AETIOLOGY, PHARMACO-THERAPEUTIC INTERVENTIONS AND CLINICAL-OUTCOME IN ACUTE DECOMPENSATED HEART FAILURE ADMISSIONS TO KENYATTA NATIONAL HOSPITAL.

BY

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A dissertation submitted in part-fulfilment of the requirement for the award of the degree of Master of Internal Medicine, University of Nairobi.

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This dissertation is my own original work and has not been presented for a degree in any other university.

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DEDICATIONS

To my father, late Dr. B. G. Parmar, mother Mrs. Neelima Parmar, my brother Rajeev Parmar, my wife Kavita and my son Yaj who gave me the inspiration to accomplish this study.

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LIST OF ABBREVIATIONS

ACC.....**American College of Cardiology**
ACE.....**Angiotensin Converting Enzyme**
ADHF.....**Acute Decompensated Heart Failure**
CCF.....**Congestive Cardiac Failure**
CRT.....**Cardiac Resynchronization Therapy**
AHA.....**American Heart Association**
ECHO.....**Echocardiogram**
EDV.....**End Diastolic Volume**
EF.....**Ejection Fraction**
FS.....**Fractional Shortening**
HF.....**Heart Failure**
HFnlEF.....**Heart Failure with normal Ejection Fraction**
IHD.....**Ischemic Heart Disease**
IVRT.....**Isovolumic Relaxation Time**
IVS.....**Inter Ventricular Septum**
KNH.....**Kenyatta National Hospital**
LVED.....**Left Ventricular End Diastolic**
LVES.....**Left Ventricular End Systolic**
LVPW.....**Left Ventricular Posterior Wall**
MI.....**Myocardial Infarction**
MR.....**Mitral Regurgitation**
MVA.....**Mitral Valve Area**
NYHA.....**New York Heart Association**
PI.....**Principal Investigator**
Pul. I.....**Pulmonary Incompetence**
QRS..... **Electrocardiographic wave**
RAAS.....**Renin-Angiotensin-Aldosterone System**
RHD.....**Rheumatic Heart Disease**
TR.....**Tricuspid Regurgitation**
USA.....**United States of America**
WHO.....**World Health Organization**

ABSTRACT

Objective: To characterize heart failure in patients admitted to medical ward at Kenyatta National Hospital, Nairobi.
Design: Prospective clinical observational register.
Setting: Medical ward, Kenyatta National Hospital.
Subjects: Two hundred and sixty one patients with acute decompensated heart failure were studied over a period of six months.
Results: 261 patients with an age range of 13-94 years and most were between 20-60 years with 59.1% females. 91% of ADHF cases were in classes III and IV. Non-compliance was the precipitating factor in 83.1% of the cases, followed by infective endocarditis at 8.5%. Out of these 40.9%, 27%, 18.9%, 18.1%, and 7.3% had dilated cardiomyopathy, rheumatic heart disease, Cor-Pulmonale, hypertensive heart disease, and Ischaemic heart disease respectively. The prevalence of heart failure with normal ejection fraction was 39.5% at an ejection fraction cut-off of 40%, average age was 49.3 ± 21.35 years with a statistical significance of $p<0.006$, with predominantly systolic heart failure in males at 61.7%. Among all the cases, 21 had a QRS duration of more than 120 milliseconds and 19 when rheumatic disease was excluded. Increase in QRS duration was noted with increase in age, with average age being 44.6 years, 62.1 years and 64·7 years in group less than 120 ms, 120 ms-160 ms and more than 160 ms, respectively ($p<0.01$) and ejection fractions were 38.5%, 43.8% and 30.05% respectively. The mean serum level of NT-proBNP among 50 cases at admission was 13752.36 pg/ml, and 369.3 pg/ml at discharge ($p<0.01$). At admission 49 out of 50 cases had serum level of more than 1800 pg/ml. At discharge, based on age related cut-offs, 10 patients were in the 'acute congestive cardiac failure likely' likely group while, 10 cases were in the 'less likely' group. On admission, Frusemide was used in 89% of cases, Digoxin in 77% and Spironolactone in 56.5% and on discharge Frusemide was used in 84.8% of cases, Digoxin in 72.3% and Spironolactone in 56% of the cases. Enalapril, Captopril and Carvedilol were used in less than 50% of the cases. The overall case fatality rate was 14.1%. The mean length of hospital stay was 7.1 days, the median was 6 days while the range was 2-29 days. The case fatality rates according to disease aetiologies were: rheumatic heart disease 14.5%; hypertensive heart disease 19.1%; ischaemic heart disease 26.3%; and dilated cardiomyopathy 9.7%.
Conclusion: Dilated cardiomyopathy was the most common cause of heart failure at 42.7%. 39.5% of the cases had heart failure with normal ejection fraction, more prevalent among older patients. Prolonged QRS duration was evident in older age groups. Enalapril, Captopril, and Carvedilol were used in less than 50% of heart failure cases. At discharge, 20% of the cases were in heart failure with a raised NT-proBNP level.

1. INTRODUCTION/LITERATURE REVIEW

Heart failure is a major public health problem worldwide. [1] The last two decades have witnessed a progressive and remarkable increase in the morbidity and mortality of heart failure, in association with a reduction in age-adjusted mortality due to cardiovascular events. This situation has been brought about by a real reduction in deaths from some cardiac diseases (acute coronary syndrome in particular), combined with increases in the number of survivors prone to develop heart failure and in the elderly population generally.

1.1 EPIDEMIOLOGY

The problem of heart failure has been recognized in sub-Saharan Africa for over sixty years.[2] Estimates of the crude incidence of heart failure in the general population of developed countries range from one to five cases per 1,000 per year, while the crude prevalence is 3–20 per 1,000.[3] No population-based incidence or prevalence studies of heart failure from Africa or other developing countries have been published.[4] Information on the epidemiology of heart failure in Africa is therefore obtained from hospital-based studies. These studies indicate that cardiovascular diseases account for 7–10% of all medical admissions to African hospitals, with heart failure contributing 3–7% of these admissions.[5] 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 diagnosis of myocardial infarction was made in only 2% of the cases, confirming the observation that coronary artery disease remains uncommon in Sub-Saharan Africa.[6,7,8] This is not surprising because the prevalence of risk factors for coronary artery disease, apart from hypertension, remains relatively low in many parts of the sub-Sahara.[9] It must be noted, however, that the diagnosis of myocardial infarction was made on the basis of electrocardiographic abnormalities in almost all the African studies done so far. This method may underestimate the true prevalence of coronary artery disease within a heart failure population.[10]

The common causes of heart failure in sub-Saharan Africa, such as rheumatic heart disease, peripartum cardiomyopathy (PCM) [11] and endomyocardial fibrosis (EMF)[12], present before middle age, whereas in developed regions of the world, heart failure is a disease of the elderly, occurring at an average age of 76 years.[13]

The Framingham Community Study identified 650 cases of heart failure over 40 years, from the late 1940s. The incidence increased steeply with age, from 3 per 1,000/ year in men aged 50-59 years to 27 per 1,000/year in men aged 80-89, with lower rates in women (age-adjusted odds ratio for men vs women : 1.7). [18]

In separate study of men born in 1913 in Gothenberg, Sweden, a prospective cohort study of men aged 50 to 67 years with a case definition unique to the study, reported an average annual incidence of manifest heart failure of 5.5 per 1,000. Case definitions were designed to be less strict than those of the Framingham study, to include milder cases of heart failure, but at the risk of including a higher rate of false positive diagnosis. [19]

In a population of 11,000 subjects aged 45 to 74 years in Eastern Finland, 60 cases of heart failure were identified over a 2-year period. Patients were diagnosed using a weighted scoring system based on symptoms, signs, and chest radiograph findings. Despite the narrow age range and small population, this study showed an increase in heart failure incidence with age and male gender, with an age-adjusted incidence ratio of 2:1.[20]

The United States National Discharge Registry Survey showed a constant increase in hospitalization due to heart failure. According to data published in 1999, covering the period from 1985 to 1995, the number of hospitalizations increased from 577,000 to 871,000 for a first-listed diagnosis, and from 1.7 to 2.6 million for any diagnosis of heart failure. Heart failure is the most frequent cause of death and hospitalization in those aged over 65.[21]

In the UK, a general practice study found a heart failure prevalence of 0.2% in the 45 to 64 age group, increasing to 1.9% in those over 65 years. An urban epidemiological study calculated a prevalence of 0.4% basing its estimates on diuretic prescribing rates. The Framingham Heart Study found a prevalence of 0.3% in subjects under 62 years old in a population of over 5,000. Follow-up over 34 years showed a prevalence of 0.8% in subjects 50 to 59 years old, and 9.1% in those over 80 years old.[22]

In Scotland, Stewart *et.al.* described heart failure hospitalization trends from 1990 to 1996, including all cases in which heart failure was the primary or secondary discharge diagnosis. Over this period the number of hospitalizations with a primary diagnosis of heart failure increased by 16% in men and 12% in women. Hospitalization with a secondary diagnosis of heart failure increased by 53%, representing 23% of all hospitalizations in 1996. A primary or secondary diagnosis of heart failure accounted for 2% of all hospitalizations in 1990, rising to 2.6% in 1996.[23]

Mayosi et. al. *Heart* 2007;93;1176-1183 . Data from 12 studies, over 50 years. 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 diagnosis of MI was made in only 2% of cases, which confirms the observation that CAD remains uncommon in Africa.

Common causes of heart failure in sub-Saharan Africa, such as rheumatic heart disease, peripartum cardiomyopathy (PCM), and endomyocardial fibrosis (EMF), 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.
The problem in Africa has been recognized for the last 60 years.[2] No population based incidence or prevalence studies of heart failure from Africa or other developing countries have been published.[3]

Amoah *et al.* in 2000, with 572 patients, using two-D, colour flow doppler, reported the incidence of hypertension (21.3%), rheumatic heart disease (20.1%) and cardiomyopathy (16.8%) with congenital heart disease (9.8%) and coronary artery disease (10%).[164] Ogola E.N, *et al.* following a study done at Kenyatta National Hospital in 1997, reported hypertension to be common among patients above 60 yrs. rheumatic disease and cardiomyopathy were uncommon.[17] In Cameroon, a hospital-based study showed that the main etiologies were: hypertension (54.49%), cardiomyopathies (26.34%) and valvular heart diseases (24.55%). Ischaemic heart disease was the fifth etiology (2.39%). Oyoo *et al.* (1993) reported in his study that and rheumatic disease was most common (29%) followed by cardiomyopathy

1.2 Pathophysiology of heart failure

Heart failure occurs when the ventricles are unable to generate sufficient pressure during contraction or when high pressures are required to fill the ventricles. It usually is the result of an index event, such as myocardial infarction, which results in impaired cardiac function and vascular congestion. Over the last 20 years, there has been an increased understanding of the pathophysiology of congestive heart failure. This coincides with a dramatic increase in the incidence of congestive heart failure that has paralleled a rise in the elderly population. The earlier hypothesis that progression of heart failure was based on impaired contractility and pump dysfunction has evolved into the current understanding of the importance of neurohormonal role in heart failure.[27]

Although impaired contractility and pump dysfunction are important to the understanding and treatment of acute decompensated heart failure, chronic progression of heart failure is now thought to be integrally linked to neurohormonal events and pathologic reflexes. Heart failure, like most clinical syndromes, is the end result of other events. LV remodelling encompasses numerous mechanisms, such as cell death, collagen break- down, and myocyte hypertrophy, and is a result of the neurohormonal response that occurs in heart failure. Activation of neurohormonal pathways is adaptive in the short term to maintain blood pressure but can promote the progression of heart failure through LV remodelling when long-term activation occurs. According to the neurohormonal concept, an index event occurs, resulting in the activation of neurohormones and cytokines, which then cause the development and progression of LV remodelling, resulting in the clinical syndromes of heart failure.[27] Studies have shown that 50% of elderly patients who present with symptoms of heart failure have preserved LV systolic function.[28]. This diagnosis appears to be most prevalent in elderly females with hypertension and coronary artery disease.[28] Heart failure with preserved systolic function may be difficult to diagnose, since a large number of patients with presumed diastolic dysfunction have other medical co-morbidities. A recent study reported that elderly patients with diastolic heart failure had pathophysiologic abnormalities similar to patients with systolic heart failure. Results of this study suggest that the underlying pathophysiology of both systolic and

diastolic heart failure may be similar, and neurohormonal therapies may be beneficial in both systolic and diastolic heart failure.[28]

Coronary artery disease leads to heart failure through multiple mechanisms. Myocardial infarction can lead to regional contractile dysfunction, myocyte hypertrophy, apoptosis, and deposition of extracellular matrix.[25] In addition, transient ischemia may result in episodic dysfunction even in the presence of normal resting ventricular function.[25] In many, we do not know the aetiology of the heart failure. Its development is a slow process, and may be multifactorial. Disorders affecting extra-cardiac organs, such as hyperthyroidism, and chronic pulmonary obstructive disease, can also cause heart failure. In the United States of America, most cases of heart failure are as a result of coronary artery disease, or a combination of hypertension, LV hypertrophy, and diabetes.[25]. The common pathway from any index event is the response that the heart takes to make effective blood circulation and productive blood flow to vital organs. [27].

At the cellular level there is often an inflammatory component after the initial index event. This can lead to hypertrophy of myocytes and apoptosis. In situations in which the index event results in a more chronic fluid overload state, such as mitral regurgitation, there is elongation of cardiac myocyte. Although initial phase elongation and resulting heart chamber dilation may maintain stroke volume, in time this leads to further impaired systolic function. In patients with excessive afterload, myocyte hypertrophy also occurs and is associated with increased cellular thickness. In these situations, the common factor is the resulting ventricular remodelling.

An index event can result in acute decompensation in a patient with chronic heart failure. Patients with previously diagnosed heart failure can have acute exacerbations as a result of conditions such as myocardial ischemia, arrhythmias, worsening renal function and natural progression of this chronic disease.[27].

