POST-DISCHARGE, SHORT TERM MORBIDITY AND MORTALITY OF CHRONIC HEART FAILURE AT KENYATTA NATIONAL HOSPITAL.

A dissertation submitted 9s part fulfillment for the degree of Master of Medicine in Internal Medicine

By

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This dissertation is my original work and has not to my knowledge been submitted for a degree in any other University.

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DEDICATION

I dedicate this work to the Lord God Almighty, for from his holy place the Lttrd has seen, looking ddwn ott the earth frottt heaVtett; Hearing the cry of the prisoner, making frfefe those htf whbm death is drdered.

ACKNOWLEDGEMENTS

I would like to thank the following for their contribution towards the success of this work

- My wife, Sylvia, for as sure as day follows night her love and support were constant and like an even keel in choppy seas her encouragement has seen me through this work.

- My family for their prayers and material support throughout the duration of my studies.

- My supervisors Dr. M. D. Joshi and Dr. E. Karari for their valued insight and direction in making this work a success

- Dr. Danstort Mukoko to whom I owe all my knowledge of statistical analysis and for professional analysis of the data.

- To the staff at the Department of clinical Medicine and Therapeutics laboratory for their hard work and cooperation.

- Members of the heart failure journal club, started by Dr. Joshi, without whom f ly understanding of heart failure would at best be rudimentary.

- John Spertus. drtd the developers of the KCCQ for allowing use of this tool free of charge for this study. (Appendix 2)

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List of Abbreviations

ACE - I - Angiotensin converting enzyme inhibitor

ARB - Angiotensin Receptor Blocker

BB - Beta blocker

CCB - Calcium Channel Blocker

CHD - Coronary Artery Disease

CHF - Chronic Heart failure

EF - Ejection Fraction

eGFR - estimated Glomerular Filtration rate

HF - Heart Failure

KCCQ - Kansas City Cardiomyopathy Questionnaire

LVEF - Left Ventricular Ejection Fraction

NYHA - New York Heart Association

KNH - Kenyatta National Hospital

ABSTRACT

Objective: To ascertain the mortality and rehospitalization rates among patients discharged with a diagnosis of congestive cardiac failure.

Design: Prospective observational study

Setting: Kenyatta National Hospital, Nairobi, Kenya.

Subjects: one hundred and fifty five (155) consecutive medical ward discharges with an admission diagnosis of congestive cardiac failure meeting Framingham criteria.

Results: The study was conducted from July 2008 to March 2009. One hundred and fifty five patients were recruited and followed up for a mean duration of 138 days. 55.3% were female and the average age of the population was 47years. On admission, 91% were in NYHA class 3 and 4, 31% had valvular heart disease, mainly rheumatic valve disease while 69% had non valvular disease. 40% had the admission during the study period as the index admission while 60% had had one or more than one previous admissions. During follow up, 35 patients died out of 138 giving a 4 - 6 months mortality rate of 25.4% (95% CI 18.1 -32.6). When 17 patients lost to follow up are included in the analysis and presumed dead, the mortality rate is 37.7% (95% CI 29.6-45.8). The average time to death was 72.2 days. When categorized into nonvalvular and valvular heart disease patients, the mortality rate is 21.3% (95% CI 13.1-29.5) and 40.5% (95% CI 28.7-52.3) respectively. Among non valvular heart disease patients, significant predictors of increased mortality were a duration of heart failure diagnosis or symptoms of less than 30days prior to admission (O.R 7.0,95% CI 2-25, p = 0.001) and an elevated urea level on admission (O.R 5.0, 95% CI 1.42-17.24, p=0.008). Among valvular heart disease patients only a NYHA class 3 or 4 at 4-12 weeks post discharge was associated with increased mortality (O.R 10, 95% CI 1.2-100, p=O.OI7). 38% of the population was rehospitalized. The rehospitalization rate per patient was 1.29. The time to rehospitalization was 69.8 days (±60.28) and 29.5% of the population was rehospitalized once while 6.2% and 2.3% were rehospitalized twice and three times respectively. The rate of death or first rehospitalization was 49.2% (95% CI 40.6-57.8). Significant predictors of increased rehospitalization among non valvular heart disease patients included a low potassium level on admission (OR 6.15, 95% CI 1.9-21.3, p=0.001), not receiving an ACE-I on clinic review at 4-12weeks (OR 4.1, 95% CI 1.3-12.9, p=0.013) and receiving aldactone on discharge (OR 3.22, 95% CI 1.12-9.23, p=0.026) and on clinic review (OR 9.3, 95% CI 1.13-76.9, p=0.015).

Conclusion-, the 4 - 6 month mortality of patients with CHF is 25-38% in our population while the rehospitalization rate is 38%. Various factors including a shorter duration of CHF prior to admission, hyperuriceamia on admission and poor NYHA score are associated with increased mortality. Factors associated with increased rehospitalization include hypokalemia on admission, not receiving an ACE-I on discharge and use of aldactone.

1. INTRODUCTION AND LITERATURE REVIEW

1.1 BACKGROUND

HF is defined as a pathophysiologic condition in which impaired cardiac performance is responsible for the heart's inability, at normal filling pressures, to increase cardiac output proportionally to meet the patient's metabolic demands.

The burden of cardiovascular disease varies considerably between regions of the world. Such disease remains the number one killer in Europe and North America, accounting for almost 50% of deaths, but in developing countries it ranks third and represents about 25% of all deaths.[1] In sub Saharan Africa, the mortality rates from cardiovascular diseases are likely to remain low but are almost equal to the mortality rates from parasitic and infectious diseases[2]. Chronic heart failure (CHF) has a very high prevalence, and is expected to double by the year 2030[J,4] and commands an increasing percentage of health care resources[5].

In the last decade, heart failure (HF) has emerged as a major health problem in developed countries imposing a major burden on the health care systems[6]. European data suggests that approximately 1% of national health care budgets is spent on individuals with heart failure[7]. Estimates of the crude incidence of heart failure in developed countries range from 1 to 5 cases per 1000 per year with a crude prevalence ranging from 3 to 20 per 1000[8]. Projections to the year 2020 predict that deaths from cardiovascular disease will exceed deaths from infectious and parasitic diseases for the first time in all world regions except sub-Saharan Africa[6,9] leading to a global cardiovascular disease epidemic. At least two forces are at work: control of infectious, parasitic, and nutritional diseases in developing countries; and demographic changes in the structure of many countries' populations with more people reaching the age at which cardiovascular disease manifests[10].

In Africa studies indicate that cardiovascular diseases account for 7-10% of all medical admissions to African hospitals, with heart failure contributing to 3-7% of these admissions". 98% of heart failure cases are due to non-ischaemic causes, with hypertensive heart disease, rheumatic heart disease, and cardiomyopathy accounting for 65% of cases. The common causes of heart failure in sub-Saharan Africa, such as rheumatic heart disease, peripartum cardiomyopathy (PCM),'" and endomyocardial fibrosis (EMF)[10] present before middle age, whereas in developed regions of the world, heart failure is a disease of the elderly, with an average age of 76 years.[1J] * The early presentation of heart failure in Africans has the potential to undermine national productivity as a consequence of the number of active life years lost by the most productive segment of the population. Infectious diseases remain a major ca^se of heart failure in Africans. The contribution of cor-pulmonale and pericarditis to about 10% of cases of heart failure reflects the continuing impact of tuberculosis on heart disease. Cor- pulmonale is

mainly related to chronic post-tuberculosis lung disease, whereas pericarditis, which has been exacerbated by the HIV epidemic, is overwhelmingly due to active tuberculous involvement". Syphilitic heart disease has accounted for an average of 7% of cases of heart failure in the hospital series.[14,16]

1.2 PATHOPHYSIOLOGY OF HEART FAILURE

It has been thought that the heart responds to the reduced cardiac output by compensatory mechanisms affecting the Frank-Starling curve relationship, the thickness of the ventricular wall, and sympathetic nervous system functionality. HF can result from systolic dysfunction or diastolic dysfunction. The former is seen in two thirds of patients and is caused primarily by ischemic heart disease; patients with this type of dysfunction demonstrate a left ventricular ejection fraction (EF) < 40%. Diastolic dysfunction is seen in one third of patients and is commonly caused by hypertension, hypertrophic and infiltrative diseases, and possibly diabetes.

Once considered a simple problem of left ventricular (LV) pump dysfunction, heart failure has now come to be understood as a highly complex clinical syndrome that is manifested by many extracardiac features, including neuroendocrine activation and cytokine release. The hemodynamic model still applies to the treatment of acute heart failure in the hospital setting, however the neurohormonal paradigm is modeled on the prevention of the progression of heart failure, which is largely an outpatient approach.[1']

Reflex activation of hormones is not simply a compensatory mechanism, but also leads to apoptosis, endothelial dysfunction, decreased vasodilator capacity, abnormal redistribution of blood, ventricular remodeling and dysfunction and many other problems that are detrimental to the patient. The major neurohormonal systems that are activated in HF are the renin-angiotensin system, the sympathetic nervous system, endothelin pathway, natriuretic peptides, and tissue necrosis factor.

Heart failure usually begins after an index event which may be defined as any event affecting the heart muscle that leads to decrease in myocyte mass, reduced contractility or change in heart morphology and eventually reduction in cardiac output.[18] The disease then progresses through an asymptomatic phase whose length is variable. Random echocardiographic screening of healthy populations of men and women aged 25 to 74 years suggests that approximately 1.5% of the general population has asymptomatic LV dysfunction,'[9] but this finding is very age dependent.

Neurohormonal activation eventually leads to a convergence of myocyte hypertrophy, myocyte slippage, reactive and reparative fibrosis, cytoskeletal alterations, and apoptosis which are believed to ultimately modulate the size, shape, and stiffness of the heart, leading to progressive remodeling and the development of the syndrome of heart failure. Arrhythmogenesis, a hallmark of heart failure, is an inevitable consequence of these structural changes. Heart failure may therefore be largely a structural disorder, leading to diminished myocardial performance and

circulatory congestion. The biologic underpinnings of the structural changes are under intense study, but altered mechanical forces, neurohormones, and cytokines appear to play a prominent role."

Excessive sympathetic drive and activation of the renin- angiotensin system promote preferential efferent glomerular arteriolar constriction in the kidney. There is increased sodium reabsorption in the proximal tubule of the kidney and release of vasopressin from the posterior pituitary gland, leading to reduced free water clearance. Increased angiotensin II stimulates aldosterone synthesis, further enhancing sodium reabsorption and potassium excretion. As cardiac output gradually diminishes, a reduction in effective arterial blood volume results in further stimulation of the sympathetic nervous and renin-angiotensin-aldosterone systems. Renal blood flow diminishes. Intraglomerular hydraulic pressure and filtration fraction are usually protected by efferent arteriole constriction even as renal blood flow diminishes, but a vicious circle ultimately develops. The kidney attempts to restore adequate perfusion by avidly retaining more salt and water, and edema begins to ensue. The patient becomes severely "congested," a hallmark of the clinical syndrome.
As the heart progressively enlarges, coronary blood flow is limited by microvascular abnormalities and the high intramyocardial wall tension caused by the altered geometry of the now dilated and hypertrophied ventricle."[0] Capillary flow per unit of myocardial mass is reduced as the physical distances between capillaries and myocytes increase and a state of energy starvation ensues in the failing heart, reducing the capacity of oxidative phosphorylation.[21] Isoform shifts also occur in the enzymes of the heart that are normally responsible for energy production, and this also likely contributes to a state of energy starvation.[22]

Other circulating peptides that are increased include the endothelins, which are likely important in the pathogenesis of heart failure.[2,24] Endothelin-l is associated with growth of both cardiac myocytes and the interstitial matrix, and engages in a complex interplay with the myocardium."[5] Inducible Nitric Oxide Synthase may be expressed in the failing heart[26] and may contribute to a reduced contractile state by increasing intracellular cyclic guanosinc monophosphate[2'] and by reducing sensitivity to beta-adrenergic stimulation. Reduced vascular NO production may contribute to impaired vasodilation and exercise intolerance. The understanding of the role that nitric oxide plays in the pathophysiology of heart failure continues to evolve but is currently not well understood.

1.3 TREATMENT OF HEART FAILURE

Surveys suggest that treatment of heart failure in daily practice differs from guidelines and is characterised especially by the underuse of recommended medications."[s] Since the publication of the first trials, the prescription rates of the various therapeutic categories demonstrated to be effective in terms of the reduction of mortality and morbidity, especially angiotensin-converting enzyme (ACE) inhibitors and beta-blockers, have gradually increased[2']. However, according to the most recent data, these prescription rates are still far from optimal.[20]

In spite of a number of trials showing significant improvements in survival and reductions in hospital admissions for patients with HF, irrespective of aetiology, who received ACE inhibitors, many clinical surveys conducted in different countries revealed that a substantial proportion of patients who should have been treated with an ACE inhibitor were not receiving that treatment, both in primary care and in hospital practice.

The aldosterone antagonist, spironolactone, has been found to be convincingly beneficial in HF. In patients with severe HF, already receiving a diuretic and ACE inhibitor, spironolactone improved survival and reduced hospital admissions, compared to placebo, irrespective of aetiology.

Four large clinical trials, published between 1999 and 2001, showed that the addition of a beta blocker to standard treatment with a diuretic and ACE-inhibitor, led to a significant increase in survival, reduction in hospital admissions and improvement in symptoms and well-being, irrespective of aetiology [33,34-35,36].

Angiotensin receptor blockers (ARBs) are the most recent class of drug to be shown to be of benefit in HF. Several large trials have shown morbidity and mortality benefits in patients with mild-moderately severe HF treated with an ARB, beta blocker and, in a minority of cases, spironolactone, irrespective of aetiology [37,38,39-40].

1.4 MORTALITY IN HEART FAILURE

A wide variety of factors are reported to be associated with an increased risk of hospital admission or death, including demographic factors (for example, male sex and single marital status), clinical characteristics (lower systolic blood pressure, renal dysfunction), history of heart failure (previous hospital admissions), and comorbidity (diabetes and depression).

