CLINICAL PRESENTATION AND IN-HOSPITAL OUTCOME OF PATIENTS WITH MYOCARDIAL INFARCTION ADMITTED IN MULAGO HOSPITAL

BY

JOSEPHINE ACHAN MBChB (MAK)

SUPERVISORS

1. DR MONDO CHARLES KIIZZA MBChB, MMED, PhD
2. DR SEBATTA ELIAS MBChB, MMED
3. DR OKELLO EMMY MBChB, MMED

A DISSERTATION SUBMITTED TO THE SCHOOL OF GRADUATE STUDIES IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF MEDICINE IN INTERNAL MEDICINE OF MAKERERE UNIVERSITY

MAY 2014
DECLARATION

I hereby declare that the work in this dissertation is a result of my own study and has not been published and/or submitted elsewhere for any award.

Signed: …………………………... Date: ……………………………

Dr. Achan Josephine

This dissertation has been submitted for the examination with the approval of the following supervisors:

1. DR MONDO CHARLES KIIZZA MBChB, MMED, PhD

   Signed: ………………………... Date: ………………………

2. DR SEBATTA ELIAS MBChB, MMED

   Signed: ………………………... Date: ………………………

3. DR OKELLO EMMY MBChB, MMED

   Signed: ………………………... Date: ………………………
DEDICATION

This book is dedicated to my mother Mrs. Agara-Oyo Christine Clare for her tireless endeavors and advice. To my late father Mr. Agara-Oyo Raymond Alex who would be proud of this success. Not to forget my sisters Eng Priscilla Clare Ayo and Don Elizabeth Lamunu, my brother Peter Okidi for their unwavering support and encouragement. Also dedicate this work to my wonderful nephews, Melvin and Raymond.
ACKNOWLEDGEMENT

I would like to acknowledge the contribution made by many people in an attempt to make this work a success.

In particular I would like to thank Dr Mondo Charles K who helped me come up with this topic and encouraged me.

My sincere thanks to Dr Okello Emmy and Dr Sebatta Elias who worked tirelessly in the development of this dissertation and they have continued to mentor me, encourage me and offer support until completion of this dissertation.

I would wish to thank Dr Kaddu Mark and Dr Musoke Charles who took time to read my proposal and advised me accordingly.

I also thank Simon Mugambe of Department of Medicine who helped me to translate the consent form into luganda and Dr Kecha D D for being a wonderful research assistant.

Above all, I thank my mother, my sisters and brother for the support and encouragement they rendered to me. Not to forget I thank God for giving me strength, courage and seeing me through during this hectic period.

Research reported in this dissertation was supported by the Fogarty International Center, the National Heart Lung and Blood Institute, and the Common Fund of the National Institutes of Health under Award Number 5R24 TW008861. The content is solely the responsibility of the Authors and does not necessarily represent the official views of the National Institutes of Health.
# TABLE OF CONTENTS

DECLARATION ............................................................................................................. i
DEDICATION ............................................................................................................... II
ACKNOWLEDGEMENT .............................................................................................. III
LIST OF TABLE ......................................................................................................... VI
LIST OF FIGURE ....................................................................................................... VI
ABBREVIATIONS ....................................................................................................... VII

OPERATIONAL DEFINITION ...................................................................................... viii

ABSTRACT ................................................................................................................ ix

CHAPTER ONE .......................................................................................................... 1
  1.0 INTRODUCTION ................................................................................................ 1
  1.1.0 Problem Statement ....................................................................................... 1
  1.2.0 Justification .................................................................................................. 2
  1.3.0 Research Questions ...................................................................................... 2
  1.4.0 Objectives .................................................................................................... 2
  1.4.1 General Objectives ...................................................................................... 2
  1.4.2 Specific Objectives ...................................................................................... 2

CHAPTER TWO ........................................................................................................ 5
  2.0 LITERATURE REVIEW ...................................................................................... 5
  2.1.0 Definition and Pathogenesis of Myocardial Infarction ................................. 5
  2.2.0 Burden of Myocardial Infarction .................................................................. 5
  2.3.0 Risk factors associated with Myocardial Infarction .................................... 6
  2.4.0 Clinical Presentations of Myocardial Infarction ......................................... 7
  2.6.0 In-hospital outcome of Myocardial Infarction .......................................... 9

CHAPTER THREE .................................................................................................... 12
  3.0 METHODS ........................................................................................................ 12
  3.1.0 Study Design ................................................................................................ 12
  3.2.0 Study Setting ................................................................................................ 12
  3.3.0 Study Population ........................................................................................ 12
  3.3.1 Target population ....................................................................................... 12
  3.3.2 Accessible population ................................................................................ 13
  3.3.3 Study population ....................................................................................... 13
  3.4.0 Study period ................................................................................................ 13
LIST OF TABLE
Table 1: socio demographic features of the patients............................Error! Bookmark not defined.
Table 2: Clinical presentation of patients admitted with myocardial infarction. Error! Bookmark not defined.
Table 3: Laboratory findings of the participants.............................Error! Bookmark not defined.
Table 4a: Echocardiograph findings of the participants .................Error! Bookmark not defined.
Table 4b: Echocardiograph findings of the participants .................Error! Bookmark not defined.
Table 3: Risk factors associated with myocardial infarction .........Error! Bookmark not defined.
Table 5: Bivariate analysis for in-hospital outcome ......................Error! Bookmark not defined.

LIST OF FIGURE
Figure 1 patient flow chart ..............................................................Error! Bookmark not defined.
Figure 5: showing the in hospital outcome by subtypes of myocardial infarction ..............Error! Bookmark not defined.
Figure 3 shows the proportion of associated symptoms of myocardial infarction .............Error! Bookmark not defined.
Figure 4: showing overall in-hospital outcomes among patients with myocardial infarction..................................................................................................................Error! Bookmark not defined.
Figure 2 showing activity at onset of symptoms .............................Error! Bookmark not defined.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACS</td>
<td>Acute Coronary Syndrome</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiography</td>
</tr>
<tr>
<td>ECHO</td>
<td>Echocardiography</td>
</tr>
<tr>
<td>HDL</td>
<td>High Density lipoprotein</td>
</tr>
<tr>
<td>LDL</td>
<td>Low Density Lipoprotein</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>Non-ST elevation myocardial infarction</td>
</tr>
<tr>
<td>OR</td>
<td>Odds Ratio</td>
</tr>
<tr>
<td>PI</td>
<td>Principle investigator</td>
</tr>
<tr>
<td>STEMI</td>
<td>ST segment elevation myocardial infarction</td>
</tr>
<tr>
<td>UA</td>
<td>Unstable Angina</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>
OPERATIONAL DEFINITION

Myocardial infarction (MI) was diagnosed based on the American College of Cardiology and the European Society of Cardiology definition. It defined MI as a typical rise in cardiac troponin T or I, or CKMB with at least one of the following: ischemic symptoms, development of pathological Q waves on the ECG, ischemic ECG changes (ST depression or elevation) or coronary artery intervention.

ST Segment myocardial infarction (STEMI) was diagnosed if ST segment elevation at the J point of ≥ 1mm occurred in two contiguous limb or precordial leads or a new left bundle branch block was found on the electrocardiograph (ECG) with biochemical evidence of myocyte necrosis.

Non ST elevation myocardial infarction (NSTEMI) was diagnosed in patients with biochemical indication of myocyte necrosis without new ST segment elevation or left bundle branch (LBBB) on electrocardiograph (ECG).

In-hospital outcome was defined as the proportion of patients alive and complications (heart failure, thrombus formation, shock, pulmonary edema and death).

Coronary heart disease is the narrowing of coronary artery due to atherosclerosis. It presents as stable angina, unstable angina, myocardial infarction, heart failure, cardiac arrhythmias and sudden cardiac death. This term can be used inter changeable with ischemic heart disease.
ABSTRACT

Background

Myocardial infarction is one of the leading causes of mortality worldwide with decreasing incidence in developed countries and increasing incidences in developing countries, Uganda inclusive. This increasing trend has been attributed to urbanization and changing life styles in developing countries. There is high burden of risk factors like hypertension and diabetes mellitus in our setting.

Objectives

To describe the clinical presentation and in-hospital outcome among patients admitted with myocardial infarction in Mulago hospital.

Methods

This was a prospective cohort study that was conducted in Mulago Hospital complex and Uganda Heart Institute. 54 subjects were recruited during the eight months study period. Data on clinical presentations, associated risk factors, laboratory and imaging findings, and complications were collected through standardized questionnaire. Blood samples were obtained for laboratory investigations. Participants were followed for minimum of two weeks and maximum of one month.

Results

A total of 54 patients were recruited, 29/54 (63%) had ST segment elevation myocardial infarction (STEMI) and 17/54(37%) had non-ST segment elevation myocardial infarction. Chest pain (66.7%) was the common presentation. Most patients in this study came to the hospital more than 72 hours with median time of presentation to the hospital from onset of symptoms 93.5 hours (SD 57.09, OR=1.002 95%CI 0.9-1.0). The mean age for the study participants was 58.7 (SD =+/10) with more males 38/54 (70.4%) than females 16/54(29.6%). Common associated symptoms were breathlessness 39/54 (54.7%), palpitations 21/54 (38.9%). Symptoms occurred at rest, with exercise and emotional stress. Only 7/59 (13%) of the participants had low systolic and 11/54 (20.4%) low diastolic blood pressure. 18/54 (33.3%) had high systolic and
20/54 (37%) diastolic pressure at admission. 19/54 (35.2%) of participants had significant pulmonary rales at admission. 34/54 (63%) had New York Heart Association class I and 40/54 (74.1%) were in Killip class I.

Risk factors include past medical history of hypertension 35/54 (OR=1.53, 95% CI=0.48-4.90), diabetes mellitus (OR=1.52, 95% CI=0.46-4.95), dyslipidaemia 7/54 (OR=1.73, 955CI=0.29-10.10), high LDL Cholesterol were higher risk for myocardial infarction. Low HDL (OR=1.9, 95% CI=0.55-6.58) confers higher risk for myocardial infarction compared to normal and high HDL cholesterol levels. Also similar patterns are seen in family history as positive for hypertension (59.3%) (OR=1.1, 955CI=0.35-3.88) and diabetes mellitus (37%).

50.9% of male and 92.9% of female participants had abdominal circumference greater than 102cm and 88cm respectively. Higher body mass index of 24.5-29.5 (OR=2.0, 95% CI 0.3-13.1) and 29.5-39 (OR=2.2, 95% CI=0.38-13.5) though the p value is not statistically significant was also a risk for myocardial infarction.

Positive history of current or former h/o alcohol consumption constituted more than half of the participants (29/54). In contrast only approximately 13/54 (24.1%) were current or previous smokers.

Over all 10/54 (24.1%) developed shock, 10/54 (18.5%) had pulmonary oedema and congestive heart failure, 6/54 (11.1%) developed arrhythmia, 6/54 (11.1%) died in the hospital, and 2/54 (3.7%) had ventricular wall aneurysm formation. 1/54 (1.9%) had stroke, re infarction and thrombus formation. No patient developed pericarditis and LV dysfunction.

Conclusion

Majority of patients admitted with myocardial infarction had STEMI and present with chest pain. Most patients are males. Hypertension, Diabetes Mellitus and dyslipidaemia were high risk factors. There was delayed time to presentation to hospital from the time of onset of symptoms. Almost half of the patients with STEMI developed pulmonary edema, shock, congestive heart failure and arrhythmia.