1.3 Renin-Angiotensin-Aldosterone-System Angiotensin

Since activation of neurohormonal pathways is a result of mechanoreceptors that sense alterations in arterial pressure, this process is present in patients with both high

output failure, such as thyrotoxicosis, and low output states as in cardiac ischemia. As cardiac output declines, there is activation of the renin-angiotensin system. This results in arteriolar constriction, which increases blood pressure and thus maintains the GFR, in part by angiotensin-II and sympathetic nervous system activity.[29] The RAAS system is a cascade of neurohormonal release that is initiated by the release of renin from the kidney. Renin cleaves angiotensinogen, which is produced primarily in the liver. Angiotensin II results from the conversion of angiotensinogen to angiotensin-I , which is then cleared by the angiotensin-converting enzyme (ACE). [29]

Aldosterone further promotes the absorption of sodium in exchange for potassium in the renal tubules. In congestive heart failure, its secretion is stimulated by angiotensin-II, elevated potassium concentrations, catecholamines, endothelins and vasopressin. Unlike angiotensin-II which promotes sodium absorption in the proximal renal tubules, aldosterone acts on the distal cortical-collecting duct to further prevent sodium reabsorption.[30] Aldosterone causes a reduction in endothelial nitric oxide, which reduces arterial compliance and results in endothelial dysfunction. Increased sympathetic activity is attributed to the blocking of catecholamine reuptake by aldosterone. The increase in sympathetic activity can result in a proarrhythmic state that can lead to sudden cardiac death.[31] In addition, aldosterone-induced increases in myocardial hypertrophy, fibrosis, and apoptosis (programmed myocellular necrosis) result in increased ventricular stiffness which can lead to LV dysfunction and worsening heart failure.[29] Activation of the RAAS therefore results in a number of pathologic consequences that lead to the progression of heart failure by attempting to restore adequate renal perfusion, which can result in salt and water retention, and by facilitating cardiac myocyte growth, therefore promoting LV remodelling.[2]

1.4 Adrenergic Nervous System

In patients with congestive heart failure, the immediate response to beta-adrenergic system activation, increased heart rate, and contractility are beneficial. A fall in cardiac output results in increased sympathetic activity. This results in increased heart rate, and force of ventricular contraction via beta-receptor activation and peripheral vasoconstriction. However, prolonged stimulation of these receptors results in beta-adrenergic desensitization. This is a function of reduced beta-1

receptor density and uncoupling of beta-2 receptors downstream, resulting in a decreased arterial baroreceptor reflex control of heart rate in patients with heart failure.[33] In addition, there is decreased ionotropic response through alteration in calcium-mediated contractile proteins that leads to decline in cardiac output, and thus worsening congestive cardiac failure. These factors ultimately result in alteration in calcium transport across sarcoplasmic reticulum, as well as release of calcium to maintain contractility.[34] It is unknown if these adrenergic signal abnormalities are an adaptive response to overstimulation or maladaptive change that depresses contractility and drives heart failure progression. These effects on adrenergic system result in upregulation of central and peripheral chemo receptors, which have been shown to contribute to the development of central sleep apnea that occurs in one-third of all heart failure patients.[35]

The release of norepinephrine, an effector of the adrenergic nervous system, contributes to the progression of heart failure by many different mechanisms. Plasma norepinephrine levels correlate with severity of heart failure and also the long term prognosis. Studies have shown that the release of norepinephrine is associated with increased pulmonary artery pressure and pulmonary wedge pressure, which lead to the hypothesis that there is a link between cardiopulmonary baroreceptors and efferent cardiac sympathetic activity.[36] The increase in norepinephrine level results in both local and systemic effects such as direct myocardial toxicity, sodium and water retention, peripheral vasoconstriction, induction of apoptosis, activation of RAAS, and stimulation of arrhythmia.[37] The presence of chronically elevated norepinephrine, along with tumor necrosis factor and inflammatory cytokines, leads to an increase in nitric oxide production that can result in cellular proliferation and apoptosis.[37]

1.5 Natriuretic Peptides

The natriuretic peptides are promising markers of myocardial infarction and HF. In 1956, the electron microscope was used to demonstrate granules present in the atria that were absent in ventricles.[160] Henry and Pearce observed an increase in urine flow when a balloon was inflated in the atrium of a dog.[161] Influenced by these initial investigations, subsequent studies have identified four natriuretic peptides: Atrial Natriuretic peptide (ANP, predominantly secreted from Atrial myocardium), brain or

B-type Natriuretic peptide (BNP, predominantly secreted from ventricular myocardium), C-type natriuretic peptide (CNP, predominantly secreted from vascular endothelium), and D-type natriuretic peptide (DNP, predominantly secreted by the kidney).

BNP has been studied the most. BNP is released under conditions of increased myocardial pressure and stretching, and possess vasodialatory and natriuretic properties. It is released as prohormone and on secretion from myocyte, is cleaved into the biologically active BNP (32 amino acids in length) and the biologically inactive NT-BNP (76 amino acids). BNP is primarily removed by natriuretic peptide receptors with a small amount of renal clearance, while the kidney primarily clears NT-BNP.

BNP and NT-BNP assays have generally proven superior in diagnostic accuracy and clinical performance as prognostic markers than AANP, or its precursor NT-ANP [162] BNP and NT-BNP have both received attention as markers useful for diagnosis or exclusion of HF in ED in patients. The European Society of Cardiology for diagnosis and treatment of chronic HF has recommended that a cardiac natriuretic hormone assay be included in the initial evaluation of HF.[163]

The natriuretic peptides, therefore, act in a counter-regulatory fashion to the detrimental neurohormonal activation of the RAAS, adrenergic nervous system, and endothelin. Natriuretic peptides cause efferent renal arterial constriction, but afferent arterial dilation, which promotes increased diuresis and sodium excretion. In addition, the natriuretic peptides inhibit aldosterone and renin secretion and serve as physiologic antagonists to angiotensin-II. They promote vascular relaxation and blood pressure reduction by reducing sympathetic tone, and decreasing synthesis of catecholamines and Endothelin-1.[37] Release of ANP and BNP may delay systemic and renal arterial vasoconstriction, venoconstriction and sodium retention, therefore balancing the activation of the RAAS.[29] Through the increased production of cyclic GMP, the natriuretic peptides decrease the activation of plasminogen activator-1 which induces the prothrombotic state of heart failure. However, in acute decompensated heart failure, the release of stored natriuretic peptides and the

production of the peptides is insufficient to balance the vasoconstriction and fluid retention of the RAAS.

Natriuretic peptides are promising markers of myocardial dysfunction and heart failure. Four natriuretic peptides are secreted: Atrial natriuretic peptide (ANP, predominantly secreted from atrial myocardium), Brain natriuretic peptide (BNP, predominantly secreted from ventricular myocardium), C-type natriuretic peptide (CNP, from vascular endothelium) and D-type natriuretic peptide(DNP, predominantly secreted by the kidney).

BNP which is released under conditions of increased myocardial pressure and stretching, possesses vasodilatory and natreuretic properties. Released as prohormone , secreted from myocyte, cleaved into biologically active BNP (32 amino acids) and inactive NT-BNP (76 amino acids). BNP is removed from body by natriuretic peptide c- receptors and NT-BNP through kidney.

BNP and NT-BNP assays have generally proven superior in diagnostic accuracy and clinical performance as prognostic markers than ANP, or its precursor NT-ANP.[46]

BNP and NT-BNP have both received attention as markers useful for diagnosis or exclusion of heart failure. The task force of European Society of Cardiology for the diagnosis and treatment of chronic heart failure has recommended that a cardiac natriuretc hormone assay be included in the evaluation.[47]

The Breathing Not Properly Multinational Study confirmed findings from pilot studies that BNP was useful as a diagnostic marker in patients presenting to the emergency department with undifferentiated dyspnoea.[48] A BNP value less than 100pg/ml frequently excludes heart failure as a cause of dyspnoea (sensitivity=90%, specificity=76%, negative predictive value, NPV=89%), while a patient with a BNP value over 400pg/ml is highly likely to have Heart Failure.[50] There is a high degree of correlation with the New York Heart Association functional.[49] The BNP Study demonstrated that those patients with diastolic dysfunction had significant elevations in BNP, compared with those patients without Heart Failure. The median BNP was 413pg/ml in diastolic dysfunction and 821 pg/ml in systolic dysfunction.[51]

In a separate analysis of BNP study, the creatinine clearance worsened the cut-off point for maximum diagnostic accuracy which increased as well.[52] While it has not been validated to the extent of BNP, especially in the emergency department setting of undifferentiated dyspnoea, recent studies of NT-BNP have confirmed earlier

findings that it is an accurate marker of left ventricular dysfunction.[53,54,55] BNP and NT-BNP correlated well with each other (r2=0.94) and were predictive of New York Heart Association functional class and ejection fraction in ambulatory patients. NT-BNP proved to be a strong marker of reduced systolic function (EF<40%) in a cohort of 2,193 patients admitted to a general hospital. Those patients with reduced systolic function were detected at a sensitivity of 78%, specificity of 76%, negative predictive value of 96%, and positive predictive value of 30%. These findings were similar to earlier studies performed with BNP.[56] One would have expected overall lower diagnostic test characteristics when assay is compared solely to LVEF because there is a large cohort of patients with diastolic dysfunction that would have elevated NT-BNP values but have normal LVEF.

In 415 ambulatory patients with possible HF, NT-BNP increased as EF worsened.[56] It was less helpful in patients with diastolic dysfunction, especially mild relaxation abnormalities. Interestingly, while NT-BNP was helpful in those subjects where the examining cardiologist felt there was a "strong" (NT-BNP= 227pg/ml) or "no" (NT-BNP=66 pg/ml) suspicion of heart failure, it was not helpful in the group with moderate clinical suspicion of Heart Failure (mean NT-BNP= 85 pg/ml vs. 73 pg/ml in normal subjects).

In summary, BNP and NT-BNP have been validated against LVEF and have both been found to have similar test characteristics. While BNP increases with age and sex, NT-BNP seems to do so at a much greater degree. Age appropriate cut-offs for NT-proBNP, is suggested. Commercially available assays suggest two cut-offs based on age (age<75 normal cut-off = 125 g/ml, age >75 normal cut-off = 450 pg/ml). Both BNP and NT-BNP are elevated in patients with renal insufficiency even though they may have no clinical or echocardiographic evidence of heart failure. While BNP's creatinine clearance is well defined (>90% chance of heart failure when BNP >500 pg/ml), the relationship with NT-BNP is less well delineated. Because only kidneys clear NT-BNP it would be expected that renal disease would have a much greater impact on interpretation of NT-BNP results than BNP results. Finally, both BNP and NT-BNP have been confirmed in emergency department population using hospital discharge diagnosis of heart failure as gold standard (capturing both systolic and diastolic dysfunction).

For interpretation of results of serum level of NT-proBNP we used a criteria forwarded by the Roche diagnostics. Age was stratified according to less than 50 yrs, 50 –75 years, more than 75 years.

Patient's Age	Acute CCF Less Likely	Acute CCF Likely
< 50 years	300–450 pg/ml	➢ 450 pg/ml
50 – 70 years	300–900 pg/ml	➢ 900 pg/ml
➢ 70 years	300–1800 pg/ml	➢ 1800 pg/ml

In my discussion, i have elaborated on above table and clinical significance of these cut-off values.

The natriuretic peptides represent a counter-regulatory system, They unload the failing heart through diuresis, natriuresis, and vasodilation by suppressing the RAAS and endothelin. These peptides are secreted in response to hemodynamic stress, with higher stress resulting in higher levels of secretion.

2.5 Cardiomyopathy

2.5.1 Dilated cardiomyopathy

DCM, a primary disorder of heart muscle, which is characterized by dilatation and impaired contraction of the chambers of the heart, accounts for 10–17% of cardiac conditions encountered at necropsy,[64] and for 20% of patients who are admitted to hospital for heart failure. Presentation is usually with heart failure, that is progressive, with a 4-year mortality of 34% after the onset of symptoms.[65]

DCM represents a final common expression of myocardial damage that can be provoked by multiple insults, including hemodynamic, infective, immunological, toxic, nutritional and genetic factors. The aetiological factors that have been examined in African subjects include unrecognized hypertension, myocarditis, autoimmune mechanisms, iron overload, excessive alcohol intake, nutritional deficiency, and pregnancy. A systematic overview of studies of these potential aetiological factors is available elsewhere.[66]

It has been established that an intensive strategy of clinical investigation that includes coronary angiography and endomyocardial biopsy, where indicated, yields a specific diagnosis in up to 50% of patients with previously unexplained DCM.[65] The cases of DCM that remain unexplained even after invasive investigation need to be considered for family and molecular genetic studies. A major advance meant in the study of the pathogenesis of unexplained DCM has been the demonstration that about 30% of cases are familial, suggesting that genetic factors may be involved in the aetiology of the condition.[67] Reported families most commonly are compatible with autosomal dominant inheritance, but some with X-linked and autosomal recessive inheritance have been documented. Familial DCM is caused by mutations at over 25 chromosome loci, where genes encoding contractile, cytoskeletal and calcium regulatory proteins have been identified, underlining the genetic heterogeneity of the condition.[68] To the best of our knowledge, the first report of familial DCM in Africa described twin brothers who were affected from Uganda.[69] Brink *et al.* subsequently documented a condition characterized by hereditary dysrhythmic congestive cardiomyopathy,[70] and Przybojewski et al described two brothers of Afrikaner ancestry from South Africa with unexplained DCM.[71] More recently, Fernandez and others have shown that familial progressive heart block type II, which was initially reported in 1977, may be associated with DCM in the late stages of the disease.[70,72] Apart from these reports, there seems to be no systematic family studies that have been conducted to establish the frequency of familial DCM in Africans [66] such as have been done elsewhere.[73] Nevertheless, several gene association studies have been conducted, which suggest that heredity may have a role in the susceptibility to DCM in Africans. An association with HLADR1 and DRw10 antigens has been reported in South African patients, implying that genetically determined immune response factors have a role in the pathogenesis of some people with DCM.[74]

A common mitochondrial DNA polymorphism (T16189C) has also been found to be a genetic risk factor for DCM in a South African cohort, with a population attributable risk of 6%.[75] These genetic associations have been replicated in other populations, suggesting that they may represent genuine genetic risk factors for DCM worldwide.[76,77] Mutation screening studies in South African patients with idiopathic and familial DCM have identified a family with early onset DCM caused by a known

mutation in the troponin T gene (Arg141Trp)[78] but failed to show mutations in the cardiac and skeletal actin genes.[79]

The management of African patients with heart failure due to DCM is similar to that for patients with other forms of heart failure.[80] There is, however, emerging clinical trial evidence from South Africa that suggests that the immunomodulating agent, pentoxifylline, may be beneficial in patients with heart failure due to cardiomyopathy.[81,82]