Despite major advances in the treatment of congestive heart failure, such as introduction of ACE-inhibitors, b-blockers, aldosterone antagonists, * and angiotensin receptor blockers, mortality and morbidity in HF remain unacceptably high. In contemporary studies of unselected HF patients admitted to hospital, the one year mortality rate was 25% or greater.[42] Hospitalisation for HF is distressing for the patient and their family.[43]

In Cameroon, in a descriptive study entitled "A new look at adult chronic heart failure in Africa in the age of the Doppler echocardiography", done at Yaunde General Hospital in Cameroon, carried out from October 1998 to November 2001, one hundred and sixty-seven patients with heart failure were enrolled. It was found that heart failure was the reason for 5.77% of all hospital admissions. Rehospitalisation rate was 8.33%, the prevalence of heart failure was 30% and overall mortality was 9.03%. 44% of patients were in class III of the NYHA and 7% in class IV.[44]

In a retrospective analysis of the medical records of 1055 consecutive patients presenting to a consultant cardiologist private practice in Jamaica between January 2002 and March 2003, most were over 65 years of age, female, never smoked cigarettes, were overweight/obese and hypertensive (82%). The main aetiologies were hypertension (54%) and ischaemic heart disease (1HD) (26%).The most commonly prescribed medications at one month were ACEIs (91%), beta blockers (88%) and loop diuretics (55%). The survival at one year was 81%.[4:>]

The EPICAL study, a prospective, observational, community-based, epidemiological evaluation of advanced CHF conducted in Lorainne region of France in 1994, identified 499 patients with a hospital admission for heart failure with NYHA class III and IV and followed them up for 18 months for re -admission to hospital or mortality. Causes of CHF included Coronary Heart Disease (CHD) (n = 231; 46.3%) (History of MI in 202 patients [87.4%]) and non-CHD (n = 268; 53.6%) (Congenital heart disease [n = 3], valvular heart disease [n = 4], dilated cardiomyopathy [n = 214], unknown causes [n = 47]). One-year mortality rate was 35.4% and the rate of mortality and/or re-admission to hospital was 81%. Patients were admitted to hospital 2.05 times per year (64% of these for worsening heart failure).[46]

In a prospective study in the Netherlands including 152 patients with heart failure, with 18 months follow up period, 51 (34%) of the 152 heart failure patients died (mortality at six and 12 months, 26 (17%)and 43 (28%), respectively). The cause of death was known in 35 of these patients: they all died from a cardiovascular cause (for example, terminal heart failure, sudden cardiac death). Independent predictors of mortality were diabetes mellitus, a history of renal dysfunction (or higher creatinine), New York Heart Association (NYHA) functional class III or IV, lower weight or body mass index, lower blood pressure, ankle oedema and higher scores on a disease specific quality of life questionnaire. The use of beta blockers was predictive of a better prognosis. The average age of the study participants was 69yrs.[4]

In the USA, heart failure mortality data were derived from the National Health and Nutrition Examination Survey- 1 (NHANES-1) Epidemiologic Follow-up Study (1982 to 1986). Mortality at 10 and 15 years for those persons with congestive heart failure was found to increase in a graded fashion with advancing age, with men more likely to die than women. In the group greater than or equal to 55 years old, the 15-year total mortality rate was 39.1% for women and 71.8% for men.[48]

In Canada, a population-based study on Life expectancy after an index hospitalization for patients with heart failure found that hospitalized patients with heart failure had a lyear mortality of 33.1% and 5-year mortality of 68.7% and a median survival of 2.4 years. When stratified as per the EFFECT HF risk score, mortality varied substantially across risk groups such that median survival was only 8 months for patients in the high-risk group and only 3 months in the very high risk group. Among patients with depressed left ventricular ejection fraction (<30%) (LVEF),

median survival was only 6 and 3 months in the high- and very high risk groups, respectively. Among patients with HF with depressed LVEF. 1-year mortality was 33.3% and 5-year mortality was 65.8%. These patients with low LVEF had a mean age of 79yrs. 33.6% were female and 46.8% had a history of a prior myocardial infarction." '

1.5 REHOSPITAL1ZATION IN HEART FAILURE

Almost one-third of patients with HF have New York Heart Association functional class III or IV heart failure, often characterized by progressive deterioration and frequent hospital admissions. In the United States of America annual expenditures for heart failure have been estimated to be as high as $38 billion, of which $23 billion is for hospital stays. The incidence of hospital admission for heart failure is increasing rapidly, and readmissions for heart failure occur within months of the index hospital period for over 30% of patients. Readmission costs consume a proportional share (30%) of the overall inpatient costs, and even more in patients with the greatest degree of chronic functional impairment[50].

Generally, 25-50% of hospitalised HF patients will be readmitted within 6 months after discharge[51]. The most frequent causes of worsening HF symptoms and readmission are poor drug compliance, poor compliance with fluid restriction, or insufficient medical therapy/[8] In addition to these findings, it has repeatedly been shown that a substantial number of patients with left ventricular dysfunction are not treated with ACE-inhibitors and b-blockers.[4C'] If the drugs are given, they are often prescribed in doses that are lower than the doses proven to be effective. In fact, it has been reported that more than 50% of acute HF admissions are preventable, at least in theory.[40] One possible explanation for under-utilisation of proven medications may be that introduction and up-titration of ACE-inhibitors and b-blockers can be time-consuming tasks that are often difficult for physicians or clinics not dedicated to treatment of cardiovascular disease.[30]-[53]-[54] Also.

it is clear that modem HF therapy is complex and may lead to unwanted side effects or interactions, especially in inexperienced hands. Such side effects are generally more common in clinical practice than reported in randomised clinical trials as recently shown for spironolactone.[55] From several non-randomised and randomised clinical studies it seems well documented that treatment in specialised HF clinics using nurse intervention reduces readmission frequency and improves quality of care for HF patients.[56]

1.6 RENAL INSUFFICIENCY AND HEART FAILURE

Patients with moderate and severe renal insufficiency have been relatively underrepresented or excluded from clinical trials. Evidence has therefore been inadequate to guide the management of patients with heart failure and renal insufficiency.[57]

Decreased renal function has consistently been found to be an independent risk factor for cardiovascular (CV) disease outcomes and all-cause mortality in a large spectrum of CV disease patients, including those with left ventricular systolic dysfunction and chronic heart failure

(CHF).[58-59] In terms of clinical application, renal function may potentially be a stronger predictor of clinical events than left ventricular ejection fraction (LVEF).

In the Candesartan in Heart FailurerAssessment of Reduction in Mortality and Morbidity (CHARM) study, North American cohort, the proportion of patients with eGFR less than 60 mL/min per 1.73 m^2 was 36.0%. There was a stepwise increase in the cumulative incidence of CV death or admission to hospital for heart failure across successively lower quintiles of eGFR. The investigators found no interaction between eGFR and LVEF. indicating that eGFR and cardiac function had effects that were independent in terms of predicting the primary end point of death or admission to hospital for heart failure.[60]

Renal dysfunction might be a marker of general vascular disease and therefore possibly reflects severity of atherosclerosis in both kidney and heart.[61,62]

In the Studies of Left Ventricular Dysfunction (SOLVD) Treatment trial, one third of outpatients with moderate heart failure had an estimated glomerular filtration rate (GFR) less than 60 mL/min per 1.73 m^2. Renal insufficiency was associated with a 40% increased risk of death in the SOLVD trials.[59]

In a study that included patients hospitalized and readmitted to a large community teaching hospital in USA for CHF exacerbation within 30 days between January 7, 2005 and June 30, 2006, a total of 58 patients were responsible for 79 readmissions. Forty-five percent (45%) of all readmitted patients had underlying chronic renal insufficiency/failure (CRI/CRF) compared with 26% of CHF patients who were not readmitted within 30 days.[6']

Renal function is important for the management of heart failure because several important medications, including ACE inhibitors, ARBs, spironolactone, and digoxin, may be associated with an increased risk for adverse effects in patients with renal insufficiency.

1.7 QUALITY OF LIFE MEASUREMENT IN HEART FAILURE

Heart failure is associated with poor health-related quality of life (HRQL) and increasing dependency. Over the past decade, there has been a growth in the use of quality-of- life measurements as an indicator of health outcome. Quality of life reflects the way a person's mental and physical well-being is evident in their everyday life. HRQL measures the effects of an illness or a treatment from the patient's perspective. Quality of life in CHF may be impaired by physical symptoms, psychological problems, adverse treatment effects and social limitations. These factors may lead to individuals withdrawing from activities and previous social contacts and losing their social relations and social support[64].

Disease specific measures of quality of life

Disease specific measures of quality of life have been used to increase the responsiveness of the measures to the patients being studied. Disease-specific instruments have been developed to be suitable to the problems associated with a specific medical condition. CHF patients usually have other comorbid conditions, which generic instruments may not detect. If only generic instruments such as the SF-36 are used to assess differences in, or changes to, HRQL in CHF patients, then the probability of making an incorrect conclusion is altered, in an unpredictable manner. The choice of quality-of-life instrument should be based on issues relating to the ability to demonstrate reliability and validity to change over time or the psychometric properties of the measure[64].

Traditionally, in CHF, the New York Heart Association (NYHA) classification system has been used to assess functional status. This scale assesses a combination of physical symptoms and limitations. The NYHA is the most widely used system, but it has been shown to be unresponsive to change, has a high degree of inter observer variability and the perspective is that of the doctor rather than of the patient[63].

Various disease-specific questionnaires have been designed to obtain information about quality of life in patients with heart failure. These include, the Quality of Life in Severe Heart Failure Questionnaire (QLQ-SHF; [66]), the Chronic Heart Failure Questionnaire (CHQ;[67]), the Kansas City Cardiomyopathy Questionnaire (KCCQ;[65]), the Left Ventricular Dysfunction Questionnaire (LVD-36;[68]) and the Minnesota Living with Heart failure Questionnaire (MLHFQ;[69]). According to the literature, the three most commonly used are the QLQ-SHF, CHQ and MLHFQ [70].

The MLHFQ was designed specifically for use in heart failure. It assesses the patients' perception of the effects of CHF on the physical, socioeconomic and psychological aspects of their life. Patients respond to 21 items using a six-point Likert scale (0-5). The questionnaire is easy to administer, short and easily understood. It can be administered by interview, self-administered or by postal questionnaire. The measure has been found to be valid in comparison with other health outcome scales[71]. It has been shown to discriminate between patients with CHF and those with symptomatic left ventricular dysfunction. However, it does not distinguish well between different severities of CHF. Test-retest technique found that initial low scores tended to increase and initial high scores tended to decrease. This suggests that regression to the mean is operating. Estimated mean MLHFQ scores were 21,37, 53 and 69 in NYHA class I, II, III and IV respectively. A study that used a new standardized questionnaire to classify patients into NYHA classes reported that class III and IV patients had mean scores of 57 and 69 respectively[72].

The Kansas City Cardiomyopathy Questionnaire (KCCQ) (appendix 1) is a, self-administered, 23-item questionnaire developed to provide a better description of HRQoL in patients with CHF. It quantifies, clinically relevant domains in a disease-specific fashion: physical limitations (question 1), symptoms (frequency [questions 3, 5, 7 and 9], severity [questions 4, 6 and 8] and

change over time [question 2]), self-efficacy and knowledge (questions 11,12), social interference (question 16) and QoL (questions 13-15). The last item of the QoL domain (question 15) was adapted from the Mental Health Inventory of the SF-36 because it is a marker of depression, an important prognostic variable in cardiovascular disease. The KCCQ has been validated and predictable and significant relationships with the best available criterion standards were demonstrated for each individual domain of the KCCQ.[63]

The KCCQ is scored by assigning each response an ordinal value, beginning with I for the response that implies the lowest level of functioning and summing items within each domain. Missing values within each domain are assigned the average of the answered items within that same domain. Scale scores are transformed to a 0 to 100 range by subtracting the lowest possible scale score, dividing by the range of the scale and multiplying by 100. To facilitate interpretability, two summary scores were developed. Combining the physical limitation and symptom domains (excluding symptom stability) forms a functional status score. A clinical summary score can be calculated by combining the functional status with the QoL and social limitation domains[65].

1.8 ASSESSMENT OF SELF CARE BEHAVIOUR IN HEART FAILURE

It is generally recognised that heart failure patients have to learn to live with the consequences of the disease and treatment, which means: comply with a regimen concerning medication, diet and exercise, monitor symptoms, and seek assistance when symptoms occur. It is believed that an improvement in outcomes depends on patients' abilities to care for themselves and manage the consequences of their condition. Heart failure management programmes aim at improving patients' self-care, making them experts in heart failure self-management and teaching them to recognise deterioration and take relevant actions in case of exacerbation.

Patients' self-care abilities are often far from optimal. Carlson et al'[3] studied 139 patients who were primarily elderly, male, retired, unmarried, and earning less than $20,000 annually. Most of the group had multiple HF symptoms during the past year, yet their knowledge of the importance of signs and symptoms was poor and many misperceptions were evident. Recognition of changes in signs and symptoms was difficult for most patients, but easier for those more experienced with HF. Experienced patients were more likely to use appropriate self-care remedies than newly diagnosed patients. Few patients were comfortable evaluating the effectiveness of their self-care actions and most had low self-confidence in their ability to perform self-care.

