Medical Journal of Zambia, Vol. 41, No. 1 (2014)

Factors Associated with Mortality in Adults Admitted with Heart Failure at the University Teaching Hospital in Lusaka, Zambia

P. Chansa¹, S. Lakhi¹, B. Andrews^{1,2}, S. Kalinchenko¹, R. Sakr¹

¹ University Teaching Hospital, University of Zambia², Vanderbilt Institute for Global Health³

ABSTRACT

Background: Heart failure is a major public health problem and has been recognized as an important cause of morbidity and mortality for several years. It is one of the leading non-infectious causes of death among hospitalized patients at the University Teaching Hospital (UTH) in Lusaka, Zambia. This study aimed to investigate the predictors of 30-day mortality in heart failure patients admitted to the medical wards at the UTH using routinely obtained clinical data.

Methods: We enrolled 390 heart failure patients and followed them up over a period of 30 days. Data collected included demographic characteristics (age, sex), medication use and co-morbidities (hypertension, diabetes mellitus, Human Immunodeficiency Virus (HIV) infection). Clinical data included vital signs, blood urea, serum sodium, serum potassium, serum creatinine, and haemoglobin level. Trans-thoracic echocardiographs and electrocardiographs were also done to determine left ventricular ejection fraction (LVEF) and to check for the presence of arrhythmias. Patients were dichotomized into those with preserved (LVEF>=40 percent) and reduced (LVEF<40 percent) systolic function. Recruited patients were then prospectively followed up to determine outcome by day 30 (i.e. dead or alive). Cox proportion Hazard regression analysis (on Epi Info software version 3.5.3) was used to analyse the effect of each of these parameters on outcome.

Results: Of the recruited patients, 59% were female (95% CI 54-64). The median age was 50 years (IQR 33-68). 138 patients (35%, 95% CI 31-40) died within 30 days of admission. 94 (68%) of these deaths occurred in-hospital.

The factors shown to be independent predictors of death onmultivariate logistic regression analysis were LVEF<40 percent (OR=2.86, 95%CI 1.68- 4.87), NYHA class IV (OR=2.15, 95%CI 1.27- 3.64),serum urea above 15mmol/L (OR=2.48, 95%CI 1.07-5.70), and haemoglobin level below 12g/dL (OR=1.79, 95%CI 1.11-2.89). The additional factor associated with increased risk of mortality on univariate analysis wassystolic blood pressure below 115mmHg (OR=1.63, 95%CI 1.05- 2.51). However, serum creatinine (OR=1.49, 95%CI 0.49-4.48) and HIV seropositivity (OR=0.96, 95% CI 0.53-1.72)had no bearing on the risk of death in this patient population.

Conclusions: Left ventricular ejection fraction <40 percent, New York Heart Association class IV, serum urea above 15mmol/L, haemoglobin level below 12g/dLand systolic blood pressure below 115mmHg are predictors of poor 30-day outcome in hospitalised heart failure patients.

BACKGROUND

The clinical syndrome of heart failure is characterised by dyspnoea, fatigue, and fluid retention which may develop as a consequence of cardiac disease.^{1, 2} The aetiology is varied³ and ultimately results in impaired diastolic or systolic function.

Heart failure is an important public healthproblem. It has been recognised for several years as one of the major causes of morbidity and mortality worldwide.^{3,4} The burden of heart disease and cost of management of patients is high.^{4,5} In North America almost 20 percent of admissions to hospital are due to heart failure and between 2.1 and 22 percent of these patients die.⁵ In Europe heart failure accounts for 5 percent of admissions to hospital medical wards and the mortality is estimated at about 13 percent.⁶ Unfortunately, the magnitude of this problem in Africa is not well studied as few population-based studies have been done on the prevalence of heart failure and the mortality rates.

The use of prognostic indicators in predicting disease progression and the probability of death is important in clinical practice. Early recognition and treatment of reversible factors indicative of a poor outcome could aid in reducing deaths.

Several studies done in different populations have reached varying conclusions regarding the most accurate predictors of mortality in heart failure patients. Some of these studies are population-based and describe the longterm probability of death. Such a study is the landmark Framingham heart failure study, a long-term follow-up study started in the 1940s. This study as well as subsequent studies which used data derived from it helped highlight the importance of prognostication in heart failure. Most recently, studies on prognosis in heart failure have focused on the identification of factors which are likely to be indicative of a poor outcome in hospitalised patients. These studies utilise data which is routinely collected on patients' admission to hospital in order to aid clinicians triage patients and provide effective intervention measures.