2.5.2 PCM

PCM, which is defined as a disorder of unknown cause in which left ventricular systolic dysfunction and symptoms of heart failure occur between the last month of pregnancy and the first 5 months post partum,[66,83] shares many features with other forms of non-ischaemic DCM. The important distinction is that women with PCM have a higher rate of spontaneous recovery of ventricular function and better survival than idiopathic DCM.[65,83] A prospective echocardiographic study of 92 women from Haiti showed partial improvement of systolic function in the first year in the majority of patients, with one third of patients recovering fully over 3–5 years.[84] The contemporary mortality rate is 15% at 6 months,[85] and 42% over 25 years of follow-up.[86] The clinical predictors of mortality are New York Heart Association functional class at presentation, radiographic cardiothoracic ratio, ECG QRS voltage based on the Sokolow-Lyon criteria and higher diastolic blood pressure.[85,86] The presence of HIV infection does not seem to have an effect on outcome at 6 months of follow-up.[87]

Rheumatic Heart Disease:

Once accounted for half of the admissions around the world, remains common in developing countries. In industrialized nations, the incidence is now 1 per 100,000 people while in endemic area, the rates are higher than 100 per 100,000 people. In industrialized nations it accounts for 1 % of cardiac admissions. WHO statistics described 39.5% of New England cardiac admissions are related to RHD in 1928 and 23.5% in 1951. By 1990, RHD had 0.5% of primary discharge diagnosis in United States.[217]

2.6 Chest Radiography

The chest radiograph initially shows upper lobe diversion as the pulmonary blood redistributes to the upper portion of the lungs. With further increase in the pulmonary capillary wedge pressure, fluid starts to accumulate in the interstitial spaces, resulting in Kerley B lines. In the final stages, fluid accumulates in the alveoli, resulting in the classic perihilar butterfly infiltrates. On the X-ray, the cardiac shadow is often enlarged (cardiothoracic ratio of more than 50%) and there may be presence of unilateral (more often on the right side) or bilateral pleural effusions. Unfortunately, 20% of cardiomegaly seen on echocardiography is missed on chest radiography,[152] the cardiothoracic ratio is poor in predicting left ventricular dysfunction,[153] and pulmonary congestion can be minimal or absent even with significantly elevated pulmonary artery wedge pressures.[154]

While radiographic findings of pulmonary congestion may precede the presence of rales, the changes sometimes do not correlate well with the patient's clinical condition. There may be a lag in radiographic development of HF by several hours, and similarly, with clinical improvement, radiographic resolution may take a few days.

2.7 Echocardiography

The causes of Heart Failure with preserved ejection fraction are acute ischemia, hypertension, myocardial hypertrophy, restrictive cardiomyopathy e.t.c., common among women and the older population.

A study in 2002, in EGYPT, on 155 patients with heart failure reported that 66% had systolic heart failure while 34% had diastolic heart failure. Diastolic failures were mostly hypertensives.

2.8 Electrocardiography

The electrocardiogram (ECG) is not helpful in diagnosing HF, although it frequently shows abnormalities. Evidence of ischemia, acute myocardial infarction, or sustained arrhythmias may help establish a precipitating cause. Atrial fibrillation develops in around one-third of patients with HF [155] is a frequent cause of decompensation, [156-157]

and represents a worse prognosis compared to those who maintain sinus rhythm.[37] Intraventricular conduction delays are present in up to 25% of patients with HF, and are also a prognostic marker of adverse outcome.[63] In chronic HF, the ECG often shows evidence of chamber hypertrophy Q waves from old myocardial infarction, dysrhythmias, or conduction defects. QRS Duration-Electrical dyssynchrony (QRS duration of 120 milliseconds or greater) is associated with increased mortality in outpatients with heart failure [5,8] During a follow up of 9.9 months all cause mortality was 18.7% in patients. with normal baseline QRS duration and 28.1% for prolonged baseline duration. The composite cardiovascular death or hospitalization for heart failure was 32.4% and 41.6% for patients. with QRS duration greater than 120 ms. (Gheorghiade M. *et al.* 2008).

Biventricular pacing has been shown to improve hemodynamics almost immediately.[21] Isovolumetric systolic dysfunction and functional mitral regurgitation were shown to improve significantly.[22,23] Myocardial Oxygen consumption is decreased. [25] Benefit of reverse ventricular remodelling has been demonstrated in long-term follow up.[9,26] Narrow QRS duration in RethinQ study resynchronization did not show difference in peak oxygen consumption. In CARE-HF study, Cardiac Resynchronization Therapy improved symptoms and reduced the risk of death in pts. with reduced ejection fraction and prolonged QRS duration in outpatient setting.[9] Contribution of prolonged QRS duration to sudden cardiac death is not well established.[30] A prolonged QRS has not been shown to be predictive of ventricular arrhythmias in patients with Coronary Artery Disease and ICD.[32] The COMPANION trial demonstrated CRT combined with ICD decreased mortality in NYHA class III and IV, Ischemic or Non-ischemic Cardiomyopathy LVEF of 35% or less and QRS duration of 120 ms or more. It was significant with QRS duration of >168 ms, almost significant with duration of >148-168 ms and virtually no effect between 120 ms and 147 ms.[33] Similar findings were noted in PATCH-CHF II trial of 86 patients who found no benefit with QRS duration of 120 ms to 150 ms. 30% of patients with low ejection fraction and NYHA functional class III and IV have wide QRS complexes.[165] QRS prolongation among ADHF resulted in two fold increase in mortality (HR-1.94 , CI: 1.22-3.07).[166] Royal Brompton Hospital Heart Failure Unit found a graded increase in mortality with QRS without any definite threshold. Mortality at 3 years was 20% at <120 ms, 36% at 120 ms-160 ms, and 58% for QRS > 160 ms.[167] In

CARE-HF trial 813 patients were analyzed. Ventricular dyssynchrony was seen with QRS >150 ms, composite death was 37% (16%) and all–cause deaths was 36% (10%).

2.10 Clinical Scoring Scales

Sole reliance on the aforementioned tools to distinguish between cardiac and non-cardiac causes of dyspnoea is often challenging. While signs and symptoms of fluid overload raise suspicion of HF, their lack of sensitivity makes them poor screening tools.[38] The frequent limitations of this information have been extensively reported.[145,152-154] As a result, tools such as the Framingham criteria,[158] and Boston criteria[159] have been used to assist in the diagnosis of HF. The Framingham criteria uses major and minor clinical findings to establish a diagnosis of HF,[18] while the Boston criteria uses a point system such that a higher score is associated with greater possibility of HF. Unfortunately, point scoring systems completely miss patients with asymptomatic HF, which is why the American Heart Association scoring system identifies asymptomatic patients with significant risk factors as in the initial stages of HF.

3.0 JUSTIFICATION

Heart failure is rapidly becoming a major public health problem worldwide.[1] Prevalence of patients with heart failure exceeded 5 million in 2002 with an incidence of 550,000 new cases a year in the United States.[14] Heart failure accounts for 47% of all deaths in Egypt.[15] Locally, in a hospital-based study done in Kenya in 1993, the prevalence of heart failure stood at 3.3%.[16] Patients aged 60 years and above had hypertension as the most common cardiovascular disease.[17] Aetiology in Africa varies in comparison to the West, but few studies have been done to substantiate. We do not have data on incidence of Heart Failure with normal ejection fraction and on pharmaco-therapeutic interventions. With emerging diseases like HIV and Diabetes Mellitus, aetiological profile may have changed. This topic has not been researched for many years in the Department of Internal Medicine, the last study having been done 15 years ago.

4.0 BROAD OBJECTIVE

To characterize heart failure according to aetiology, pharmaco-therapeutic interventions and clinical outcome in patients admitted to medical wards at Kenyatta National Hospital over 6 months.

4.1 (i) Primary objective

1. To document the aetiological basis of heart failure.
2. To document the prevalence of diastolic heart failure in patients who do not have valvular heart disease.
3. To document QRS duration on electrocardiogram.
4. To document anti-failure pharmacotherapy utilized in terms of:
   (a) class of drug
   (b) dose: (i) at admission.
   (ii) at discharge.
5. To document the case fatality rate and duration of hospital stay.

(ii) Secondary specific objective

To determine BNP levels on admission and at discharge on a randomly selected 10% sub-set of patients with heart failure.

5.0 Outcome variables

1. **Hypertensive Heart Disease** – Left ventricular hypertrophy with preserved/ enhanced systolic function or left ventricular dilatation with decreased ejection fraction on echo and in the presence of hypertension (diastolic blood pressure >90 mmHg and systolic blood pressure >160 mmHg) or in a normotensive patient currently or previously on treatment with no other cardiac disease determined.
2. **Rheumatic Heart Disease** - Heart failure with valvular lesion(s) clinically and echocardiographically suggestive of rheumatic origin with no other cardiac disease present.
3. **Rheumatic:** Multiple valvular lesions, suggestive of rheumatic changes echocardiographically with no other cardiac disease present.

4. **CARDIOMYOPATHY**

4.1 **Dilated Cardiomyopathy**–Biatrial and biventricular dilation with global hypocontractility, no regional wall motion abnormality or thinning of wall and without compensatory hypertrophy.

4.2 **Alcoholic Dilated Cardiomyopathy**– Above parameters with history of > 2 units of alcohol per day >10 years.

4.3 Idiopathic Dilated Cardiomyopathy– Of unknown cause.

4.4 Hypertrophic Cardiomyopathy – hypertrophy of predominantly left ventricle in the absence of cardiac or systemic cause. M mode: septal to posterior wall thickness ratio to >1.5. Two-D echo : thickness of anterior septum or posterior wall ≥15mm or a posterior septum or free wall thickness ≥17mm on echo.

4.4 **Ischaemic Cardiomyopathy** – Dilation with global hypocontractility, no regional wall motion abnormality and thinning of wall and without compensatory hypertrophy, with no history or ECG evidence of Coronary Artery Disease.

4.5 **Peripartum Cardiomyopathy**–a) women developing heart failure in last month of pregnancy or within 5 months of delivery, b) absence of an identifiable cause for heart failure, c) absence of a recognizable heart disease prior to last month of pregnancy, d) left ventricular systolic dysfunction-depressed shortening fraction or ejection fraction with dilatation of all four cardiac chambers.

4.6 **Restrictive Cardiomyopathy**–Reduced ventricular diastolic compliance and restricted blood inflow (small LV cavity, with large atria) due to pathological conditions of endocardium and myocardium.

5. **Congenital Heat Disease**--Heart failure with a known and/or confirmed congenital heart disease on echo with no other heart disease present.
6. **Pericardial Disease** - Presence of pericardial thickening and/or pericardial effusion with no other cardiac disease present.
7. **Cor-Pulmonale**--Enlargement of right ventricle and right atrium on echo with intrinsic pulmonary disease.
8. **Ischemic Heart Disease** --History of myocardial infarction or systolic wall thickening , diastolic wall thickness with a wall thickness <7 mm or 30% less

than adjacent myocardium. With wall motion abnormality: hypokinesia, akinesia, dyskinesia on echo.

9. **Systolic HF**-- Ejection fraction of less than 40% and Framingham criteria.
10. **Diastolic HF**--Left ventricular ejection fraction of ≥ 40% and evidence of abnormal LV diastolic function: mitral inflow doppler examination.
11. **EKG**–QRS duration >120 ms, 120–160 ms, >160 ms.
12. **Case fatality rate**–Total number of deaths/ total number of heart failure patients admitted over the time period.
13. **Class of drug**:-
    (a) Diuretics: loop, thiazide, K+ spairing
    (b) β-blockers: β1, β2, both β1and β2
    (c) Angiotensin receptor blockers-specific
    (d) Angiotensin converting enzymes-specific
    (e) Digoxin
    (f) Statins specific
14. **Dose of medication**--At initiation of therapy and before discharge.
15. **Discharge**--Once decided by attending team of physicians and documented in file.
16. **Duration of hospital stay**–Counted from first day of admission to the day patient is discharged by the team or death.
17. **Serum level of N-terminal pro-Brain Natriuretic Peptide**–In picograms per millilitre at admission and at discharge.

The aetiologic basis of HF was determined on the basis of history, physical examination findings, and investigations including chest radiograph, Electrocadiogramme and Echocardiography. These were reviewed and consensus reached by the principal investigator in-conjunction with two supervisors. Wherever the two disagreed a third cardiologist was asked to adjudicate.

All the tests necessary for the above diagnostic consideration are an essential part of usual evaluation. However, the study will facilitate wherever a timely electro cardiograph, chest radiograph and Echocardiography will be needed.

6.0 METHODOLOGY

1. STUDY DESIGN

Prospective clinical observational register.

2. STUDY SITE

Medical wards, Kenyatta National Teaching and Referral Hospital, Nairobi, Kenya.

3. STUDY POPULATION AND DURATION

Consecutive prospective medical wards and ICU admission with heart failure over a period of 6 months.

4. CASE DEFINITION

A diagnosis of heart failure was be made based on Modified Framingham Clinical criteria for diagnosis of heart failure with at least two major or one major and two minor criteria required to establish a clinical diagnosis of heart failure.

(i) MAJOR CRITERIA

- Orthopnea
- Pulmonary Rales
- Pulmonary Oedema on Chest X-ray
- Third heart Sound
- Elevated Jugular Venous Pressure
- Positive Hepatojugular reflux
- Cardiomegally on Chest X-ray
- Paroxysmal Nocturnal Dyspnoea
- Weight loss $\geq$4.5 kg in 5 days in response to treatment of presumed heart failure.

(ii) MINOR CRITERIA

- Bilateral Leg Oedema
- Nocturnal Cough
- Dyspnoea on Ordinary Exertion
- Hepatomegaly
- Pleural Effusion

- Tachycardia (≥120 bpm)
- Weight loss of more than or equal to 4.5 kg over 5 days of treatment

When available, echocardiographic data was added to support diagnosis.

5. INCLUSION CRITERIA

- Age equal to or above 13 years.
- Established diagnosis based on Modified Framingham criteria for diagnosis of heart failure.
- Informed written consent or assent from patient or next of kin where the patient is unable to give consent. For those under the age of 18 years both the patients assent and the caretaker consent were taken. For those without formal education a left thumb print were taken instead of their signatures.

6. EXCLUSION CRITERIA

- Ongoing or recent onset of Acute Myocardial Infarction in last one week.