Ni and co-workers assessed self-care needs in 113 patients with heart failure. Approximately 40% of the patients did not recognise the importance of weighing themselves daily and 27% weighed themselves twice per month. 37.2% of patients with HF did not recognize weight gain as a problem. Although 80% of the patients knew they should limit their salt intake, only one-third always avoided salty foods. Additionally, 36% believed they should drink a lot of fluids.[74]

Krumholz HM et al[7]" found that a formal education and support intervention substantially reduced adverse clinical outcomes and costs for patients with HF. Among the 88 patients (44 intervention and 44 control) in the study, 25 patients (56.8%) in the intervention group and 36 patients (81.8%) in the control group had at least one readmission or died during one-year follow-up (relative risk = 0.69, 95% confidence interval [CI]: 0.52, 0.92; p = 0.01). The intervention was associated with a 39% decrease in the total number of readmissions (intervention group: 49 readmissions; control group: 80 readmissions, p = 0.06). After adjusting for clinical and demographic characteristics, the intervention group had a significantly lower risk of readmission compared with the control group (hazard ratio = 0.56, 95% CI: 0.32, 0.96; p = 0.03) and hospital readmission costs of $7,515 less per patient.

2.0 STUDY JUSTIFICATION

Heart failure is a common condition leading to high rates of morbidity and mortality worldwide. It is a chronic condition whose mainstay of treatment should be in the outpatient setting. Data is lacking concerning the management of chronic heart failure in our cardiology clinic. In addition, we have no information on the incidence of morbidity and mortality in our setting. Data on CHF morbidity and mortality in our clinic would be vital tool in planning for enhanced health service delivery to CHF patients.

3.0 OBJECTIVES

Broad objective

To determine the ambulatory mortality and morbidity of patients discharged with a diagnosis of heart failure from Kenyatta National Hospital.

Primary Specific Objectives

1. To determine the four to six months all cause mortality rate in patients discharged from the KNH with a diagnosis of heart failure.
2. To determine the 4 - 6 months all cause hospitalization rate.

Secondary Specific Objectives

1. To determine levels of sodium, potassium, creatinine and urea at on clinic review.
2. To determine health related quality of life at 4-12 weeks post discharge using Kansas City Cardiomyopathy Questionnaire domains (KCCQ)
3. To relate the QOL with mortality at 4 - 6 months.
4. To relate QOL with re-hospitalization at 4 - 6 months.
5. To ascertain physician prescribing habits in terms of heart failure medication type and dose at 4 - 12 weeks post discharge and compare with medication type and dose at discharge and correlate these to adherence to the European Society of cardiology drug and optimal dose guidelines on treatment of heart failure.
6. To ascertain patient adherence to non pharmacological management of heart failure at 4 - 12 weeks

4.0 METHODOLOGY

4.1 Site

The cardiology clinic at Kenyatta National Hospital, a National Referral Hospital in Nairobi, Kenya

4.2 Study population

Medical ward discharges with a primary diagnosis of heart failure and on follow up at K.NH cardiology clinic

43 Study design

Prospective observational study

4.4 Case definition

Inclusion criteria

- Patient discharged from KNH medical wards with a primary diagnosis of Heart Failure
- Patient electing to undergo follow up at the K.NH cardiology clinic
- Patients with a reliable telephone contact
- Age equal to or above 13 yrs
- Informed written consent or ascent

Exclusion criteria

- Patients with co-morbid illnesses who may not survive till study completion or are at risk of re admission due to the cormobidity, including, advanced cancer, severe liver disease, severe chronic obstructive pulmonary disease, end stage renal disease an stroke
- Patients not able to co-operate in data collection including those with dementia and psychosis.

4.5 Sampling Technique

Consecutive sampling technique was utilised.

4.6 Screening and Recruitment

Patients who were admitted to the medical wards with a diagnosis of heart failure and survived until discharge were recruited for this study. Patients were screened by the primary investigator once the ward doctor certified them to be discharged. If they met the inclusion criteria, they were recruited after obtaining written informed consent. Telephone contacts of the patient and any other relatives or caregivers were then collected and recorded on the data collection card. Upon discharge, the principal investigator established telephone contact and followed the patients through the cardiology clinic from 4-12 weeks post discharge where relevant data was collected as per the study proforma. Mortality and rehospitalization were assessed at 4- 6 months post discharge by telephone contact.

4.7 Sample Size

Sample size was calculated according to the following equation for sample size in a prevalence study:

$$n = \frac{Z^2 P (1 - P)}{d^2}$$

where:

n = sample size required

Z = Z score of required confidence interval (1.96)

P = likely value of parameter

d = relative error

therefore, substituting for an assumed mortality of 10% and 95% confidence interval, the calculated sample size was 138 patients. It was assumed that 15% of patients recruited would be lost to follow up and adjusting for this loss brought the sample size to 160 patients.

4.8 Study Feasibility

Given an approximate 4% (1993 study prevalence 3.3%, Oyoo G. O et al) prevalence of heart failure in patients admitted to KNH medical wards and with an average daily admission of 50 patients, and allowing for 20% in hospital mortality, it was estimated that we could recruit 50 patients every month and achieve the sample size in 3 months.

5.0 DATA COLLECTION

Patient telephone contact or that of a reliable guardian was obtained and recorded by the Primary Investigator (PI) prior to the patient leaving the hospital at discharge. At 3 weeks post discharge, the PI contacted the patient or their guardian via telephone, reminded them to attend their scheduled clinic at **4 - 6** weeks and obtained information on any readmissions to any hospital for any cause or information on mortality where applicable.

At 4-12 weeks post discharge, the PI confirmed the attendance of the patient to the cardiology clinic by telephone contact and proceeded to trace the patient at the clinic. After being seen by the doctor at the clinic the PI would see the patient and the following would be recorded in the study proforma

1. Patient weight - taken with the patient standing on a weighing scale without heavy attire. The weight obtained will be approximated to the nearest 0.5kg.

2. Heart rate - measured using a stethoscope placed on the patient's precodium. This was done 5 min after any activity and was repeated twice at 5 min intervals and the average recorded.

3. Blood pressure - measured using a validated sphygmomanometer. It was taken on the right arm with the patient lying supine and propped up in bed with arms at the same horizontal level as the trunk. An average of 3 readings were recorded.

4. Information of the duration of heart failure diagnosis or symptoms, present cormobidities, number of previous hospitalizations for heart failure (prior to the hospitalization at which the patients were recruited), aetiology of heart failure, ejection fraction and NYHA class, level of Na^+, K^+ and creatinine at the previous admission and medication at discharge was obtained from the patient file or discharge summary and recorded in the study proforma.

5. The K.CCQ was administered (appendix 1). The questionnaire was administered by the PI by face to face interview and in the language most understood by the patient. Relatives will not be allowed to contribute to the responses.

6. Open ended questioning was used to assess current NYHA class.

7. Information was obtained from the prescription issued or patient file on medication prescribed on this visit.

8. Questionnaire was administered to find out level of adherence to non pharmacological management of heart failure (appendix 2). The questionnaire was administered by the PI by face to face interview and in the language most understood by the patient. Relatives were not allowed to contribute to the responses.

9. Blood was drawn to measure Na\ K\ urea and creatinine levels. 2 mis of blood were drawn into a plain bottle and carried to the Department of Medicine and Therapeutics clinical chemistry laboratory.

10. It was confirmed that telephone contact had not changed, and the patient and guardian were informed to alert the PI by telephone in case of death of the patient or readmission to any facility.

At 12 weeks post discharge the PI would establish contact the patient and collect information on death or readmission, and encourage them to attend clinic at the appointed time.

At 16, 20 and 24 weeks post discharge, the PI would call the patient / guardian and obtain information on death or readmission.

6.0 DATA MANAGEMENT

Data prospectively recorded into the study proforma was entered into computerized data entry sheets. Cleaning and verification was done on a weekly basis to ensure validation and completeness of the information. Data analysis will be presented in tables, graphs and pie charts. Statistical analysis will be conducted using the statistical package for social sciences version 11.5 software for windows.

Descriptive statistics such as frequency, proportions, mean, variance and standard deviation will be used in result presentation. Chi square will be used in the analysis of categorical variables and cross tabulation was used to compare differences between categorical variables and correlate these variables to the outcomes of mortality and rehospitalization. Student t-test will be used for analysis of continuous variables. To compare differences between levels of serum parameter at admission and on clinic review, paired t-test was used. Non parametric statistical methods were used when continuous data grossly deviated from normal.

Differences were considered significant when p value was <0.05.

7.0 ETHICAL CONSIDERATIONS

1. Approval was obtained from the Department of clinical Medicine and Therapeutics of the University of Nairobi and the Kenyatta National Hospital Scientific and Ethical committee before data collection begun.
2. Informed consent was obtained from all participants at the point of obtaining their telephone contact. For patients below 18yrs of age, an ascent was obtained
3. Patients were free to withdraw from the study at any point and were not discriminated against in any way.
4. Confidentiality was maintained by excluding patient names from the computerized data entry sheets and storing proforma in a secure location

5. Results of laboratory measurements were communicated back to the patient and inserted in the patient file.
6. Non pharmacological management of heart failure was taught to all patients and reinforced at all encounters after administering the relevant questionnaires.

8.0 STUDY FLOWCHART

PATIENTS WITH DISCHARGE DIAGNOSIS OF HEART FAILURE

Meet inclusion criteria; telephone follow up

Do not meet inclusion criteria; excluded

REVIEW IN CARDIOLOGY CLINIC AT 4 • 12WKS AND ADMINISTER QUESTIONAIRES. DRAW BLOOD

TELEPHONE FOLLOWUP AT 16, 20 AND 24 WEEKS POST DISCHARGE TO ASSESS MORTALITY AND REHOSPITALIZATION

9.0 RESULTS

Recruitment for the study begun on 14/07/2008 and ended on 14/12/2008. The mean duration of follow up was 138.69 (±54.57) days and a mode of 180 days.

During the study period, a total no. of 4380 patients were admitted to the medical wards. Of these patients 248 were discharged from the hospital with a primary diagnosis of congestive cardiac failure. Those who met study inclusion criteria were 155 and were recruited into the study. 17 patients were subsequently lost to follow up of which 11 seemed to have had their telephones disconnected. 4 did not pick up their telephones despite repeated attempts at calling and in 2 cases, the person on the other side claimed not to know the patient. For those lost to follow up, 13 did not attend first scheduled clinic visit while 4 were lost to follow up after attending the clinic visit. Of the patients for whom follow up for primary objectives was achieved 35 (25.4%) did not attend scheduled follow up clinic and therefore data is missing for their secondary outcomes.

9.1 Socio-demographic profiles

a. Sex

Males comprised 44.7% and females were 55.3% of the study population.

figure 1: Population Sex distribution

b. Age

The mean age of the study population was 47.06 years. Males had a mean age of 47.88 years while females had a mean age of 46.31 years. The mean ages between the two sexes was not

significantly different, p=0.662. The peak age group was from 20 to 29 years and comprised 17.2% of the total population while 33.5% of the population was more than 60 years of age. The bar graph below (figure 2) illustrates the age distribution according to age group and sex. There was a bimodal age distribution with an early peak at 20 - 40 years and a later peak at 50 - 70 years of age. Both peaks were dominated by female gender while the second male peak is seen to occur about 10 years later than the female peak.

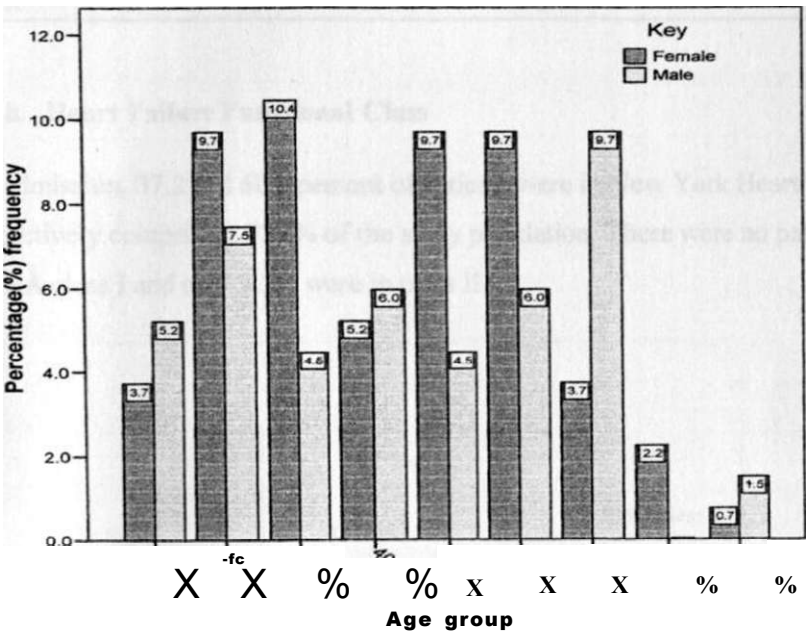

Figure 2: Distribution of study population by Age group and Sex.

9.2 Clinical Profile

a. Heart Failure Aetiology

The aetiology of heart failure could be classified as shown in table 1. It is notable that non valvular heart disease comprised 68% of the population studied. Rheumatic heart disease was the most prevalent single aetiology of heart failure comprising 31.8% of the population.

Table 1: Aetiology of Heart Failure

Aetiology	Frequency (%) n = 129
Rheumatic Heart Disease	31.8
Cardiomyopathies	26.4
Hypertensive Heart disease	21.7
Cor - pulmonale	8.5
Ischemic heart disease	3.9
Congenital	0.02
Other	0.06

b. **Heart Failure Functional Class**

On admission, 37.2 and 60.5 percent of patients were in New York Heart Class III and IV respectively comprising 97.7% of the study population. There were no patients admitted in NYHA class I and only 2.3% were in class II.

