Prognostic Significance of ECG Abnormalities for Mortality Risk in Acute Heart Failure: Insight From the Sub-Saharan Africa Survey of Heart Failure (THESUS-HF)

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ABSTRACT

Objective: The aim of this study was to assess the predictive utility of 12-lead electrocardiogram (ECG) abnormalities among Africans with acute heart failure (HF).

Methods and Results: We used the Sub-Saharan Africa Survey of Heart Failure, a multicenter prospective cohort study of 1,006 acute HF patients, and regression models to relate baseline ECG findings to all-cause mortality and readmission during a 6-month follow-up period. Of 814 ECGs available, 523 (49.0% male) were obtained within 15 days of admission, among which 97.7% showed abnormalities. Mean age was 52.0 years and median follow-up was 180 days, with 77 deaths (Kaplan-Meier 17.5%) through day 180 and 63 patients with death or readmission to day 60. QRS width, QT duration, bundle branch block, and ischemic changes were not associated with outcomes. Increasing ventricular rate was associated with increasing risk of both outcomes (hazard ratio [HR] 1.07 per 5 beats/min increase for 60-day death or readmission, 95% confidence interval [CI] 1.02−1.12; $P = .0047$), and the presence of sinus rhythm was associated with lower risk (HR 0.58, 95% CI 0.34−0.97; $P = .0385$). There was a strong association between survival and heart rate in patients in sinus rhythm, with heart rate >119 beats/min conveying the worst mortality risk.

Conclusions: ECG abnormalities are almost universal among Africans with acute HF, which may add to the immediate diagnosis of patients presenting with dyspnea. Although some ECG findings have prognostic value for risk of adverse outcomes, most of them are nonspecific and add little to the risk stratification of these patients. (*J Cardiac Fail 2014;20:45−52*)

Key Words: Africa, heart failure, electrocardiogram, outcome, prognosis.

The 12-lead electrocardiogram (ECG) represents a widely available test, relatively inexpensive, simple to perform, and yields an instant result. It is objective and reproducible. ECG is recommended by the American Heart Association and European Society of Cardiology as initial test in patients with heart failure (HF).[1,2] Indeed, most patients with HF due to systolic dysfunction have a significant abnormality on ECG.[3]

However, the grade and spectrum of ECG abnormalities may differ by HF etiology and possibly other factors, such

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as ethnicity.[4] The current knowledge on 12-lead ECG abnormalities in HF is largely derived from North American and European cohorts (predominantly white men).[4] Sliwa et al recently reported findings from a large study of electrocardiographic findings among heart disease−free Africans,[5] showing that some of the suggested ECG criteria may be less applicable in this population; eg, up to 13% of the studied population presented with significant Q waves in the absence of myocardial ischemia.

To our knowledge, the diagnostic and prognostic utility of the ECG in Africans with acute HF was not been reported. The Sub-Saharan Africa Survey of Heart Failure (THESUS-HF)[6] prospectively collected clinical data in an African cohort of patients with HF during admission and after follow-up, and thus gives the first unique opportunity to study the utility of ECG in patients admitted with HF in this part of the world.

Methods

Study Design and Clinical Setting

THESUS-HF was a prospective, multicenter, international observational survey conducted in 12 cardiology centers from 9 countries in the southern, eastern, central, and western regions of sub-Saharan Africa, as described in detail elsewhere.[6] Ethical approval was obtained from the Ethical Review Boards of the participating institutions, and the study conformed to the principles of the Declaration of Helsinki.

Inclusion and Exclusion Criteria

Patients admitted with dyspnea as the main complaint to each of the participating centers from July 2007 to June 2010 were screened for inclusion in the study. Patients ≥12 years of age with clinical signs and symptoms consistent with congestive heart failure (ie, pedal edema, elevated jugular venous pressure, pulmonary congestion, and tender hepatomegaly) and patients that were prepared to continue follow-up, including visits over 6 months, were included. Exclusion criteria included acute ST-segment elevation myocardial infarction (but not chronic ischemic cardiomyopathy), severe known renal failure (patients on dialysis or creatinine >4 mg/dL), nephrotic syndrome, and chronic liver disease or other cause of hypoalbuminemia. Written informed consent was obtained from each subject.