The clinical prognostic determinants in heart failure can be categorised into the following: (1) patient characteristics and co-morbidities; (2) laboratory parameters; (3) functional parameters and ventricular function; and (4) interventions received.^{7,8}

Some of the documented independent factors associated with high risk of death in heart failure patients include the patient's age, sex, heart rate, blood pressure, New York Heart Association (NYHA) class, haemoglobin (Hb) level, serum sodium, serum urea, serum creatinine, presence of arrhythmias, and the left ventricular ejection fraction (LVEF). Recently, the use of biomarkers (Brain Natriuretic Peptide, Cystatin C, Troponin T and Growth differentiation factor 15) to predict mortality has been investigated, particularly in the Western world with varying conclusions regarding the biomarker with the most accurate prediction of poor outcome. For several years, heart disease has been one of the major causes of morbidity and mortality at the University Teaching Hospital (UTH). Hospital records indicate that the proportion of deaths has increased from 30 percent to 44percent in the last ten years. The aim of this study was to establish some of the factors associated with 30-day mortality in patients admitted with heart failure at the UTH using routinely collected clinical data in order to aid reduce deaths.

METHODOLOGY

Study Design

This was was a cohort study with a follow-up period of 30 days.

Study setting and population

This study was conducted in the admitting medical wards at the UTH in Lusaka. Enrolment and follow-up of patients was done between November 2010 and September 2011. Patients above the age of 18 years who gave written consent and met the case definition of heart failure according to the European Society of Cardiology guidelines were recruited into the study. Patients with pericardial effusion and constrictive pericarditis were excluded (definitive management these patients would involve either drainage of fluid from the pericardial space or pericardectomy, and treating the underlying cause as opposed to administering standard anti-failure medication).

Clinical Procedure

Study patients were recruited from the admitting medical wards. Written consent was then obtained from eligible patients who met the diagnostic criterion of heart failure. Information gathered included patient demographic characteristics (age, sex, contact phone number), previous and current anti-failure medication use, past medical history, functional status (NYHA class) and admission vital signs (blood pressure (BP), heart rate, respiratory rate, and axillary temperature). Blood was collected from each patient for blood urea, electrolytes (sodium, potassium), creatinine, and Hb level soon after hospitalisation for analysis.

LVEF was determined using 2-dimensional transthoracic echocardiography (ECHO) set in motion-mode from left parasternal long axis views. Patients were dichotomised as either having reduced systolic function (LVEF<40 percent) or preserved systolic function (LVEF>=40 percent). Electrocardiography (ECG) were also done on admission and reported on independently by two clinicians. Arrhythmias and conduction defects such as atrial fibrillation (AF) or flutter, left bundle branch block (LBBB), right bundle branch block (RBBB), premature ventricular contractions (PVCs), and Long QTc were documented.

Outcome

Patients were followed up on a daily basis while in hospital. Discharged patients were reviewed in the outpatient clinic and/or contacted by phone at day 30 to determine outcome (i.e. dead or alive). Data collected was then entered into *epi info* software and analysed.

Data analysis

All statistical analysis was done using epi info version 3.5.3 dataset at 95 percent confidence interval.Continuous variables with Gaussian distribution were expressed as means and standard deviation. The student t-test was used to test for statistical significance. Continuous variables with non-Gaussian distribution were expressed as medians and inter-group differences. Kruskal-Wallis test was used in this group to test for statistical significance. Categorical variables were expressed as percentages or proportions. Chi-square test was used to measure the effect of each categorical variable (e.g. LVEF, sex) on the outcome (dead or alive at day 30). Some of the continuous variables (e.g. Hb, systolic blood pressure (SBP), diastolic blood pressure (DBP), serum creatinine) were dichotomised before analysis based on values obtained from previous studies. Univariate and multivariate logistic regression analysis was used to analyse the effect of each of the prognostic features (e.g. age, sex, LVEF) on outcome in heart failure patients (i.e. dead or alive). Cox proportion hazard regression analysis was also used for time-to-event analysis and to determine crude hazard ratios.

Study definitions

For the purpose of this study the following definitions were used.