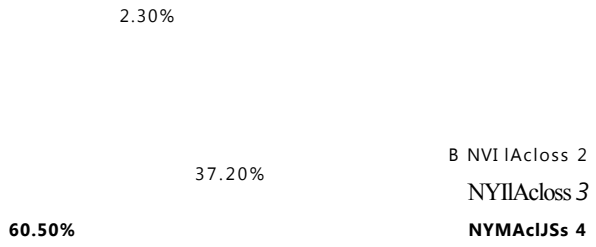

Figure 3: New York Heart association Class on admission

c. Ejection Fraction

62.8% had an ejection fraction more than 40% while 37.2 % had an ejection fraction less than 40%. When only non-valvular heart disease cases are considered, the ejection fraction was less than 40% in 50% of this population.

d. Renal Function tests, electrolytes and haemoglobin levels

The mean levels of sodium, potassium, creatinine and urea were 134.6mmol/l (±10.8), 4.1mmol/l (±0.92), 135.8 mmol/1 (142.6) and 11,05mmol/l (±12.6) respectively. The average Haemoglobin (Hb) level on admission was 12.3g/dl (±2.6). When stratified by sex, males had an average Hb of 13.1 g/dl while females had an average Hb of 11.9g/dl, and this difference in heamoglobin levels was significant, $p < 0.05$. A large proportion of the study population had deranged serum parameters as illustrated in table 2 below.

Table 2: Table showing levels of serum parameters on admission for the whole population

	Mean (mmol/1)	On admission, n (%)
Creatinine (n=113)	**135.81(±142.6)**	
Creatinine > 100mmol/l		65 (57.5)
Urea (n=109)	**11.05 (±12.6)**	
Urea > 8.3mmol/1		48 (44.0)
Sodium (n=lll)	**134.6 (±10.81)**	
< 135mmol/l		45 (40.5)
135-145 mmol/1		53 (47.7)
> 145mmol/l		13(11.7)
Potassium (n=110)	**4.1 (±0.9)**	
< 3.5mmol/l		20(18.2)
3.5-5.5mmol/l		88 (80.0)
> 5.5mmol/l		2 (1.8)

9.3 Morbidity characteristics

The mean duration of hospital stay was 10.4 days with a mode of 4 days and a range of 1 to 55 days.

a. Prior hospitalizations for heart failure

Of the patients recruited in this study 40.9 percent had the admission to hospital as the index hospitalization for heart failure, 28.3% had 1 previous hospitalization for heart failure, 12.6% and 18.1% had two and more than two previous hospitalizations for heart failure respectively.

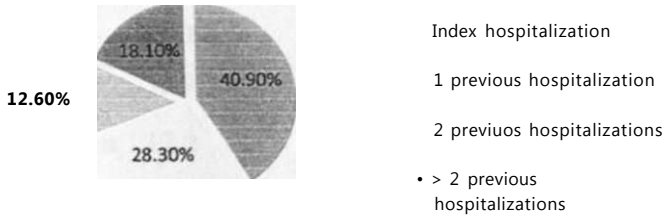

Figure 4: Relative distribution of previous hospitalizations for the whole population

When only patients with non-valvular heart disease are considered, 47.1% had no previous hospitalizations for heart failure while 25.3%, 11.5% and 16.1% had one, two and more than two previous hospitalizations for heart failure.

b. Duration of diagnosis or symptoms of heart failure

The average duration of heart failure diagnosis or symptoms for this population was 30.3 months with a mode of 1 month. Patients who had symptoms of heart failure for 30 days or less comprised 33.9% of the population while those with symptoms or a diagnosis of heart failure for more than 30 days to 90 days was 12.2%. The table below (table 3) illustrates the stratification of the study population according to the duration of heart failure diagnosis.

Table 3: Table showing stratification of study Population according to the duration of Heart Failure.

Duration	Population (%)	Valvular heart disease n=35(%)	Non-valvular Heart disease n=79(%)	P value
Less than 30 days	33.9	7 (20.0)	31 (39.2)	
30 days to less than 90 days	12.2	3 (8.6)	11 (13.9)	
90 days to less than 180 days	8.7	2 (5.7)	8(10.11)	Y p=0.06
180 days to less than 360 days	3.5	1 (2.9)	3 (3.8)	
More than 360	41.7	22 (62.9)	22 (32.9)	

When divided into valvular disease and non-valvular disease populations, the proportion of patients with valvular heart disease with duration of heart failure diagnosis or symptoms of less than 1 year was 37.1% compared to 67.1% in the non-valvular heart disease population. This difference was statistically significant, p=0.003 (table **4).**

Table 4: Table showing stratification of study Population according to a duration of Heart Failure less than or more than one year.

	Valvular heart disease, n =35 (%)	Non valvular Heart disease, n=79 (%)	P value
Less than 1 year	13 (37.1)	53 (67.1)	Y p = 0.003
More than 1 year	22(62.9)	26(32.9)	

c. Presence of Cormobities

54.7% of the population did not have any cormobidities as ascertained from history given by the patients. 32.8 % had one cormobidity while 12.5 % had at least 2 cormobities. Diabetes was

present in 15(11.7%) patients while hypertension was seen in 35 (27.3%) patients in the population. Other cormobities were as represented in table 5 below.

Table 5: Table showing prevalent Cormobities in the study population

Cormobidity	**Frequency (%)**	**Valvular heart disease (%); n=39**	**Non valvular heart disease**
No cormobidity	54.7	76.9	43.7
Hypertension	27.2	2.6	39
Diabetes Mellitus	11.7	0	17.2
HIV	9.4	10.3	9.2
Asthma/COPD	2.3	0	2.3
Malignancy	0.8	0	1.1
Others	4.7	10.3	2.3

10.0 PRIMARY OUTCOMES- MORTALITY

10.1 Follow up Period

The average duration of follow up was 138.7 days (± 54.57) with a median of 144.5 days and a mode of 180days. The minimum duration was 3 days and the maximum was 250 days giving a range of 247 days.

10.2 Mortality rate

Mortality rate for the whole study population, based on 52 patients presumed dead over four to six months of follow up was 33.5% (95% CI 26.6-41.3). When 17 patients who were lost to follow up are excluded from this analysis, mortality rate becomes 25.4% (95% CI 18.1-32.6). The average duration from hospital discharge to death was 72.2(±54.1)days, with a mode of 30 days, median of 52.5 days and a range of 3 to 205 days.

Non valvular heart disease patients had a mortality rate of 21.3%(95% CI 13.1-29.5) while valvular heart disease patients had a mortality rate of 40.5%(95% CI 28.7-52.2).

78.6% of valvular heart disease patients died within the first 120 days after discharge and this was comparable to 73.7% of non valvular heart disease deaths within the same period.(Table 6)

Table 6: Table showing duration from discharge to death according to type of heart disease

	<30	30-90	90-120	120-150	>150
Valvular (n =19)	**7 (36.8%)**	**3 (15.8%)**	**4(21.1%)**	**3(15.8%)**	**2(10.5%)**
Non Valvular (n=14)	**4 (28.6%)**	**7(50.0%)**	**0**	**2(14.3%)**	**1(7.1%)**

A duration of heart failure diagnosis or symptoms prior to hospitalization of 30 days or less was associated with a 4.2 times increased mortality compared to those with longer duration of heart failure diagnosis or symptoms and this association was significant, 95% CI 1.67-11.11, p=0.032. 59% of deceased patients had heart failure symptoms for less than 30 days prior to hospitalization.

A serum Urea level of more than 8.3mmol/l was associated with 2.5 times increased mortality compared to those with a serum urea of less than 8.3mmol/l on admission and this association was significant, 95% CI 1.05-5.9, p=0.036. This was more evident in patients with non valvular heart disease where urea of more than 8.3mmol/l was associated with 38.2% mortality (OR 5.0, 95% CI 1.42-17.24, p=0.008) while in valvular heart disease the association between increased urea levels and mortality did not reach statistical significance (OR 1.38, 95% CI 0.32-8.09, p=0.667).

In both valvular and non valvular disease categories there was no association between mortality and age, duration of hospital stay, heart failure aetiology, number of previous admissions, number of cormobidities, ejection fraction, NYHA class on admission, type of drug prescribed at discharge, levels of creatinine, sodium, potassium and haemoglobin on admission.

The NYHA class on clinic review was significantly associated with outcome of mortality in patients with valvular heart disease. Being in NYHA class III or IV on clinic review was associated with a 10 times increased risk of mortality compared to being in NYHA class I or II (OR 10, 95% CI 1.2-100.0,p = 0.017).

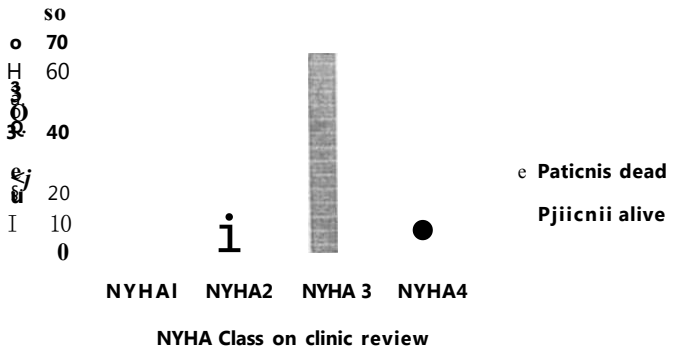

Figure 5: Relationship between NYHA on clinic review and Survival

11.0 PRIMARY OUTCOMES -REHOSPITALIZATION

A total of 49 (38%, 95% CI 29.62-46.38) patients were rehospitalized during the follow up period and these accounted for 63 rehospitalizations. The average number of rehospitalizations per patient rehospitalized was 1.29. Among patients studied, 38 (29.5%) were rehospitalized once while 8 (6.2%) and 3 (2.3%) patients were rehospitalized twice and three times respectively. The average duration from discharge to first rehospitalization was 69.8(±60.28) days.

When the rate of death or first rehospitalization are analyzed as a composite endpoint, we found that 49.2%(95% CI 40.6-57.8) of the population studied either died or were rehospitalized within the first 4 - 6 months after discharge.

Among non valvular heart disease patients, the rate of rehospitalization was 33 %(95% CI 23.18-42.82). The mean duration from discharge to first rehospitalization was 83.93(±64.83) days with a median of 83 days and a range of 201 days.

In valvular heart disease patients, 47.4%(95% CI 23.18^2.82) were rehospitalized. The mean duration from discharge to rehospitalization was 50(±44.12) days with a median of 34 days, mode 24 days and a range of 142 days.

For the combined population, there was no association between rehospitalization and the variables of age, duration of heart failure symptoms or diagnosis, aetiology of heart failure, number of previous admissions, presence and number of cormobities, renal function, electrolytes, ejection fraction, NYHA at admission or drugs given at discharge.

However, when looking at the non valvular heart disease population, a potassium level on admission of < 3.5mmol/l was associated with increased rehosp^alization at a rate of 61.5% vs 24.1% for those with normal potassium levels (OR 6.15, 95% CI 1.9-21.27,p=0.001). A sodium level on clinic review but not on initial admission, of < 135mmol/l was also associated with increased rehospitalization whereby, all 3 patients who had a sodium level on clinic review of < 135mmol/l were rehospitalized while of the patients with normal sodium levels, only 25% were rehospitalized. This association between sodium level and rehospitalization was statistically significant, p=0.02. Receiving aldactone at discharge tended to be associated with increased rehospitalization at a rate of 76.9% versus 23.1% for patients not receiving aldactone (OR 3.22 95% CI 1.12-9.23,p=0.026). On clinic review, this association was even stronger, whereby receiving aldactone on clinic review was associated with a significant increase in rehospitalizations (OR 9.3, 95% CI 1.136-76.19, p=0.015). Of the patients who did not receive an ACE-I on clinic review 55.6% were rehospitalized versus only 23.4% of those who received an ACE-I (OR 4.1, 95% CI 1.29-12.98,p=0.013).

Table 7: Factors significantly associated with increased rehospitalization among non-valvular heart disease patients

K^r < 3.5 on adm.	6.15	0.001	1.9-21.27
No ACE-I on review	4.1	0.013	1.29-12.98
Rec. aldactone on discharge	3.22	0.026	1.12-9.23
Rec. Aldactone on review	9.3	0.015	1.136-76.9
Na* < 135 on review	N/A	0.02	N/A

For the patients with valvular heart disease only a urea level of > 8.3mmol/l at both admission and clinic review were associated with an increase in the rehospitalization rate. Of the patients with an elevated urea on admission 75.0% were rehospitalized compared to 35.0% of those with normal urea level on admission(OR 5.6, 95% CI 1.128-27.52,p=0.028). On clinic review 83.3% of patients with a high urea level of > 8.3mmol/l were rehospitalized compared to 33.3% of patients with a normal urea level of < 8.3mmol/l. This difference represented a 10.0 times increased risk of rehospitalization for patients with high urea levels on clinic review compared to those with normal urea levels and this was statistically significant, 95% CI 0.9-100,p=0.027.

12.0 SECONDARY OUTCOMES

The secondary outcomes are based on 103 (74.6%) patients who attended clinic for follow up. Those who attended clinic at 4 - 8 weeks post discharge were 78 (75.7%) patients while those who attended clinic at more than 8 weeks post discharge were 25 (24.35%) patients.

12.1 Measures of Creatinine, Urea, Sodium and Potassium levels on follow up

The mean creatinine level on clinic review was 108.8mmol/l while the mean urea, sodium and potassium levels were 7.09mmol/l, 139.8 mmol/1 and 4.2mmol/l respectively. On comparing these means with the mean levels at admission, there was a statistically significant difference in the levels of creatinine ($p < 0.01$), urea ($p < 0.05$) and sodium ($p < 0.01$)with the mean levels on review being less as illustrated in table 8 below.

Table 8: Table showing Mean change in serum parameters

	On admission (mmol/n	On clinic Review fmmol/h	P value (t - test)
Creatinine	139.42	109.65	0.001 (3.546)
Urea	10.37	7.47	0.031 (2.198)
Sodium	134.92	139.48	0.003 (-3.108)
Potassium	4.32	4.21	0.407 (0.835)

When considering only non valvular heart disease cases, the drop in creatinine level was from a mean of 130.7mmol/l on admission to a mean of 103.9mmol/l on clinic review ($p < 0.001$). The

mean urea levels for this subgroup was 10.73mmol/l on admission versus 7.7mmol/**l** on discharge (p=0.153) while the mean sodium levels were 134.8mmol/l on admission versus 139.4mmol/l on discharge (p=0.027).