Data Collection

A comprehensive range of clinical data were collected on a standardized case report form. This included demographic data and detailed medical history. A detailed echocardiographic assessment of ventricular contractility, valvular structure and function, and regional wall abnormalities was performed. All echocardiographic procedures were undertaken by trained physicians and measurements made according to the American Society of Echocardiography guidelines.[7]

Laboratory evaluations provided by the local institutions and intravenous and oral medications were recorded at admission and days 1, 2, and 7 (or at discharge if earlier). Vital signs (blood pressure, heart rate, respiratory rate, and temperature) and signs and symptoms of HF (including oxygen saturation, intensity of edema and rales, body weight, and levels of orthopnea) were assessed at the same time points. Changes in dyspnea and well-being relative to admission were assessed on days 1, 2, and 7 (or at discharge if earlier). Investigators provided the final diagnosis of HF and its cause. Cardiomyopathy was classified according to the position statement from the European Society of Cardiology.[8] Hypertensive HF, right-sided HF, and ischemic HF were defined by standard criteria.[9]

Study Outcome

Subjects who were discharged after admission were evaluated at 1 and 6 months. At these time points, patients were evaluated for signs and symptoms of HF, laboratory evaluations were performed, and oral medications were recorded. Readmissions and death, with reasons and cause, respectively, through 6 months of follow-up were collected.

ECG Data Acquisition and Interpretation

A 12-lead ECG was performed within 15 days of admission. All ECGs were read centrally by Momentum Research by one cardiologist (O.M.) and reviewed another (G.C.). ECGs were analyzed for conduction or rhythm disturbances, evidence of myocardial ischemia/infarction, or hypertrophy. The following parameters were entered in the database registry together with other clinical data: heart rate; length of QRS, QT, and QTc intervals; type of rhythm (sinus rhythm, atrial fibrillation/flutter, other supraventricular tachycardia, ventricular tachycardia, junctional rhythm, ventricular pacing, 1st-, 2nd-, or 3rd-degree atrioventricular block); Q wave compatible with myocardial infarction as well as all other forms of Q waves; and ST-T segment changes. ST-T segment changes were noted as ST-segment elevation at the J point in 2 contiguous leads of 0.2 mV in men or 0.15 mV in women in leads V2−V3 or 0.1 mV in other leads, as ST-segment depression of horizontal or down-sloping depression of 0.05 mV in 2 contiguous leads, as nonspecific ST-T abnormality where changes did not meet the above criteria for ST-segment elevation or depression, or as changes due to bundle branch block. Left ventricular hypertrophy (LVH) was determined with the use of the Sokolow-Lyon index (SV1 + RV5/6 > 35 mm), and all of other described ECG parameters were defined and analyzed according to the standard international criteria and definitions.[10,11] All ECG abnormalities were classified into major and minor with the use of the Minnesota code classification system which allows a systematic approach and facilitate an easy appreciation of the prognostic value of ECG findings for total cardiovascular disease.[12]

Statistical Analyses

All data were processed at the central coordinating center at Momentum Research, Durham, North Carolina, USA. Data were analyzed with the use of SAS version 9.2 (SAS Institute, Cary, North Carolina). Summary statistics (mean, SD, median, and 25th−75th percentiles) are provided for continuous variables and frequencies for categoric variables. Some parameters could not be estimated or interpreted by the central reader from the ECG provided. These were treated as missing in the analyses.