7. SAMPLING TECHNIQUE

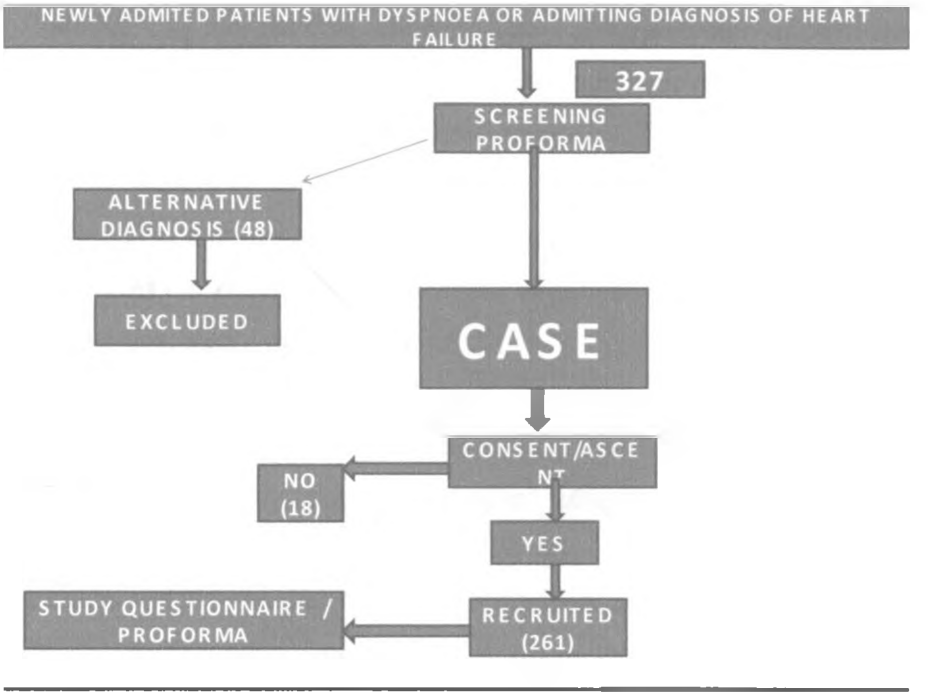
Consecutive convenient sampling technique was be utilized.

8. SCREENING AND RECRUITMENT

The Principal Investigator and Study Assistant reviewed the admission records of patients admitted the previous day with dyspnoea or heart failure. The patients were identified, a rapport established with them and their caretakers, and a questionnaire with Framingham criteria administered to determine eligibility for inclusion into the study. The objectives of the study were clearly explained. Patients who agreed to participate in the study were enrolled. A through history was taken, physical examination carried out and the tests/investigations carried out: urea, electrolyte, creatinine, electrocardiogrammes, and echocardiography. Those patients who declined consent were not discriminated against in any way but were left to undergo the routine ward evaluation and management. Recruited patients (Cases) were

followed up from admission until discharge or death. Data collection was done through electrocardiogram, echocardiography and NT-proBNP.

NEWLY ADMITTED PATIENTS WITH DYSPNOEA OR ADMITTING DIAGNOSIS OF HEART FAILURE



9. DATA COLLECTION

The following clinical procedure was done as part of the clinical evaluation of the patient and the findings recorded in the study pro forma:

1. Heart rate was measured using a stethoscope placed on patient's precordium. The rate was observed for a minute. This was repeated three times in 5 minutes of interval and the average recorded.
2. Blood pressure was measured using a manual blood pressure recording machine (Mercury Sphygmomanometer). It was taken on both arms with patient lying supine and propped up in bed, and arms at the same horizontal level as the trunk. Three readings were taken and their average recorded. Heart rate was recorded first followed by blood pressure measurement within

5 minutes of time interval. One study assistant and I were involved in this data collection.

3. A plain chest radiograph was done for each patient within 24 hours of admission. Every effort was made to take a standard good quality posterior/anterior (PA) view. All films were taken at KNH at the radiology department using a specific machine: *Medio 50 CP Model 14490206001003.* Interpretation of the film was undertaken by a consultant radiologist on duty 'blinded' to the patients clinical state. Interpretation of AP view was conducted with caution with respect to cardiothoracic ratio (CTR); choosing to 'eyeball' for evidence of gross cardiomegally. Cephalization of pulmonary vessels, Kerley B lines, alveolar oedema and pleural effusion were the other radiological features of interest. No attempts were made to correct intra or inter-observer variations. The presence of pulmonary oedema, pleural effusion and cardiomegally were noted.

4. A standard, resting, 12- lead electrocardiogramme was recorded at a speed of 25 mm/sec. QRS duration was measured using leads V_3-V_6. Prolonged QRS interval was defined as duration > 120 ms. With callipers used on printed ECG, each QRS interval with longest duration, and clearest onset and end was measured, beginning from the point where the first wave of the complex deviated from the baseline. The point at which the last wave of complex leveled out at, above, or below the baseline (visual returning of S wave to isoelectric line) marked the end of QRS complex. The information was passed on to the team in charge for the management.

5. Echocardiography was carried out with the patient positioned in standard echocardiographic views. Two-dimensional, M-mode and doppler examinations were carried out on all patients. Measurements were taken as per the recommendations of the American Society of Echocardiography. The procedure was done by a cardiologist. In case of unavailability, an echocardiographer did the recordings and the report adjudicated by two cardiologists.

   I facilitated the procedure by organizing through usual procedure. However, in circumstances where a patient could not finance the same or in

case of mechanical problems with the echo machine, I used the echo machine from the Department of Medicine.

6. A blood sample of 6 ml was obtained from the patient's forearm and was collected in a plastic tube. The sample was centrifuged at 3,000 rpm for 15 minutes and stored at 20^0C, for evaluation at a later date for N-terminal proBrain Natriuretic Peptide.

   SERUM N-terminal proBNP, an enzyme immunoassay for the measurement of N-terminal proBNP, developed by Roche elecsys random access analyzer, was used. Second reaction step: the analytical sensitivity was determined as 5 pg/ml with a dynamic range of up to 35,000 pg/ml.

The patients were followed up until discharge or death. During this period the PI or his assistant collected data on medication instituted after admission to the hospital and upon discharge, as well as the clinical outcome including new presentation of signs and symptoms after admission. This were recorded by name and were coded during analysis.

Data prospectively recorded into the study pro forma were entered into data entry sheets. Cleaning and verification was done using Statistical Package for Social Sciences version 14.2 software for windows on a weekly basis to ensure validation and completeness of the information.

Quality assurance

Aseptic technique was used for specimen collection to minimize pre-analytical errors. Procedures for specimen handling and storage were also adhered to. All equipments were calibrated according to the manufacturer's specifications. Commercial control materials were used to validate the calibrations. These were included in all analytical runs. Results were only accepted if the control values were within the expected ranges.

10. DATA ANALYSIS:

Descriptive statistics such as frequency, mean, variance and standard deviations were used for categorical variables e. g. Age, gender . For associations, cross tabulation with Pearson's Chi-square statistics e.g. mortality was used.

Means were computed by student *t*-test. Paired *t*-test was used for NT-proBNP levels at admission and at discharge. Chi-square was used in the analysis of categorical variables.

Independent *t*-test was used for length of stay in the hospital.

Two-tailed *t*-test was used for analysis of continuous variables. Outcomes were considered significant with a 95% Confidence Interval and a p-value of <0.05.

11. ETHICAL CONSIDERATION

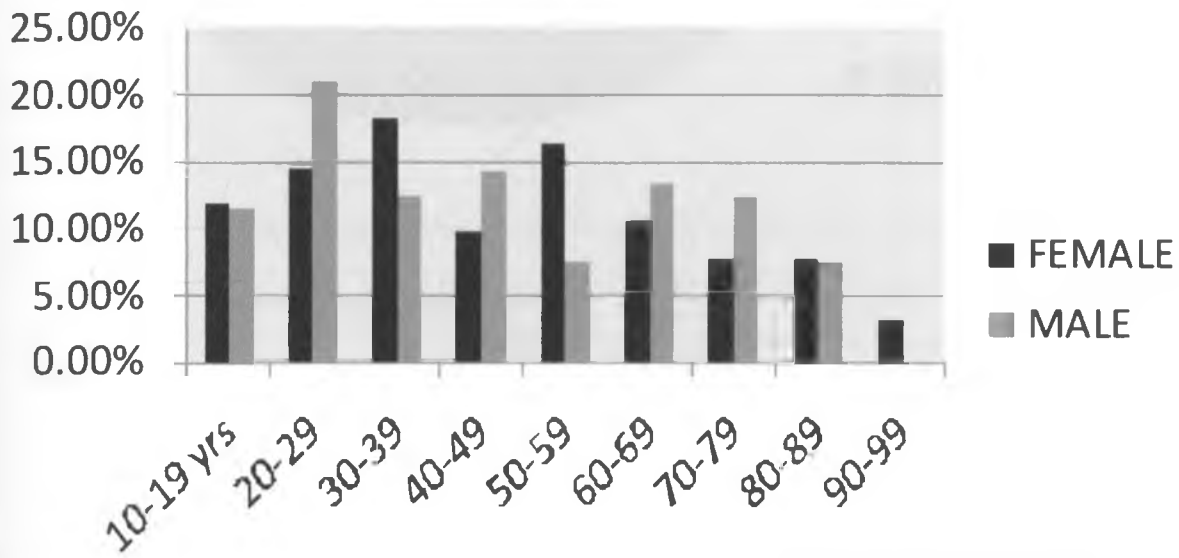
1. The study was conducted after approval by the Department of Clinical Medicine and Therapeutics University of Nairobi, and Kenyatta National Hospital Scientific Committee.
2. Before recruitment into the study, all eligible patients received information concerning the purpose of the study in lay terms and signed consent obtained. For those under the age of 18 years assent from care takers was obtained.
3. Recruited patients were free to withdraw from the study at anytime without being discriminated against in any way.
4. Patients who declined to consent were offered standard hospital care.
5. The investigations carried out on the patients were relevant based on current scientific understanding of the pathophysiological basis of heart failure.
6. All information obtained from patients was kept confidential.
7. Data sheets contained only patient's study information and numbers were provided in each sheet.
8. Results of the study were communicated back to the medical team managing the patients.

7.0 RESULTS

The study was carried out over 6 months, from 5th February, 2008 to 6th August, 2008. During this time 327 patients were screened for dyspnoea with a diagnosis for heart failure in the medical wards. Out of these, 48 had alternative diagnosis and were excluded. 279 cases satisfied the criteria for Heart Failure. 18 patients did not consent. 261 patients were recruited into the study.

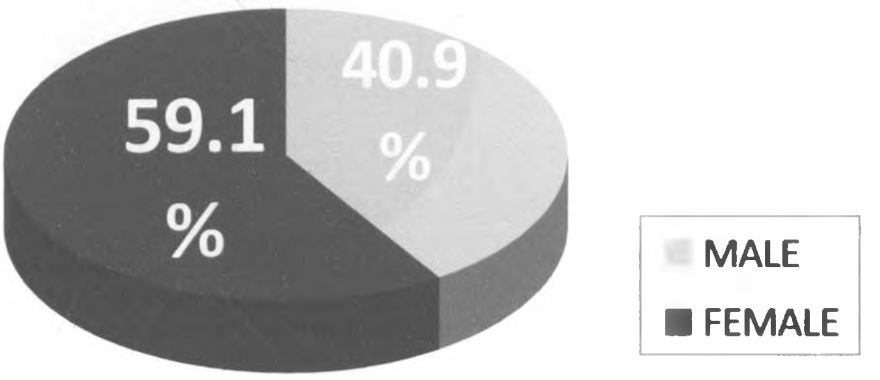
7. 1. DEMOGRAPHIC PROFILE

Distribution by age and gender in patients with Heart Failure



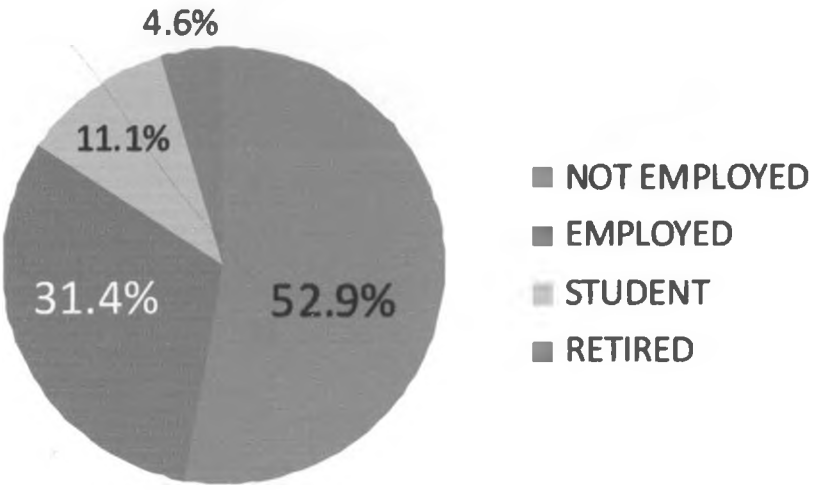
A total of 261 patients with a range of 10-94 years, with distribution of more than 10% in each sub-group of 10 years starting at 13 years. The distribution is more between 20 and 60 years. The majority were young and a good percentage were elderly.

7. 2 GENDER DISTRIBUTION IN STUDY POPULATION



There were 107 males (40.9 %) and 154 females (59.1%).

7.3 OCCUPATION



More than one half of the patients (52.9%) were unemployed. 31.4% of study participants were employed. 11.1% of study participants who were students were dependent upon their parents.

7.4 SOCIAL / LIFESTYLE

21.9% of the respondents had consumed alcohol at some point in time. The most common were local brews e. g. chang'aa, followed by bottled beer.

19.5% of study respondents had smoked cigarettes. The average number of cigarettes smoked by current smokers was 6 sticks a day.

7.5 DISTRIBUTION ACCORDING TO SEVERITY OF HEART FAILURE

Functional Class	n	%
I	1	0.4%
II	22	8.5%
III	123	47.5%
IV	113	43.6%

NYHA III & IV: 91%

Very few were from Class I and Class II, constituting 0.4% and 8.5% respectively. The majority 91% were class III and Class IV.