For patients with valvular heart disease the changes in serum sodium, creatinine and urea were in a similar direction as that depicted above.

When comparing the proportion of patients with deranged serum parameters, patients with creatinine level more than 100mmol/l accounted for 60.5% of patients at admission but only 39.4% on clinic review. Patients with urea more than 8.3mmol/l were 41% versus 22.3% on admission and clinic review respectively. (Table 9).

Table 9: **Changes in serum parameters when categorized**

	On admission, n (%)	**On clinic review, n (%)**
Creatinine > 100mmol/l	49 (60.5)	37 (39.4)
Urea > 8.3mmol/l	32(41.0)	21 (22.3)
Sodium		
< 135mmol/l	29 (36.7)	5 (5.3)
135-145 mmol/1	38 (48.1)	79 (84.0)
> 145mmol/l	12(15.2)	10(10.6)
Potassium		
< 3.5mmol/l	11 (13.9)	12(12.8)
3.5 — 5.5mmol/l	66 (83.5)	78 (83.0)
> 5.5mmol/l	2(1.9)	4(4.3)

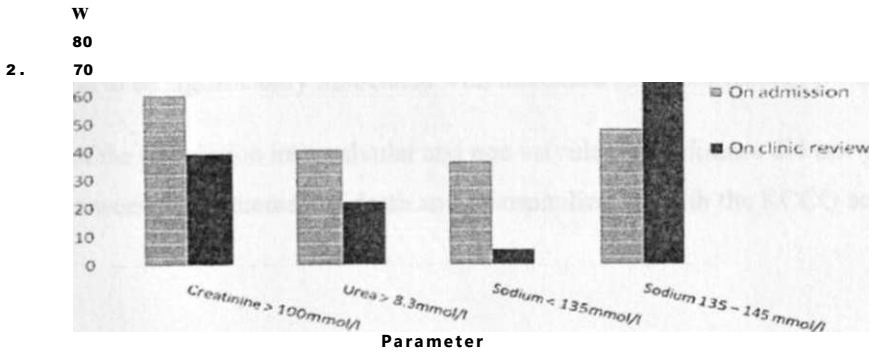

Figure 6: Change in population distribution within various serum parameter categories.

12.2 Health Related Quality of Life

The mean Kansas City Cardiomyopathy Questionnaire (KCCQ) overall summary score and overall clinical score for the population reviewed in the clinic were 50.35 and 59.13 respectively. When the overall summary score was categorized into good (>75), fair (50 - 74), poor (25 - 49) and worst (<25) scores, 17.2% had a good score while 28.3, 40.4 and 14.1 percent had a fair, poor and worst score respectively. (Table 10)

Table 10: Distribution of KCCQ scores by category

Category	0 - 25(worst) (%population)	>25 - 50(poor) (%population)	>50-75(Fair) ("/..population)	> 75 (good) (%population)
Overall summary score	14.1	40.4	28.3	17.2.
Overall Clinical score	9.1	29.3	32.3	29.3

The various KCCQ scores did not differ statistically with whether a patient was seen within 8 weeks after discharge or after 8 weeks post discharge.

KCCQ scores were not significantly associated with the outcome of mortality.

In comparing KCCQ scores with the rehospitalization rate, only a low KCCQ physical limitation score was found to be significantly associated with increased rehospitalization, p= 0.034.

Stratification of the population into valvular and non valvular heart disease did not yield any associations between the outcomes of death and rehospitalization with the KCCQ scores.

12.3 Drugs Prescribed

In the whole population Betablockers were prescribed to 49.6 % of patients on discharge as compared to 70.8% of patients on clinic review. A different trend was observed for ACE-I where they were prescribed to 78% of patients on discharge but only to 74% of patients on clinic review (table 11)

Table 11: Table showing classes of drugs prescribed for whole population

	On discharge (%)	On clinic review (%)
Beta Blockers	**49.6**	**70.8**
ACE - 1	**78**	**74**
ARB	**3.3**	**11.5**
Aldo - antagonist	**63.4**	**72.9**
Digoxin	**56.1**	**72.9**
Frusemide	**93.5**	**94.8**
CCB	**4.1**	**5.2**

When only patients with non - valvular heart disease are analyzed, those on betablockade at discharge were 51.2% while those on betablockade after first clinic review were 76.8%. A similar trend was observed for ACE-I, ARB and aldactone. (Figure 7)

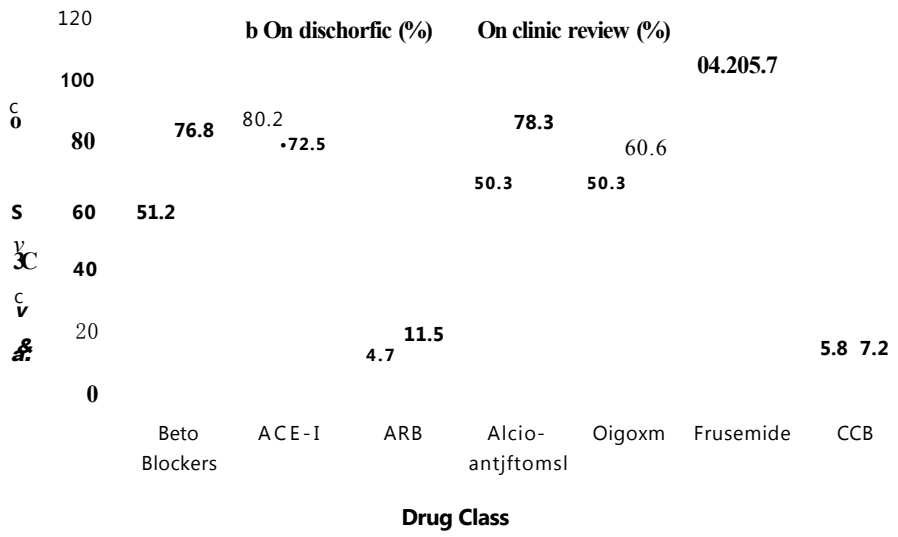

Figure 7: Drugs prescribed to patients with non-valvular heart disease

Among those for whom beta blockers were prescribed, 92.5% received carvedilol while 7.5% received atenolol while for those •» **ACfc-l were** prescribed, 85.7% received enalapril while only 14% received captopril. Those for whom an ARB was prescribed received lorsatan, while those on an aldosterone antagonist received aldactone and those on a diuretic received frusemide.

12.4 Drug doses in non valvular heart disease patients

Among those for whom carvedilol was prescribed, most (25.58%) received a total daily dose of 6.25mg upon discharge from hospital. However, upon clinic review, the majority of patients (36.23%) received an increased dose of 12.5mg per day. The doses of enalapril and captopril however remained almost unchanged between discharge and clinic review.(Figures 8,9 and 10)

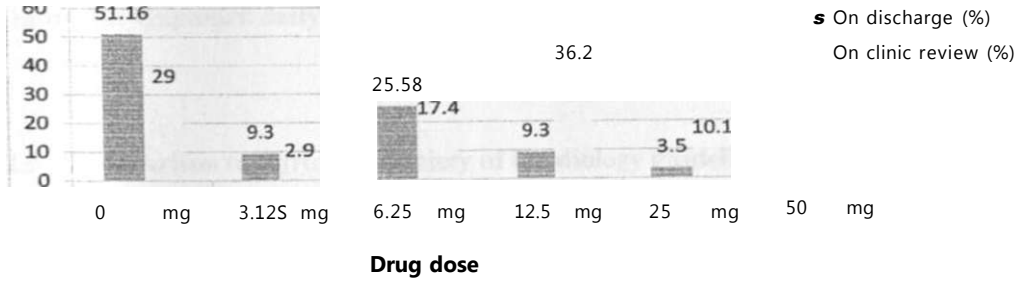

Figure 8: Carvedilol daily doses

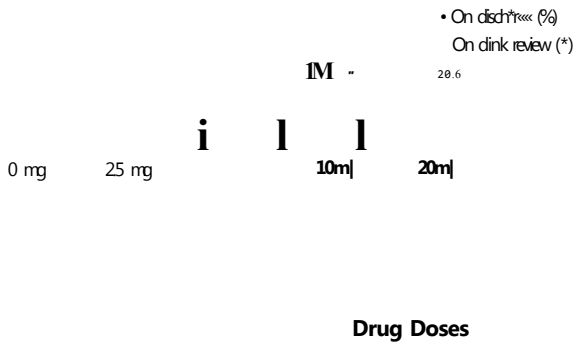

Figure 9: Enalapril daily doses

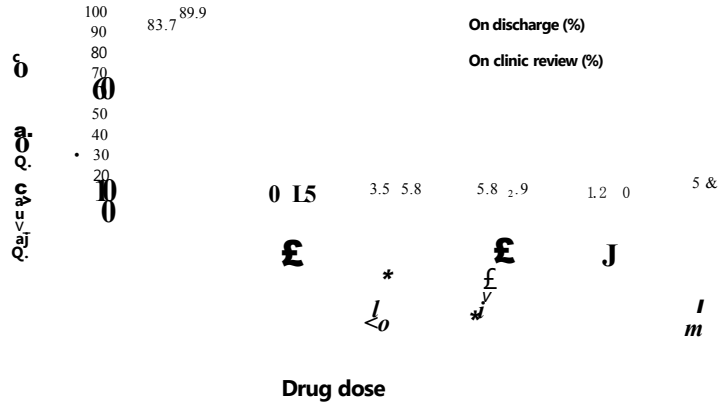

Figure 10: Captopril daily doses against percent population

12.5 Comparison to European Society of Cardiology guidelines

When comparing the drugs prescribed to non valvular heart disease patients to the European society of cardiology guidelines we find that beta blockers were prescribed to only 48.8% of the population therefore leaving a large proportion without the benefit of beta blockade. Of those for whom carvedilol was prescribed 52.3% received the recommended starting dose of 6.25mg per day. On clinic review, the proportion of patients on carvedilol had increased to 71%. On clinic review 24.5% of the patients were on 6.25mg of carvedilol per day representing 25% of the target dose while 50.9% of these were on 12.5mg per day which represents 50% of the target dose as per the ESC guidelines. Only 20.4% of those receiving carvedilol on clinic review had achieved the target dose as per the ESC guidelines.

According to ESC guidelines, all patients with heart failure are supposed to be on an ACE-I. In this study 78% received an ACE-I on discharge while this decreased to 74% on clinic review. For non valvular heart disease, this figures were 80.2% on discharge and 72.5% on clinic review. In non valvular heart disease patients, only 29.1% of those receiving an ACE-I on discharge received a dose equivalent to the ESC recommended starting dose of 5mg per day. 7.4% received

a total dose of 2.5mg per day while 36.4% and 27.2% received lOmg and 20mg per day respectively. On clinic review, only 33.3% had attained the target dose of 20-40mg per day.

This study had majority of patients in NYHA class 3 and 4 and as such, the ESC guidelines recommend that these patients should be on an aldosterone antagonist. On discharge, the patients on an aldosterone antagonist were only 59.3% while on clinic review this number was 78.3%. The doses prescribed ranged from 25mg to 50mg per day and this was in compliance to the ESC guidelines.

12.6 Adherence to non- pharmacological therapy

In the population that had a clinic review, 91.8% considered it fairly important or important to attend follow up appointments. Patients also attached great importance to use of medication, diet, cessation of smoking and cessation of alcohol use with 95.8%, 85%, 95% and 94% respectively reporting these activities to be important to very important. Exercise ranked somewhat lower with only 79% reporting that they considered exercise to be important in the management of heart failure.

In compliance to attendance of follow up appointments, 77% reported attending all follow up appointments while 15% and 8% reported their attendance to be most of the time and half the time respectively. Of those who did not attend all follow up appointments, 50% reported that lack of money was the main reason for non attendance while 41 % had other reasons of which the most common was that they did not know about the clinic date that they (patient) were supposed to attend.

67% of the population studied reported using medication all the time while 23% reported using medication most of the time. 60% of patients who reported not using medications all the time sited the high cost of medications as a main cause of non use, while 16.7% and 6.7% gave presence of side effects of the medicines and natural forgetfulness as the reasons for not taking medication.

As regards diet restrictions, 74% reported that they had reduced their salt intake while only 59% reported a reduction in their fluid intake.

As regards regular exercise, 75.3% reported that they never or seldom did any exercise. 98% of the population reported that they were not smokers and did not take alcohol. As regards weight monitoring, 93.8% rarely checked their weight while of those who checked their weight 98% took no action on finding that they had gained weight.

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DISCUSSSION

The prevalence of patients admitted with acute decompensated heart failure in this study was 7.5%. This figure represents a big proportion considering that communicable diseases are the main causes of hospitalization in our set up. Regionally, a study done in Lusaka Teaching hospital in Zambia in 1976 found a prevalence of 10.6%, while unpublished data by Baraza et al[7]" of a study done in KNH in 2008 gave a prevalence of 5.7%.

In this study, females comprised 55.3% of the population therefore indicating that acute decompensated heart failure is slightly more prevalent in the female gender. This was in keeping with studies done regionally in South Africa, *the Heart of Soweto study*, and by Barasa et al at KNH (unpublished data)[76]. Though the differences in sex distribution did not reach statistical significance, the higher female preponderance could be explained by several factors. One hypothesis could be that females in our population are more predisposed to heart failure and may be having more risk factors for development of heart disease. This may need to be studied further but is suggested by data from the heart of Soweto study[77]. Another reason for more female patients may be the fact that most patients attending KNH are females therefore causing a bias towards most CHF patients being female. The *Heart of Soweto study* \ a prospective cohort study carried out in South Africa between Jan 2006 and Dec 2006, found that female sex comprised 59% of the heart failure patients presenting to their hospital. Generally, females were found to have more risk factors for cardiovascular disease with obesity being highly prevalent in females.[78] Data from developed countries shows that heart failure is more predominant in the male population[79] and this may be in keeping with higher rates of hypertension and atherosclerosis in their male population when compared to their female population.