Heart failure case definition:

As adapted from the 2008 European Society of Cardiology (ESC) Guidelines²

- 1. Symptoms (and signs) of heart failure at rest or during exercise. These include fatigue, breathlessness and congestion of systemic veins (orthopnoea, paroxysmal nocturnal dyspnoea, bilateral fine basal crepitations, dependent peripheral oedema, raised jugular venous pressure, and hepatomegaly), and
- 2. Objective evidence (preferably by echocardiography) of cardiac dysfunction (systolic or diastolic) at rest.

Reduced- or preserved systolic function

The cut-off of 40 percent for LVEF used in this study to categorise heart failure patients as having either reduced or preserved systolic function was adopted from a validated multicentre risk stratification study done in more than 100,000 hospitalised heart failure patients in the United States of America.^{9,10} One of the few documented African heart failure studies on prognosis also utilised the LVEF cut-off of 40 percent.¹¹

Reduced systolic function: LVEF less than 40 percent; Preserved systolic function: LVEF greater than or equal to 40 percent.

Low systolic blood pressure, high serum urea and creatinine

Similarly, the cut-offs for systolic blood pressure, serum urea, and serum creatinine were adopted from the validated risk stratification study mentioned above. High risk patients were identified as having the following abnormal admission parameters:¹⁰

Low systolic blood pressure: Systolic blood pressure less than 115mmHg

High serum urea: Serum urea above 15mmol/L.

High serum creatinine: Serum creatinine above 243µmol/L.

Hyponatremia and hypokalemia (2008 ESC Guidelines)²

Serum sodium below 135mmol/L.and serum potassium below 3.5mmol/L respectively.

Anaemia(2008 ESC Guidelines² and W.H.O definition)¹²

Haemoglobin level below 12g/dL in females and less than 13g/dL in men.

New York Heart Association functional classification

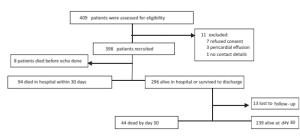
Adapted from the recommendations made by the Criteria Committee of the New York Heart Association of 1994:¹³

- 1. Class I: No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnoea.
- Class II: Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
- 3. Class III: Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnoea.
- Class IV: Unable to carry out any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.

RESULTS

A total of 409 patients were approached as potential candidates for inclusion into the study. Of these, 7 refused to give consent. 3 patients were excluded on the basis of having pericardial disease and 1 had no contact details for follow-up (see *Fig. 1*). 94 patients died while in hospital. Of the discharged patients, 13 were lost to follow-up and 44 were dead by day 30 with a median time-to-death of 14 days (IQR=7-25 days).

Fig 1: Recruited patients



Baseline characteristics of the participants

Patient demographics and clinical findings are shown in *Table 1* and described briefly here. Majority of the patients were female (59%; 95% CI 54-64). The patients' mean age was 48years and 50 years for males and females respectively (p=0.927). 88 patients were HIV positive (23%; 95% CI 19-27).

Table 1: Baseline characteristics of heart failure patients and laboratory measurements

Characteristic	Total n=390	LVEF<40% n=163	LVEF >= 40% n=227	P value
Age, yrs	50 (33-68)	48 (32-69)	52 (34-69)	0.865
Females (No (%))	231 (59)	133 (58)	97 (42)	<0.001*
NYHA class IV (No (%))	119 (31)	118 (72)	153 (67)	0.292
Hypertension (No (%))	130 (33)	49(30)	81 (38)	0.246
Diabetes mellitus (No (%))	19 (5)	8(4.9)	11 (4.8)	0.978
HIV (No (%))	88 (23)	42 (26)	46 (20)	0.200
SBP, mmHg	110 (90-130)	100 (90-124)	110 (100-140)	0.001*
DBP, mmHg	70 (60-80)	68 (60-80)	70 (60-81)	0.009*
Heart rate, beats/min	90 (80-100)	93 (80-102)	90 (80-100)	0.040*
Temperature, ⁰ Celsius	36.1(36-36.8)	36 (36-36.8)	36.3 (36-37)	0.093
Respiratory Rate, breaths/min	24 (20-28)	24 (20-28)	22 (20-26)	0.013*
Underlying cardiac pathology (No (%)):			
Dilated cardiomyopathy Corpulmonale Hypertensive heart disease Valvular heart disease	180 (46) 42 (11) 112 (29) 45 (12)	118 (72) 6 (4) 28 (17) 10 (6.1)	62 (27) 36 (16) 84 (37) 35 (15)	<0.001* <0.001* <0.001* 0.005*
Urea, mmol/L	6.7 (4.8-10.1)	7.0 (4.8-10.3)	6.3 (4.8-10)	0.506
Creatinine, µmol/L	100 (73-131)	98 (71-118)	102 (74-143)	0.076
Sodium, mmol/L	135 (132-140)	135 (131-139)	136 (132-141)	0.123
Potassium, mmol/L	3.86 (3.40-4.42)	3.73 (3.31-4.37)	3.91 (3.46-4.50)	0.267
Anaemia (Hb <12g/ dL)	166 (43)	69 (42)	97 (43)	0.937