The associations of ECG findings with the composite end point of all-cause death or readmission to 60 days and all-cause death to day 180 were evaluated in those subjects with ECGs administered within 15 days of the admission. Univariable and multivariable Cox regression models were constructed considering the time from admission to the first event; times for patients without the

event of interest were censored at the earlier of the last date the patient was known to be alive or the period of interest. Unadjusted Kaplan-Meier survival estimates are presented in the figures.

Multivariable models were constructed from ECG parameters with values available for sufficient subjects. None of the associations between continuous ECG predictors and each outcome was found to be significantly nonlinear, as assessed by examining the significance of the nonlinear terms of restricted cubic splines (RCSs) with 4 "knots." Multivariable models were adjusted for clinical covariates included in multivariable prognostic models in the overall THESUS-HF registry.[6] For the outcome all-cause death or readmission to 60 days, these included history of hyperlipidemia, malignancy, or cor pulmonale, systolic BP, left ventricular ejection fraction, oxygen saturation, rales, blood urea nitrogen, and region. For the outcome all-cause death to day 180, clinical covariates included sex, history of malignancy, cor pulmonale, smoking, or human immunodeficiency virus, systolic blood pressure (BP), heart rate, oxygen saturation, orthopnea, rales, edema, creatinine, and hemoglobin. The ECG parameters QRS duration, QT, QTc, ventricular rate, sinus rhythm, atrial fibrillation/flutter, bundle branch block, left ventricular hypertrophy, T-wave inversion in ≥2 contiguous leads, ST-segment depression ≥1 mm, ST-segment elevation ≥1 mm, nonspecific ST-T changes, isolated pathologic Q waves and Q waves compatible with myocardial infarction (MI) were then added to the multivariable models to see if any had a significant association after adjusting for the clinical covariates.

Approximately 40% of subjects were missing ≥1 of the predictors. Multiple imputations with the use of 7 imputed datasets and assumption of multivariate normality were used to handle missing values. The Rubin algorithm was used for averaging parameter estimates across the imputed datasets. Backward selection of the ECG parameters was performed for each imputed dataset, with predictors with $P < .1$ kept in the model. ECG parameters that remained in a majority (≥4) of the models were then included in the final model. c-Statistics are reported for the models with and without the ECG parameters to examine the change in model discrimination with the addition of ECG information.

Results

Of the 1,006 patients enrolled in the THESUS-HF study, 813 patients had an available 12-lead ECG, 523 of which were obtained within 15 days of admission and were included in the analyses.[6] Characteristics of these patients were similar to the overall study population (Supplementary Table 1). The majority of included patients were enrolled in Nigeria (32.1%), Uganda (21.0%), and South Africa (18.9%). Table 1 depicts the baseline characteristics of the 523 subjects (49% male, overall mean age 52 ± 18.4 years) included in the analysis. The etiologies of HF were dominated by hypertension (43.2%), idiopathic dilated cardiomyopathy (21.0%), rheumatic heart disease (17.2%), and ischemic heart disease (7.7%).

Table 1. Baseline Characteristics for Subjects With an Electrocardiogram Within 15 Days of Admission (n = 523)