The mean age of the population studied was 47.06 yrs with there being no statistical difference in the age of the males versus the females at recruitment. This is similar to other studies done regionally including the *heart of Soweto study* where the mean age was 52.8 years. The population in this study was characterised by a bimodal age distribution with two peaks at 20 - 30 yrs and at 50 - 60 yrs with both age groups having a predominantly female composition. This bimodal age distribution can be explained by the fact that we have rheumatic heart disease as a major cause of heart failure in the younger age group of 20- 30 years while non valvular heart disease, mainly cardiomyopathy and hypertensive heart disease are more prevalent in the older

age group. In this study, the mean age of rheumatic heart disease patients was 28.83±10.9 years and 85.4% of them were aged below 39 years. The mean age of non valvular heart disease patients was 54.84 ±18.6 years with only 21.7% of the population being aged below 39 years. In comparing with western studies, the average age of the population with heart failure in Europe tends to be at least 2 decades older than that of our population[80]. This could be explained by the fact that with increasing age, risk factors for cardiovascular disease become more prevalent. However, in Africa, infection related causes of cardiac disease are still highly prevalent and more common in the younger population. Black Africans have also been shown to be more susceptible to hypertension induced cardiovascular disease as seen in the *heart ofSoweto study,* and this may explain the younger age of heart disease onset in our population.

In this study, rheumatic heart disease was the single main aetiological factor with a prevalence of 31.8%. Cardiomyopathy comprised 26.4% of the population while hypertensive heart disease comprised 21.7% of the population. The contribution of ischemic heart disease remained low at 3.9%. These features were comparable to those in a study done by Oyoo et al'° in Kenyatta National Hospital in 1999 where the prevalence of rheumatic heart disease was 32%. Cardiomyopathy 25.2%, hypertensive heart disease 17.6% and ischemic causes comprises 2.2%.

It is instructive to note that rheumatic heart disease (RHD) remains the single most prevalent aetiology. It is worrying that the prevalence of rheumatic heart disease has remained relatively constant in our population at 31.8% in this study, 31.9% in the study by Oyoo et al in 1993 and 27% in a study by Parmar et al in 2008 (unpublished data). This is a direct reflection of the failure of our health care systems to rein in rheumatic heart disease and to treat subsequent valve pathology that is usually amenable to surgery.

Meanwhile, the prevalence of cardiomyopathy and hypertensive heart disease far surpasses that of RHD. This could be due to effects of increasing urbanization and increasing prevalence of diabetes, hypertension and other lifestyle diseases in our population. The prevalence of ischemic heart disease may be grossly under reported in this study and indeed in most studies done regionally. This is because echocardiographic findings are mostly used to report on ischemic changes and this tool is not sensitive in diagnosing ischemic heart disease.

Regionally, the prevalence of rheumatic heart disease ranges from 38.3% as seen by *Baldachin el aF* in Zimbabwe in 1962 to the low of 24.6% as seen by *Kingue et at"* in Cameroon in 2005. In these same studies, that by *Baldachin et al* showed a 22.2% prevalence of hypertensive heart disease and a 7.3% prevalence of dilated Cardiomyopathy while that by *Kinque et al* showed a 54.5% prevalence of hypertensive heart disease and 26.3% prevalence of dilated cardiomyopathy.

60.5% of the patients admitted belonged to NYHA functional class 4 while 37.2% belonged to NYHA class 3 on admission. Oyoo et al in 1993 found that 52.6% of patients were in NYHA class 4 while 31.9% were in class 3. Similar findings are reported from Ghana.*[2] This is representative of patients having acute decompensated heart failure requiring hospital admission. These patients are usually very sick and require hospital admission to enhance bed rest and administer intravenous medications, mainly diuretics.

When the population of patients with non valvular disease is considered, 50% had an ejection fraction of less than 40%. This is a reflection of a high rate of diastolic heart failure in our population which may suggest that hypertension and by extension hypertensive heart disease may be a major cause of heart failure in our population.

In this study, derangements in serum electrolytes, urea and creatinine were common on admission with 57% of the population studied having elevated creatinine level, 44% having elevated urea, 40.5% having low sodium levels while • ;rangements in potassium levels were uncommon. These derangements were found to correct to normal levels for a large proportion of the patients on clinic review. *Heywoodet al"*[3]using data from the ADHERE registry found that 56.6% of patients admitted with a diagnosis of heart failure had elevated creatinine that represented moderate to severe renal dysfunction.

The population of patients recruited in this study was characterised by a high rate of previous hospitalizations with 59.1% having had previous hospitalization with a diagnosis of heart failure. This may have impacted on the results. Studies have shown that patients who have been rehospitalized before for heart failure are more likely to suffer subsequent rehospitalizations and that each subsequent rehospitalization increases the risk of death.[84]

46% of the population had had heart failure symptoms or diagnosis for less than 3 months. When stratified into valvular and non valvular disease cases, it becomes apparent that the non valvular heart disease patients had a shorter duration of illness prior to recruitment into this study with 67.1% having had disease for less than 1 year compared to 37.1% for valvular heart disease. This would be expected due to the fact that rheumatic heart disease tends to be more gradual in onset and is more chronic in nature, characterised by slow gradual deterioration.

23.1% of valvular heart disease patients had Cormobities compared to 56.3% of those with non valvular heart disease. The major Cormobities in non valvular heart disease patients were hypertension which comprised 39% of the population and diabetes mellitus in 17.2% of the population. HIV had a prevalence of 10%. Hypertension and diabetes are major risk factors for heart disease and their prevalence in our population is high. Without measures to control these diseases, it is likely that their prevalence will continue to grow leading to even greater incidence of heart disease in our population. In *the heart of Soweto study,* 44% of the population had hypertension while 10% had type 2 diabetes mellitus. The prevalence of HIV was 5%. Using western data from ADHERE registry, hypertension comprises 73% of cormobidity for patients with heart failure while Diabetes and Coronary artery disease constitute 44% and 57% respectively[85]. These data further explains the differences in aetiology and demographics of heart failure between Africa and in the developed countries.

This study reports a prevalent 5 month mortality of 37.7%. Mortality for non valvular heart disease was 21.3% while that for valvular heart disease was 40.5%. The higher mortality in valvular heart disease patients may be due to the fact that by the time valvular heart disease patients develop acute decompensated heart failure of NYHA grade III or IV requiring hospital admission, their cardiac function has deteriorated greatly and they may even be considered to be in end stage cardiac disease. Valvular heart disease patients in this population also had more prior hospitalizations and a longer duration of heart disease diagnosis or symptoms prior to recruitment thus increasing their risk of mortality.

Regionally, data on heart failure related mortality is mainly that of in hospital mortality and ranges from a high of 67.1% in a Nigerian population that was highly selected to involve patients with LVEF less than 25%[86] to low of 9.03%[92] in a Cameroonian population with 55%

Hypertensive heart disease and 26% and 25% consisting of cardiomyopathy and valvular heart disease respectively.

The 5 month overall mortality rate of 35% and the 21.3% mortality rate for non valvular heart disease in this study is high given the short duration of follow up. The 5 month mortality for non valvular heart disease in this study is higher than the 6 month mortality of 19.6% reported in a European study.*[7] The overall 5 month mortality in our setup of 35% approaches that of one year mortality of 33.1% given in a study conducted in Canada[50] which was representative of western data. This high mortality in our population could be reflective of multiple factors including greater severity of heart disease in our set up and the fact that being in a resource poor setting, care for heart failure patients is far from optimal. In the Canadian study, patients classified as having poor and very poor prognosis had a median survival of 3 to 8 months. A review of European data in the EPICAL study[4,] (1999) shows a one year mortality of 35.4%. This European population had a mean age of 64.6 yrs and had mainly coronary artery disease and cardiomyopathy with a mean LVEF of 22.4%.

The duration from discharge to death was 72.2 days with a mode of 30 days and median of 52.5 days. The difference in duration from discharge to death for valvular and non valvular heart disease patients was not statistically significant though on average valvular heart disease patients died 12 days earlier than non valvular heart disease patients. This may be indicative of the greater morbidity associated with the valvular heart disease patients at onset as described above.

Variables significantly associated with increased mortality included a shorter duration of heart failure diagnosis or symptoms, elevated urea levels and worse NYHA class on clinic review.

The association between a shorter duration of heart failure diagnosis or symptoms and increased mortality is puzzling and needs to further study. Possible reasons may be that these patients could be having undetected ischemic or thromboembolic events that lead to sudden death soon after discharge. This study did not analyse ECG characteristics and it is therefore hard to tell whether these patients who died soon after development of heart failure had ECG characteristics that predispose them to fatal arrhythmias. However, in studies done in Europe, patients with incident heart failure were found to have a high 1 month and 3 month mortality of 20% and 30% respectively. In this study, patients with a short duration of heart failure diagnosis or symptoms

were also more likely to have been having an index admission for heart failure at the point of recruitment. Studies have shown that heart failure related mortality is highest after the index admission. A patient with an index admission for heart failure is six times more likely to die immediately post discharge compared to a patient who has never been admitted but with similar class of heart failure. This risk reduces to 2 times increase at the end of 2 years without subsequent rehosp- alization. Subsequent rehospitalizations increase risk of death by 30%.[86] In this study, a large number of patients had prior hospitalizations thus putting them at reduced risk of death in this study as they can be classified as "long term heart failure survivors". This could mean that the death rate for incident heart failure may be much higher.

Elevated urea levels have been associated with increased mortality in other studies such as those by *Aronson el al*™ and *Douglas et a f*. Elevated blood urea nitrogen levels probably reflect the cumulative effects of hemodynamic and neurohormonal alterations that result in renal hypoperfusion.

The association between worse NYHA class on clinic review and mortality may indicate the progressive nature of heart failure. This association can easily be used to detect patients at high risk of death during clinic visits and therefore be able to offer more intensive therapy to this group. This relationship was also found to hold true in a study of mortality in unselected heart failure patients attending primary care physician offices in Switzerland[90].

This study reports a 5 month rehospitalization rate of 38%. The average duration from discharge to first rehospitalization was 69.8 days. Valvular heart disease patients had a 47.4% rehospitalization rate compared to 33% for non valvular disease. The possibility that valvular heart disease patients may have been at end stage cardiac disease, have had the illness for a longer period, and therefore have improved care seeking behavior with increased likelihood of seeking medical attention when they start feeling worse may explain this difference. Various factors can explain the increased rehospitalization rate in our study including a low level of formal education and the low social economic status of the study population[91], lack of adherence to medication and to non pharmacological management.

A European study[92] found a 36% annual rate of rehospitalization. Other studies give a range of 25 - 50% rehospitalization over a 6 month duration[52,93].

Among non valvular heart disease patients, a low level of potassium on admission, a low sodium level on clinic review but not on admission, not receiving an ACE-1 on clinic review and receiving aldactone at discharge and at clinic review were all associated with an increased rate of rehospitalization. The hypokalemia and hyponatremia may be an indicator of increased renin-angiotensin activation. It may be that these were an indicator of a greater reduction in cardiac output in those with hypokalemia and hyponatremia compared to those with normal potassium and sodium levels. Patients with congestive heart failure have marked deficits of total body and intracellular potassium, which may or may not be reflected by a measurable decrease in serum potassium concentration. The administration of diuretic drugs further depletes body stores of potassium (as well as magnesium) by promoting the renal excretion of these predominantly intracellular cations, an effect potentiated by coexistent hyperaldosteronism and- metabolic alkalosis. Furthermore, the high levels of circulating catecholamines in patients with heart failure may enhance the movement of potassium into cells thereby exacerbating the hypokalemic state and potentiating its arrhythmogenic effects[94]. A study by *Ali et* a/[95], also showed an association between low potassium levels and increased hospitalization and mortality in a group of outpatient clinic attendees with heart failure.

ACE-I have been shown to reduce mortality and rehospitalization in various randomized control trials[96]. It is therefore expected that failure to receive this all important class of drugs would be associated with increased morbidity.

The association between use of aldactone (spironolactone) both at discharge and on clinic review with increased rehospitalization is puzzling and warrants further study. In the *RALES*[32] study, aldactone was compared to placebo and found to reduce relative risk of mortality by 30% and that of rehospitalization by 35%. Studies done after the publication of the *RALES* study[97] showed increased rates of hyperkalemia that led to increased rehospitalizations therefore necessitating a dose of 25mg of spironolactone to be set as the optimum dose for patients with heart failure. In this study 88% of the patients were on 25mg per day dose. It could be that our population is more sensitive to the potassium retaining effects of aldactone and therefore remain susceptible to hyperkalemia even at this recommended dose. There may also be other poorly understood mechanisms in action.

Deranged levels of creatinine, urea, sodium and potassium have been associated with poor outcomes in patients with heart failure. In this study, we demonstrate a gradual correction of serum levels of these parameters when comparing values measured while in hospital and those measured post discharge. This trend is expected because the drugs prescribed are known to have a positive modulating effect on these parameters in the setting of heart failure. A study by *Ajayi et al* in Nigeria showed a gradual correction in sodium levels when patients were put on enalapril[9]". On further scrutiny of the results of this study, the proportion of patients with high potassium levels on admission (1.9% whole group and 2.7% non valvular group) is shown to more than double (4.3% whole group and 6.3% non valvular group) on clinic review. This may indicate that the use of combination treatment with ACE-1 and aldosterone antagonist may be causing hyperkalemia that may not have been statistically significant in this study due to the small number of patients involved[99]"*'

Instruments to measure quality of life are increasingly being used in clinical trials to assess patient well being from their own perspective. A disease specific quality of life instrument was utilized in this study to assess the patients' quality of life as relates to their heart failure. Studies done in Africa and assessing disease specific quality of life are very few and as such there are no questionnaires validated for that purpose in our setting. In this study, quality of life was measured using the disease specific Kansas City Cardiomyopathy Questionnaire (KCCQ). Poorer scores on this tool have been shown in the western countries to be associated with inflammatory activation and to predict worse long term outcome in patients with congestive cardiac failure. Furthermore KCCQ functional score has been shown to be independently associated with long term outcome[101].