Data are expressed as median (IQR) unless stated otherwise * Statistically significant

163 (42%, 95% CI 37-47) of the patients had an LVEF below 40% on trans-thoracic echocardiography. There was no statistically significant difference in age, NYHA class, HIV status or comorbidities such as diabetes or hypertension between those with reduced and preserved LVEF. As expected, patients with reduced LVEF had lower blood pressures and higher heart rates on admission. A significant proportion of patients with reduced LVEF had dilated cardiomyopathy as the underlying cardiac pathology. Those with preserved LVEF had either hypertensive heart disease, corpulmonale or valvularheart disease.

The median values for blood urea, creatinine, sodium, potassium and Hb were comparable between the heart

failure patients with reduced LVEF and those with preserved LVEF.

Table 2: Previous and admission medication

LVEF<40%	LVEF>=40%	P value				
Previous medication (n=142)						
Loop diuretic	65 (40)	95 (42)	0.696			
Angiotensin Converting Enzyme Inhibitor(ACEI) or Angiotensin Receptor Blocker55 (34)68 (30)0.428(ARB)						
Aldosterone antagonist	21 (13)	25 (11)	0.573			
β Blockers	3 (1.8)	5 (2.2)	0.804			
Admission medication (n=3	90)					
Loop diuretic	120 (74)	188 (83)	0.028*			
ACEI or ARB86 (53)	144 (63)	0.035*				
Aldosterone antagonists	31 (19)	37 (16)	0.486			
β Blockers	2 (1.2)	5 (2.2)	0.475			
Digoxin	52 (32)	52 (23)	0.048			
Aspirin	49 (30)	76 (34)	0.476			
Dopamine	39 (24)	22 (10)	< 0.001*			

Data are expressed as number (percent) *Statistically significant

Most of the previously diagnosed heart failure patients were on a combination of loop diuretics with ACEI (or ARBs) and/or aldosterone antagonists. Only 8 patients were on β blockers (Carvedilol) for chronic heart failure. Loop diuretics and ACEIs/ARBs were more commonly prescribed in patients with LVEF>=40 percent on admission. Those with LVEF<40 percent were more likely to receive dopamine and digoxin than those with preserved systolic function.

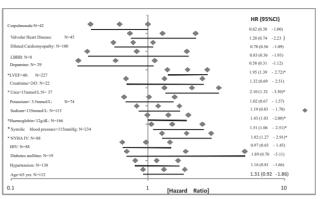
Outcome and Predictors of 30 day mortality

Table 3: Outcome of heart failure patients within 30days of admission

	Total n=390	LVEF<40% n=163	LVEF >= 40% n=227	P value
Death from any cause:	138 (35)	77 (56)	61 (44)	< 0.00 1*
In -hospital Mortality	94 (68)	57 (61)	37 (39)	0.006 *
Discharged then died	44 (32)	20 (21)	24 (79)	0.181
Alive at day 30: Alive in hospital Discharged, alive at day 30	252 (65) 14 (6) 238 (94)	86 (34) 3 (35) 83 (45)	166 (66) 11 (65) 155 (55)	<0.001* 0.006 0.18 1

Data are expressed as number (percent); *Statistically significant

Figure 2: Predictors of death (Crude Hazard Ratios on Cox Proportion Logistic Regression)

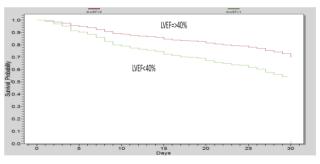


N=number; *Statistically significant

Table 4: ECG findings and the probability of death

Variable	Crude OR	95% CI	Adjusted OR	95% CI
A trial fibrillation or flutter	1.25	0.58-2.67	1.24	0.46 - 3.33
PVCs	1.37	0.77 - 2.43	1.40	0.65 -2.98
RBBB	2.99	0.42 - 21.4	0.26	0.03 - 2.13
LBBB	0.83	0.36 - 1.93	1.86	0.46-7.59
Long QTc	0.50	0.07-3.5	0.45	0.05 - 4.07