7.6 CLINICAL SYMPTOMS

Symptoms	Frequency
Shortness of Breath	100%
Easy Fatigability	100%
Palpitation	96.9%
Night Cough	95.4%
Orthopnea	94.3%

Shortness of breath and easy fatigability were present in all cases, followed closely by Palpitations reported by 96·9% of study participants. 95·4% had night cough and 94·3% had orthopnoea.

7.7 CLINICAL SIGNS

Signs	Frequency (%)
Raised JVP	86%
Tender Hepatomegaly	80%
Bilateral Leg Oedema	80%
Murmurs	64%
Ascites	41.2%
Displaced Apex	62.2%
Diffuse Apex	24.7%

Raised Jugular Venous Pressure was the most common sign present in 86% of study participants, followed by tender Hepatomegaly and bilateral leg oedema (80%). A

cardiac Murmur was auscultated in over 50% of the cases. Diffuse apex beat was found in 24.7 % while displaced apex was present in 62.2%

7.8 AETIOLOGY

	Number	PERCENTAGE	
Rheumatic Heart Disease	**70**	**27.0%**	
Idiopathic Dilated Cardiomyopathy	**56**	**21.6%**	**40.1%**
Alcoholic Cardiomyopathy	**48**	**18.5%**	
Hypertensive Heart Disease	**47**	**18.1%**	
Cor-Pulmonale	**47**	**18.9%**	
Ischemic Heart Disease	**19**	**7.3%**	
Pericardial Disease	**14**	**5.4%**	
Congenital Heart Disease	**3**	**1.2%**	
Hypertrophic Cardiomyopathy	**2**	**0.8%**	
Restrictive Cardiomyopathy	**2**	**0.8%**	
Ischemic Cardiomyopathy	**1**	**0.4%**	
Peripartum Cardiomyopathy	**1**	**0.4%**	

Dilated cardiomyopathy comprising of idiopathic and alcoholic cardiomyopathy accounted for 40.1% of cases of heart failure were the most common aetiological class. Rheumatic Heart disease was present in 70 patients (27%). Hypertensive Heart disease and Cor-Pulmonale had an equal distribution of 47 patients each class.

Ischemic Heart Disease was present in 19 patients (7.3%) while Pericardial disease was detected in 14 patients (5.4%). Congenital heart disease was detected in 3 patients (1.2%). Two (0.8%) patients had Hypertrophic Cardiomyopathy and two others had Restrictive Cardiomyopathy. Ischemic Cardiomyopathy and Peripartum Cardiomyopathy each had one patient constituting 0.4%.

7.9 AETIOLOGY AND AGE RELATIONSHIP

Age	HHD n (%)	RHD n (%)	Per. D n (%)	IHD n (%)	Cor.PUL. n (%)
10-29	6, (12.5%)	42, (60%)	8, (57.1)%	2, (10.5%)	20, (40.8)%
30-49	11, (22.9%)	25, (35.7%)	3, (21.4)%	1, (5.3%	13, (26.5%
50-69	20, (41.7%)	2, (2.9%)	2, 14.3%	7, (36.8%)	7, (14.3%)
> 70	11, (22.9%)	1, (1.4%)	1, 7.1%	9, (47.7%)	9, (18.4%)
P value	<0.01	<0.001	= 0.105	< 0.01	= 0.135

Contd.

Age	Alcoholic Cardiomyopathy n (%)	Idiopathic Cardiomyopathy n (%)
10-29	3, (6.3%)	6, (10.9%)
30-49	14, (29.2%)	13, (23.6%)
50-69	14, (29.2%)	20, (36.4%)
> 70	17, (35.4%)	16, (29.1%)
P value	< 0.001	

87.5% of Hypertensive Heart Disease cases are more than 30 years of age (p<0.01), 20 cases of them aged 50-69 years. Rheumatic Heart Disease is more common in the younger age group (95%) (p<0.001), most of them below the age of 30 years. Pericardial disease was more common in those aged less than 50 (78.5%). 78.5% (16 cases) of subjects with Ischemic Heart Disease cases were more than 50 years of age (p<0.01), statistically significant. Cor-Pulmonale was more common among those aged less than 30 years but this association was not statistically significant (p<0.135). 93.8% of patients with Alcoholic Dilated Cardiomyopathy were more than 30 years old (p<0.001). 65.5% of patients with Idiopathic Cardiomyopathy were more than 50 years of age (p<0.001).

7.9 CLINICAL OUTCOMES

The mean duration of hospital stay was 7.1 days with a median of 6 days and a range of between 2 and 29 days. The Case Fatality rate was 14.1%.

7.10 CASE FATALITY RATE ACCORDING TO DISEASE AETIOLOGY.

Case Fatality Rate According to Disease Aetiology								
Aetiology	**RHD**		**HHD**		**IHD**		**DCM**	
	%		**%**		**%**		**%**	**(n)**
	(n)		**(n)**		**(n)**			
Case Fatality	**14.5%**	**(70)**	**19.1%**	**(47)**	**26.3%**	**(19)**	**9.7%**	**(104)**

11 patients (14.5%) Rheumatic heart disease, 9 patients (19·1%) Hypertensive heart disease, 5 patients (26·3%) Ischemic Heart Disease and 10 patients (9.7%) dilated Cardiomyopathy died during the study period.

7.11 PRECIPITATING FACTORS CAUSING ACUTE DECOMPENSATION.

Precipitating factors	Frequency	Percentage
Non-Compliance	216	83.1%
Infective Endocarditis	22	8.5%
Hyper/Hypothyroidism	4	1.5%
Acute Rheumatic Fever	2	0.8%

Non-compliance was the most common precipitating factor for acute cardiac decompensation accounting for 83% of all cases, followed by infective endocarditis, which was responsible for 22 (8.5%) cases of heart failure. Thyroid disorder precipitated 4 cases of heart failure, while rheumatic fever was responsible for 2 cases of acute heart failure. The reason for non-compliance is not elucidated further as well as endocarditis..

7.13. NORMAL EJECTION FRACTION IN PATIENTS WITHOUT VALVULAR HEART DISEASE. (Ejection Fraction Cut-off of 40%, 45% and 50%)

jection Fraction cuff-off 40			
<40 (Systolic HF)		≥40 (NEF HF)	
	(%)	n	(%)
8	(60.5%)	77	(39.5%)

Ejection Fraction cut-off 45			
<45 (Systolic HF)		≥45 (NEF HF)	
n	(%)	n	(%)
124	(63.6%)	71	(36.4%)

Ejection Fraction cuff-off 50			
<50 (Systolic HF)		≥50 (NEF HF)	
n	%	n	%
139	(71.3%)	56	(28.7%)

I used three cut-off points since, there is no consensus on the cut-off points. Secondly, studies have used one of the three values for their research, and I will be able to compare these studies using any of three.

When different cut-off points of Ejection Fraction were used: At 40%, 51 (39.5%) cases of had HFnlEF, while 78 cases (60.5%) had systolic HF. When 45% was used as cut-off: 71 (36.4%) cases had HFnlEF and 124 (63.6%) cases had systolic HF. At 50% cut-off, 56 (28.7%) cases had HFnlEF, and 139 (71.3%) cases had systolic HF.

7.13.1 EF HF Classification by Age & Gender

	Systolic HF (n=100)	Normal EF HF (n=95)	p value
Age	41.14yrs ± 19.08	49.3yrs ± 21.35	$p < 0.006$
Male	61.7% (n 37)	39.3% (n 24)	$p< 0.05$
Female	53.2% (n 41)	46.8% (n 36)	p=0.420
LVH (40)	47.5% (n 19)	52.5% (n 21)	p=0.281
AF (43)	65.1% (n 28)	34.9% (n 15)	p=0.140

The total number of systolic heart failure cases were 100 while cases with HFnlEF were 95. Patients with HFnlEF, were older compared to systolic HF and this was statistically significant (p<006). As in the western world, HFnlEF is common in females, and have a higher ejection fraction. Atrial fibrillation was more common in systolic heart failure but not statistically significant.

7.13.2 MEAN DAYS OF HOSPITALIZATION ACCORDING TO EJECTION FRACTION. (ALL CASES).

MEAN DAYS OF HOSPITALIZATION according to EF.

Ejection Fraction	RHD No. Of days (n)	HHD No. Of days (n)	IHD No. Of days (n)	DCM No. Of days (n)
EF = <40	7.45 (22)	6.63 (8)	6.5 (4)	8.08 (59)
EF = > 40	8.07 (29)	5.96 (23)	7.25 (8)	7 (17)

At 40% cut-off, 22 cases of rheumatic heart disease had systolic heart failure with a mean days of hospital stay of 7.45 days, while 29 cases with HFnlEF had a mean of 8.07 days of hospital stay, and is longer in rheumatic disease. 8 cases of hypertensive heart disease with systolic heart failure had a mean of 6.63 days of hospital stay, while 23 cases with HFnlEF had 5.96 days. 4 cases in ischemic heart disease with systolic heart failure had 6.5 days, while 8 cases with HFnlEF had 7.25 days and this was statistically not significant. Dilated cardiomyopathy constituted 59 cases of systolic HF with a mean of 8.08 days, while 17 cases with HFnlEF had 7 days, and in this systolic heart failure, subjects had longer hospital stay although not statistically significant.

7.13.3 MORTALITY RATE ACCORDING TO EJECTION FRACTION

MORTALITY RATE ACCORDING TO EF.

Ejection Fraction	RHD		HHD		IHD		DCM	
	%	(n)	%	(n)	%	(n)	%	(n)
EF = <40	0%	(22)	12.5%	(8)	25%	(4)	4.5%	(67)
EF=> 40	15.2%	(33)	4.2%	(23)	0%	(8)	5.6%	(17)

The mortality, when the EF cut-off is 40%: out off 22 cases, none had died in rheumatic heart disease but was higher (15.2%) with HFnlEF. Similarly, there was no death of patients with IHD with HFnlEF, but are not statistically significant. The reasons are not apparent and to be explored further. 8 cases (12.5%) with hypertensive heart disease died with systolic heart failure, while 23 cases (4.2%) in HFnlEF. 4 cases (25%) died with systolic HF in ischemic heart disease, none in HFnlEF. 67 cases (4.5%) died in the Dilated cardiomyopathy group from systolic HF, with 17 cases (5.6%) in HFnlEF.

7.14 QRS DURATION IN PATIENTS WITH HEART FAILURE EXCLUDING RHEUMATIC HEART DISEASE.

23.14.1 CLASSIFICATION OF QRS DURATION ACCORDING TO GROUPS.

	< 120 ms		120 ms-160 ms		>160 ms	
All	**224**	**(91.4%)**	**18**	**(7.3%)**	**3**	**(1.2%)**
Ex-RHD	**157**	**(89.2%)**	**16**	**(9.1%)**	**3**	**(1.7%)**
Age	**44.6 yrs**		**62.1 yrs**		**64.7 yrs**	**p<0.01**
EF – (Ex-RHD)	**Mean**	**(SD)**	**Mean**	**(SD)**	**Mean**	**(SD)**
	38.5%	**(14.9)**	**43.8%**	**(20.4)**	**30.05%**	**(8.4)**
	(n 123)		**(n 12)**		**(n 2)**	

224 cases (91.4%) had QRS duration of less than 120 milliseconds. 18 cases (7.3%) had a duration of between 120 ms-160 ms. 3 cases had QRS duration of more than 160 ms.

When Rheumatic heart disease cases were excluded, 157 cases had a QRS duration of less than 120 ms. 16 cases (9.1%) had QRS duration of between 120milliseconds to 160 ms and 3 cases (1.7%) had QRS duration of more than 160 milliseconds.

The mean age of the group with QRS duration of less than 120 ms was 44.6 years, while those in the 120 ms-160 ms group was 62.1 years and it was 64.7 years for cases in the group with QRS duration of more than 160 ms, which was statistically significant ($p<0.01$).

The mean EF in the group with less than 120 ms, is 38.5% with SD of 14.9, while the mean EF in 120 ms−160 ms, is 43.8% with SD of 20.4 and in more than 160 ms, mean is 30.05% with a SD of 8.4.

7.14.2 QRS DURATION AND GENDER.

	QRS duration	<120 ms	120-160 ms	> 160 ms	P value
SEX	Male	97, (43.7%)	4, (22.2%)	3, (100%)	=0.027
	Female	125, (56.3%)	14, (77.8%)	0	

The sex distribution: Male; 97 cases (43.7%) in the group with less than 120 milliseconds while 4 cases (22.2%) were in the group of 120 ms – 160 ms and 3 cases all (100%) were in the group of more than 160 ms.

Female; 125 cases (56.3%) were in the group of less than 120 ms while 14 cases (77.8%) were between 120 ms – 160 ms, and none in more than 160 ms group. This was statistically significant.

7.15.1 USE OF ANTI-FAILURE PHARMACOTHERAPY AT ADMISSION AND AT DISCHARGE

DRUGS	Admission		Discharge	
	n	%	n	%
ENALAPRIL	94	(49%)	83	(43%)
CAPTOPRIL	70	(36.6%)	69	(36.1%)
LORSATAN	9	(4.7%)	9	(4.7%)
CARVEDILOL	65	(34.0%)	72	(37.7%)
FRUSEMIDE	170	(89.0%)	162	(84.8%)
DIGOXIN	147	(77.0%)	138	(72.3%)
SPIRONOLACTONE	108	(56.5%)	107	(56.0%)

Enalapril was instituted in94 cases (49%) at admission while 83 cases (43%) received enalapril at discharge. Lorsatan was instituted in 9 cases (4.7%) at admission and the same number of patients at discharge.

Carvedilol: the most common beta-blocker used both at (34%) admission and discharge (37·7%).

Frusemide: 170 (89%) cases received frusemide at admission and cases (84.8%) at discharge.

Digoxin: 147 cases received digoxin at admission (77%), while 138 (72.3%) cases were discharged on Digoxin.

Spironolactone: 108 cases (56.5%) received aldactone at admission, while almost similar number 107 received at discharge (56%).

Atorvastatin and aspirin were studied as part of pharmacotherapy armamentum, although they do not belong to anti-failure medications:

Atorvastatin: 43 cases (22.5%) received at admission while 48 cases (25.1%) at discharge.

Aspirin: 22 cases (11.1%) received Aspirin at admission while 27 case (14.1%) at discharge.