In this study, the KCCQ scores were not predictive of outcome except the KCCQ physical limitation sub score in which lower scores were associated with increased rehospitalization. The lack of strong associations may be due to the fact that the KCCQ tool used had not yet been validated for our population. The KCCQ physical limitation sub score measures aspects such as dyspnoea and difficulty in completing various tasks due to dyspnoea or fatigue therefore lower scores indicate reduced physical functioning and this may be what precipitates rehospitalization.

Studies have shown that patients with heart failure benefit most from drug regimens that contain a beta blocker, an angiotensin converting enzyme inhibitor or angiotensin receptor blocker and an aldosterone antagonist among other drugs.[102]

In this study only 51.2% of non valvular heart disease patients received a beta blocker on discharge and this figure increased to 70.8% on clinic review. This shows that physician compliance to the ESC guidelines is extremely low as far as beta blocker use is concerned. The dose of beta blockers administered to the majority of patients is also low compared to the ESC recommended doses. The low rate of beta blocker prescription could be because physicians are usually hesitant to start beta blockers on in patients especially if limb oedema is still present. Studies have however shown that starting beta blockers in hospital before discharge increases the chances of continuing them post discharge without any increase in adverse effects.

An ACE-I was prescribed to 80.2% of patients with non valvular heart disease on discharge and to 72.5% on clinic review. This rate of prescription is encouraging and approximates complete physician adherence to the ESC guidelines. The dose of ACE inhibitor used was also appropriate in that most patients (92.7%) received either a dose equivalent to or more than the recommended doses on discharge. However, upon clinic review only 33.3% had attained the target doses of ACE-I as per the ESC guidelines. The reduction in prescription rate of ACE-I on clinic review could be due to patients being switched to an ARB due to side effects of the ACE-I, of which troublesome cough and angioneurotic edema tended to be the most common. However, despite this, the fact that of those who remained on ACE-I only 33.3% had attained the target dose after 8 - 1 2 weeks post discharge displays a very low rate of dose escalation among physicians. Our outpatient cardiology clinic is very busy and physicians may be tempted to just rewrite prescriptions rather than do a full evaluation of each patient and adjust doses accordingly. On the other hand, the population of patients we have may have been too sick to tolerate ACE-I dose escalations. Lack of knowledge among physicians that these drugs are supposed to be titrated upwards to a certain recommended dose may also have contributed to suboptimal dose escalation. Further studies can be done in this area to assess the exact reasons for suboptimal dose escalation.

Keeping in mind that most of the patients in this study were in NYHA class 3 and 4, an aldosterone antagonist has been shown to reduce mortality and hosj alizations and is therefore

recommended. Only 59.3% of patients with non-valvular heart disease received an aldosterone antagonist on discharge and this increased to 78.3% on clinic discharge. The reasons for non use of ACE-I may also apply in explaining the non use of aldactone.

Most patients (95%) received a diuretic (frusemide) and this is expected due to the fact that the most common signs of decompensation are those related to fluid overload. Digoxin was prescribed to 59.3% on discharge and 69.6% on clinic review. This study did not collect data on the reasons for digoxin prescription.

This study did not seek to ascertain the reasons for sub optimal dose escalation. It may well be that the doses achieved were the maximal tolerated doses for these patients. The ESC guidelines recommend that the dose of ACE-I and beta blockers should be gradually escalated every 2 - 4 weeks until the maximum tolerated dose or the target dose is achieved.

Regionally, a study conducted in Ghana in 2004[s4] showed the rate of ACE-I, beta blocker and aldosterone antagonist prescription on discharge to be 56%, 17% and 18% respectively. These rates are much lower than those seen in our study. Various factors may explain this including differing aetiologies of heart failure and inaccessibility of some drugs like ACE-I in Ghana at the time. Similarly, a study conducted in Malaysia in 2003[1W] only 47% of patients received ACE-I on discharge while less than 5% received spironolactone. At clinic follow up at 12 weeks, attendance was only 85% despite a telephone reminder and the prescription rate for ACE-I and spironolactone were significantly lower than at discharge. The extremely low use of beta blockers and aldosterone antagonists was attributed to limited medical experience and lack of funding. Patient factors contributed to the reduced clinic attendance.

A European study conducted from Nov 2002 to Jan 2004 showed that at discharge 63%, 67% and 29% of patients received a prescription for ACE-I, Beta blocker and aldosterone antagonist respectively[104]. These rates remain low compared to our study. However, the European study did not document the reasons for not receiving these drugs and we therefore cannot make a direct comparison.

Non pharmacological therapy is considered a mainstay in the management of heart failure. At recruitement into this study patients received verbal and written advice on reduction of salt

intake, reduction of fluid intake to 1.5 to 2 litres per day, to stop smoking, to stop alcohol use, maintain an exercise regime and to regularly weigh themselves with an aim of noting any rapid increase in weight that may be attributed to fluid overload. They were also adviced on how to notice early signs of decompensation and to take their medications all the time and attend clinic regularly.

Despite these instructions, only 77% reported attending all follow up appointments. Reasons for missing appointments were mainly lack of money to attend clinic and miscommunication during the discharge process as to when they were supposed to attend clinic. It is important that the discharge process be more regularized to avoid such miscommunication that ends up being very costly to the patient. Only 67% reported using medication all the time. Lack of money to buy the medicines was the main reason for not being adherent to medication. As regards the diet restrictions only 74% reduced their salt intake while only 59% reduced their fluid intake. The high rate of diuretic continuation after discharge from hospital and at relatively high doses may have contributed to difficulty in adhering to the recommendation on fluid intake reduction. Recommendations to have an exercise regime and to weigh themselves on a regular basis were not heeded by 75.3 and 98% of the patients respectively. This could be due to the fact that these patients had poor exercise tolerance and poor quality of life scores that could have inhibited their participation in exercise. Lack of structured programs to show such patients how to exercise may also contribute to this. The concept of exercising may also not be widely acceptable in the African culture. Most patients did not weigh themselves because they did not have access to a weighing scale.

A formal education and support intervention substantially reduces adverse clinical outcomes and costs for patients with Heart Failure.'[6]

Studies done in the area of non pharmacological management have focused on the effect of enhanced patient education and follow up on the outcomes of mortality, rehospitalization and quality of life. This study shows that despite patient education at recruitment, the level of adherence to non pharmacological management is low in our setting. This can be explained by various factors as outlined above. A system that encourages and allows comprehensive

management that includes formal patient education sessions would greatly impact positively on the mortality and morbidity associated with heart failure.

CONCLUSION

This study shows that the mortality and morbidity associated with chronic heart failure at Kenyatta National Hospital, Nairobi is high and exceeds that seen in the western countries. Factors shown to significantly increase mortality include a short duration of heart failure diagnosis or treatment, elevated urea levels and poor NYHA class o clinic review. Factors associated with increased rehospitalization include hypokalemia on admission, non use of an ACE-**I,** use of an aldosterone receptor blocker and hyponatremia on clinic review. Despite availability of evidence based medication, the utilization remains suboptimal. Non pharmacological management of heart failure is grossly underutilized in our set up and strategies need to be put in place to ensure patients are well educated about their condition and the information re-enforced at each clinic visit.

RECOMMENDATIONS

1. A larger cohort of patients needs to be assembled for follow up over a longer period of time to ascertain the long term outcomes of patients diagnosed with heart failure in our set up. This would also allow for a more comprehensive study that would identify predictors of short term and long term morbidity and mortality and therefore guide specialized care to high risk individuals..
2. A strategy looking at ways to enhance patient education and adherence to non pharmacological management needs to be devised and tested.
3. Questionnaires to assess quality of life in our set up need to be devised and validated as this may be an important and easy clinical tool that may predict outcomes.
4. A study assessing reasons for sub optimal prescription and dose escalation of evidence based medication should be carried out.
5. We need to devise strategies to minimize the impact of hypertension and diabetes as these are major aetiological factors for heart failure in our population.

LIMITATIONS

1. Data on the primary outcomes of death and hospitalization was based on telephone report from the patient or caregiver. The scope of the study was such that it was not possible to verify each report of death or hospitalization. However, it was assumed that no patient or their relative would intentionally give false information as regards the above two outcomes.
2. This was a study based in a referral hospital that is also a teaching hospital with most of the heart failure medication being available and accessible to the patients. As such, these results cannot be extrapolated to other hospitals within Kenya.
3. The fact that most patients are referred to KNH would mean that the population studied represents the most severe cases of heart failure in the region. However, KNH also serves as a primary care centre for Nairobi and its environs.
4. Patients received constant reminders on non pharmacological management whenever they had physical or telephone contact with the study investigator. This means that the rates of adherence to non pharmacological management may be grossly overstated compared to actual rates that may prevail in the population of patients with heart faihn^
5. This study focused on only a few of the possible predictors of a poor outcome once a patient is discharged from hospital.
6. The quality of life instrument utilized though modified for our situation was not validated for our population.

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'« Martin Scherer. Cordula Sobek, Dirk Wetzel. Janka Koschack
Changes in heart failure medications in patients hospitalised and discharged, *BMC ham.ly Practice* **2006, 7:69**

SCREENING PROFOMA

Hospital No.....**Ward— Age:** ____ **Sex:** ____ **Date**

Tick appropriately if present. Cross if absent

History

Orthopnea*

Paroxysmal nocturnal dyspnea*

Dyspnea on ordinary exertion

Nocturnal cough

Physical Findings

Heart rate > 120/min

Bilateral leg oedema

Raised jugular venous pressure*

S3 gallop*

Pulmonary rales *

Tender Hepatomegally

CXR

Cardiomegally*

Pulmonary oedema*

Pleural effusion

At least 2 major or 1 major (*Italic with **) and 2 minor criteria cannot be attributed to another medical condition = Heart Failure

Reliable telephone contact

CONSENT FORM

I, Dr Kuria Kamau. a postgraduate student in the department of Clinical Medicine and Therapeutics of the University of Nairobi, am conducting a study on chronic! art failure patients. This is a non interventional study looking at how patients with heart failure fair up to 6 months after discharge. We will specifically be concerned with finding out how often you are re admitted to hospital within this time period, we shall assess your quality of life using a questionnaire and by measuring your blood to assess your kidney function and we will also be finding out whether you will be alive at the end of the six months. Other things we will be looking at include how our doctors prescribe medication for heart failure and also how well you adhere to instructions on non pharmacological management of heart failure that you are given in the ward or clinic.

If you agree to participate in this study, I will access your file and get information on the cause of your heart failure and other clinical parameters related to your admission. I will also obtain a telephone contact from you or any relative or guardian (with your permission) and will use this telephone number to contact you while you are at home. I will also encourage you to attend clinics. At the clinic visits, I will see you and ask you questions regarding your condition using standard questionnaires. At 4 -12 weeks after your discharge, I will draw 2 mis of blood from your arm and sent it to the laboratory to evaluate your kidney function. There will be some pain when drawing the blood and you may experience some slight bleeding at the needle puncture site. It is not envisaged that you will suffer any severe adverse effects from this procedure. The results of this investigation will be availed in your file at the next visit.

Participation is free and you are free to withdraw at any time during the course of the study period. Your refusal to participate or withdrawal from the study at any point will not in any way affect the quality of your treatment.

All the information obtained will be treated confidentially.

I……of……understand the above consent explanation and voluntarily accept to participate in the study.

Signed
I confirm that I have explained to the patient the above statement
Signed……(interviewer) Name

)R KAMAU D.I

STUDY PROFORMA

PATIENT CODE:

1. **Patient details**
   a. **Name ...** **Telephone contact 1.....2.**
   b. **Date of discharge.....Today's date**
   c. **Duration post discharge (weeks)**
   d. **Hospital no**
   e. **Sex : Male. . . . . .Female**
   f. **Age**
   g. **District of birth**
   h. **Residence within past 5 years**
   i. **Formal education level (tick one)**
      i. **None**
      ii. **Primary**
      iii. **Secondary**
      iv. **Tertiary**
2. **Employment status**
   a. **Unemployed** I——I
   b. **Unskilled manual labourer** i I
   c. **Skilled labourer (carpenter, technician, fitter, driver etc)** I I
   d. **Trained, clerical, teacher, supervisor** I I
   e. **Professional (manager, lawyer, doctor ect)** I_____I
3. **File information at discharge**
   a. **aetiology of heart failure**

   i. ischaemic • ,ii. Hypertensive •, iii. dilated cardiomyopathy • , iv. alcoholic cardiomyopathy •, v. rheumatic heart disease •, vi. hiv cardiomyopathy • , vii. other Q

   b. **level of creatinine**
   c. **urea level**
   d. **level of sodium**
   e. **potassium level**
   f. **ejection fraction ,(tick as appropriate)**
      a. **< 40% (=1**
      b. **>40%** 1=11
   g. **Weight on admission.....On day 5_____ON discharge**
   h. **Leg oedema.....(+).....(-)**
   i. **Jaundice.....(+).....(-)**
   j. **Wasting.....(+).....(-)**

k. Medication prescribed at discharge

Class	P blocker	ACE-I	ARB	Digoxin	CCB	Aldo-antagoni St	Nitrate	diuretic	other
Drug									
dose									

4. Information at Current visit. Date

a. clinical parameters

i. Weight (kg)

ii. Heart rate (beats/min)

iii. Blood pressure (mmHg) systolic……diastolic

iv. Respiratory rate

b. New York Heart Association Classification

c. KCCQ administration (as per appendix 1)

d. current medications prescribed:

Class	P blocker	ACE-I	ARB	Digoxin	CCB	Aldo-antagonist	Nitrate	diuretic	other
Drug									
dose									

e. Adherence to non pharmacological management

Adherence questionnaire (adapted from heart failure compliance questionnaire)