Figure 3: Kaplan Meier curve: Survival probability based on LVEF



All-cause mortality was 35 percent with most of these deaths occurring in hospital (see *Table 3*). The factors shown to be predictive of death on both univariate and multivariate logistic regression analysis, and cox proportion hazard regression analysis (see *Figure 2* and *3*) were LVEF<40 percent (OR=2.86, 95%CI 1.68- 4.87), NYHA class IV (OR=2.15, 95%CI 1.27- 3.64), serum urea above 15mmol/L (OR=2.48, 95%CI 1.07-5.70), and Hb below 12g/dL (OR=1.79, 95%CI 1.11-2.89). The additional factor associated with increased risk of mortality on univariate analysis was SBP below

115mmHg OR=1.63, 95%CI 1.05- 2.51). However, serum creatinine (OR=1.49, 95%CI 0.49-4.48), the use of dopamine on admission (OR=1.52, 95% CI 0.79-2.91), and HIV status (OR=0.96, 95% CI 0.53-1.72) had no bearing on the risk of death in this patient population. The underlying cardiac abnormality was not associated with increased risk of death.

A few patients had conduction defects and arrhythmias. The most frequent findings on ECG were PVCs (n=30, 14 percent) and AF (n=18; 8 percent). Only 8 of the patients we studied had LBBB. However, these findings were not associated with an increased risk of death (see *Table 4*).

DISCUSSION

Our patient population was mostly female and younger by almost 20 years compared to other cohorts studied in the North America.¹⁰ This finding was similar to other studies done in Uganda and Nigeria^{11, 14} and was probably due to the higher proportion of valvular heart disease and dilated cardiomyopathy (presumed to be of infective cause) in our patient population compared to coronary artery disease which is found predominantly in males (and postmenopausal females) in western settings.¹HIV seroprevalence in our study population at 23 percent was higher than in the general Zambian population. HIV seropositivity however, was not associated with increased risk of death. The underlying cardiac condition was not associated with increased risk of death. The proportion of patients with ischaemic heart disease was small and this could be an underestimate. Some patients may have erroneously been labelled as having cardiomyopathy. With the lifestyle changes noted amongst the affluent African population, ischaemic heart disease needs to be thoroughly explored in our patient population.

The proportions of deaths in our patient population during the follow-up period was quite high at 35 percent (n=138). This was much higher than the in-hospital mortalities recorded in other studies (i.e. 3 to 4.1 percent). The magnitude of deaths was also 3 fold higher than that documented in a Nigerian study in patients who were followed-up for a period of 6 months.¹¹ However, the authors acknowledged that the study's sample size of 79 patients was quite small. Being a tertiary health institution, most of the patients who present to the University Teaching Hospital are critically ill patients referred from primary health care centres and secondlevel hospitals. In addition, most of the patients present late to health care facilities further contributing to a delay in diagnosis and treatment. Finally, most of our heart failure patients, including those who could benefit from cardiac surgery, have limited access to anti-failure medication as the only readily available treatment option. None of the heart failure patients followed up with poor systolic function had access to intracardiac defibrillators or cardiac transplant. This could explain the higher mortality in patients with poor systolic function in our setting compared to the rest of the world where patients have better access to optimal medical therapy and potentially life-saving surgical interventions.

The independent prognostic predictors of mortality determined from our study included LVEF<40 percent, NYHA class IV, serum urea above 15mmol/L, and Hb below 12g/dL. SBP below 115mmHg were also indicative of poor outcome within the 30 day period of follow-up.

The proportion of heart failure patients with LVEF<40 percent from our study was 42 percent (n= 163). This finding was similar to studies done in the developed world^{10, 15} which found that about half of the admitted heart failure patients had reduced systolic function on transthoracic echocardiography. The majority of the long-term follow-up studies done in North America as well as two studies done in Brazil and Nigeria found that patients with LVEF below 40 percent had worse outcomes. However, unlike our study, these studies had varied periods of follow-up ranging between three and six months. Most of these studies recruited chronic stable heart failure patients who were monitored and treated on out-patient basis. Despite the differences in patient populations and periods of follow-up, we found that LVEF less than 40 percent was an important independent prognostic factor in the patients we studied.