Age, y	52.0 ± 18.4
Gender, male	256 (49.0%)
Systolic blood pressure, mm Hg	132.9 ± 34.3
Heart rate, beats/min	105.1 ± 21.9
O_2 saturation, %	93.6 ± 7.4
Ejection fraction, %	39.9 ± 16.5
Rales (2/3 vs 0/1)	255 (58.4%)
Orthopnea (2/3 vs 0/1)	388 (89.2%)
Edema (2/3 vs 0/1)	328 (63.7%)
Medical history	
Diabetes mellitus	55 (10.5%)
Hypertension	291 (55.8%)
Hyperlipidemia	44 (8.6%)
Malignancy	8 (1.5%)
Cor pulmonale	38 (7.3%)
Smoking	45 (8.7%)
HIV positive	45 (8.8%)
Etiology of heart failure	
Hypertensive CMP	226 (43.2%)
Idiopathic dilated CMP	110 (21.0%)
Rheumatic heart disease	90 (17.2%)
Ischemic heart disease	40 (7.7%)
Peripartum cardiomyopathy	33 (6.3%)
Pericardial effusion/tamponade	29 (5.5%)
HIV CMP	18 (3.4%)
Endomyocardial fibroelastosis	10 (1.9%)
Laboratory values	
Creatinine, mg/dL	1.43 ± 1.09
Hemoglobin, g/dL	12.4 ± 2.4
eGFR, mL min^{-1} 1.73 m^{-2}	81.7 ± 49.1
BUN, mg/dL	33.3 ± 33.5
Electrocardiographic parameters	
QRS duration, ms	73.9 ± 26.7
QT duration, ms	348.3 ± 52.3
QTc, ms	274.4 ± 71.3
Ventricular rate, beats/min	104.1 ± 26.0
Sinus rhythm	392 (75.1%)
Atrial fibrillation/flutter	121 (23.6%)
Left bundle branch block	42 (8.1%)
Right bundle branch block	26 (5.0%)
Left ventricular hypertrophy	158 (30.6%)
Other electrocardiographic abnormalities	
T-wave inversion in ≥2 contiguous leads	163 (31.2%)
ST-segment depression ≥1 mm	13 (2.5%)
ST-segment elevation ≥1 mm	17 (3.3%)
Nonspecific ST-T changes	124 (23.7%)
Isolated pathologic Q waves	9 (1.7%)
Q-waves compatible with MI	90 (17.2%)

Values are presented as mean ± SD for continuous variables and n (%) presented for categoric data (percentages of nonmissing values). CMP, cardiomyopathy; HIV, human immunodeficiency virus; eGFR, estimated glomerular filtration rate; BUN, blood urea nitrogen; MI, myocardial infarction.

Electrocardiography findings

The mean ventricular rate was 104 beats/min, with a ranged of 40−200 (Supplemental Table 1). Abnormal ECG was detected in 511 out of 523 patients (97.7%). Figure 1 shows the ECG abnormalities in these patients, with major alterations similar in men and women. The distribution of subtypes of minor abnormalities in men and women is depicted in Figure 2.

ECG Abnormalities and Morbidity and Mortality Risks

Patients included in the analyses were followed for a median of 180 days. There were 77 deaths to day 180, with a Kaplan-Meier event rate of 17.5%. There were 63 patients with a death or readmission to day 60, for a rate of 13.5%. Eighty subjects (15.3%) died without completing a 6-month assessment, 261 (49.9%) had a 6-month assessment, and 156 (29.8%) had a last known

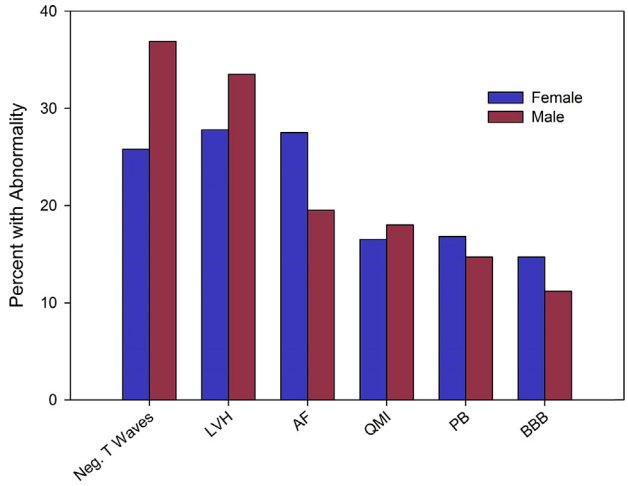

Fig. 1. Major electrocardiographic abnormalities in male and female patients. LVH, left ventricular hypertrophy; AF, atrial fibrillation/flutter; QMI, Q wave compatible with myocardial infarction; PB, premature beats; BBB, bundle branch block.

date alive. The remaining 26 patients (5.0%) were considered to be lost to follow-up.