A. As relates to your heart failure condition, how much importance do you attach to the following factors

	Not important			very important
1. Follow up appointments	**0**	**1 2**	**3**	**4**
2. Medications	**0**	**1 2**	**3**	**4**
3. Diet	**0**	**1 2**	**3**	**4**
4. Exercise	**0**	**1 2**	**3**	**4**

5. Smoking cessation	**0**	**1 2**	**3**	**4**
6. Alcohol cessation	**0**	**1 2**	**3**	**4**

B. How do you estimate your compliance to be in the following areas

1.	**None of the time**	**seldom**	**About half the time**	**Most of the time**	**All the time**
1 attend all Follow up appointments	**0**	1	**2**	**3**	**4**

What reasons would you give for not attending all follow up appointments?

a. I had no one to bring me i___I
b. I had no money 1 1
c. I was too sick I___I
d. I was feeling well I___I
e. 1 was admitted elsewhere l___I
f. Other

2.	**None of the time**	**seldom**	**About half the time**	**Most of the time**	**All the time**
1 use all Medication as required	**0**	**1**	**2**	**3**	**4**

What are some of the reasons for not taking medication? YES NO

a. Cost am czzi
b. Convenience 1 1 1 1
c. Side effects 1___I I___I
d. Too many drugs I 1 I 1
e. Satisfaction with current medicationl I I___I
f. Medication is not working I 1 I___1
g. Do not trust the doctor I___I I___1
h. Forgetfulness 1 1 I 1
i. Complicated instructions l___I I___I
j. Other

3.	**None of the time**	**seldom**	**About half the time**	**Most of the time**	**All the time**
1 follow the	**0**	**1**	**2**	**3**	**4**

Diet

restrictions

a. Have you reduced your salt intake YES |_____| NO

b. Have you reduced your fluid intake YES I 1 NO

	None of the time	seldom	About half the time	Most of the time	All the time
4.1 Exercise regularly	**0**	**1**	**2**	**3**	**4**
5.1 still Smoke	**4**	**3**	**2**	**1**	**0**
6.1 take Alcohol	**4**	**3**	**2**	**1**	**0**
7a. 1 check my weight	**Weekly**	**Every 2 weeks**	**Monthly**	**Every 2 months**	**rarely**

7b. what do you do when you find that you have gained weight?

a. Nothing
b. Increase my diuretic dose
c. Decrease my water intake
d. Decrease my salt intake
e. See a doctor |___1
f. Other_

THE KANSAS CITY CARDIOMYOPATHY QUESTIONNAIRE:

The following questions refer to your heart failure and how it may affect your life. Please read and complete the following questions. There are no right or wrong answers. Please mark the answer that best applies to you.

I. Heart failure affects different people in different ways. Some feel shortness of breath while others feel fatigue. Please indicate how much you are limited by heart failure (shortness of breath or fatigue) in your ability to do the following activities over the past 2 weeks

Place an X in one box on each line

Activity	Extremely	Quite a bit	Moderately	Slightly	Not at all	Limited for other reasons
	Limited	Limited	Limited	Limited	Limited	or did not do the activity
Dressing yourself	•	●	●	●	●	●
Showering/Bathing	•	●	●	●	●	Q
Walking 1 block on level ground	●	●	●	●	●	●
Doing yardwork, housework or carrying groceries	●	●	●	●	●	●
Climbing a flight of stairs without stopping	•	●	●	●	•	●
Hurrying or jogging (as if to catch a bus)	●	●	●	●	•	●

2. Compared with 2 weeks ago, have your symptoms of heart failure (shortness of breath, fatigue

Much worse	Slightly worse	Not changed	Slightly better	Much better	I have had no symptoms over the last 2 weeks
●	●	●	●	●	●

3. Over the past 2 weeks, how many times did you have swelling in your feet, ankles or legs when you woke up in the morning?

Every morning	3 or more times a week, but not every day	1-2 times a week	Less than once a week	Never over the past 2 weeks
●	●	●	●	●

4. Over the past 2 weeks, how much has swelling in your feet, ankles or legs bothered you? It has been ...

Extremely bothersome	Quite a bit bothersome	Moderately bothersome	Slightly bothersome	Not at all bothersome	I've had no swelling
●	●	●	●	●	●

5. Over the past 2 weeks, on average, how many times has fatigue limited your ability to do what you want?

All of the time	Several times per day	At least once a day	3 or more times per week but not every day	1-2 times per week	Less than once a week	Never over the past 2 weeks
●	•	●	●	●	●	●

6. Over the past 2 weeks, how much has your fatigue bothered you? It has been ...

Extremely bothersome	Quite a bit bothersome	Moderately bothersome	Slightly bothersome	Not at all bothersome	I've had no fatigue
●	●	Q	●	●	●

7. Over the past 2 weeks, on average, how many times has shortness of breath limited your ability to do what you wanted?

All of the time	Several times per day	At least once a day	3 or more times per week but not every day	1-2 times per week	Less than once a week	Never over the past 2 weeks
•	●	●	●	●	●	●

8. Over the past 2 weeks, how much has your shortness of breath bothered you?
It has been ...

Extremely bothersome	Quite a bit bothersome	Moderately bothersome	Slightly bothersome	Not at all bothersome	I've had no shortness of breath
●	●	●	●	●	●

9. Over the past 2 weeks, on average, how many times have you been forced to sleep sitting up in a chair or with at least 3 pillows to prop you up because of shortness of breath?

Every night	3 or more times a week, but not every day	1-2 times a week	Less than once a week	Never over the past 2 weeks
●	●	●	●	●

10. Heart failure symptoms can worsen for a number of reasons. How sure are you that you know what to do, or whom to call, if your heart failure gets worse?

Not at all sure	Not very sure	Somewhat sure	Mostly sure	Completely sure
●	●	●	●	●

11. How well do you understand what things you are able to do to keep your heart failure symptoms from getting worse? (for example, weighing yourself, eating a low salt diet, etc.)'

i Do not understand at all	Do not understand very well	Somewhat understand	Mostly understand	Completely understand
●	●	●	●	●

12. Over the past 2 weeks, how much has your heart failure limited your enjoyment of life?

It has extremely limited my enjoyment of life	It has limited my enjoyment of life quite a bit	It has moderately limited my enjoyment of life	It has slightly limited my enjoyment of life	It has not limited my enjoyment of life at all
●	●	●	●	● W 1

13. If you had to spend the rest of your life with your heart failure the way it is right now. how would you feel about this?

Not at all satisfied	Mostly dissatisfied	Somewhat satisfied	Mostly satisfied	Completely satisfied
●	Q	●	●	●

14. Over the past 2 weeks, how often have you felt discouraged or down in the dumps because of your heart failure?

I felt that way all of the time	I felt that way most of the time	I occasionally felt that way	I rarely felt that way	I never felt that way
●	●	●	●	●

15. How much does your heart failure affect your lifestyle? Please indicate how your heart failure may have limited your participation in the following activities over the past 2 weeks. Please place an X in one box on each line

Activity	Sever cty limite d	Limited quite a bit	Moderate iy limited	Slightly limited	Did not limit at all	Does not apply or did not do for other reasons
Hobbies, recreational activities	●	●	●	●	●	●
Working or doing household chores	●	●	●	●	●	●
Visiting family or friends out of your home	●	●	●	●	●	●
Intimate relationships with loved ones	●	●	●	●	●	●

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-i-- M ^m^m Cardiovascular
« . B WW M Outcomes Inc.

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10. Indemnification of Licensor. Licensee hereby agrees to hold Licensor harmless of and from and indemnifies it against any and all losses, liabilities, claims, damages and expenses (including attorneys' fees and expenses) which Licensor may incur or be obligated to pay, or for which it may become liable or be compelled to pay in any action, claim or proceeding for or by reason of any acts, whether of omission or commission, that may be claimed to be or are actually committed or suffered by Licensee arising out of Licensee's use of the Licensed Properties. The provisions of this paragraph and Licensee's obligations hereunder shall survive the expiration or termination of this Agreement.

XI. Indemnification of License**. Subject to Section 9 hereof. Licensor hereby agrees to hold Licensee harmless of and from and indemnifies it against any and all losses, liabilities, claims, damages and expenses (including attorneys' fees and expenses) which Licensee may incur or be obligated to pay, or for which it may become liable or compelled to pay in any action, claim or proceeding for or by reason of any breach of any representation, warranty or agreement on the part of Licensor under this Agreement.

12. Nondisclosure. During the term of this Agreement, the parties may have access to trade secrets, proprietary information, or other sensitive materials belonging to the other which are not generally known to the public ("Confidential Information"). During the term of this Agreement and for a period of five (5) years after termination or expiration hereof, the receiving party ("Recipient") agrees to maintain in trust and confidence all Confidential Information of the other party (the "Disclosing Party"). The Recipient agrees to safeguard the Confidential Information using the same standard of care it uses to protect its own Confidential Information. The Recipient will not disclose any Confidential Information to any third party, or make any use thereof other than as expressly permitted hereby, without the prior written consent of the Disclosing Party. As used herein. Confidential Information does not include any Information which the Recipient can demonstrate (i) was known to the Recipient or to the general public at the time of disclosure; (ii) was independently developed by the Recipient without the use of any of the Confidential Information; or (iii) was disclosed by a third party without violating any restriction or duty to the Disclosing Party.

13. Publications. Notwithstanding the general restrictions set forth in Section 12 above, the parties agree that publication of the results of research activities serves their mutual interests in improving the quality of health care. Accordingly, Licensee shall be free to publish the results of its research and development activities carried out with respect to the Licensed Properties and the Subject Study. Licensee agrees to refer to Licensor and the Licensed Properties in the bibliography section of the publication.

14. Term. Subject to the provisions of Section 15 hereof, this Agreement shall remain in effect from May 1, 2008 to March 1, 2009. Subsequent renewal of this Agreement shall be optionally available through application through the web site.

15. Licensor's Right to Terminate. Licensor shall have the right to immediately terminate this Agreement by giving written notice to Licensee in the event Licensee: (I) fails to perform any of its duties and obligations set forth herein, and the continuation thereof for thirty (30) days after notice; (II) files a petition in bankruptcy or is adjudicated a bankrupt or insolvent, or makes an assignment for the benefit of creditors; (iii) makes any use of the Licensed Properties not otherwise expressly permitted herein or (iv) the Subject Study is cancelled, abandoned, withdrawn or suspended. In such event. Licensee shall immediately cease and terminate its use of any of the rights granted hereby and shall, upon the request of Licensor, return to Licensor all records, copies, documents, media and files making use of the Licensed

Properties, or furnish evidence, satisfactory to licensor, of the destruction thereof.

16 Equitable Remedies. The parties further acknowledge that the breach, whether threatened or actual, of any of the terms hereof by Licensee shall result in immediate. Irreparable injury to Licensor and its goodwill and that accordingly. Licensor shall be entitled to apply for a preliminary and/or permanent Injunction to restrain the threatened or actual violation of the terms hereof by the Licensee or to compel specific performance of the terms and conditions of this License Agreement. Nothing set forth herein shall be construed as prohibiting the Licensor from pursuing any other remedies available for such breach or threatened breach, including the recovery of damages and costs incurred, together with attorneys' fees.

17. Miscellaneous.

a. This Agreement together with the exhibits hereto constitutes the entire understanding between the parties with respect to this Agreement. No change or modification of any of the provisions of this Agreement shall be effective unless memorialized by an instrument in writing signed by the parties hereto. All notices required or permitted to be given hereunder shall be given in writing, to the parties at their addresses set forth herein, or to such other address with respect to which notice has been given In accordance herewith. Whenever possible, each provision of this License Agreement shall be interpreted in such a manner as to be effective and valid under applicable law. If any covenant or other provision of this Agreement, or portion thereof, under circumstances not now contemplated by the parties, is Invalid, illegal or incapable of being enforced, by reason of any rule of law, administrative order, judicial decision or public policy, all other conditions and provisions of this Agreement shall, nevertheless, remain in full force and effect, and no covenant or provision shall be deemed dependent upon any other covenant or provision unless so expressed herein. The parties desire and consent that the court or other body making such determination shall, to the extent necessary to avoid any unenforceability, so reform such covenant, term, condition or other provision or portion of this Agreement to the minimum extent necessary so as to render the same enforceable in accordance with the intent herein expressed.

b. This Agreement shall inure to the benefit of Licensor, its successors and assigns. Licensee shall not have the right to assign this Agreement, or delegate its duties, by operation of law or otherwise, without first obtaining the written consent of Licensor.

c. This Agreement shall be governed by and construed in accordance with the laws of the State of Missouri.

IN WITNESS WHEREOF, the parties hereto have caused this Agreement to be duly executed as of the day and year first above mentioned.

CV **Outcomes, Inc.**

By: John Spertus
Title: President

University of Nairobi

By: DAVID KAMAU
Title: PRINCIPLE INVESTIGATOR
"Licensee"

SCHEDULE A: LICENSED PROPERTIES

KCCQ - English (UK)

This version of the KCCQ has been designed for English-speaking patients in the UK. This zip file includes two PDF files: the KCCQ itself and scoring instructions.

SCHEDULE B: DESCRIPTION OF STUDY

Project Name

Mortality and morbidity of patients with congestive heart failure at Kenyatta National Hospital

Project Type

Student Research

Project Dates

Start: May 1, 2008
End: March 1, 2009

Study duration: 304 days

Enrollment

Number of Sites: 1
Number of Subjects per Sue. 150
Total Enrollment: 150

Schedule of Use

Administer to subjects every 3 month
Total uses per subject: 3
Total uses during study: 450

Sponsor Type

non sponsored

Sponsor Name

david kamau

SCHEOULE C: LICENSE FEES & PAYMENT TERMS

Instrument License Fees

$0.00

Total License Fees for Study

S0.0C

Payment Terms

Payable on Receipt