The proportion of the heart failure patients recruited with anaemia was slightly lower than was estimated in other studies on hospitalised patients.¹⁶ Anaemia however, was an independent prognostic determinant of 30-day mortality in our patient population.Even mild degrees of anaemia are associated with worsened symptoms, functional status and survival particularly in patients with poor myocardial reserve.^{17,18}

The prognostic value of high NYHA class has mainly been echoed in long-term follow-up studies on heart failure and not in studies on in-hospital mortality. However, this factor could be of prognostic significance as NYHA class IV on admission indicates poor functional status and significant pulmonary congestion in patients who present with acute decompensation of chronic heart failure.

According to a study done by Fonarow *et al*¹⁰ in-hospital mortality was higher in heart failure patients who had systolic blood pressure below 115 mmHg, serum urea above 15mmol/L and serum creatinine above 243µmoL/L. The prognostic value of these factors was independent of the patients' LVEF. The predictability of high blood urea in prognosis was also in keeping with the findings in Petersen's study.¹⁵ However, high serum creatinine was not found to be an important prognostic factor in our study on analysis, even after adjusting for other variables. The contribution of poor renal function to worsening cardiac function and increased risk of death, however, was not fully explored as estimation of creatinine clearance or glomerular fitration rates were not done due to logistical problems. It is therefore difficult to conclude from these findings that cardio-renal syndrome does not play a role in the deaths of these patients.

The median serum sodium level of 135 mmol/L and serum potassium level of 3.8mmol/L in our patients was comparable to the findings in other Western studies^{10, 19}. Some of our patients however, had deranged electrolyte profiles. 19 percent of the patients in this study had hyponatremia and 30 percent had hypokalaemia of varying degrees. Despite these findings, hyponatremia and hypokalaemia did not contribute to increased risk of death in the patients we studied.

The low number of patients found to have arrhythmias or conduction defects may not be reflective of the actual magnitude of the problem in our heart failure patients as none of the patients we studied had access to continuous cardiac monitoring. Potentially fatal arrhythmias may not have been detected during their hospitalisation. Therefore, the data obtained refuting an association between an increased risk of death in patients with atrial fibrillation and LBBB, in line with major studies, may be inaccurate.

STUDYLIMITATONS

None of the patients had continuous monitoring of cardiac rhythm even whilst in hospital due to the limited number cardiac of monitors for inpatients and lack of ambulatory ECG devices for outpatients in order to detect arrhythmias.

Considering that all-cause mortality was used as one of the end-points and that the study protocol did not include post-mortems, it is possible that some deaths may not have been due to cardiovascular cause, contributing to the high mortality reported in this study.

Some important prognostic indicators were not tested for in this study due to limited resources (i.e. cardiac biomarkers such as BNP, cystatin c and troponins).

The information regarding the predictors of long term prognosis in the heart failure patients we studied was not obtained due to the limited follow-up period.

CONCLUSION AND RECOMMENDATIONS

In conclusion, mortality amongst hospitalised heart failure patients at the University Teaching Hospital in Lusaka, Zambia is high.

LVEF less than 40 percent was found to be a predictor of 30 day mortality in hospitalised heart failure patients at the UTH. Other factors such as NYHA class IV, low systolic blood pressure, high serum urea and anaemia also increased the risk of death of 30 day mortality.

In view of the high mortality, we recommend that patients be triaged according to risk utilising the factors identified. In order to improve survival, heart failure patients who are at low- or high risk of death need regular monitoring so that potentially life-saving measures can be instituted when feasible.

Patients should have ready access to echocardiography and laboratory services as soon as they are admitted to the emergency medical wards. LVEF, Hb levels, and serum urea should be routinely measured in all heart failure patients. NYHA class must be documented and accurate blood pressure measurements taken on all in-patients in order to assess the risk of mortality Patients with LVEF less than 40 percent with refractory heart failure and cardiogenic shock should have access to implantable cardioverter defibrillators and cardiac transplant in order to aid improve survival.

Early referral of heart failure patients to tertiary health institutions for evaluation and management should be encouraged as late presentation to hospital could have contributed to the high mortality reported in this study. We also recommend that public health awareness be increased on the need for patients to seek medical help early in order to reduce mortality rates.