Without adjustment, QRS width and QT duration were not associated with either the composite outcome of death or readmission through 60 days or death through 180 days (Tables 2 and 3). Bundle branch block and ischemic changes were not independently significantly associated with either outcome.

Corrected QT intervals were calculated for all patients, and after dividing patients into male and female cohorts only 7 out of 254 male patients (2.8%) had a borderline to abnormal QTc (>430 ms) and only 6 out of 267 female patients (2.2%) had a borderline to abnormal QTc (>450 ms).

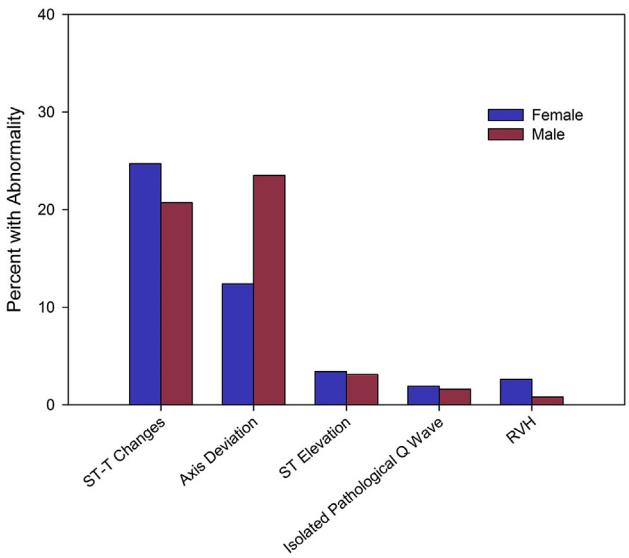

Fig. 2. Minor electrocardiographic abnormalities in male and female patients. RVH, right ventricular hypertrophy.

Increasing ventricular rate was associated with increasing risk of both outcomes, whereas the presence of sinus rhythm was associated with lower risk (Fig. 3). Heart rate analysis when patients were divided into quartiles based on the heart rate showed an association with survival probability. Further analysis based on the heart rate and sinus rhythm/atrial fibrillation showed strong association between survival and heart rate specifically in patients in sinus rhythm. Patients in sinus rhythm with heart rate of >119 beats/min fared the worst (Fig. 3).

After multivariable adjustment, lack of sinus rhythm and Q waves associated with MI were the only ECG variables associated ($P < .10$) with the risk of death or readmission within 60 days (Table 2) or death within 180 days (Table 3). The addition of these ECG parameters did not improve the multivariable models' discrimination (the ability to correctly classify patients with versus without an event) over a model including clinical and laboratory characteristics for either 60-day readmission or death (c-statistic 0.74, 95% CI 0.67−0.80, versus 0.70, 95% CI 0.65−0.75) or 180-day mortality (c-statistic 0.77, 95% CI 0.72−0.82, versus 0.72, 95 CI 0.68−0.75).[13]

Discussion

In this large cohort study of acute HF in 9 sub-Saharan African countries, we found a high prevalence (97.7%) of ECG abnormalities. Also, our analysis indicated that some of these abnormalities are associated with a higher risk for adverse outcome in these patients. The predominant abnormalities include increased heart rate, lack of sinus rhythm, and Q waves, all of which were associated with a higher risk of adverse outcomes. This study confirms the previously reported high prevalence rates of abnormal ECG[3] and the fact that some of these findings are associated with more adverse outcome.[14] However, when adjusted for additional baseline characteristics, ECG findings did not improve the ability of the model to predict adverse outcome and therefore added little to the risk stratification of patients with AHF.