**Recommendations**

There is need for increased sensitization and awareness about myocardial infarction, it’s presenting symptoms and risk factors among the population at risk and encourage patients to come early to the hospital. In particular aggressive risk reduction and treatment for hypertension diabetes mellitus and dyslipidaemia should be emphasized.

Due to high risk of developing shock, pulmonary edema and congestive heart failure during admission, early intervention should be strongly emphasized.

There is need for further studies with larger sample size that can answer more questions about acute myocardial infarction.
CHAPTER ONE

1.0 INTRODUCTION

Myocardial infarction and ischemic heart disease is on the rise in Africa. Currently it’s the second leading cause of death in Africa preceded by HIV and accounts for more 60% of the mortality in developing countries. The rapid western cultural adaptation (mainly smoking cigarettes, drinking alcohol, high intake of fast foods, sedentary life style), coupled with increasing prevalence of diabetes mellitus, hypertension, and obesity have been noted as the key risk factors in the increasing incidence of CHD in developing countries[1, 2].

The INTERHEART Africa study identified five major risk factors in a study of 12 African countries. These included hypertension, diabetes mellitus, abdominal obesity, smoking and elevated apolipoproteinB100/apolipoproteinA1 ratio[3].

In Kenya, perspective study showed majority of patients presented late, greater than 12 hours and this had an impact on their outcome. Further it was noted most patients have chest pain but 20% did not have chest pain. In-hospital outcome included death, heart failure, and arrhythmias. [4]

Cardiac enzymes like, creatinine kinase-MB (CK-MB) and troponin I or T serum level measurements correlates with the presence and extent of myocardial infarction. Studies have shown the higher the levels of these cardiac enzymes, the greater the extent of infarction. These tests can be done even in low income countries, Uganda inclusive.

In Uganda, 7% of the outpatients seen in 2009 were due to ischemic heart disease. Studies by kayima et al (2010) further noted increasing trend of IHD accounting for 10.1% admissions in the cardiology ward of Mulago Hospital[5]. There is however limited current data on clinical pattern and in hospital outcome of myocardial infarction and other coronary heart disease at large in Uganda. This study described the clinical presentation and in-hospital outcome of 54 patients admitted and treated for acute myocardial infarction in Mulago Hospital.

1.1.0 Problem Statement

Several studies have been done in developed countries about the clinical presentations, risk factors and outcome. High prevalence of hypertension, diabetes mellitus, dyslipidaemia, age and abdominal obesity are among the risk factors for MI. Most patients develop heart failure,
arrhythmias and death. However, not many of these have been done in resource limited countries. This situation might be worse or similar in sub-Saharan African countries, let alone in Uganda. It is not known if the clinical presentation, risk factors and outcome is the same as indigenous Ugandan population and thus no national guidelines for early intervention in Uganda.

1.2.0 Justification

With the increasing incidence of ischaemic heart disease in developing countries, little is still known about the extent of myocardial infarction in Uganda. This study therefore determined the clinical, laboratory and imaging presentations and associated risk factors of patients with myocardial infarction admitted in Mulago Hospital. It also determined the in-hospital outcome and factors associated with mortality among patients with myocardial infarction. This help stratify patients at admission to which clinical and laboratory presentations and risk factors are associated with adverse outcome. This information will guide clinicians to formulate guidelines for the management of myocardial infarction. Data generated from this study will also influence public policy on myocardial infarction and cardiovascular disease at large.

1.3.0 Research Questions

What are the clinical presentations and in-hospital outcome of patients with myocardial infarction admitted in Mulago Hospital?

1.4.0 Objectives

1.4.1 General Objectives

To determine the clinical presentations and in-hospital outcome of patients with myocardial infarction admitted in Mulago Hospital.

1.4.2 Specific Objectives

Primary secondary

I. To describe the clinical presentations of patients with myocardial infarction admitted in Mulago Hospital.

II. To determine the proportion of known risk factors for myocardial infarction among patients admitted in Mulago Hospital.
III. To determine the in-hospital outcome of patients admitted with myocardial infarction in Mulago hospital.

Secondary Objectives

I. To describe the factors associated with in-hospital outcome among patients admitted with myocardial infarction in Mulago Hospital.
CONCEPTUAL FRAMEWORK

MEDICAL FACTORS
- Hypertension
- Diabetes mellitus
- Dyslipidaemia

LIFE STYLE/SOCIAL FACTORS
- Smoking
- Sedentary life style
- Age
- Obesity

ATHEROSCLEROSIS

MEDICAL FACTORS

MYOCARDIAL INFARCTION

Heart Failure
- LV Dysfunction
- Arrhythmias
- Post MI Reinfarction

MECHANICAL COMPLICATIONS
- Ventricular rupture
- VSD
- Papillary muscle rupture

Stroke

Death

Family history of coronary artery disease
CHAPTER TWO

2.0 LITERATURE REVIEW

2.1.0 Definition and Pathogenesis of Myocardial Infarction

Myocardial infarction is cardiac muscle death that occurs as a result of poor blood supply. Common risk factors like age, male sex, hypertension, diabetes mellitus and dyslipidaemia work either independently or dependently to cause atheroma formation in the coronary arteries. An atheroma is lipid laden plaque that can remain stable causing no symptoms. However this plaque can rupture with thrombus formation which can occlude the coronary vessels. Rupture tends to occur at sites of maximal mechanical stress and may be triggered by a surge in blood pressure like during exercise heavy meal and emotional stress (14).

Persistent vessel occlusions cause ischemia with eventual tissue damage to the myocardium due to lack of adequate oxygen and sufficient metabolite delivery to the myocardium. Death of cardiac muscle cells slowly propagates to involve the whole wall thickness and leads to release of lactate dehydrogenase (LDH), myoglobin, cardiac specific cardiac markers like creatinine kinase MB and troponin T or I into the blood which act as markers of cardiac cell death. Death of conduction tissue commonly seen in right ventricular wall infarct can lead to conduction defect. Also oxygen free radicals are released from damaged cells and coupled with vagal efferent discharge lead to bradycardia and various degrees of arrhythmias. (14)

The different types is classified according to the electrocardiographic changes which include ST segment elevation in two contiguous limb or precordial or presence of new left bundle branch block to give what is called ST segment elevation myocardial infarction. Or depression of the ST segment in two contiguous limb or precordial leads which is called Non-ST segment myocardial infarction

2.2.0 Burden of Myocardial Infarction

Globally, myocardial infarction and ischemic heart disease is one of the leading causes of death with approximately 3.8 million men and 3.4 million women who die each year from IHD[1]. More than 60% of these deaths occur in developing countries[1]. In developed countries, there
has been a decreasing mortality rates due to IHD with approximately 23-47% decline attributed to new modality of treatment[6].

However, in developing countries mortality due to ischemic heart disease is projected to increase in 2020 by 120% in women and 137% in men[1]. In Africa, cardiovascular disease is the second leading cause of death preceded by HIV contributing about 10% mortality rate[7]. In South Africa mortality rate is at 40% among the adult populations[8].

In East Africa, a prospective study carried out in Kenya, in-hospital mortality was 10%, 20% patients have atypical presentations and most patients present more 12hours from time of onset of symptoms[4].

In Uganda, myocardial infarction and coronary heart disease(CHD) was previously deemed rare as shown by Professor Hutt who found at autopsy CHD contributed less than 1% of cardiac death[9].

Currently there is an increasing incidence in ischemic heart disease in Uganda (10). Mulago National Referral Hospital registered an increase in the cases of coronary heart disease from 1.8% outpatients in 2002 to 7% in 2009[10]. Studies by kayima et al (2010) further noted increasing trend of IHD accounting for 10.1% admissions in the cardiology ward of Mulago Hospital[5]. Anecdotal data from Uganda heart institute records (2011) indicated myocardial infarction accounted for 4.4% for the total admission.

2.3.0 Risk factors associated with Myocardial Infarction

There are various modifiable (history of smoking cigarette, diabetes mellitus, hypertension, dyslipidaemia, obesity, sedentary life style) and non-modifiable risk factors (sex, age, family history of premature coronary artery disease and men-baldness pattern) associated with myocardial infarction.

The INTERHEART Africa study identified five modifiable risk factors that contributed to 89.2% of the risk for first time MI. These risk factors include history of smoking tobacco, abdominal obesity as measured by waist-to-hip ratio, elevated apolipoproteinB100/apolipoprotein A1 ratio, self reported hypertension and diabetes mellitus[11]. Hypertension and diabetes mellitus was considered the most important due to the higher population attributable risk they contributed[11].
Hypertension was associated with a higher acute MI risk at OR 3.44: 95%CI 2.64 to 4.48, as well as abdominal obesity (OR 2.99, 95%CI 2.20 to 4.07)[11]

A study of 405 black African participants and 510 colored African participants in 52 countries found elevated Apolipoprotein B100/Apolipoprotein A1 ratio as the most important lipid marker for increased risk for acute MI[12]. Norman et al found high cholesterol level accounted for 59% and 29% of IHD and ischaemic stroke respectively in adults age 30 and above.

Higher socio-economic status (high levels of education and income) among black Africans was associated with higher risk of acute MI compared to other Africans with high education among women contributing to the risk[11].

In Africa morbidity and mortality due to MI was higher among males compared to females.

In the developed countries, MI is seen in older patients compared to Africans who are young with mean age of 54.3 years about 3.8 years younger.

A meta-analysis has shown that calcium supplementation without co administration of Vitamin D leads to increased risk of myocardial infarction[13]. Low Vitamin D has been demonstrated to be associated with cardiovascular events.

Other risk factors include elevated homocysteine levels, vasculitis, and coronary vasospasm.

Modifiable risk factors like tobacco use still remains one of the risk for MI. A study done in Dakar showed 40% of patients admitted with MI in a hospital were smokers.

All the above mentioned risk factors work either independently or dependently to cause atheroma formation in the coronary arteries

**2.4.0 Clinical Presentations of Myocardial Infarction**

In myocardial infarction chest pain is the cardinal symptom[14]. It’s a retrosternal chest pain/discomfort that last more than 30 minutes not relieved by rest. It radiates to the neck, jaw and left arm. However a section of individuals especially elderly and diabetes have chest heaviness or painless presentations. A study done in Kenya showed for every one in five (20%) patients, the presenting symptom was other than chest pain[4]. These include: nausea, vomiting,
epigastric pain, dyspnea or syncope. Nausea and vomiting have been documented due to vagal stimulation and is particularly common in patients with inferior wall MI, [4, 14].

For most patients breathlessness is a common finding. It has been noted 10% of patients with MI develop cardiogenic shock which might be the presenting feature and 2/3 of these are expected to die within a few weeks [15-17].

The use of New York Heart Classification of heart failure and Killip classification to measure the severity of heart failure in patients with myocardial infarction helps to risk stratify the patients and improve management of patients.

The physical findings include raised heart rate and sweating due to sympathetic activation, signs of impaired myocardial function (low blood pressure, cold extremities, narrow pulse pressure, raised jugular venous pressure, third heart sound, quiet first heart sound, and diffuse apical impulse and lung crepitation)[14]. Fevers occur due to tissue damage. Others present with complications like pericarditis, mitral regurgitation, and stroke[14].

Laboratory findings include raised cardio specific enzyme CK-MB and the cardio specific proteins, troponin T and I. This rise may depend on the extent and duration of infarct[14]. Cardiac troponins, especially troponin I, are preferred biomarker and its elevated level correlates with an increased risk of death. It is useful in the diagnosis, risk stratification and determination of prognosis. The greater the rise in cardiac troponins the greater the risk of adverse outcome[18].