A long term follow-up study on outcome in our patient population with heart failure needs to be done. The state of debility and outcome of these patients post-discharge remains largely unknown. With the current pressure on the few available medical resources in the healthcare system and the human resource crisis, it would be prudent to conduct a long-term follow-up study in order to determine ways of improving healthcare delivery for our heart failure patients in Zambia.

Further studies on cardiac biomarkers such as brain natriuretic peptide, troponins, and cystatin C ought to be done in order to determine the contribution of these factors to prognosis in our patient population.

ACKNOWLEDGEMENTS

This research would not have been possible without the contribution of Dr L. Hachaambwa and the staff at the UTH HIV Medicine Teaching Laboratory who provided the necessary laboratory support. We would also like to thank those working in the ECHO/ECG lab at the UTH. Finally, we greatly appreciate the participation of the heart failure patients we enrolled and their relatives for graciously accepting to be part of this study.

REFERENCES

- NHS Guidelines: Management of Chronic Heart Failure in Adults in Primary and Secondary care. <u>www.nice.orgc.uk</u>. 2010
- ESC Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart failure. *Eur Heart J 2008*. 29:2388-2442

- 3. American Heart Association: American Stroke Association. Heart disease and stroke statistics update. *www.americanheart.org*. 2004.
- Ho K, Pinsky J, Kannel W, Levy D. The Epidemiology of Heart Failure: the Framingham study. J Am Coll Cardiol 1993; 22:6A
- 5. Jessup M. Heart Failure. *NEJM 2003*; 348:2007-2018.
- 6. Velavan P, Khan N, Goode K. Predictors of Short-term Mortality in Heart Failure- Insights from the Euro Heart Survey. *Int J Cardiol 2010*; 138:63-69.
- 7 Levy W, Mozaffarian D, Linker D, Sutradha S, Anker S, Cropp A, *et al.* The Seattle Heart Failure Model. *Circ 2006*; 113: 1424-1433.
- 8. Mosterd A, Hoes A. Clinical epidemiology of heart failure. *Heart 2007;* 93: 1137-1146.
- Somaratne J, Berro C, Mc Murray J, Poppe K, Doughty R, Whalley G. The Prognostic Significance of Heart Failure with Preserved Left Ventricular Ejection Fraction. *Eur J of Heart Fail 2009:* 11; 855-862.
- Fonarow G, Adams K, Abraham W, Yancy C, Boscardin W. Risk stratification for in-hospital mortality in acutely decompensated heart failure: classification and regression tree analysis. *JAMA* 2005; 293: 572–580.
- Karaye K, Sani M. Factors associated with poor prognosis among patients admitted with Heart Failure in a Nigerian tertiary medical centre. *BMC Cardiovasc disord 2008;* 8:16.
- 12. Worldwide Prevalence of Anaemia 1993-2005.W.H.O global database on anaemia. *ISBN 978 92 4* 1596657.
- 13. The Criteria Committee of the New York Heart Association. Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. 9th ed. *Little Brown & Co1994*. p 253–256
- Amoah A, Kallen C. Aetiology of Heart Failure as seen from a Cardiac Referral Centre in Africa. *Cardiology 2000*; 93:11-8.
- 15. Peterson P, Rumsfeld J, Liang L, Albert L, Hernandez A, Petersen E, *et al.* A Validated Risk Stratification Score for In-Hospital Mortality in Patients with Heart Failure from the American Medical Association Get With The Guidelines Programme. *Circ 2010*; 3:25-32

- 16. LindenfeldJ. Prevalence of anemia and the effects on mortality in patients with heart failure. Am Heart J 2005; 149:391-401
- 17. Silverberg D, Wexlerb D, Iaina A, Schwartz D. *The interaction between heart failure and other heart disease and anemia. Semin Nephrol 2006; 26: 296-306.*
- 18. Horwich T, Fonarow G, Hamilton M, MacLellan W, Borenstein J. Anemia is associated with worse

symptoms, greater impairment in functional capacity and a significant increase in mortality in patients with advanced heart failure. *Circ* 2006; 113: 2713-2723.

19. Abraham W, Fonarow G, Albert N, Stough W, Gheorghiade M, Greenberg B, et al. Predictors of inhospital mortality in patients hospitalized for heart failure: insights from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients With Heart Failure (OPTIMIZE-HF). J Am Coll Cardiol 2008; 52: 347–356