The high prevalence of ECG abnormalities is probably because a significant number of these patients suffered from structural cardiac abnormalities such as reduced EF and valvular diseases as well as comorbidities such as hypertension (56%) and diabetes mellitus (DM) (11%). This finding would tend to suggest, as reported elsewhere, that a normal ECG is rare in patients with suspected HF and is clinically useful in confirming the presence of heart disease without a need to resort to other tests, such as B-type natriuretic peptide. The almost universal presence of ECG abnormality implies that a patient with signs and symptoms of acute HF and a normal ECG has a noncardiac cause of the presentation until proven otherwise. Interestingly, and consistent with the main results of the THESUS-HF study, only 90 patients (17.2%) were found to have Q waves compatible with MI, which, again, is in line with earlier studies showing that ischemic heart disease is an uncommon cause of acute HF in sub-Saharan Africa.[6,15−18]

Table 2. Univariable and Multivariable Models of All-Cause Death or Readmission Through Day 60

Baseline Characteristic	HR for Change of …	Univariable Models		Full Model		Multivariable Model	
		HR (95% CI)	*P* Value	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value
History of hyperlipidemia	Yes/No	0.32 (0.07−1.44)	.1381	0.38 (0.08−1.91)	.2407	0.32 (0.07−1.59)	.1642
History of malignancy	Yes/No	5.79 (2.11−15.87)	.0007	7.72 (2.54−23.48)	.0003	6.76 (2.30−19.87)	.0005
History of cor pulmonale	Yes/No	2.60 (1.31−5.14)	.0062	1.76 (0.82−3.79)	.1453	1.64 (0.78−3.46)	.1937
Systolic blood pressure	10	0.93 (0.86−1.00)	.0634	0.95 (0.87−1.04)	.2621	0.96 (0.88−1.04)	.2831
Ejection fraction	50 vs 28	0.78 (0.55−1.12)	.4099	0.79 (0.52−1.20)	.5261	0.81 (0.55−1.21)	.6129
O_2 saturation	5	0.89 (0.77, 1.01)	.0798	0.86 (0.73−1.00)	.0528	0.88 (0.75−1.02)	.0929
Rales	(2/3 vs 0/1)	1.81 (1.00−3.28)	.0499	1.73 (0.85−3.52)	.1329	1.78 (0.88−3.59)	.1095
Laboratory tests							
BUN (log 2)	Doubling	1.37 (1.10−1.72)	.0059	1.46 (1.12−1.91)	.0053	1.42 (1.10−1.85)	.0077
Region*	(South vs West)	1.66 (0.98−2.82)	.0602	2.94 (1.56−5.55)	.0009	2.77 (1.49−5.17)	.0014
Region	(East vs West)	0.50 (0.22−1.14)	.0993	0.89 (0.35−2.30)	.8129	0.83 (0.33−2.05)	.6825
Electrocardiographic parameters							
QRS duration	10	0.99 (0.90−1.09)	.8998	0.97 (0.85−1.10)	.6453		
QT	20	0.94 (0.85−1.03)	.2076	1.07 (0.83−1.37)	.6189		
QTc	10	0.97 (0.93−1.01)	.1002	0.96 (0.88−1.05)	.3558		
Ventricular rate, beats/min	5	1.07 (1.02−1.12)	.0047				
Sinus rhythm	Yes/No	0.58 (0.34−0.97)	.0385	0.55 (0.30−1.01)	.0557	0.52 (0.30−0.90)	.0200
Atrial fibrillation/flutter	Yes/No	1.59 (0.92−2.75)	.0941				
Left bundle branch block	Yes/No	1.55 (0.71−3.41)	.2726	2.27 (0.77−6.65)	.1356	1.89 (0.82−4.32)	.1335
Right bundle branch block	Yes/No	1.31 (0.48−3.60)	.5974	1.82 (0.53−6.22)	.3376		
Left ventricular hypertrophy	Yes/No	0.54 (0.29−1.02)	.0578	0.82 (0.40−1.68)	.5820		
Other electrocardiographic abnormalities							
T-wave inversion in ≥2 contiguous leads	Yes/No	0.71 (0.40−1.26)	.2399	1.18 (0.58−2.38)	.6453		
ST-segment depression ≥1 mm	Yes/No	1.29 (0.32−5.29)	.7206	2.72 (0.58−12.72)	.2027		
ST-segment elevation ≥1 mm	Yes/No	0.91 (0.22−3.72)	.8937	0.93 (0.21−4.16)	.9240		
Nonspecific ST-T changes	Yes/No	0.95 (0.53−1.73)	.8744	0.74 (0.36−1.53)	.4210		
Isolated pathologic Q waves	Yes/No	2.36 (0.58−9.63)	.2341	2.03 (0.44−9.26)	.3621		
Q waves compatible with MI	Yes/No	1.41 (0.78−2.56)	.2561	1.74 (0.85−3.56)	.1297	1.86 (0.97−3.54)	.0608
c-Statistic				0.7448 (0.6782−0.8113)		0.7387 (0.6749−0.8024)	