Other biomarkers of MI include LDH, hypersensitivity C reactive protein and myoglobin. However they are not specific to myocardial injury though is a sensitive marker. More specific marker includes microRNAs. Studies done in both humans and animals found up-regulated miR-1,-133a,-133b, and -499-5p plasma levels only in STEMI patients[19].

In transmural (full thickness) lesions the electrocardiograph shows ST segment elevation and later development of pathological Q wave which defines ST segment elevation type of MI. In contrast, partial thickness or subendocardial (partial thickness) infarction causes ST/T wave changes without Q waves or prominent ST elevation. This is called the non-Q wave or non-ST segment elevation MI[14].
ST segment elevation myocardial infarction constitute ST elevation at the J point of ≥ 1mm in two contiguous limb leads or precordial leads or new left bundle branch block (LBBB) as seen on electrocardiogram with biochemical evidence of myocyte necrosis. In non-ST segment elevation myocardial infarction, patients have biochemical indication of myocyte necrosis without new ST segment elevation or LBBB on ECG [20]. A prospective review study on ACS in Kenya found most of the patients had STEMI (62%) and less NSTEMI (44%) [4]. The site of infarction of those with STEMI by ECG was highest in the anterior wall at 41%, followed by inferior wall infarct 37%, anterolateral 10%, and at 6% were lateral and septal infarcts [4].

Coronary angiogram provides better analysis of the extent of coronary artery involvement. Among patients studied in a prospective review in Kenya, 40% of those with STEMI had left anterior descending artery occlusion, followed by 33% in the right coronary artery and 7% in the left circumflex artery [4]. About 56% of the NSTEMI/UA subgroup had double or triple vessel disease [4].

2.6.0 In-hospital outcome of Myocardial Infarction

Arrhythmias

Nearly all patients with acute MI have some form of arrhythmia ranging from sinus bradycardia, sinus tachycardia, atrioventricular block to ventricular fibrillation (5-10%). Prompt pre-hospital resuscitation and defibrillation for VT has a good prognosis [14]. VT is one of the causes of sudden death. In the GISSI-2 trial ventricular arrhythmias were present in 64.1% of the patients studied with a total of 256 deaths.

Left ventricular dysfunction

As sequelae of acute MI, the damage undergoes remodeling with subsequent dilatation of the ventricles and about 10% form ventricular aneurysm. This greatly reduces the function of the ventricles and patients present with heart failure over time [14]. Anecdotal data at UHI indicate most patients have heart failure followed by arrhythmias, mitral regurgitation and death as the major complications due to MI.
Mortality

Studies have shown varying rates of mortality among patients with STEMI and NSTEMI. There is higher mortality rate among those with NSTEMI at 6-month (8.8% versus 5.0%) and 1-year (11.1% versus 7.0%) than unstable angina [21-23].

In the GRACE study 8 independent factors accounted for 89.9% of the prognostic information. Cardiac arrest during presentation accounted for the highest rate of mortality at odds ratio of 4.3. Other factors included age (OR 1.7 per 10 years), Killip class (OR 2.0 per class), systolic pressure (OR 1.4 per 20mmHg decrease), STEMI (OR 2.4), serum creatinine level (OR 1.2 per 1mg/dl micromol/l increase), positive initial cardiac enzyme findings (OR 1.6), and heart rate (OR 1.3 per 30beats/minute increase)[24].

Mortality among MI patients is highest in the first 30 days of onset. Factors associated with high mortality include cardiogenic shock, age, hypertension, heart failure, DM, angina and in-hospital reinfarction[25]. Thrombolytic therapy has been noted to reduce risk of dying[25].

Cardiogenic shock complicates about 7-10% patients with acute MI and when left untreated it accounts for 80% of mortality [17, 26].

The TRACE study done in Denmark found 30-day mortality of 62% with cardiogenic shock compared to 9% among non-shock patients[25]. It also noted that cardiogenic shock carried the single most important risk of mortality with risk ratio of 2.6 and CI 2.3-3.0[25].

In hospital mortality of 51% was found in a retrospective analysis of a cohort of 113 patients undergoing coronary angiography and attempted PCI for cardiogenic shock complicating MI[27].

In patients with MI admission glucose has been shown to be more strongly associated with mortality than antecedent diagnosis of diabetes. In a retrospective cohort study of UK population with STEMI and NSTEMI, there was a 5-7% increased risk of post discharge mortality per mmol/l increase in glucose[28].

Other factors strongly contributing to mortality are hypertension, age, history of smoking. However reperfusion therapy reduced mortality[25].
In Africa a prospective study of ACS done in Aga Khan University Hospital, Kenya found in-hospital mortality rates were 9.7% for those with STEMI and 6.7% among NSTEMI/unstable angina with total of 10% mortality rate for both [4].

**Stroke** can occur as a result of thrombolytic therapy with subsequent intracranial bleeding[4] or due to brain infarct.

**Mechanical complications** may include rupture of the papillary muscles, rupture of the ventricular septum and ventricular wall rupture causing tamponade and death if not intervened early[14].

**Post MI re-infarction or ischemia**

According to GUSTO-IIB trial the rates of reinfarction at 6 months was higher in the NSTEMI than those with unstable angina (9.8% versus 6.2%)[21].

**Stroke**

Ischaemic stroke is due to embolism of thrombi to the intracranial arteries. Common cardiac cause is the mural thrombus. Intracranial bleeding can occur following use of fibrinolytic therapy. More intensive fibrinolytic regimens using recombinant tissue plasminogen (rtPA) and heparin pose greater risk of intracranial bleeding as compared to streptokinase and aspirin[29].
CHAPTER THREE

3.0 METHODS

3.1.0 Study Design
This was a prospective cohort study done within a period of 8 months.

3.2.0 Study Setting
The Study was carried out in Mulago Hospital complex including the 4C cardiology in patient ward and Uganda Heart Institute. Mulago Hospital is located in Kampala the capital city of Uganda. It is Uganda`s largest national referral, teaching and research healthcare facility with a bed capacity of over 5000 operating on a 24 hour basis. It provides specialist services to all patients referred in from all parts of Uganda and neighboring countries. Patients were enrolled from Uganda Heart Institute and 4C cardiology. Patients come from all over the country either as formal referral or self referral and are admitted on a 24 hour service through casualty to either Uganda heart institute (UHI) or to the general medical wards (4C Cardiology ward) as general patients.

Uganda Heart Institute is an autonomous institute with highly equipped state of the art machines that include cardiac catheterization laboratory, echocardiography laboratory, and well equipped chemistry laboratory. It has highly qualified personnel including interventional cardiologists, adult cardiologists, pediatric cardiologists, cardiothoracic surgeons, medical officers, trained nurses and other personnel.

4C cardiology is a ward in the fourth floor of Mulago hospital allocated for patients admitted with cardiology related conditions. It also operates on 24 hours basis, run by cardiologists, resident students on rotation, intern doctors, nurses and other allied health workers.

3.3.0 Study Population

3.3.1 Target population
All adult patients with myocardial infarction admitted in Mulago hospital
3.3.2 Accessible population
All adult patients with myocardial infarction admitted in 4C cardiology and Uganda heart institute.

3.3.3 Study population
All adult patients with myocardial infarction admitted in 4C cardiology and Uganda Heart Institute who met the eligibility criteria.

3.4.0 Study period
The study was carried out for 8 months during the year 2013/2014

3.5.0 Eligibility Criteria

3.5.1 Inclusion criteria
1. Individuals of age ≥18 years

2. All patients with confirmed diagnosis of myocardial infarction (positive cardiac enzymes and electrocardiographic findings) admitted to the ward 4C cardiology and UHI during the study period.

3. Provide informed consent.

3.5.2 Exclusion criteria
No subjects were excluded.

3.6.0 Subject Recruitment and Sample Size Estimation

3.6.1 Sampling and recruitment
Subjects were recruited consecutively on ward 4C cardiology and UHI during the study period.

3.6.2 Sample size estimation
Using the formula for studies of proportions $n = Z^2 p(1-p)/e^2$, and prevalence of average outcome of 7.7% according to Shavadia J et al., 2012, the estimated sample size at 95% confidence interval was
\[N = 1.96^2 \times 0.077(1-0.077)\]

\[0.05^2\]

\[N = 109\]

Assuming 10% non response \(n=110\)

### 3.7.0 Study Variables

#### 3.7.1 Independent variables

Clinical history: Age (years), occupation, history of smoking, alcohol intake, physical activity, history of hypertension, diabetes, drug history and family history of coronary heart disease.

Physical examination: abdominal obesity (waist circumference (cm)), BMI (measure weight and height), blood pressure (mmHg), pulse rate (beats/min), pulse rhythm, heart sounds, breath sounds and liver size.

Laboratory measurements: serum cardiac enzymes (troponin I and CK-MB), serum creatinine (mg)/GFR, serum urea (mg), Random Blood Sugar (mmol/l), HBA1C and fasting lipid profiles (serum total cholesterol (mmol/l), serum HDL (mmol/l), serum LDL (mmol/l), serum triglycerides (mmol/l))

Imaging: Electrocardiograph, echocardiograph

### 3.8.0 Data Management and Analysis

#### 3.8.1 Data Collection Tool

A pre-tested standardized questionnaire was used as a data collection tool.

Other data source documents included: Clinical form; Laboratory report forms to capture information on cardiac enzymes (troponin I and CKMB), fasting lipid profile, random blood glucose, HBA1C, RFTs and electrolytes; ECHO report form capturing information on structural and functional heart abnormalities and ECG report form capturing information on diagnostic criteria for myocardial infarction. A research assistants at the level of medical officers was
trained according to protocol to conduct data collection. The principle investigator (PI) supervised daily data collection.

3.8.2 Data Collection Procedures

Patients who met the inclusion criteria were requested to participate in the study through a written informed consent. Subjects were recruited consecutively on 4C cardiology ward and UHI. Interviews were conducted by the PI and/or the research assistants using a pre-designed questionnaire. Questions were numbered, and semi-structured responses pre-coded. The questionnaire assessed risk factors such as age, history of smoking and drinking alcohol, personal history of diabetes, history of hypertension, and history of dyslipidaemia. Clinical history to focus on the presentation of myocardial infarction: the onset of chest pain, the nature of pain, the site of chest pain, any pain radiation, associated relieving and aggravating factors.

Waist circumference was measured with a flexible non-stretchable tape (Seca 200) following the WHO STEPS protocol. Weight was measured, corrected to 1 decimal point and Height was also measured (to the nearest cm). BMI was then calculated for all study subjects.

Pulse rate (bpm) was assessed by palpating the radial pulse for 60 seconds, after at least 5 minutes of rest. Pulse rhythm noted as regular or irregular. For an irregular pulse, the rate was be determined by auscultation over the precordium and an ECG done to confirm any arrhythmia. Blood pressure was measured with a standard mercury sphygmomanometer, as per the JNC VII criteria.

Venous blood samples (8ml) were drawn aseptically. Random blood sugar, HBA1C, fasting lipid profile, serum creatinine and urea, serum electrolytes were measured in the clinical chemistry laboratory.

A 12 lead rest electrocardiogram (ECG) was performed by an experienced technician and interpreted by a senior cardiologist.

An Echocardiography (ECHO) done by a senior cardiologist assisted by the principle investigator to determine structural changes and left ventricular ejection fraction of the heart.