7.15.2 ANTI-FAILURE DRUGS USED AS PER THE AETIOLOGY.

DRUGS	RHD % (n)	HHD % (n)	IHD % (n)	DCM % n
ACEIs/ARBs	77.1% (54)	89.6% (43)	0	87.5% (91)
β blockers	44.3% (31)	41.7% (20)	36.8% (7)	46.2% (48)
Diuretics	88.6% (62)	93.8% (45)	84.2% (16)	93.2% (96)
Spironolactone	65.7% (46)	58.3% (28)	63.2% (12)	73.1% (76)
Digoxin	68.6% (48)	47.9% (23)	63.2% (12)	62.5% (65)

This table explains the drugs used in 4 aetiological class:

ACEIs/ARBs: were used in 91 cases (87.5%) of Dilated cardiomyopathy, 54 cases (77.1%) in rheumatic heart disease, 43 cases (89.6%) in Hypertensive heart disease.

Beta- blockers: were used in 31 cases (44.3%) of rheumatic heart disease,48 cases (46.2%) in Dilated cardiomyopathy, 20 cases received (41.7%) in Hypertensive heart disease, and 7 cases (36.8%) in Ischemic heart disease. In spite of known advantage of the drug, use is less than 50%, reason needs to be explored.

Diuretics: 96 cases (93.2%) received furosemide, 62 cases (88.6%) in rheumatic heart disease, while 45 cases (93.8%) in Hypertensive heart disease and 16 cases (84.2%) in Ischemic heart disease.

Spironolactone: 76 cases (73.1%) received spironolactone, followed by 46 cases (65.7%) in rheumatic heart disease, while 28 cases (58.3%) in hypertensive heart disease and 12 cases (63.2%) in ischemic heart disease.

Digoxin: 65 cases (62.5%) received digoxin, followed by 48 cases (68.6%) in rheumatic heart disease, and 12 cases (63.2%) in ischemic heart disease. And in HHD

the use was les than 50%.

7.15.3 MEAN DOSE OF ANTI FALURE MEDICATION AT ADMISSION AND AT DISCHARGE.

MEAN DOSE 0F ANTIFAILURE MEDICATION AT ADMISSION AND AT DISCHARGE.

	At Admission	At Discharge
	Mean Dose (mg)	Mean Dose (mg)
Captopril	42	39.4
Enalapril	13.3	10.24
Carvedilol	7.1	8.3
Frusemide	114.8	75.3
Digoxin	0.150	0.151
Spironolactone	26.6	26.4

Captopril: mean dose at admission was 42 mg and at discharge -39.4 mg.

Enalapril: at admission is 13.3 mg while at discharge it was 10.24 mg

Carvedilol: 7.1 mg at admission and 8.3 mg at discharge.

Frusemide: mean dose at admission- 114.8 mg and 75.3 mg at discharge, and this was statistically significant ($p<0.001$).

Digoxin : at admission the mean dose was 0.150 mg while at discharge it was 0.151 mg.

Spironolactone: mean dose at admission was 26.6 mg and at discharge, 26.4 mg.

Except Frusemide no other drug reached statistical significance.

7.16.1 SERUM N-TERMINAL pro BRAIN NATRIURETIC PEPTIDE LEVEL IN 50 CASES OF HEART FAILURE PATIENTS.

No. patients	Mean Serum levels at Admission	Mean serum levels at Discharge	P value
50	13752.36 pg/ml	369.3 pg/ml	P< 0.001
Range	(1550-35000) 33450 pg/ml	(15.38-1905) 1889.6 pg/ml	
Median	9637.5	200.2	
Mode	35000	15.4	
1st Quartile	4485.8	55.5	
2nd Q	9637.5	200.2	
3rd Quartile	20217.8	541.6	

The mean serum level at admission was high with many cases levels were higher than 35,000 pg/ml. While at discharge some of them had still high levels. The mean levels were statistically significant.

7.16.2 INTERPRETATION OF NT-proBNP LEVEL.

	<125 pg/ml CCF Excluded	125pg/ml- 300 pg/ml ACUTE CCF UNLIKELY	300 pg/ml-1800 pg/ml AGE RELATED CUT-OFF VALUE REQUIRED. ****	>1800 pg/ml ACUTE CCF LIKELY
Admission	0	0	1	49
Discharge	19	11	19	1

The interpretation of NT-proBNP level at admission and at discharge was done according to the above table. 19 cases were discharged with less than 125 pg/ml and CCF was excluded. At serum level of 125 pg/ml-300 pg/ml 11 cases had the diagnosis of Acute CCF unlikely at discharge, and none at admission. 49 cases had serum level more than 1800 pg/ml at admission. At levels between 300 pg-1800 pg/ml the age related cut-off value was required (see Table. No.23.16.3) and in this group one case was noted in admission while 19 cases were in this group.

7.16.3 BNP levels and Functional Class at Admission and at Discharge Age related cut-off value (300 pg/ml-1800 pg/ml).

Cut off by age & BNP levels.	Admission		Discharge	
	Acute CCF Less Likely	Acute CCF Likely	Acute CCF Less Likely	Acute CCF Likely
<50 yrs 300-450 pg/ml	0		3	
>450 pg/ml		48% (24)		6
50-75 yrs 300-900 pg/ml	0		4	
> 900 pg/ml		38% (19)		4
>75 yrs 300-1800 pg/ml	0		3	
>1800 pg/ml		14.35 (7)		0

At admission, 24 cases who were less than 50 years of age had >450 pg/ml and were in the group of Acute CCF likely and 6 cases at discharge while in the same age 3 cases had acute CCF less likely at discharge.

At admission, 19 cases who were between 50-75 years of age had >900 pg/ml and were in the group of Acute CCF likely and 4 cases at discharge while in the same age 4 cases had acute CCF less likely at discharge.

At admission, 19 cases who were between 50-75 years of age had >900 pg/ml and were in the group of Acute CCF likely and 4 cases at discharge while in the same age 4 cases had acute CCF less likely at discharge.

At admission, 7 cases who 75 years of age and above had >1800 pg/ml and were in the group of Acute CCF likely and while in the same age 3 cases had acute CCF less likely at discharge.

8. 0 DISCUSSION

A total of 261 patients aged between 13−94 years old were included in the study. 40.9% were males. The previous study by Oyoo had 48.4% males. Most patients were between 20 and 60 years of age. The peak was between 20 and 40 years. Males had peak between 20-30yrs, then decline was gradual.

Most of the patients were of the low income group: either unemployed or unskilled workers. 52.9% were unemployed, 31.4% were employed (professionals and unskilled), 11% were students and 4.6% were retired. In the previous study by Oyoo, whose heart failure cases were in the following groups. 39.6% unemployed, 28.6% employed skilled professionals and 16.5% unskilled workers.[16] The cases who were unemployed has increased in last 15 years.

91% of study subjects were in classes III and IV. This is similar to the finding of Oyoo (1995) who reported that 94.5% of patients with heart failure were in classes III and IV. In the frequency of symptoms, shortness of breath and easy fatigability were 100%. Palpitation, night cough and orthopnoea were all more than 90%. The clinical signs raised jugular venous pressure, tender Hepatomegaly, were found in more than 80% of the cases. This observation may be related to the fact that Kenyatta is a referral hospital and hence one is more likely to encounter sicker patients, and constraints of in-patient beds necessitates the most sick to be admitted.

Cadiomyopathy was present in 42.7% of the study subjects making it the most common cause of admission for patients in congestive heart failure. This may be related to site of study, older and are more decompensated thus likely to get admitted. Community prevalence among compensated heart failure cases may be different. Oyoo G.O.,[16] in 1995, found the prevalence to be 25.2%. This represents almost double the prevalence (1.8 times) over a 13 year period. Mayosi *et al.* in 2007, showed the incidence of cardiomyopathy to be 20% as a cause of heart failure in

Africa.[214] Parkar, in Nairobi and Nhonoli in Dar-es-Salam, found that 10% and 11% respectively, of cardiovascular admissions were due to cardiomyopathy.[206,210] Barr reported cadiomyopathy to be the third most common cause of emergency cardiovascular admissions accounting for 8 out of 115 admissions.[206]

Among cardiomyopathies, dilated cardiomyopathy was the most common with a prevalence of 40.9%. Idiopathic and alcoholic dilated cardiomyopathy accounted for 21.6% and 18.5% respectively. The majority of patients with dilated cardiomyopathy were above the age of 30 years. Rees *et al.* found that eight of the thirty one patients were under the age of 45 years [212] Nhonoli reported that two out of twenty-five patients were under 20 years old, the rest being over 30 years old. [210] Ogola[17], found a mean age of 42.4 years. with a range of 18-78 years. It is more common than RHD and this change is significant as the previous study was by Oyoo et al. was hospital based study.

Rheumatic heart disease (RHD) was the second most common aetiology associated with heart failure and was seen in 27% of all patients. This finding is comparable to that reported by Oyoo[16] Barr[205] Parkar[206] and Gikonyo[207]. Oyoo G. O., in 1995, found that rheumatic heart disease, at 31.9%, was the most common aetiology among patients admitted with congestive heart failure at Kenyatta National Hospital. Mayosi (2007) published data of 12 hospital based studies over the period 1957 to 2005 involving 4,549 patients in eight countries, and showed that rheumatic heart disease was responsible for 22% of heart failure cases in Africa[215] Barr R.D., in 1972, reported that rheumatic heart disease, at 26.1%, was the most common cardiovascular cause of emergency admission to adult medical ward at Kenyatta National Hospital. While analyzing notes on 500 patients attending the special cardiac clinic in Nairobi during 1969, 1970 and 1971, Parkar found that rheumatic heart disease was the most common cardiovascular pathology in the patients, at 50%. Gikonyo[207] observed that RHD accounted for 74.6% of cardiac clinic attendants between 1975 and 1980 at KNH paediatric cardiac clinic. A similar pattern was observed in Ethiopia.[208]

The predominance of rheumatic heart disease among younger members of our population is not new and has been observed before. The disease affects a younger age group in the developing world as opposed to developed world. [209,207,208,210] That it

is rare above 50 years may be an indication of high mortality associated with this condition. In the previous study done by Oyoo G. O [16], it was rare above 30 years of age. Advances in drug accessibility and surgical treatments may be responsible for this favourable change in outcome.

Hypertensive heart disease was present in 18.1% of patients. In 1995, Oyoo found that hypertension accounted for 17.6% of all cases of heart failure.[16] In 1972, Brar found that hypertension accounted for 25% of cardiovascular admissions being the second most common cause of admission.[205] Nhonoli, in Dar-es-Salam, found that hypertensive heart disease was the most common heart disease.[210]

Reviews on cardiovascular diseases from many parts of Africa have reported that hypertension accounts for a considerable proportion of cardiovascular admissions. Hypertensives and Rheumatic Heart disease jointly account for 70% of all cardiovascular admissions including heart failure.[213,208] In this study, the three conditions accounted for 87.8% of all admissions due to congestive heart failure. Mayosi from Capetown, noted that Hypertensive heart disease accounted for 23% of all heart failure admissions in Sub-Saharan Africa.

Previous studies in East Africa raised concerns of rise in blood pressure in older age groups and the presence of hypertension.[213] More recent data from hospital admissions suggest that hypertension is a major problem.[213] In this study, cases of hypertensive heart disease above the age of 30 years accounted for 85.5% of the total cases of hypertensive heart disease. Although patients characteristics differ in these studies, the prevalence of HHD has not differed significantly. The shows that there is now more cases of hypertension than was before.

Cor-pulmonale accounted for 18.9% of congestive heart failure admissions to adult medical wards at Kenyatta National Hospital. In 1995, Oyoo G.O. , found the figure to be 7.7%.[16] In 2007, Mayosi found the figure to be 2%[214] Previous studies, done prior to the 1980s by Barr, show 3.5%[205] In 1976, Obienche found the figure to be 5.3% of all cardiovascular admissions in Lusaka, Zambia.[209] The higher prevalence of Cor-pulmonale in our study can be explained by the increased incidence of

pulmonary diseases and its sequelae, increase in longevity of patients with easy accessibility to drugs. Although the causes of Cŏr-Pulmonale were not looked into and this needs another study to evaluate.

Ischemic heart disease accounted for 7.3% of the congestive heart failure admissions to adult medical wards at Kenyatta National Hospital. Oyoo G.O., in his study found the figure to be 2.2%.[16] Previous study in 1972, IHD was not reported among the 115 emergency cardiovascular admissions.[205] Mayosi, in his publication in 2007, reported the figure to be 2%.[214] Parkar reported 7 cases (1.5%) out of 523 patients attending specialised cardiac clinic [206] Nhonoli reported 1 case (0.5%) out of 226 patients in Dar-es-Salam.[210] Obienche reported 1.8% of 'Diabetic' CHF in Lusaka.[209] This increase in Ischemic heart disease can possibly be explained as being due to: (a) change in lifestyle, i.e. food habit, towards western diet; (b) increase in availability of diagnostic gadgets and; (c) physician awareness. However, patients had other factors such as hypertension and obesity, which could have also contributed. The risk factors for this increase in incidences is needed to be looked into.

16 out of 19 patients were more than 50 years of age, which implies that other risk factors for coronary artery disease play a role. 2 patients who were less than 30 years had obesity but other risk factors could not be ascertained.

Prevalence of Pericardial disease in this study was 5.4% of the total congestive heart failure patients. Oyoo found the figure to be 12%.[16] Mayosi from Capetown found the figure to be 3% from the 12 studies.[46] In Nairobi, Barr reported two cases (1.7%) out of 115 emergency cardiovascular admissions.[205] In 1973, Parkar reported [206] that pericardial disease accounted for 1.5% of cardiovascular disorders in Nairobi while Obienche (1976) reported a prevalence of 2.9% in Lusaka.[209] Nhonoli (1968) reported a prevalence of 0.9% in Dar-es-Salam.[210] In Ethiopia, pericardial diseases increased from 1.7% in 1963 to 6.2% in 1993.[40] There is an apparent decrease in pericardial disease. Although there is improvement in diagnostic methods, increase in tuberculosis cases and possibly increase in HIV and HIV related infection, but this requires further study to know the apparent reason.

Congenital Heart Disease accounted for 1.2% of the admissions. Oyoo found a figure of 2.2%[16], Barr found 1.7%[205] while Obienche found 1.2%.[209] Nhonoli reported that congenital heart disease accounted for 1.3% of heart disease in Dar-es-Salam, in 1968.[210] Mayosi, in his pooled data, found the prevalence of Congenital Heart Disease to be 3%. However, in 1973, Parkar reported that congenital heart disease accounted for 20% of diagnoses among patients attending specialised cardiac clinic, both paediatric and adults. Congenital Heart Disease is not a significant cause of congestive cardiac failure in adult patients in our set up because most cases died earlier in life or corrected earlier in life.