MI, myocardial infarction.
*Regions: East: Ethiopia, Kenya, Sudan, and Uganda; South: Mozambique and South Africa; West: Cameroon, Nigeria, and Senegal.

Most other ECG findings were nonspecific. These observations are in agreement with the recent study that was conducted by Khan et al[3]—drawing from the Euro Heart Failure survey of 11,327 patients admitted for acute HF—suggesting that most ECG findings in these patients are nonspecific and that ECG criteria alone were not accurate for the diagnosis or exclusion of specific cardiac abnormalities in patients with acute HF.

Similarly, the results of the present study suggest that although some ECG variables (especially rate and rhythm and the presence Q waves) are associated with worse outcomes, these factors do not increase the predictive value of the models for readmission or mortality. Untreated patients with HF usually exhibit a high heart rate that results from hyperadrenergia; however, we can not rule out that the association of higher heart rate with mortality may indeed be due to more severe neurohormonal activation in these patients.[19] The rate of left bundle branch block (LBBB) in our study was low (8%), compared with 16%−22% in other populations admitted for HF in European countries.[20,21] Moreover, LBBB was not associated with increased mortality, in contrast to associations reported in the European population; differences between populations with different age and etiology of HF (ie, less ischemic cardiomyopathy, more hypertensive and rheumatic heart disease) might account for these findings. Similar results were reported by Hebert et al, in Hispanics with systolic HF.[22] Even though they found higher prevalence of paced rhythm, LBBB, and abnormal QT intervals, in univariate and multivariate analysis corrected for other characteristics (age, sex, coronary artery disease, hypertension, ejection fraction, medications) ECG findings added little prognostic information. Therefore, it seems that although ECG is an important assessment that should be performed routinely in patients with acute HF, its value lies mostly in excluding some specific causes of acute HF such as MI or life-threatening arrhythmias. Other electrocardiographic findings do not substantially help in the risk stratification of patients with acute HF.

Although found previously to be independently associated with adverse outcomes in 2 recent studies—in 872 patients with preserved systolic function[23] and in 4,133 patients enrolled in EVEREST trial[24] which included acute HF patients with ejection fraction ≤40%—QRS duration was nonsignificantly associated with adverse outcome in the present study. Regarding QTc abnormalities, Kolo