Follow up

Patients were followed up at interval of every two days until discharge to document complications the patient developed. Minimum length of stay in the hospital expected to be
approximately one week at UHI and two weeks at 4C cardiology ward. For participants who were hospitalized for more than two weeks, the maximum period of follow up in the hospital was done until one month from the date of admission.

**PATIENT FLOW CHART**

- **Confirmed Myocardial Infarction using cardiac enzymes and ECG**
  - Get informed consent
    - Interview with questionnaire
      - Physical exam, ECG, ECHO, RFT, LFT, lipid profile, RBS
        - Follow up for in-hospital outcome
  - Decline consent
3.8.3 Data extraction
Data was captured using EpiData entry version 3.1. Data was also cleaned for missing entries, duplicate entries and illogical entries.

3.9.0 Data analysis plan

General analysis
Data entered into EPI-DATA (version 3.1) was then exported to STATA version 12 for analysis. Continuous variables summarized in mean (standard deviation), median (inter quartile range) and presented in histograms. Categorical data summarized using frequency and percentages and results presented in frequency tables, pie and bar charts.

Objective 1
Myocardial Infarction presentation was described among different demographic, clinical, laboratory, electrocardiographic and echocardiography characteristics.

Objective 2
The known risk factors were analyzed as a proportion and significant odds ratio and p values were obtained.

Objective 3
In hospital outcome; the proportion of patient with identified outcome was described as number of patient with a complication out of the total number of patients in the study. Significant odds ratios were obtained.

Secondary objective
No factors associated with the different outcomes were analyzed.

3.10.0 Quality Control
The questionnaire was standardized and pre-tested. A research assistant was trained on data collection. Data was cleaned every week to ensure accuracy and consistence. Before the study commenced the instruments were pre tested, standardised and piloted. Echocardiograms were performed, recorded and interpreted by an experienced cardiologist. The laboratory procedures
were carried out by a competent and experienced laboratory technologist. Blood parameter results were recorded in standard reporting forms by the laboratory technologist.

3.11.0 Ethical Considerations
Institutional approval for the study was obtained from the Department of Medicine, Makerere University College of health Sciences School of Medicine Research and Ethic committee and the Mulago hospital ethics committee.

Informed consent for all the patients was obtained. Patients were identified through study numbers and not by their names to ensure confidentiality.

3.12.0 Study Limitations
The standard of care in 4C cardiology ward was not the same as Uganda Heart Institute. This thus affected identification of complications. This study only recruited participants who come to Mulago Hospital.

3.13.0 Dissemination of Results
The results of the study will be availed and Published in indexed peer reviewed journals, the Department of Medicine library and book bank, the Uganda Heart Institute Library, Albert Cook Library, School of Graduate studies Makerere University, the Ministry of Health, Non communicable diseases sector.
CHAPTER FOUR

4.0 RESULTS

4.1 Patient profile during the study

Between August 2013 and April 2014, 60 patients were approached and screened for enrolment into the study. 6 patients did not qualify due to other causes of chest pain. Therefore, only 54 patients qualified and were enrolled. They were then followed up from day of admission to date of discharge with minimum duration of two weeks and maximum of one month. Data from 54 patients were analysed. During hospital stay, 10/54 (24.1%) of the patients developed shock, followed by 10/54 (18.5%) who had pulmonary oedema and congestive heart failure. 6/54 (11.1%) developed arrhythmia, 11.1% died in the hospital, and 2/54 (3.7%) had ventricular wall aneurysm formation. Only 1/54 (1.9%) had stroke, re infarction and thrombus formation. No patient developed pericarditis and left ventricular dysfunction. Other complications that contributed about 1.9% included acute renal failure, sepsis, hyperkalemia and hyponatremia.

The patient flow chart during recruitment is as shown below
Figure 1 patient flow chart

Expected N=110 patients

Only 60 patients were enrolled over 8 months period

6 patients were not eligible

54 patients were analysed

No loss to follow up

13/54 (24.1%) developed shock

10/54 (18.5%) had pulmonary oedema and congestive heart failure

6/54 (11.1%) developed arrhythmia

6/54 (11.1%) died in the hospital

2/54 (3.7%) had ventricular wall aneurysm formation.

1/54 (1.9%) had stroke, reinfarction and thrombus formation

No patient developed pericarditis and LV dysfunction.
Table 1: socio demographic features of the patients

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total Frequency</th>
<th>STEMI N=34(%)</th>
<th>NSTEMI N=20(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=54(%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>WARD</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UHI</td>
<td>46(85.2)</td>
<td>29(63)</td>
<td>17(37)</td>
</tr>
<tr>
<td>4C</td>
<td>8(14.8)</td>
<td>5(62.5)</td>
<td>3(37.5)</td>
</tr>
<tr>
<td><strong>GENDER</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MALE</td>
<td>38(70.4)</td>
<td>24(63.2)</td>
<td>14(36.8)</td>
</tr>
<tr>
<td>FEMALE</td>
<td>16(29.6)</td>
<td>10(62.5)</td>
<td>6(37.5)</td>
</tr>
<tr>
<td><strong>RACE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BLACK</td>
<td>47(87)</td>
<td>29(61.7)</td>
<td>18(38.3)</td>
</tr>
<tr>
<td>ASIAN</td>
<td>7(13)</td>
<td>5(71.4)</td>
<td>2(28.6)</td>
</tr>
<tr>
<td><strong>LEVEL OF EDUCATION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary</td>
<td>13(24.1)</td>
<td>10(76.9)</td>
<td>3(23.1)</td>
</tr>
<tr>
<td>Secondary</td>
<td>13(24.1)</td>
<td>10(76.9)</td>
<td>3(23.1)</td>
</tr>
<tr>
<td>Tertiary</td>
<td>18(33.3)</td>
<td>7(38.9)</td>
<td>11(61.1)</td>
</tr>
<tr>
<td>University</td>
<td>10(18.5)</td>
<td>7(70)</td>
<td>3(30)</td>
</tr>
<tr>
<td><strong>RELIGION</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catholic</td>
<td>12(22.2)</td>
<td>8(66.7)</td>
<td>4(33.3)</td>
</tr>
<tr>
<td>Protestant</td>
<td>20(37)</td>
<td>12(60)</td>
<td>8(40)</td>
</tr>
<tr>
<td>Islam</td>
<td>14(25.9)</td>
<td>11(78.6)</td>
<td>3(21.4)</td>
</tr>
<tr>
<td>Orthodox</td>
<td>2(3.7)</td>
<td>1(50)</td>
<td>1(50)</td>
</tr>
<tr>
<td>SDA</td>
<td>6(11.1)</td>
<td>2(33.3)</td>
<td>4(66.7)</td>
</tr>
<tr>
<td><strong>MARITAL STATUS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>1(1.9)</td>
<td>1(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>Married</td>
<td>46(85.2)</td>
<td>28(60.9)</td>
<td>18(39.1)</td>
</tr>
<tr>
<td>Divorced</td>
<td>3(5.6)</td>
<td>3(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>Widow/widower</td>
<td>4(7.4)</td>
<td>2(50)</td>
<td>2(50)</td>
</tr>
</tbody>
</table>

The mean age for the study participants was 58.7(SD=+/10). There were more male (70.4%) than female (29.6%). Most patients had up to tertiary level of education. There were few participants of Asian origin (13%). protestants (37%) had higher cases of myocardial infarction however among those with STEMI; there were more Islam (78.6%).

21
Table 2: Clinical presentation of patients admitted with myocardial infarction.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total Frequency</th>
<th>STEMI N=34 (%)</th>
<th>NSTEMI N=20 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=54 (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest pain</td>
<td>36(66.7)</td>
<td>23(63.9)</td>
<td>13(13)</td>
</tr>
<tr>
<td>Chest heaviness</td>
<td>18(33.3)</td>
<td>11(61.1)</td>
<td>7(38.9)</td>
</tr>
<tr>
<td>SITE OF PAIN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>35(64.8)</td>
<td>20(60.6)</td>
<td>13(39.4)</td>
</tr>
<tr>
<td>Right</td>
<td>3(5.6)</td>
<td>1(33.3)</td>
<td>2(66.7)</td>
</tr>
<tr>
<td>Both</td>
<td>6(11.7)</td>
<td>0(100)</td>
<td>6(100)</td>
</tr>
<tr>
<td>None</td>
<td>2(3.7)</td>
<td>0(100)</td>
<td>2(100)</td>
</tr>
<tr>
<td>Retrosternal</td>
<td>5(9.3)</td>
<td>2(40)</td>
<td>3(60)</td>
</tr>
<tr>
<td>Epigastric</td>
<td>3(5.6)</td>
<td>3(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>PAIN RADIATION</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left arm</td>
<td>10(18.5)</td>
<td>8(80)</td>
<td>2(20)</td>
</tr>
<tr>
<td>Jaw</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
</tr>
<tr>
<td>Right arms</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
</tr>
<tr>
<td>Arms and neck</td>
<td>7(13)</td>
<td>5(71.4)</td>
<td>2(28.6)</td>
</tr>
<tr>
<td>Neck</td>
<td>6(11.1)</td>
<td>2(33.3)</td>
<td>4(66.7)</td>
</tr>
<tr>
<td>Shoulders</td>
<td>5(9.3)</td>
<td>3(60)</td>
<td>2(40)</td>
</tr>
<tr>
<td>Both arms</td>
<td>3(5.6)</td>
<td>3(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>None</td>
<td>23(42.6)</td>
<td>13(56.5)</td>
<td>10(43.5)</td>
</tr>
<tr>
<td>PULSE RATE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;60</td>
<td>5(9.3)</td>
<td>2(40)</td>
<td>3(60)</td>
</tr>
<tr>
<td>60-100</td>
<td>33(63)</td>
<td>23(69.7)</td>
<td>10(30.3)</td>
</tr>
<tr>
<td>&gt;100</td>
<td>15(27.7)</td>
<td>8(53.3)</td>
<td>7(46.7)</td>
</tr>
<tr>
<td>BLOOD PRESSURE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>11(20.4)</td>
<td>6(54.5)</td>
<td>5(45.5)</td>
</tr>
<tr>
<td>Normal</td>
<td>23(42.6)</td>
<td>13(56.5)</td>
<td>10(43.5)</td>
</tr>
<tr>
<td>High</td>
<td>20(37)</td>
<td>15(75)</td>
<td>5(25)</td>
</tr>
<tr>
<td>Diastolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>7(13.2)</td>
<td>2(28.6)</td>
<td>5(71.4)</td>
</tr>
<tr>
<td>Normal</td>
<td>28(52.8)</td>
<td>20(71.4)</td>
<td>8(28.6)</td>
</tr>
<tr>
<td>High</td>
<td>18(33.9)</td>
<td>11(61.1)</td>
<td>7(38.9)</td>
</tr>
<tr>
<td>CREPITATIONS</td>
<td>19(35.2)</td>
<td>11(57.9)</td>
<td>8(42.1)</td>
</tr>
<tr>
<td>NYHA CLASS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>34(63)</td>
<td>23(67.6)</td>
<td>11(32.4)</td>
</tr>
<tr>
<td>II</td>
<td>6(11.1)</td>
<td>3(50)</td>
<td>3(50)</td>
</tr>
<tr>
<td>III</td>
<td>8(14.8)</td>
<td>3(37.5)</td>
<td>5(62.5)</td>
</tr>
<tr>
<td>IV</td>
<td>6(11.1)</td>
<td>5(83.3)</td>
<td>1(16.7)</td>
</tr>
<tr>
<td>KILLIP CLASS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>40(74.1)</td>
<td>26(65)</td>
<td>14(35)</td>
</tr>
<tr>
<td>II</td>
<td>8(14.8)</td>
<td>3(37.5)</td>
<td>5(62.5)</td>
</tr>
<tr>
<td>III</td>
<td>4(7.4)</td>
<td>4(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>IV</td>
<td>2(3.7)</td>
<td>1(50)</td>
<td>1(50)</td>
</tr>
</tbody>
</table>
66.7% of participants presented with complaints of chest pain, of which the major site of pain being the left side (64.8%). Common associated symptoms were breathlessness (54.7%), palpitations (38.9%) as seen in figure 3. Symptoms occurred at rest, with exercise and emotional stress as shown in the figure 2. Only 54.5% of the participants had both low systolic and diastolic Blood pressure and 70% had both high systolic and diastolic pressure at admission. 35.2% of participants had significant pulmonary rales at admission. 63% had New York Heart Association class I and 74.1% were in Killip class I.

