous studies that implementation of interventions even in the developed world may not be successful if there is no buy-in from all stakeholders (71) Multi-disciplinary teams composed of nursing, A&E doctors, registrars, cardiologists, hospital management and finance departments are required to analyze the provided guidelines then develop and implement customized protocols. The necessity of a multidisciplinary effort was further highlighted in this study when the intervention team experienced technical challenges with the ECG machine and required the expertise of the biomedical team to restore the function of the ECG machine. During this time lapse, 6.4% of the patients did not have a screening ECG done at the triage. Sustainability of such interventions has to be evaluated with regular reviews and feedback mechanisms are required to ensure that the gains made are not lost subsequently. Overall, several key factors have been identified as central to the improvement of ACS care delivery and this include a commitment to the goals which must be clearly defined, development of cohesive teams with empowered clinical leaders to champion the cause and develop a working environment that culture that encourages data feedback, continuous development and resilience to the challenges that definitely arise as changes to improve care are implemented (72)

ECG Features

A key finding in this study was that only 12.4% of the patients who obtained an ECG had a normal ECG. 87.6% had some abnormalities and of these abnormalities 25% were consistent with myocardial ischaemia. This subset of patients requires further evaluation with cardiac biomarkers to make a definitive diagnosis of myocardial infarction. In a prospective study done 8 years ago among diabetic patients presenting to Kenyatta National Hospital A&E department, it was found that 30% had an Acute coronary syndrome based on ECG findings and positive cardiac biomarkers(16) These findings also highlight the utility of rapid ECG acquisition for the diagnosis of other cardiac abnormalities, especially conduction abnormalities that may require rapid action to reduce cardiovascular morbidity and mortality.
6.0 CONCLUSION

The current screening and diagnosis of acute coronary syndromes is sub-optimal. The institution of an ECG triage tool and free ECG service can significantly bridge this gap. However, no single intervention is adequate to reach the desired goals.

7.0 RECOMMENDATIONS

1. Implementation of an ECG triage protocol with empowerment of the triage nursing staff to do the ECG prior to doctors’ evaluation.

2. ECG to be considered an emergency service administered for free or pre-payement for patients who meets a triage criteria of high probability of ACS.

3. Training on the implementation of ECG triage pathway for all relevant staff doctors, nurse, auxiliary staff

4. More studies are required to determine if this tool results in increased diagnosis, treatment and better patient outcomes

5. More studies to determine the cost effectiveness of free ECG service or indirect payment of the ECG service in the ER

6. Establishment of audit systems to determine continued utilization and impact on patient care

8.0 STUDY LIMITATIONS

The implementation of our protocol was a study directed activity and may not fully represent the timelines achievable once a chest pain protocol is implemented by the triage A&E staff.

There was no objective assessment of the clinicians knowledge attitude and practice that may inform the screening practices observed.

This was a single centre, non randomized study.
9.0 REFERENCES


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14. Shavadia J, Yonga G, Otieno H. A prospective review of acute coronary syndromes in an urban hospital in sub-Saharan Africa: cardiovascular topics. 2012 [cited 2015 Dec 4]; Available from: http://reference.sabinet.co.za/proxy/DocumentView/aHR0cDovL3JlZmVyZW5jZmVpbmV0Ln hsvL2RVy3VtZW50L0VKQzEyMjcyNA%3D%3D/a%3A1%3A7Bs%3 A6%3A%22source%22%3B%3A6%3A%22browse%22%3B%7D


Chest Pain at the Emergency Department, a Prospective Cohort Study. PloS One [Internet]. 2015 [cited 2016 Jan 7];10(1). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4295862/


1. INFORMATION SHEET

Introduction

I, Julia Mbithe Ndawa, am a postgraduate student at the University of Nairobi, currently doing a masters’ degree in Internal medicine. I am conducting my research project for which I request your participation. As you read this form you may ask any questions on what you do not understand.

Purpose of the study

I am carrying out a study on the care of patients with acute coronary syndromes to evaluate how they are screened and diagnosed and also to evaluate how to shorten the time it takes. The study is part of my university requirements but the results of the study will be used to offer recommendations which, if implemented, may lead to improved management and quality of life of patients with acute coronary syndromes.

Procedures to be followed in the study

Pre-intervention: Once you agree to participate in the study, you will have to answer questions of a personal nature as outlined in the study questionnaire, undergo a physical examination. We may call you or your next of kin to interview you concerning the investigations and tests you will receive while in the Accidents and Emergency unit.

*Post intervention: Once you agree to participate in the study, you will have to answer questions of a personal nature as outlined in the study questionnaire, undergo a physical examination and provide a blood sample. The results of your ECG together with the other information obtained above shall be used to for the study.
Confidentiality

All the information you provide will be handled in a confidential manner and will not be divulged to any other person without your consent. Your individual responses will be stored in a locked place under my control and will only be seen by my statistician and I.

Voluntariness of participation

Your participation in this research is voluntary and in the event that you refuse to participate in this study, your treatment will not be affected. If you choose to participate and not answer certain questions, you are free to do so. You are free to terminate the interview and withdraw from the study at any time. You are free to ask questions before signing the consent form.

Benefits

Your participation in the study and the tests will be used for your individual benefit. Information obtained will improve knowledge to health care givers at Kenyatta National hospital.

Risks

You may feel slight pain/ discomfort when the blood sample is drawn. There may be slight swelling at the site of the needle prick, but this will disappear on its own after a few days. The amount of blood that will be drawn will not affect your health.