Our study reported one case of Peripartum cardiomyopathy. Cases may have missed as maternity ward was not included.

Looking at the factors predisposing patients with heart disease to develop congestive heart failure, non-compliance came out as the main reason for decompensation in our patients, with a figure of 83.1%, followed by infective endocarditis 8.5%. Oyoo found the figure to be 27%[16] and Infective Endocarditis to be 3.3%. The reasons for this may be :

(a) Most of our patients are from low income groups or are dependents and may lack the resources to purchase drugs or visit hospitals.

(b) The low level of education among our patients would make it difficult to understand the disease process and the need for compliance.

(c) There is irregular drug availability at the Kenyatta National Hospital pharmacy.

(d) The number of cases who were employed stood at a figure of 31.4%. This high level of unemployed (52.9%) pool of cases suggest as factor for non-compliance.

(e) The number of patients seen at clinics are so high that patients do not get enough time to be explained to the nature of disease and the need for compliance.

(f) The patients get long periods of time between the booking dates. This is another reason that they run short of medication or cannot renew the prescription.

Since heart disease is a chronic disease, it needs firm commitment and resolve towards drug compliance. The medical team should stress the importance of compliance.

Bacterial Endocarditis was present in 8.5% of cases. Oyoo, in 1995, found the prevalence to be 3.3% while Barr, in 1972, recorded 0.9%[205] Ochieng noted that infective endocarditis contributed to 1.5% of cardiovascular admissions at Kenyatta National Hospital. The proportion is higher in the present study, possibly because not all cardiovascular diseases were considered. The study recruited only patients in congestive cardiac failure. The diagnosis of endocarditis among all these patients was made by echocardiography. All blood cultures were negative. The study may have missed some cases since Mintz and co-workers reported that M-Mode echocardiography only detects 35% to 55% of vegetations.[215]

The mean length of hospital stay was 7.1 days. The median was 6 days while the range was 2 to 29 days. There is no data available in Kenya or Africa on in-patient mortality and duration of hospital stay. The mean length of hospital stay in Europe ranges from 9 to 11 days and if they were admitted to ICU/CCU then the mean length of stay was 15 days.[219] In the United States, the mean length of stay ranges from 4.3 days to 6.4 days, and mean ICU/CCU length of stay is 2.4 days.[220,221] Although, the length of stay is longer in this study it is comparable to some European studies as above. But it is far from data from USA.

The Case fatality rate in our study stands at 14.1%. There is no data to compare locally or in Africa. The data available from studies outside Africa, estimate a case fatality rate of between 4% and 7%. In the OPTIME-HF study, which studied 949 cases, the mortality was 3%. The IMPACT-HF registry studied 567 cases and found a mortality of 2.6%. The ADHERE registry which studied 187,565 cases, had a case fatality rate of 3.8%. The OPTIMIZE-HF study studied 41,267 cases had a case fatality rate of 3.8%. European studies had higher mortality rates: An Italian study by Tavazzi, with 2,807 cases, found a mortality rate of 7.3%; EHFS studied 3,580 cases and found a mortality rate of 6.7%. The case fatality rate is high in our study. There is a need to study the factors that are responsible for higher mortality. The possible

explanations which can be forwarded in our institution are: (a) lack of work force; (b) availability of drugs and ; (c) availability of equipment.

Fonarrow G.C., in 2003, presented the CART methodology, where risk stratification for hospital mortality in heart failure, using Classification and Regression Tree methodology was used. Mortality was high (22%) when all the three parameters (creatinine exceeded 2.75 mg/dl, BUN was more than 43 mg/dl and Systolic Blood Pressure was lower than115 mmHg) were increased simultaneously. In the absence of all three, mortality was 2%.[216]

Ischemic Heart Disease had the highest case fatality (26.3%), followed by Hypertensive heart disease (19.1%), rheumatic heart disease (14.5%) and Dilated cardiopmyopathy (9.7%). Mortality rate due to Cardiovascular diseases in Sub-Saharan Africa is 9.7%.[217] Mortality rate is 3.2% for Ischemic heart disease, 0.2% for rheumatic heart disease and 0.6% for hypertensive heart disease.[217] The overall high mortality rate may be explained by factors similar to the case fatality rate. However, these common four diseases need to be treated more vigorously.

Preserved Left Ventricular Ejection Fraction (PLVEF) / Heart Failure with Normal Ejection Fraction (HFnlEF):

The frequency of systolic heart failure and heart failure with normal ejection fraction was assessed using different cut-off levels, in patients without valvular heart disease. At a cut-off of 40%, 39.5% of cases had HFnlEF, at 45% cut-off, it was 36.4% and at 50% cut-off, the percentage of cases with HFnlEF was 28.7%. So, the higher the cut-offs the less number of cases that fall under HFnlEF.

Prevalence of HFnlEF among patients admitted for HF at single large institution had increased dramatically over a 15 year period from 1987 to 2001.[176] Thirteen epidemiological studies have defined the prevalence of HFnlEF in various HF population and have documented a prevalence of 50% to 55%.[176,177,178,179]

The prevalence of Systolic heart failure and Normal ejection fraction heart failure at cut-off of 40%, and stratification by gender, ventricular hypertrophy and Atrial Fibrillation. Patients tend to be of older in the HFnlEF, which is in keeping with the

trend that it is more common with older age groups.[180] The prevalence increases more dramatically with age and is much more common in women than in men at any age.[180] Males were 61.7% in systolic HF while in HFnlEF they were 39.3%, which was statistically significant. This is in keeping with the trend that HFnlEF is less common in males.

In the present study, females were 46.8% in HFnlEF while males were 53.2%. This was not statistically significant (p=0.420) and HFnlEF is much more common in women than in men at any age.[180] More studies are needed to support this finding.

Left ventricular Hypertrophy was equally prevalent in both groups, 47.5% in syst. HF and 52.5% in HFnlEF. This was statistically not significant (p=0.281).

Although, HFnlEF has been thought to occur primarily in patients with LVH, criteria for LVH is met in less than 50% of patients.[182,183,184,185] LVH is not invariably present in HFnlEF, in which cardiac phenotype is variable.[186] There is some evidence that prevalence of LVH may be higher in African American HFnlEF patients.[188] Since LVH is present in more than 50% of cases, this needs to be supported by further studies. The mortality for HFnlEF is similar to HF with reduced EF.[177,178,179,180] Survival for HF patients with reduced EF has improved, it has not changed for patients with HFnlEF.[181] In our study, Atrial Fibrillation was present in 34.9% of cases in HFnlEF and 65.1% in systolic HF. However, this was statistically not significant (p=0.140). Atrial Fibrillation is recognized as a frequent precipitant of acute decompensation in patients with HFnlEF. Atrial arrhythmias are recognized precipitant of acute decompensation in HFnlEF. Bradyarrhythmia, caused by first degree heart block may adversely affect LV filing in some patients. AF may cause decompensated HF in patients with diastolic dysfunction. Diastolic dysfunction (in the absence of heart failure) is also a risk factor for AF.[188] Thus, diastolic dysfunction, AF and HFnlEF share a common pathogenic mechanism in the elderly.

The mean duration of length of stay, taking into account all cases, according to EF, was analyzed. Patients with systolic heart failure had a mean of 7.85 days, median of 7 days and range of 2–29 days, of hospital stay, while in HFnlEF, it was 7.52 days, 6 days and 2–28 days respectively. This was not statistically significant (p=0.663). When we analyzed the data excluding rheumatic heart disease in systolic HF, the mean length of stay was 8 days, median being 7 days, and the range was 2–29 days, while in HFnlEF, the mean was 7.29 days, median 6 days and the range 2–20 days.

This was statistically not significant (p=0.430). There is no difference in length of stay between systolic and diastolic HF. There was no statistical difference in mean length of stay in hospital with or withought HFnlEF.

The mortality in systolic HF in hypertensive heart disease is 12.5% while in HFnlEF it is 4.2%. In Ischemic heart disease with systolic HF, mortality was 25% while in HFnlEF there was none. Patients with IHD were more prone to die if they had systolic HF. Patients with Dilated cardiomyopathy and systolic heart failure had a mortality of 4.5%, while those with HFnlEF had a mortality rate of 5.6%. Mortality was not different for both groups of heart failure. Mortality rate was 3% in systolic heart failure while in HFnlEF it was 8.6% (p=0.097). When rheumatic disease was excluded, the mortality in systolic heart failure was 3.9% while it was 5% for patients with HFnlEF (p=0.754). There is no difference in mortality between systolic and diastolic heart failure.

A total of 21 patients had QRS duration of more than 120 ms. When rheumatic disease was excluded 19 patients had QRS duration of more than 120 ms. Increase in QRS duration in older age group needs to be looked for as cardiac resynchronization therapy is of a choice.

Serial increases in QRS duration lead to an increase in adverse cardiac events. This we did not intend to do. Aranda *et al.* Demonstrated that QRS duration in patients with HF has greater variability than patients withought HF, leading them to conclude that repeat measurement of duration should be performed in the outpatient to evaluate whether prolonged QRS duration during hospitalization remain candidates for CRT.

Pharmaco-therapeutics:

Digoxin

Digoxin was used in 77% of cases at admission and 72·3% at discharge. Although more than 90% of cases belonged to NYHA Class III and IV, the use was not commensurate with the current guidelines. Digoxin prevents hospitalization and worsening of Heart Failure, but does not alter mortality. Clinical benefit has been observed from RADIANCE trial and PROVED trial. Digoxin Investigator Group (DIG) trial, a prospective study, showed decrease in death due to progressive pump failure. The use of digoxin was most common among patients, with rheumatic heart disease at 68.6%, followed by 63.2% in Ischemic Heart disease, 62.5% in Dilated cardiomyopathy and was least in Hypertensive Heart disease. The mean dose at admission was 0.150 μg and at discharge 0.151 μg. There was a negligible difference in the dose at admission and discharge.

ACEIs

49% of patients at admission and 43% at discharge received enalapril, which explains that either they were stopped due to side effect or there was change to another ACEIs. Possible cause maybe unavailability of particular drug. The mean dose of enalapril at admission was 13.3 mg, while at discharge it was 10.24 mg. The dose decreased may be due to patient improvement or drug intolerance. Captopril was used less frequently. At admission it stood at 36.6% while at discharge it was 36.1%. The mean dose per day was 40 mg and 39.4 mg at discharge. So , there was neither difference of frequency of use nor any difference in mean dose. CONSENSUS 1 showed a 31% reduction in mortality, SOLVD Treatment Trial, 21%. ACEIs reduce mortality in direct relationship to degree of Heart Failure.

ARBs

Lorsatan was used in 9 cases at admission and discharge. Candesartan was used in 4 cases at admission and at discharge. In CHARM-Alternative and CHARM-Added , Candesartan reduced all cause mortality, cardiovascular deaths and hospital admission.

ACEIs/ARBs

Those were used 89.6% of patients with hypertensive heart disease, closely followed by 87.5%. 77.1% in rheumatic heart disease received either of them. So, the use of

these medication was common across the disease aetiologies, although nobody in Ischemic heart disease group received this medication.

Diuretics (Frusemide)

Frusemide use was quite common, 89% at admission and 84.8% at discharge. There was no difference at admission and at discharge. Frusemide was use in more than 80% of cases in all aetiological classes. The mean dose at admission was 114.8 mg compared to 75.3 mg at discharge. This shows that the use is less once the decompensation is dealt with.

β-blockers

Carvedilol was used most frequently. Reduced mortality at one year by 38% and relative risk of death or Heart Failure hospitalization by 31% in COPERNICUS study. The use of carvedilol was 34% at admission and 37.7% at discharge suggesting that more patients received carvedilol at discharge although overall use was less. 46.2% received in dilated cardiomyopathy group with 44.3% in RHD and 41.7% in hypertensive heart disease group. The average dose was 7.1 mg at admission and rose to 8.3 mg at discharge. This suggests that doses were stepped up at discharge. In ANZ-Carvedilol trial, there was 26% risk reduction of death or hospitalization at 19 months. Rates of hospitalization were 10% lower than placebo.

Aldosterone Antagonist (Spironolactone)

56.5% received this at admission and equally at discharge. 73.1% were in dilated cardiomyopathy, 65.7% in rheumatic heart disease and 58.3% in hypertensive heart disease. The average dose was 26.6 mg at admission and 26.4 mg at discharge. RALES study, involving 1663 pts, with NYHA Class III & IV showed a 25% reduction in mortality from cardiovascular causes.

Aspirin

11% of cases received this at admission and 14.1% at discharge. Although Aspirin decreases incidence of myocardial infarction, and reduces stroke and death in patients with transient ischemic attacks, prescribing in heart failure awaits clarification from a conclusive study.[218]

Statins

In our study, 22.5% received it at admission while 25.1% received it at discharge. The use at discharge was more and possible explanation may be due to other risk factors but not for heart failure although there is benefit in reducing the number of hospitalizations, in particular to rosuvastatin. Nobody received this drug in our study population. Advantages in Heart Failure : Given Coronary Artery Disease is main cause of Heart failure in the West, prevention of one should prevent the other.

NT-proBNP

49 out of 50 cases had high level of NT-proBNP level at discharge and this is in keeping with the sensitivity of the test as an acute care unit lab test for heart failure. At discharge one case still had high level NT-proBNP and possibly had not improved with management or had other reasons for increase in level of NT-proBNP.

When the age and blood level of NT-proBNP was used 10 cases (the above one included) were in failure at discharge. And at admission all had high levels indicating of acute CCF likely. This is in concordance with many studies.[169, 170, 171, 172, 173,174, 175].

9. CONCLUSION:

Heart failure is no longer just a disease of young but with data from this study it is now common in older age group as cardiomyopathy is the commonest cause of heart failure contributing to 42.5%. Non-compliance is the commonest cause of acute decompensation. The aetiology of heart failure cases at KNH has evolved over the last 15 years since previous study so that dilated cardiomyopathy has become the most common (40.1%) followed by rheumatic heart disease (27%), hypertensive heart disease (18.1%). Ischemic heart disease has increased from 2.2% (1993) to 7.3% today, a very significant increase. The prevalence of heart failure with normal ejection fraction stands at 39.5%. Serum level of NT-proBNP is a sensitive marker of acute heart failure at admission and response to therapy at discharge. QRS duration of >120 ms, was observed in the older age group. Patients had average duration of hospital stay of 7.1 days and case fatality rate of 14.1% and this is higher compared to western studies.