**Figure 2 showing activity at onset of symptoms**
Figure 3 shows the proportion of associated symptoms of myocardial infarction.

More patients with STEMI had associated symptoms as compared to NSTEMI. However there were almost equal possibly of presenting with palpitations, 47.6% for STEMI and 52.4% for NSTEMI. 69% of patients with STEMI as compared to 31% of those with NSTEMI had breathlessness as one of the major associated symptoms. 70.8% of those with STEMI had epigastric pain as compared to 29.2% for NSTEMI. Also more patients with STEMI had nausea(90%) and vomiting(78.6%), dizziness light headacheness(81.2%) and loss of consciousness(75%)
### Table 3: Laboratory findings of the participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>Frequency N=54(%)</th>
<th>STEMI N=34(%)</th>
<th>NSTEMI N=20(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>White Blood Cell Count</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>6(11.1)</td>
<td>1(16.7)</td>
<td>5(83.3)</td>
</tr>
<tr>
<td>Normal</td>
<td>29(53.7)</td>
<td>22(75.9)</td>
<td>7(24.1)</td>
</tr>
<tr>
<td>High</td>
<td>17(31.5)</td>
<td>10(58.8)</td>
<td>7(41.2)</td>
</tr>
<tr>
<td><strong>Haemoglobin</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.0 – 11.9</td>
<td>11(20.4)</td>
<td>6(54.5)</td>
<td>5(45.5)</td>
</tr>
<tr>
<td>12 – 16</td>
<td>35(64.8)</td>
<td>24(68.6)</td>
<td>11(31.4)</td>
</tr>
<tr>
<td>16.1 – 20</td>
<td>7(13)</td>
<td>4(57.1)</td>
<td>3(42.9)</td>
</tr>
<tr>
<td><strong>HbA1C</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6.1</td>
<td>23(40)</td>
<td>22(95.7)</td>
<td>1(4.3)</td>
</tr>
<tr>
<td>&gt;6.1</td>
<td>25(60)</td>
<td>20(80)</td>
<td>5(20)</td>
</tr>
<tr>
<td><strong>Creatinine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>5(9.3)</td>
<td>1(20)</td>
<td>4(80)</td>
</tr>
<tr>
<td>Normal</td>
<td>28(51.9)</td>
<td>18(64.3)</td>
<td>10(35.7)</td>
</tr>
<tr>
<td>High</td>
<td>20(37)</td>
<td>15(75)</td>
<td>5(25)</td>
</tr>
<tr>
<td><strong>Total cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>17(31.5)</td>
<td>10(58.8)</td>
<td>7(41.2)</td>
</tr>
<tr>
<td>Normal</td>
<td>34(63)</td>
<td>23(67.6)</td>
<td>11(32.4)</td>
</tr>
<tr>
<td><strong>Triglyceride</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>38(70.4)</td>
<td>25(65.8)</td>
<td>13(34.2)</td>
</tr>
<tr>
<td>High</td>
<td>13(24.1)</td>
<td>8(61.5)</td>
<td>5(38.5)</td>
</tr>
<tr>
<td><strong>HDL Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>22(40.7)</td>
<td>16(72.7)</td>
<td>6(27.3)</td>
</tr>
<tr>
<td>Normal</td>
<td>24(44.4)</td>
<td>14(58.3)</td>
<td>10(41.7)</td>
</tr>
<tr>
<td>High</td>
<td>5(9.3)</td>
<td>3(60)</td>
<td>2(40)</td>
</tr>
<tr>
<td><strong>LDL Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>22(40.7)</td>
<td>12(54.5)</td>
<td>10(45.5)</td>
</tr>
<tr>
<td>High</td>
<td>29(53.7)</td>
<td>21(72.4)</td>
<td>8(27.6)</td>
</tr>
<tr>
<td><strong>CKMB</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>5(9.3)</td>
<td>5(100)</td>
<td>0(0)</td>
</tr>
<tr>
<td>High</td>
<td>48(88.9)</td>
<td>28(58.3)</td>
<td>20(41.7)</td>
</tr>
<tr>
<td><strong>Troponin I</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>27(50)</td>
<td>15(55.6)</td>
<td>12(44.4)</td>
</tr>
<tr>
<td>High</td>
<td>24(44.4)</td>
<td>18(75)</td>
<td>6(25)</td>
</tr>
</tbody>
</table>

! There were no patients with high values

Most patients had normal values across most laboratory investigations: white blood cell count (total 53.7%), Creatinine 51.9%, total cholesterol 63%, HDL cholesterol 44.4%, and haemoglobin 64.8%. However there were more patients with high 53.7% LDL cholesterol, of which majority were STEMI 72.4% compared to NSTEMI 27.6%. 88.8% had high creatinine kinase MB (STEMI 51.7, NSTEMI 41.7%). Troponin I levels were almost equally distributed
between normal (50%) and high (25%). STEMI 58.8% compared to NSTEMI who had more in low values (83.3%).

Table 4a: Echocardiograph findings of the participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Frequency</th>
<th>STEMI N=34(%)</th>
<th>NSTEMI N=20(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRESENCE OF HYPERTROPHY</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVH</td>
<td>13(24.1)</td>
<td>9(69.2)</td>
<td>4(30.8)</td>
</tr>
<tr>
<td>RVH!</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRESENCE OF REGURGITATION</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>27(50)</td>
<td>16(59.3)</td>
<td>11(40.7)</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
<td>21(38.9)</td>
<td>12(57.1)</td>
<td>9(42.9)</td>
</tr>
</tbody>
</table>

Table 4b: Echocardiograph findings of the participants

<table>
<thead>
<tr>
<th>LEFT VENTRICULAR FUNCTION</th>
<th>FREQUENCY</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=54(%)</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>13(24.1)</td>
</tr>
<tr>
<td>Normal</td>
<td>41(75.9)</td>
</tr>
<tr>
<td>Fractional shortening</td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>20(37)</td>
</tr>
<tr>
<td>Normal</td>
<td>34(63)</td>
</tr>
<tr>
<td>E/A ratio</td>
<td></td>
</tr>
<tr>
<td>0.5 – 1</td>
<td>36(66.7)</td>
</tr>
<tr>
<td>1.1 – 2.8</td>
<td>17(31.5)</td>
</tr>
</tbody>
</table>
At admission, 13/54 (24.1%) of the participants had already Left ventricular hypertrophy (LVH), 27/54 (50%) and 21/54 (38.9%) had mitral regurgitation and tricuspid regurgitation respectively. No patient had right ventricular hypertrophy at admission. 13/54 (24.1%) had systolic dysfunction at admission.

Majority of patients had normal ejection fraction 75.9% (41/54) and fractional shortening 34/54 (63%). Only 13/54 (24.1%) and 20/54 (37%) had low ejection fraction and fractional shortening at admission which is significant of a left ventricular dysfunction.

**Medications used**

Common drugs used include aspirin 53/54 (98.1%), clopidogrel 46/54 (85.2%), 15/54 (27.8%) amlodipine, ACEIS/ARBS (commonly 12/54(22.2%) lisinopril, 8/54(14.8%) captopril and 9/54(16.7%) Losartan), beta blockers (13/54(13%) carvedilol and 17/54(31.5%) bisoprolol), 33/54 (61.1%)artovastatin, morphine. 74.1% (40/54) received enoxaparin, 22.2% (12%) patients needed inotrope therapy of which 10 patients received dopamine and 2 patients received dubutamine. Only 5 patients (9.3%) got thrombolytic therapy (mainly 3 got streptokinase and 2 got tenectaplase), and only 1 patient (1.9%) required temporary pace marker insertion.

14 (25.9%) patients had angiography done, with findings being 6 (62.8%) with triple vessel disease, 3 (21.4%) patients with double vessel disease, and 4 (28.5%) patients had single vessel disease. 2 patients of those who had angiography had stent insertion done in the same sitting.
Table 3: Risk factors associated with myocardial infarction

<table>
<thead>
<tr>
<th>RISK</th>
<th>STEMI N=34</th>
<th>NSTEMI N=20</th>
<th>OR/ 95% CI</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GENDER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>male</td>
<td>24(63.2)</td>
<td>14(36.8)</td>
<td>1.029(0.30-3.44)</td>
<td>0.96</td>
</tr>
<tr>
<td>Female</td>
<td>10(62.5)</td>
<td>6(37.5)</td>
<td>0.6</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>EXERCISE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>14(70)</td>
<td>6(30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18.5-24.5</td>
<td>8(80)</td>
<td>2(20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24.5-29.5</td>
<td>10(66.7)</td>
<td>5(33.3)</td>
<td>2(0.30-13.1)</td>
<td>0.47</td>
</tr>
<tr>
<td>29.5-39.5</td>
<td>14(63.6)</td>
<td>8(36.4)</td>
<td>2.2(0.38-13.5)</td>
<td>0.362</td>
</tr>
<tr>
<td><strong>Abdominal Circumference</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;88cm</td>
<td>4(66.7)</td>
<td>2(33.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>89-102cm</td>
<td>16(84.2)</td>
<td>3(15.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;102cm</td>
<td>12(52.2)</td>
<td>11(47.8)</td>
<td>1.02(0.98-1.05)</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>PAST MEDICAL HISTORY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction (heart attack)</td>
<td>3(60)</td>
<td>2(40)</td>
<td>0.9(0.13-5.91)</td>
<td>0.9</td>
</tr>
<tr>
<td>Hypertension</td>
<td>23(65.7)</td>
<td>12(34.3)</td>
<td>1.53(0.48-4.90)</td>
<td>0.47</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>14(70)</td>
<td>6(30)</td>
<td>1.52(0.46-4.95)</td>
<td>0.49</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>5(71.4)</td>
<td>2(28.6)</td>
<td>1.73(0.29-10.10)</td>
<td>0.53</td>
</tr>
<tr>
<td>Heart surgery</td>
<td>0(0)**</td>
<td>1(100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ALCOHOL CONSUMPTION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current drinker</td>
<td>4(40)</td>
<td>1(20)</td>
<td>NS</td>
<td>1.0</td>
</tr>
<tr>
<td>None drinker</td>
<td>14(56)</td>
<td>11(44)</td>
<td>NS</td>
<td>1.0</td>
</tr>
<tr>
<td>Former drinker</td>
<td>16(66.7)</td>
<td>8(33.3)</td>
<td>NS</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>SMOKING HABITS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>0(0)**</td>
<td>1(100)</td>
<td>0.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Non smoker</td>
<td>25(61)</td>
<td>16(39)</td>
<td>0.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Former smoker</td>
<td>9(75)</td>
<td>3(25)</td>
<td>1.61</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>FAMILY HISTORY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1(25)</td>
<td>3(75)</td>
<td>0.19(0.12-1.99)</td>
<td>0.16</td>
</tr>
<tr>
<td>Hypertension</td>
<td>20(62.5)</td>
<td>12(37.5)</td>
<td>1.16(0.35-3.88)</td>
<td>0.802</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>10(50)</td>
<td>10(50)</td>
<td>0.46(0.81-1.17)</td>
<td>0.10</td>
</tr>
<tr>
<td>Obesity</td>
<td>3(50)</td>
<td>3(50)</td>
<td>0.64(0.12-3.57)</td>
<td>0.61</td>
</tr>
<tr>
<td>Premature/sudden Death</td>
<td>0(0)</td>
<td>1(100)</td>
<td>1.2(0.84-3.5)</td>
<td>0.23</td>
</tr>
<tr>
<td><strong>HDL Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>16(72.7)</td>
<td>6(27.3)</td>
<td>1.9(0.55-6.58)</td>
<td>0.30</td>
</tr>
<tr>
<td>Normal</td>
<td>14(58.3)</td>
<td>10(41.7)</td>
<td>1.7(0.23-13.40)</td>
<td>0.57</td>
</tr>
<tr>
<td>High</td>
<td>3(60)</td>
<td>2(40)</td>
<td>0.375</td>
<td></td>
</tr>
<tr>
<td><strong>LDL Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>12(54.5)</td>
<td>10(45.5)</td>
<td>1.2</td>
<td>0.67</td>
</tr>
<tr>
<td>High</td>
<td>21(72.4)</td>
<td>8(27.6)</td>
<td>2.18(0.67-7.04)</td>
<td>0.19</td>
</tr>
</tbody>
</table>
NS-not significant