Rights

You may choose to withdraw from the study at any time whatsoever with no consequences to your treatment.
2. PATIENT CONSENT FORM

I ……………………………………………… of ………………………….. hereby consent/decline to participate in this study, of which I have fully read and understood the explanation given to me. All my questions have been satisfactorily answered by the investigators.

Signed ……………………… Thumb Print ………………… Date ……………………….
Witness ………………………….. (PI/Assistant) Date ……………………….

CONTACTS

For further information, you may contact any of the following:

1. Dr. Julia Mbithe Ndawa (Principal investigator)
   University of Nairobi
   Department of Clinical Medicine and Therapeutics
   P.O Box 2515 00200 Nairobi
   Tel 0723 339 253

2. Professor A. N. Guantai,
   Chairman of Kenyatta National Hospital/University of Nairobi Ethics and Research Committee,
   P.O Box 20723, Nairobi.
   Tel 020-2726300, extension 44102.
2. FOMU YA MAELEZO YA UTAFITI

Utangulizi

Mimi, Julia Mbithe Ndawa, ni mwanafunzi katika chuo kikuu cha Nairobi. Ninatarajia kufanya uchunguzi wa ugonjwa wa moyo na ningependa wewe uhusike. Fomu hii ni ya maelezo yote utakayohitaji ukiamua kama utajiunga na utafiti huu. Unapoisoma na baada ya kusoma fomu hii, uko huru kuuliza maswali yoyote kama kuna sehemu hujaelewa vyema.

Je, utafiti huu unalenga kutambua nini?

Ninafanya utafiti kati ya wagonjwa wenyewe dalili za ugonjwa wa moyo ili kuchunguza wangapi kati yao huchunguzwa kikamilifu ili kudhibitisha shida yao na pia nitachunguza jinsi ya kufupisha muda unaotumika kudhibitisha hiyo shida ya moyo. Utafiti huu unalenga kutambua nini? Ninajifanya utafiti kati ya wagonjwa wenyewe dalili za ugonjwa wa moyo ili kuchunguza wangapi kati yao huchunguzwa kikamilifu ili kudhibitisha shida yao na pia nitachunguza jinsi ya kufupisha muda unaotumika kudhibitisha hiyo shida ya moyo. Utafiti huu unalenga kutambua nini?

Utaratibu wa utafiti:

Pre-intervention : Utakapokubali kujiunga na utafiti huu, utahitajika kujibu maswwali ya kibinafsi kama yalivyodekezwa katika karatasi ya maswali na utapimwa kimwili. Baadaye, tunaweza kukupigia simu wewe au jamaa yako ili tukuhoji kuhusu vipimo ulivyofanyiwa katika hospitali.

**Hatari na gharama inayohusika**


**Haki zako**


**Manufaa ya utafiti huu**

Kujiunga na utafiti huu na vipimo vya maabara vitatumika kwa manufaa yako. Matokeo ya utafiti yatasaidia wauguzi katika hospitali ya Kenyatta.
FOMU YA IDHINI

Mimi ........................................... kutoka ...........................................

Sahihi/Alama ya kidole gumba cha kushoto : .................................

Tarehe ..................................................

Shahidi.................................(mtafiti mkuu/msaidizi) Tarehe ............................................

MAWASILIANO

Ukiwa na maswali yoyote ya ziada, unaweza kuwasiliana na wafuatao:

1. Dkt. Julia Mbithe Ndawa (Principal investigator)

SLP 2515 00200 Nairobi.

Simu 0723 339 253

2. Prof. A.N. Guantai

Kamati ya Maadili ya Hospitali ya Kenyatta na Chuo kikuu cha Nairobi

SLP 20723 NAIROBI

Simu: 020 726300
DATA COLLECTION FORM

(PRE-INTERVENTION)

(POST INTERVENTION)

STUDY NO

AGE……………………………………..CAS/IP NO

SEX ……………………………………….TEL NO

RESIDENCE……………………ETHNICITY…………………COUNTY ………………….

ECG PRIORITIZATION TOOL

AGE >30 WITH CHEST PAIN, DIABETES OR HYPERTENSION → TRIAGE ECG

AGE >50 WITH DYSPNOEA ALTERED MENTAL STATUS UPPER LIMB PAIN SYNCOPE WEAKNESS → TRIAGE ECG

AGE >80 WITH ABDOMINAL PAIN NAUSEA VOMMITING → TRIAGE ECG

PROBABILITY FOR ACS (LOW)

(HIGH)

Exclusion: PSYCHIATRIC PATIENT

CONSENT
STUDY PROFORMA

DATE ..................................................DAY OF WEEK (WEEKEND) (WEEKDAY)

STUDY NO

DOOR TIME (24HR) □□□

PRESENTING SYMPTOMS

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<td>NAUSEA AND VOMITTING</td>
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RISK FACTOR PROFILE: History of

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PHYSICAL EXAMNINATION

BP ........................................ PULSE ...................... BMI ........................................

SHOCK ................................... HEART FAILURE .....................................
**ECG**
DONE (YES) (NO)
TIME OF ECG 24 HOUR

**ECG FINDINGS**
STEMI (YES) (NO)
N-STEMI (YES) (NO)
OTHERS (Arrhythmias) ....................................................
Conduction Abnormalities ..................................................
Other ..............................................................................

**TROPOinin**
DONE (YES) (NO)
LEVELS (NORMAL) (ELEVATED)

**ACS DIAGNOSIS**
YES STEMI
NON STEMI
UNSTABLE ANGINA
NO