10. RECOMMENDATION:

1) Heart failure now affects the all age groups and preventive measures should aim at not only rheumatic heart disease but at reducing cardiovascular risk factors such as excessive alcohol intake and hypertension
2) Routine use of serum NT-proBNP levels at emergency department would of immense help.
3) Policy measures to address availability and affordability of drugs need to be taken to reduce non compliance, the main precipitating factor for heart failure in our set up.
4) Further, studies directed to address the long duration of hospital stay and high mortality would help to justify.

11. STUDY LIMITATIONS

1) This was a hospital based study with biased sampling including only patients with severe heart disease.
2) Kenyatta National Hospital being referral institution cases with decompensated heart failure might not have been admitted due to shortage of beds.
3) This study could not elucidate exact cause, as myocardial biopsy, coronary angiography could not be done.
4) We did not have a baseline ECG nor a follow up one. So, it is uncertain whether the prolonged QRS duration was new or a pre-existing one at admission. Our findings may not be applicable to an unselected population, given the specific inclusion and exclusion for the study. Delta QRS (the difference in QRS duration)
5) Tissue doppler imaging was not used, as this is more sensitive in detecting heart failure with normal ejection fraction and to detect dyssynchrony, which we could not do to compare in our population.
6) Since, this is a tertiary care hospital, the findings cannot be generalized.

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APPENDIX – I

CONSENT FORM

(To be administered to patients)

CHARACTERIZATION OF HEART FAILURE ACCORDING TO AETIOLOGY AND ITS ASSOCIATION WITH THERAPEUTIC INTERVENTION AND OUTCOME IN IN-PATIENTS WITH HEART FAILURE AT KENYATTA NATIONAL HOSPITAL.

Name: ..... Ward..... Study No.....
Patient No:..... Room..... Date .....

I, Dr. Sanjeev Parmar am a postgraduate student at University Of Nairobi. I am studying type of heart failure and the various medication commonly used and status of patients at discharge.

Basis of participation

Your participation will be purely voluntary. If you feel uncomfortable with some questions you are free to decline them. 5cc of blood shall be drawn for analysis of NT ProBNP level at admission and at discharge.

Benefits

The results of this study may be published in a medical book or journal or for teaching purposes and will be given back to the community for better understanding of heart failure. However, your name or other identifiers will not be used in any publication or teaching materials.

Risks

There will no risks directly or indirectly whatsoever associated with this study.

Privacy and Confidentiality

I shall neither use your name in any of my reports.

Request for more information

You may ask more questions about the study at any time or at this moment. You will be informed of any significant findings discovered during or after the study.

Consent to participate

I have read this consent form, all my questions have been answered. My signature below indicates my willingness to participate in this study and my authorization to use and share with others.

SIGNATURE:

---------- ----------
Patients/Guardians/Caretakers Date and Time

I confirm that explained to the patient the above statement.
Signed..... Date..... (Interviewer)

Next of kin / Caretakers (at least two): **Contact 1** **Contact 2**

1. Name:
2. Relationship:
3. Tel. Contact:
4. Workplace Contact: a) Company Name-
   b) Tel. No.-
   c) Physical Add.-

SCREENING PROFOMA

Hospital No.....Ward---- Age:----- Sex:-------Date------

Patient Status: Available..... Deceased.....

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

CASE	NOT CASE

Study No. ☐☐☐☐☐☐☐☐
Initials. ☐☐☐☐

STUDY No.....WARD.....

STUDY PROFOMA

A. PATIENT DETAILS

i. Date..... A

ii. Hospital no..... B

iii. .Sex..... MALE = 1 FEMALE = 2 C

iv. Age..... D

v. District of birth..... E

vi. Residence within past 5 years..... F

vii. Formal Education level (Tick one) G

A. NONE =0

B LOWER PRIMARY = 1

C UPPER PRIMARY=2

D. SECONDARY=3

E COLLEGE/DIPLOMA=4

F GRADUATE =5

Study No. ☐☐☐☐☐☐☐

Initials. ☐☐☐

B. **HISTORY**

(i) Symptoms present (=1) absent (=0)

A. HShortness of breath on exertion…….. ……..

B. IEasy fatiquability…….. ……

C. JPalpitations…… ……

D. KNight cough…… ……..

E. LLeg swelling…… ……..

F. MHaemoptysis…….. ……

G. NOrthopnea…… ……..

H. ONight sweats…….. ……

I. PAbdominal swelling…… ……..

J. QFever…….. ……..

K. RWeight loss…….. ……..

L. SAnorexia…….. ……

M. TYellowness of eyes…….. ……

N. UVomiting…….. ……..

(ii) NYHA Functional class……

Study No. ☐☐☐☐☐☐☐

Initials. ☐☐☐

V (iii) Past history of admission for heart failure

YES=1	NO=0

W (iv) Social/occupational history

A1 . History of past or current smoking (Tick one)

YES=1	NO=0

A2 If yes No. of pack years

X

<1/4	¼-1/2	1/2 – 3/4	¾-1
1- 5	6 -10	11- 20	>20

Y B1 History of past or current alcohol consumption

YES =1	NO =0

B2. If yes, what is the type of alcohol?

CHANG'AA	OTHER LOCAL BREWS	COMMERCIAL BEER	SPIRITS

B3 What is the unit of measure……..

B4 How many units per week ……

B5 For how many years……

Z C. Occupation/ employment status (Tick one)

STUDENT =1	EMPLOYED =2	RETIRED =3	NOT EMPLOYED =4

AA D. Marital status (Tick one)

SINGLE=0	MARRIED =1	SEPARATED/DIVORCED =2	WIDOWED =3

Study No. ☐☐ ☐☐☐ ☐☐

Initials. ☐ ☐ ☐

C. PHYSICAL FINDINGS AT RECRUITMENT

General examination at recruitment:

i. AB Systolic Blood Pressure……..

ii. AC Diastolic Blood Pressure……

iii. AD Heart Rate……..

iv. AE Respiratory rate……

v. AF Temp……

vi. AG Weight…… On admission……..Height……

AH B…On day 5……

AI C... Weight difference……

AJ D…On discharge……

vii. AK Leg oedema …… (1) …….(0)

viii. AL Jaundice…….... (1) …….(0)

ix. AM Wasting…… (1) …….(0)

Cardiovascular system findings

i. AN JVP……... RAISED 1 | NOT RAISED 0

ii. AO Pulse: volume.. HIGH 2 | NORMAL 1 | LOW 0

iii. AP Pulse rhythm … REGULAR 1 | IRREGULAR 0

iv. AQ Apex beat…… DISPLACED 1 | NORMAL 2 | DIFFUSE

Study No. ☐☐☐☐☐☐☐

Initials. ☐☐☐

v. AR Parasternal heave..... (1) .....(0)

vi. AS Murmurs..... (1) .....(0)

vii. AT Gallop..... (1) .....(0)

Abdominal findings

i. **AU** Ascites ... . (1) .....(0)

ii. **AV** Tender hepatomegally.... (1) .....(0)

iii. AW Hepatojugular reflux..... (1) .....(0)

Respiratory findings

i. **AX** Respiratory distress.... (1) .....(0)

ii. AY Basal rales ..... (1) .....(0)

iii. **AZ** Pleural effusion ..... (1) .....(0)

Significant findings in other systems

....

D. LABORATORY/RADIOLOGICAL FINDINGS

i. Haemogram

A. BA Hb.....g/dl

B. BB MCV.....fl

Study No. ☐☐☐☐☐☐☐

Initials. ☐☐☐

ii. Renal Function

BC A. Creatinine.....

BD B. Na+.....

BE C. K+.....

iii. Chest X-Ray findings

BF A_1 Gross cardiomegally	YES	NO
BG A_2 Cardiothoracic Ratio.....		
BH B..Kerly B lines	YES =1	NO =0
BI C..Pleural effusion	YES =1	NO =0
BJ D..Alveolar oedema	YES =1	NO =0
BK E . Cephalization	YES =1	NO =0

(c) EKG REPORT

BL**(1) QRS duration:**

1)	< 120ms,	1
2)	120 – 160ms	2
3)	> 160ms	3

BM (2) QRS – LBBB [1] RBBB [2]

BN (3) NSR (Normal Sinus Rhythm) (Present=1,Absent=0) [ ]

BO (4) AF (Atrial Fibrillation) (Present=1,Absent=0) [ ]

BP Any other positive findings on EKG:.....

Study No. ☐☐☐☐☐☐☐☐

Initials. ☐☐☐

(iv) ECHOCARDIOGRAPHY REPORT

a) BQ Echocardiographic Diagnosis : 1) Dialated Cardiomyopathy

2) Restrictive Cardiomyopathy

3) Valvular Heart Disease

4) Hypertrophic Cardiomyopathy

5) Ischaemic Heart Failure

6) Hypertensive Heart Disease

b) L.V Dimensions :

Ejection Fraction..... BR- LVSD ☐☐☐

BS-LVDD ☐☐☐

BT c) LVES.....

BU d) LVED.....

BV e) EDV.....

BW f) FS.....

BX g) Pulmonary Artery Pressure.....

LV inflow:

BY Mitral E.....m/s BZ Ratio----------

CA Deceleration time----------

CB IVRT----------

CC LV Wall thickness:

CD IVS(Dias).....

CE LVPW(Dias).....

CF Aortic Root.....

CG Aortic Cusp Sep.....

Study No. ☐☐ ☐☐ ☐☐ ☐☐

Initials. ☐☐☐

CH Left Atrium.....

CI Rt Ventricle.....

Aortic Valve:

CJ Peak velocity----------

CK Max. Pressure Gradient----------

CL Mean Pressure Gradient.....

CM AOV area----------

CN AI---------- Present 1

Absent 2

Mitral Valve:

CO Mean pressure gradient----------

CP MVA----------

CQ MR----------

TricuspidValve:

CR TR----------

CS Max. TR Velocity----------

CT Max PA Velocity----------

Pulmonary Valve:

CU PI----------

Study No. ☐☐☐☐☐☐☐

Initials. ☐☐☐

(v) AETIOLOGICAL CLASSIFICATION OF CCF : Yes = 1, No = 0

CV (i)Hypertensive Heart Disease ☐

CW (ii) Rheumatic Heart Disease ☐

CX (iii) Congenital Heart Disease ☐

CY (iv) Pericardial Disease ☐

CZ (v) Ischemic Heart Disease ☐

DA (vi) Cor-Pulmonale ☐

DB (vii) Cardiomyopathy: DC 1) Dilated Cardiomyopathy: 1) Alcoholic ☐

2) Idiopathic ☐

DD 2) Hypertrophic Cardiomyopathy ☐

DE 3) Restrictive Cardiomyopathy ☐

DF 4) Peripartum Cardiomyopathy ☐

DG 5) Ischemic Cardiomyopathy ☐

(v) ACUTE DECOMPENSATING HEART FAILURE;

PRECIPITATING FACTORS:

DH (i) Non-Compliance (Anti-Failure therapy)

DI (ii) Anaemia

DJ (iii) Infective Endocarditis

DK (iv) Hyper or Hypothyroidism

DL (v) Acute Rheumatic Fever

DM (vi) Systemic Infection

DN (vii) Others Specify: .....

Study No. ☐☐☐☐☐☐☐
Initials. ☐☐☐

(Increased=1) (Decreased=0)

DO (vi) NT-proBNP:

DP a) Serum Level on Admission--------- ☐ ☐

DQ b) Serum Level on Discharge---------- ☐ ☐

(vii) DRUGS:

Class	**Specific**	**Dosage**		**(OD=A, BD=B, TDS=C, IV=D)**
			0	
DR(ACEnal) ACEs	Enalapril	2.5mg	1	☐
		5 mg	2	
		10mg	3	
		20mg	4	
DS (ACECACaptopril)		6.25mg	1	☐
		12.5mg	2	
		25mg	3	
		50mg	4	

Class	**Specific**	**Dosage**		
			0	
DT (ARBLORS)	Lorsatan	50mg	1	☐
		+HCTZ	2	
DU (ARB Irb)	Irbesartan	150mg	1	☐
		+HCTZ	2	
DV (ARBscand)	Candesartan	8mg	1	☐
		16mg	2	☐

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		+HTCZ	3		☐
			0		☐
	DW (ARBTel)	Telmesartan	80mg	1	
	DX (Bel Ate)	Beta blockers Atenolol	50mg	1	☐
			100mg	2	
			0		☐
	DY (Belicar)	Carvedilol	3.125mg	1	
			6.25mg	2	
			12.5mg	3	
			25mg	4	
			50mg	5	
				0	
Diuretics	DZ (Diurfru)	Frusemide	40mg	1	☐
			80mg	2	
				0	☐
	EA (DiurDig)	Digoxin	0.0625mg	1	
			0.125mg	2	
			0.25mg	3	
			0.5mg	4	
	Class	**Specific**	**Dosage**		
				0	
	EB(statNo)	Statins Atorvastatin	10mg	1	☐
			20mg	2	
			40mg	3	
				0	
	EC (Stat Prava)	Pravastatin	10mg	1	☐
	ED(Stat Rosu)	Rosuvastatin	5mg	1,0	☐

Initials. ☐☐☐

	10mg	2	☐
	20mg	3	☐
EE (Stat Al) Aldactone	25mg	1 ,0	☐
	50mg	2	☐
		0	
EF Aspirin	75mg	1	☐
	100mg	2	☐

(viii) Exit Interview

Health Education

Have you received information from the medical team while in the Ward concerning the following as pertains to your heart problem:

(Received=1). (Not Received=0)

EG i. Weight Monitoring..... ☐

EH ii. Alcohol abstinence..... ☐

EI iii. Avoidance / Quit Smoking..... ☐

EJ iv. Regular Follow up at out-patient clinic..... ☐

EK v. Drug Compliance..... ☐

EL vi. Role of Exercise..... ☐

EM vii. Reduction of Salt intake..... ☐

EN viii. Reduction of Fluid Intake..... ☐

(ix) Disposition

EO a. Discharged on.....

EP b. Died on.....