** No significant p values

Though not statistically significant those with past medical history of hypertension 35/54, 64.8% (OR=1.53, 95% CI=0.48-4.90), diabetes mellitus (OR=1.52, 95% CI=0.46-4.95), dyslipidaemia 7/54, 13% (OR=1.73, 95%CI=0.29-10.10) were higher risk for myocardial infarction. Also similar patterns are seen in family history as positive for hypertension (59.3%) (OR=1.1, 95%CI=0.35-3.88) and diabetes mellitus (37%). 50.9% of male and 92.9% of female participants had abdominal circumference greater than 102cm and 88cm respectively. Higher body mass index of 24.5-29.5(OR=2.0, 95%CI 0.3-13.1) and 29.5-39(OR=2.2, 95%CI=0.38-13.5) though the p value is not statistically significant was also a risk for myocardial infarction.

Positive history of current or former h/o alcohol consumption constituted more than half of the participants (29/54). In contrast only approximately 13/54(24.1%) were current or previous smokers.

Low HDL (OR=1.9, 95% CI=0.55-6.58) confers higher risk for myocardial infarction compared to normal and high HDL cholesterol levels.
Over all 24.1% (10/54) developed shock, 18.5% (10/54) had pulmonary oedema and congestive heart failure, 11.1% (6/54) developed arrhythmia, 11.1% (6/54) died in the hospital, and 3.7% (2/54) had ventricular wall aneurysm formation. 1.9% (1/54) had stroke, reinfarction and thrombus formation. No patient developed pericarditis and LV dysfunction.
Figure 5: showing the in hospital outcome by subtypes of myocardial infarction

Generally across all in-hospital outcome there with more patients in the STEMI arm as compared to NSTEMI. Commonly patients developed pulmonary oedema, arrhythmia, congestive heart failure, shock and dead. Fewer patients developed aneurysm, stroke thrombus and re infarction.
<table>
<thead>
<tr>
<th>In hospital outcome</th>
<th>STEMI</th>
<th>NSTEMI</th>
<th>OR/95% CI</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary edema</td>
<td>8(80)</td>
<td>2(20)</td>
<td>2.76(0.53-15.5)</td>
<td>0.23</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>5(83.3)</td>
<td>1(16.7)</td>
<td>3.28(0.35-30.27)</td>
<td>0.29</td>
</tr>
<tr>
<td>Congestive Heart failure</td>
<td>7(70)</td>
<td>3(30)</td>
<td>1.46(0.33-6.47)</td>
<td>0.61</td>
</tr>
<tr>
<td>Stroke</td>
<td>1(100)</td>
<td>0!</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Re infarction</td>
<td>1(100)</td>
<td>0!</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shock/hypotension</td>
<td>10(76.9)</td>
<td>3(23.1)</td>
<td>2.36(0.56-9.88)</td>
<td>0.24</td>
</tr>
<tr>
<td>Aneurysm formation</td>
<td>2(100)</td>
<td>0!</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombus formation</td>
<td>1(100)</td>
<td>0!</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>5(83.3)</td>
<td>1(16.7)</td>
<td>3.28(0.35-30.27)</td>
<td>0.29</td>
</tr>
</tbody>
</table>

! no statistical significance

This is no statistical significant association between myocardial infarction and the documental outcome. However at there is 2.76 increase risk of pulmonary oedema (OR=2.76, 95% CI 0.53-15.5), 3.28 increase risk of arrhythmia and death (OR=3.28, 95% CI 0.35-30.27), and 2.36 increase risk of shock (OR=2.36, 95% CI 0.56-9.88)
CHAPTER FIVE

5.0 DISCUSSION

5.1.0 Clinical presentations

Most patients presented with ST elevation myocardial infarction (63%) as compared to ST segment elevation. This is in contrast to studies such as GRACE registry from North America and Europe that reported 30% due to STEMI.[30] This difference can be attributed to delay in seeking medical attention. Most patients in this study came to the hospital more than 72 hours with median time of presentation to the hospital from onset of symptoms 93.5 hours (SD 57.09, OR=1.002 95%CI 0.9-1.0). Such late presentation to the hospital were observed in a prospective studies done in Kenya (13 hours) and Dakar (14.3 hours). [31, 32]. Besides patients who are admitted to Mulago Hospital complex are either self referral or referred from other hospitals and health centres, thus most patients arrive late for proper cardiac care and treatment.

Chest pain (66.7%) was the major presenting complaints with major site being left side (64.8%). The rest had chest heaviness, epigastric pain, and breathlessness as presenting symptoms. This non specificity of presenting symptoms affect patient initial diagnosis and management as some might be treated for non cardiac diseases and thus loss of time. Several studies have also shown chest pain as the major symptom.[31, 32].

Only 54.5% of the participants had both low systolic and diastolic (OR=12.7, 95% CI 1.0-157) blood pressure and 70% had both high systolic and diastolic pressure at admission. These are the group of patients with high risk of mortality. This is in agreement with the TRACE study where cardiogenic shock solely contributed more than 62% cause of death and GRACE study which emphasized systolic pressure(OR 1.4 per 20mmHg decrease) as a risk for death.(Lindholm et al., 2003, Granger Cb and et al., 2003)

35.2% of participants had significant pulmonary rales at admission. 63% had New York Heart Association class I and 74.1% were in Killip class I.

At admission, 13/54(24.1%) of the participants had already Left ventricular hypertrophy (LVH), 27/54(50%) and 21/54(38.9%) had mitral regurgitation and tricuspid regurgitation respectively.
24.1% had systolic dysfunction at admission. This can be explained by the fact that majority (64.8%) of the patients had a self reported history of hypertension.

5.1.2 Risk factors of myocardial infarction

The mean age was 58.7(SD=+/-10) which was close the global INTERHEART study where the mean age was 58.1. however for the INTERHEART study for Africa the mean age was 54.3.[33]. There were more males (70.4%) than females (29.6%).

Most patients 18/54(58.9%) had tertiary level of education (p=0.043, OR=5.23 95% CI 1.05-25.9) which was statistically significant. This further illustrates myocardial education is among the elite community. In the INTERHEART study those with tertiary education have increased risk for AMI (OR 1.86; 95% CI 1.06 to 3.25).[33]

50.9% of male and 92.9% of female participants had abdominal circumference greater than 102cm and 88cm respectively. Self reported history of hypertension 23/54(64.8%) and diabetes with similar patterns seen in family history as positive for hypertension (59.3%) and diabetes mellitus (37%). Positive history of current or former h/o alcohol consumption constituted more half of the participants. These factors are in agreement with studies done across 52 African countries by the INTERHEART study. Also in Kenya similar association between abdominal circumferences, self reported history of hypertension and diabetes mellitus as major risk of acute myocardial infarction were reported. In contrast only approximately 24.1% were current or previous smokers compared to the INTERHEART study. (Steyn et al., 2005b, Shavadia J et al., 2012). Higher body mass index 24.5-29.5(OR=2, 95%CI 0.3-13.1) and 29.5-39(OR=2.2, 95%CI=0.38-13.5) though the p value is not statistically significant.

Sixty three percent (63%) of patients had normal total cholesterol and 44.4% normal HDL cholesterol 44.4%. However there were more patients with high (53.7%) LDL cholesterol, of which majority were STEMI 72.4% compared to NSTEMI 27.6%. This is in line with other studies that show high LDL cholesterol 2.18(0.67-7.04) as one of the risk for myocardial infarction.
5.1.3: In-hospital outcome.

During hospital stay 24.1% (10/54) of the patients developed shock (OR=2.36, 95% CI 0.56-9.88), followed by 18.5% (10/54) who had pulmonary oedema (OR=2.76, 95%CI 0.53-15.5), and congestive heart failure. 11.1% (6/54) developed arrhythmia (OR=3.28, 955 CI 0.35-30.27), 11.1% (6/54) died in the hospital, and 3.7% (2/54) had ventricular wall aneurysm formation. Only 1.9% (1/54) had stroke, re infarction and thrombus formation. No patient developed pericarditis and left ventricular dysfunction. Though there is no statistical significant association between myocardial infarction and the documented outcome, this confers risk of such outcome among patients with myocardial infarction.

5.2 LIMITATION OF THE STUDY

Despite long period of study (8months) we were still unable to meet the required by sample size formula. We thus enrolled only 54 patients instead of 110

5.3 CONCLUSION

Majority of patients admitted with myocardial infarction have STEMI and present with chest pain. Most patients are males. Hypertension was a high risk. There was delayed time to presentation to hospital from the time of onset of symptoms. Almost half of the patients with STEMI developed pulmonary edema, shock, congestive heart failure and arrhythmia.

5.4 RECOMMENDATION

There is need for increased sensitization and awareness about myocardial infarction, it’s presenting symptoms and risk factors among the population at risk and encourage patients to come early to the hospital. In particular aggressive risk reduction and treatment for hypertension diabetes mellitus and dyslipidaemia should be emphasized.

Due to high risk of developing shock, pulmonary edema and congestive heart failure during admission, early intervention should be strongly emphasized.

There is need for further studies with larger sample size that can answer more questions about acute myocardial infarction.
REFERENCES


### APPENDIX I: BUDGET

<table>
<thead>
<tr>
<th>Item</th>
<th>Quantity</th>
<th>Unit cost (shillings)</th>
<th>Total cost (shillings)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BLOOD INVESTIGATIONS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting blood sugar test</td>
<td>110</td>
<td>5000</td>
<td>550,000</td>
</tr>
<tr>
<td>RFTs and serum electrolytes</td>
<td>110</td>
<td>30,000</td>
<td>3,300,000</td>
</tr>
<tr>
<td>Lipid profile</td>
<td>110</td>
<td>30,000</td>
<td>3,300,000</td>
</tr>
<tr>
<td>Cardiac enzymes</td>
<td>110</td>
<td>30,000</td>
<td>3,300,000</td>
</tr>
<tr>
<td>Full blood count</td>
<td>110</td>
<td>15,000</td>
<td>1,650,000</td>
</tr>
<tr>
<td><strong>IMAGING</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECG papers</td>
<td>1000(1 pack)</td>
<td>760</td>
<td>760,000</td>
</tr>
<tr>
<td>ECHO sono papers</td>
<td>6</td>
<td>100,000</td>
<td>600,000</td>
</tr>
<tr>
<td>ECHO gel</td>
<td>4</td>
<td>100,000</td>
<td>400,000</td>
</tr>
<tr>
<td><strong>STATIONARIES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tape measure</td>
<td>1</td>
<td>3,500</td>
<td>3,500</td>
</tr>
<tr>
<td>Weighing scale</td>
<td>1</td>
<td>25,000</td>
<td>25,000</td>
</tr>
<tr>
<td>BP machine</td>
<td>1</td>
<td>350,000</td>
<td>350,000</td>
</tr>
<tr>
<td>Vacutainer tubes(4mls)</td>
<td>300</td>
<td>1,000</td>
<td>300,000</td>
</tr>
<tr>
<td>Gloves</td>
<td>500(in 1box)</td>
<td>5,000</td>
<td>5,000</td>
</tr>
<tr>
<td>Alcohol swab</td>
<td>110</td>
<td>300</td>
<td>33,000</td>
</tr>
<tr>
<td>Printing draft proposal</td>
<td>1</td>
<td>5,000</td>
<td>5,000</td>
</tr>
<tr>
<td>Photocopying proposal for dept presentation</td>
<td>20</td>
<td>100</td>
<td>2,000</td>
</tr>
<tr>
<td>Printing final copy of proposal</td>
<td>1</td>
<td>5,000</td>
<td>5,000</td>
</tr>
<tr>
<td>Photocopying approved proposal</td>
<td>15</td>
<td>100</td>
<td>1,500</td>
</tr>
<tr>
<td>Photocopying questionnaires</td>
<td>110</td>
<td>50</td>
<td>5,500</td>
</tr>
<tr>
<td>Photocopying consent forms</td>
<td>110</td>
<td>50</td>
<td>5,500</td>
</tr>
<tr>
<td>ID labels</td>
<td>3</td>
<td>3,500</td>
<td>10,500</td>
</tr>
<tr>
<td>Pens</td>
<td>20</td>
<td>300</td>
<td>6,000</td>
</tr>
<tr>
<td>Ink pad for thumb print</td>
<td>2</td>
<td>1,500</td>
<td>3,000</td>
</tr>
<tr>
<td>Printing draft dissertation</td>
<td>2</td>
<td>5,000</td>
<td>10,000</td>
</tr>
<tr>
<td>Printing final dissertation</td>
<td>10</td>
<td>5,000</td>
<td>50,000</td>
</tr>
<tr>
<td>Binding final dissertation</td>
<td>10</td>
<td>10,000</td>
<td>100,000</td>
</tr>
<tr>
<td>Data analysis and statistician</td>
<td>1</td>
<td>500,000</td>
<td>500,000</td>
</tr>
<tr>
<td>Personnel facilitation</td>
<td></td>
<td>500,000</td>
<td>500,000</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td></td>
<td><strong>15,780,500</strong></td>
</tr>
</tbody>
</table>
## APPENDIX II: TIME FRAMEWORK

<table>
<thead>
<tr>
<th>ACTIVITY</th>
<th>TIME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proposal presentation at department of medicine</td>
<td>February 2013</td>
</tr>
<tr>
<td>Proposal presentation at IRB</td>
<td>June 2013</td>
</tr>
<tr>
<td>Collection of data</td>
<td>September 2013 to April 2014</td>
</tr>
<tr>
<td>Data analysis</td>
<td>April 2014</td>
</tr>
<tr>
<td>Writing dissertation</td>
<td>April 2014</td>
</tr>
<tr>
<td>Defense of dissertation</td>
<td>June 2014</td>
</tr>
</tbody>
</table>
APPENDIX III: CONSENT FORM

Study title: clinical presentation and in hospital outcome of patients admitted with myocardial infarction.

Principle investigator: Achan Josephine. Makerere University P O BOX 7062, Kampala, Uganda
Telephone +256752141310 Email chanajosephine@yahoo.com

Introduction
My name is Dr Achan Josephine; I am requesting you to participate in this study about patients with myocardial infarction admitted in Mulago Hospital.
This study is conducted to determine clinical presentation and in-hospital outcome of patients admitted with myocardial infarction in Mulago Hospital. This will provide a better understanding of the pattern of disease and its complications. It will also help in establish management plan and early intervention among patients with myocardial infarction.

Background of the study
Myocardial infarction is a non-infectious disease commonly called heart attack that is gradually increasing in our setting. It is the second cause of death worldwide and cause of disability. It is characterized by the chest pain or discomfort that radiates to the neck, jaw and left arm. It can result in many complications like stroke, heart failure etc

Participant’s rights as a research volunteer
Participation in this research study is voluntary and one can decide to withdraw from the study at any time. You should know such a decision will not affect your medical care or possible participation in the future research studies in anyway.

Study procedures
If one decides to participate in this study, you will be expected to answer questions about your personal data, current and past medical history and will undergo physical examination.
About 5mls of blood will be collected from the participants arm to test for complete blood count, blood sugar, enzymes that show heart attack, level of body fat, and the function of the kidneys. An ECG and echocardiography will also be done.
Potential benefit to the participant

1. The participant will be examined and investigated for the above baseline tests for myocardial infarction at no cost.
2. Results of the above investigation will be given to the attending doctors for management of their conditions.
3. They will be closely monitored and referred appropriately in case of any complication.

From the results of this study, it may be possible to design prevention and interventional programmes/strategies in management of patients with myocardial infarction.

Risk and discomfort to the participant

The procedure of drawing blood may cause some pain and discomfort. The participant may also bleed a little from the site; however, the amount of blood drawn will be too small to cause any health hazard.

Questions or Concerns

For any further questions about the study, the participant may contact Dr Achan Josephine at the Department of Medicine Mulago Hospital on telephone number +256752141310. In case of any concerns regarding your rights and participation in this study, you can contact the chairperson school of medicine research and ethics committee, Prof James Tumwine on telephone number +256414533541.

Informed consent

I understand that participation in this study is voluntary and that no consequences will result from my withdrawal from it. I have read the information provided and had time to discuss with above named doctor

Name of patient/next of kin…………………………….Signature…………….Date…………….

Name of investigator…………………………………………Signature…………….Date…………….
APPENDIX IV: CONSENT FORM IN LUGANDA

Study title: CLINICAL PRESENTATION AND IN HOSPITAL OUTCOME OF PATIENTS ADMITTED WITH MYOCARDIAL INFARCTION.

OBUBONERO N’OKUWONA KW’ABALWADDE B’OMUTIMA ABAWELEDWA EBITANDA EMULAGO

Akulira okunoonyereza: Achan Josephine. Makerere University P O BOX 7062, Kampala, Uganda
Telephone +256752141310 Email chanajosephine@yahoo.com

Principle investigator: Achan Josephine. Makerere University P O BOX 7062, Kampala, Uganda
Telephone +256752141310 Email chanajosephine@yahoo.com

Enyanjula
Nze Dr Achan Josephine, nkusaba wetabe mukunoonyereza kuno okukwata ku balwadde b’omutima abaweledwa ekitanda kudwaliro e Mulago
Okunoonyereza kuno kukoldwa okumanaya obubonero ne mpona yabalwadde b’omutima abaweledwa ekitanda eMulago. Kino kijja kutuyamba okumanya obulwadde buno n’obuzibu bwabwo. Era kijja kutuyamba okusawo enettekka wamu n’enjijanjaba yabwo nga bukyali/tebunakula mubalwadde b’omutima.

Ebyafaayo mu kunoonyereza kuno:
Eddembe lyo mukunoonyereza kuno:
Nga tonasalawo kwetaba oba obutetaba mukunoonyereza kuno, olina eddembe okumanya bino:

1. Ebigendelebwa by’okunoonyereza kuno n’engeri janaganyulwaamu, obuzibu obunaamutuukako n’ebyo ebimusubirwamu nga yetabye mukunoonyereza kuno.
2. Okwetaba mukunoonyereza kuno kwa kyeyagalire. Osobola okusalawo okuvaamu ekisera kyona era olina okumanya nti kino tekijja kulemesa bujjaniyali bwolina kufuna oba okwetaba mukunoonyereza okulala kwona.

Ebinagobelebwa mukunoonyereza kuno:
An’etaba mukunoonyereza kuno asubilwa okudamu ebibuuzo ebimukwatako, ku bulamu bwe kati n’ekubiseera ebyemabega era ajja kukebelebwa.

Ajjia kugibwaako omusayi okuva ku mukono gwo gukebelebwe okumanya obungi bwago, sukali, ekibuumba, omutima n’ensigo yo bimanyibwe nga bwe bikola era ojja kukubwa ekifananyi, otekebwe mu kativi okumanya omutima gwo bwegukola.

Ebinaganyulwa mukunoonyereza kuno

1. Akwetabyemu wakukebelebwa ebyo wagulu ebitandikirwako okumanya embera omutima gyegukolamu nga tasasudde.
2. Ebinazulibwa byakuwebwa omusawo amujjanjaba asobole okumujjanjaba obulungi.
3. Ajja kkekenezebwa era qwelezebwe mubasawo abakuggu singa wanabawo obuzibubwona. Ebinazulibwa mukunoonyezebwa kuno byinza okweyanbisibwa mukusawo enkola eyokuziyiza n’okujjanjaba endwadde y’mutima.s

Obuzibu n’obutawulira bulungi mukunoonyereza kuno

1. Oyinza okusiyibwa, okulumizibwa oba okuvamu omusayi omutonotononu nga ogibwako omusayi naye kino sikyamanyi/kitono nyo tekilina bulabe eri bulamu bwo.
2. Oyinza okujayo ebyama byo nga wetabye mukunoonyereza kuno naye abakukulira bajja kubikuuma nga bya kyaama era elinya lyo silykulagibwa wantu wona..

Ebibuuzo mukunoonyereza kuno
Bwoba n’ebibuuzo byona ebikwata ku kunoonyereza kuno laba Dr Achan Josephine mu Department ya Medicine kudwaliro e Mulago ku ssimu eno 0752141310.
Bwoba n’bibuuzo ebikwata ku dembe lyo n’obuvunanyizibwa bwo mukunoonyereza kuno, laba akulira akakiiko k’empisa n’okunoonyereza, Prof James Tumwine ku ssino eno 0414533541.

Ekiwandiiko ky’okukiriza
Manyi nti okwetaba mukunoonyereza kuno kwa kyeyagalire era tewali buzibu bunabawo nga kuvudemu. Nsomye ekiwandiiko kino era nsobodde okukyogerako n’omusawo oyo wagulu. Akwetabyemu........................omukuno/ekinkumu..................Enaku z’omwezi.........Anoonyereza......................... omukuno/ekinkumu..................Enaku z’omwezi.........
APPENDIX V: QUESTIONNAIRE

CLINICAL PRESENTATION AND IN-HOSPITAL OUTCOME OF PATIENTS WITH MYOCARDIAL INFARCTION ADMITTED AT MULAGO HOSPITAL

PI: DR ACHAN JOSEPHINE

IDENTIFICATION NUMBER………………………….

IP NUMBER……………………………….WARD……

A. DEMOGRAPHIC DATA

1) Age (years) □
2) Gender: 1) Male □ 2) Female □
3) District ……………………………………....
4) Race………………………………………….
5) Level of education
   1) Primary □  2) Secondary □  3) Tertiary □
   4) University □  5) others please specify…………………………..
6) Occupation…………………………………..
7) Religion
   1) Catholic □  2) Protestant □  3) Islam □
   4) Orthodox □  5) SDA □
   6) Others……………………………………………………
8) Marital status
   1) Single □  2) Married □
   3) Divorce □  4) Widow/widower □
   5) Others………………………………………………..

B. PRESENTING COMPLAINTS

9) Time of onset of symptoms to presentation to hospital.(hours) □
10) Chest heaviness: 1) Yes □  2) No □
11) Chest pain: 1) Yes □  2) No □
12) If yes (10 or 11) specify which side:
1) Left □ 2) Right □  
3) Both □ 4) None □  
5) Others ……………………………………….. 

13) To which part does the pain radiate? 
1) Left arm □ 5) Neck □  
2) Jaw □ 6) Shoulders □  
3) Right arms □ 7) Both arms □  
4) Arms and neck □ 8) None □  
9) Others………………………………………………… 

ASSOCIATED SYMPTOMS AT ADMISSION

14) Palpitations □ □  
15) Nausea □ □  
16) Vomiting □ □  
17) Breathlessness □ □  
18) Cough □ □  
19) Epigastric pain □ □  
20) Dizziness, light headedness, collapse □ □  
21) Loss of consciousness □ □  
22) Activity at onset of symptoms  
1) Emotional stress □ □ 3) Exercise □ □ 5) Heavy meal □ □  
2) Alcohol intake □ □ 4) Rest □ □ 6) Driving □ □  

C. PAST MEDICAL HISTORY  

Have you ever suffered from any of the following? 

1) Yes 2) No 3) Unknown  
23) Chest pain/discomfort > 30minutes □ □ □  
24) Epigastric discomfort with vomiting □ □ □  
25) Myocardial infarction (heart attack) □ □ □  
26) Hypertension □ □ □  
27) Diabetes Mellitus □ □ □  

47
28) Dyslipidaemia
29) Chronic kidney disease
30) Heart Surgery
31) If yes to (30) please specify……………………………………
32) Heart Failure
33) If yes to (32) please state the NYHA class
   1) I  2) II  3) III  4) IV
34) HIV status
   1) Reactive  2) Non reactive  3) Unknown

D. CURRENT MEDICATIONS
Tick the medications patient has been taking
   1) Yes  2) No  if yes please specify
35) Beta blockers………………………………………
36) ARBs/ACEIs………………………………………
37) Calcium channel blockers…………………………
38) Diuretics…………………………………………
39) Nitrates…………………………………………
40) Antiplatelet………………………………………
41) Anticoagulant……………………………………
42) Morphine…………………………………………
43) Statins…………………………………………
44) Antidiabetes……………………………………
45) Others ……………………………………………

E. LIFE STYLE AND SOCIAL ACTIVITIES
46) Smoking habits
   1) Current smoker  3) Former smoker
   2) Non smoker  4) Unknown
47) Duration of smoking(years)
48) Alcohol consumption
   1) Current drinker □  3) Former drinker □
   2) Non drinker □  4) Unknown □

49) Duration of drinking alcohol (years) □

50) Substance use
   1) Current user □  3) Former user □
   2) Non user □  4) Unknown □

51) Duration of substance use(years) □

52) Physical exercise
   1) Yes □  2) No □

53) Means of transport
   1) Walk □  4) Bicycle □
   2) Drive □  5) Motorcycle (bodaboda) □
   3) All □  6) Uses Taxi □

I) FAMILY HISTORY
   Does any of your relatives suffer from or have suffered from the following conditions
   1) Yes □  2) No □  3) Unknown □

54) Myocardial infarction □
55) Hypertension □
56) Diabetes Mellitus □
57) Obesity □
58) Premature/sudden death □
59) Other heart diseases □

60) If yes to (59) please specify…………………………………………

J) PHYSICAL EXAMINATION AT ADMISSION
   61) Weight (kg) □
62) Height (m)
63) Body Mass Index
64) Abdominal circumference (cm)
65) Presence of edema: 1) Yes  2) No
66) Pulse Rate (b/m)
67) Pulse rhythm: 1) Regular  2) Irregular
68) Jugular Venous Pressure: 1) Normal  2) Raised
Blood pressure (mmHg)
69) Systolic Bp(mmHg)
70) Diastolic Bp(mmHg)
71) Precordium:
   1) Heave  2) Thrill  3) None
72) Apex beat Displaced:
   1) Yes  2) No
73) Heart Sounds S1 S2:
   1) Present  2) Absent
Added sounds present:
   1) YES  2) NO
74) S3
75) S4

Murmurs heard:
   1) YES  2) NO
76) MR
77) TR
78) VSD
79) Crepitations:
   1) Present  2) Absent
80) New York Heart Association Class
   1) I  2) II  3) III  4) IV
81) Killip Classification
   1) I □  2) II □
   3) III □  4) IV □

82) Liver enlargement (cm) □

K) LABORATORY FINDINGS

83) White cell count x 10^3 □

84) Platelet count x 10^3 □

85) Haemoglobin (g/dl) □

86) Haematocrit (%) □

87) Fasting blood sugar (mmol/l) □

88) HBA1C (%) □

Renal function test and serum electrolytes

89) Creatinine (umol/l) □

90) Urea (mmol/l) □

91) Sodium (mmol/l) □

92) Potassium (mmol/l) □

93) Chloride (mmol/l) □

94) Calcium (mmol/l) □
95) Magnesium (mmol/l)

**Lipid profile**

96) Total cholesterol (mmol/l) [ ] [ ]

97) Triglyceride (mmol/l) [ ] [ ]

98) HDL Cholesterol (mmol/l) [ ] [ ]

99) LDL cholesterol (mmol/l) [ ] [ ]

100) Non-HDL cholesterol (mmol/l) [ ] [ ]

**Cardiac enzymes**

101) CK-MB (u/l) [ ] [ ]

102) Troponin I (ng/dl) [ ] [ ]

**L) IMAGING**

**Electrocardiography**

Tick the type of MI as seen on ECG

1) Yes  2) No

103) ST segment elevation [ ] [ ]

104) Non ST segment elevation [ ] [ ]

If yes to (104 and 103) which wall(s) are affected?

1) YES  2) NO

105) Anterior [ ] [ ]

106) Posterior [ ] [ ]
107) Lateral  
108) Septal   
109) Inferior  

110) Rhythm  
      1) Sinus Rhythm  
      2) Sinus Bradycardia  
      3) Sinus Tachycardia  
      4) Junctional Rhythm  
      5) AFib  
      6) AFlutter  
      7) SVT  
      8) VTach  
      9) VFib  
     10) Others………………………………………

111) Conduction abnormally  
      1) LBBB  
      2) RBBB  
      3) None  
      4) Others please specify………………………………………

112) AV block present:  
      1) First degree  
      2) Morbitz I  
      3) Morbitz II  
      4) Third degree  
      5) None  
      6) others ………………………………

Echocardiography

113) Regional wall abnormalities  
      Normal  
      Hypokinesia  
      Dyskinesia  
      Akinesia  
      Aneurysmal  

114) Which wall(s) is affected?  
      Anterior  
      Posterior  
      Inferior  
      Lateral  
      Apical  
      Rt Ventricular wall  
      IVS  
      Others specify………………………………………

Chamber size (cm)  
Left ventricle:  
53
115) Diastole □
116) Systole □

117) Left atrium □
118) Right ventricle □

119) Presence of hypertrophy
1) LVH □  2) RVH □  3) None □

Presence of Regurgitation
1) Yes  2) No

120) Mitral regurgitation □ □
121) Tricuspid regurgitation □ □

Left ventricular function
122) Ejection fraction (%) □
123) Fractional shortening (%) □
124) E/A ratio □

Presence of ventricular wall rupture
1) Yes  2) No

125) VSD □ □
126) Free wall □ □
127) Papillary muscles □ □

128) LV thrombus:
1) Present □  2) Absent □

M) FOLLOW UP DURING ADMISSION
Tick conditions patient develop during admission
1) Yes  2) No  If yes please specify
129) Pulmonary edema
130) Arrhythmia
131) Congestive heart failure
132) Stroke
133) Re infarction
134) Infarct extension
135) Shock/hypotension
136) Pericarditis/tamponade
137) LV dysfunction
138) Aneurysm
139) Wall Rupture
140) Thrombus
141) Others

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>N) TREATMENT GIVEN</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tick the treatment(s) provided to the patient

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>2) No</td>
</tr>
<tr>
<td>142)</td>
<td>Aspirin</td>
<td></td>
</tr>
<tr>
<td>143)</td>
<td>Clopidogrel</td>
<td></td>
</tr>
<tr>
<td>144)</td>
<td>ACEIs/ARB</td>
<td></td>
</tr>
<tr>
<td>145)</td>
<td>Diuretics</td>
<td></td>
</tr>
<tr>
<td>146)</td>
<td>Calcium channel blockers</td>
<td></td>
</tr>
<tr>
<td>147)</td>
<td>Beta blockers</td>
<td></td>
</tr>
<tr>
<td>148)</td>
<td>Nitrate</td>
<td></td>
</tr>
<tr>
<td>149)</td>
<td>Heparin</td>
<td></td>
</tr>
<tr>
<td>150)</td>
<td>Morphine</td>
<td></td>
</tr>
<tr>
<td>151)</td>
<td>Statins</td>
<td></td>
</tr>
<tr>
<td>152)</td>
<td>Fibrates</td>
<td></td>
</tr>
<tr>
<td>153)</td>
<td>Inotropes</td>
<td></td>
</tr>
<tr>
<td>154)</td>
<td>Pacemaker</td>
<td></td>
</tr>
<tr>
<td>155)</td>
<td>Cardioversion</td>
<td></td>
</tr>
</tbody>
</table>
156) Thrombolytic therapy ☐ ☐ .................................
157) PCI ☐ ☐ .................................
158) Others please specify.................................................................

**O) OUTCOME AT DISCHARGE**

Tick any that apply

<table>
<thead>
<tr>
<th></th>
<th>1) Yes</th>
<th>2) No</th>
<th>if yes specify</th>
</tr>
</thead>
<tbody>
<tr>
<td>159) Left ventricular dysfunction</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>160) Heart Failure</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>161) Stroke</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>162) Mitral Regurgitation</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>163) Mural thrombus</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>164) Re infarction</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>165) Arrhythmia</td>
<td>☐ ☐</td>
<td>.................................................</td>
<td></td>
</tr>
<tr>
<td>166) Status at discharge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1) Alive ☐</td>
<td>2) Dead ☐</td>
<td></td>
</tr>
</tbody>
</table>

---

56
APPENDIX VI

KILLIP CLASS AS A MEASURE OF SEVERITY OF HEART FAILURE AMONG PATIENTS WITH MYOCARDIAL INFARCTION

Class I: No clinical signs of heart failure
Class II: Crackles, S 3 gallop and elevated jugular venous pressure
Class III: Frank pulmonary oedema
Class IV: Cardiogenic shock-hypotension (systolic < 90mmHg) and evidence of peripheral vasoconstriction (oliguria, cyanosis, sweating)

NEW YORK HEART ASSOCIATION FOR CLASSIFICATION OF HEART FAILURE

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I (Mild)</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.</td>
</tr>
</tbody>
</table>