Table 3. Univariable and Multivariable Models of All-Cause Death Through Day 180

Baseline Characteristic	HR for Change of …	Univariable Models		Full Model		Multivariable Model	
		HR (95% CI)	*P* Value	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value
Male sex	Yes/No	1.05 (0.67−1.64)	.8454	1.14 (0.68−1.91)	.6209	1.04 (0.63−1.72)	.8657
History of malignancy	Yes/No	6.05 (2.21−16.59)	.0005	5.16 (1.69−15.79)	.0041	5.43 (1.83−16.08)	.0023
History of cor pulmonale	Yes/No	2.71 (1.45−5.05)	.0018	2.11 (1.03−4.32)	.0414	2.09 (1.04−4.23)	.0395
History of smoking	Yes/No	1.12 (0.51−2.44)	.7842	1.35 (0.56−3.25)	.5086	1.31 (0.56−3.08)	.5330
HIV positive	Yes/No	1.30 (0.62−2.72)	.4820	0.92 (0.39−2.19)	.8579	0.99 (0.43−2.27)	.9741
Systolic blood pressure	10	0.85 (0.79−0.92)	.0001	0.87 (0.80−0.95)	.0018	0.88 (0.81−0.95)	.0019
Heart rate	118 vs 90	1.84 (1.22−2.78)	.0016	1.40 (0.88−2.24)	.0582	1.43 (0.93−2.19)	.0483
O_2 saturation	5	0.92 (0.80−1.07)	.2927	0.86 (0.72−1.04)	.1205	0.86 (0.72−1.04)	.1238
Orthopnea	(2/3 vs 0/1)	2.36 (0.83−6.71)	.1063	2.07 (0.64−6.73)	.2237	2.22 (0.72−6.89)	.1657
Rales	(2/3 vs 0/1)	2.03 (1.15−3.59)	.0148	1.27 (0.65−2.50)	.4848	1.37 (0.71−2.65)	.3462
Edema	(2/3 vs 0/1)	2.34 (1.34−4.08)	.0028	2.25 (1.19−4.25)	.0124	2.20 (1.17−4.14)	.0144
Laboratory tests							
Creatinine	1.60 vs 0.90	1.62 (1.11−2.35)	.0412	1.39 (0.96−2.02)	.1698	1.33 (0.93−1.91)	.2640
Hemoglobin	1	0.92 (0.84−1.01)	.0676	0.94 (0.84−1.05)	.2693	0.94 (0.84−1.04)	.2198
Electrocardiographic parameters							
QRS duration	10	0.94 (0.86−1.03)	.1785	0.92 (0.80−1.05)	.2224		
QT	20	0.95 (0.88−1.04)	.2844	1.15 (0.91−1.46)	.2486		
QTc	10	0.98 (0.95−1.01)	.2250	0.97 (0.89−1.06)	.4883		
Ventricular rate, beats/min	5	1.04 (1.00−1.09)	.0572				
Sinus Rhythm	Yes/No	0.56 (0.35−0.90)	.0159	0.64 (0.37−1.13)	.1226	0.61 (0.36−1.02)	.0618
Atrial fibrillation/flutter	Yes/No	1.53 (0.94−2.51)	.0892				
Left bundle branch block	Yes/No	1.00 (0.43−2.32)	.9930	2.18 (0.64−7.39)	.2111		
Right bundle branch block	Yes/No	0.77 (0.24−2.45)	.6565	0.71 (0.18−2.85)	.6324		
Left ventricular hypertrophy	Yes/No	0.52 (0.29−0.93)	.0286	0.71 (0.37−1.35)	.2953		
Other electrocardiographic abnormalities							
T-wave inversion in ≥2 contiguous leads	Yes/No	0.55 (0.31−0.97)	.0393	0.85 (0.44−1.64)	.6259		
ST-segment depression≥1 mm	Yes/No	0.48 (0.07−3.45)	.4649	1.35 (0.17−10.85)	.7780		
ST-segment elevation ≥1 mm	Yes/No	1.90 (0.77−4.71)	.1646	2.05 (0.74−5.68)	.1659		
Nonspecific ST-T changes	Yes/No	1.45 (0.89−2.37)	.1340	1.62 (0.87−3.03)	.1276	1.57 (0.91−2.72)	.1057
Isolated pathologic Q waves	Yes/No	1.90 (0.47−7.73)	.3719	2.97 (0.65−13.46)	.1587		
Q waves compatible with MI	Yes/No	2.19 (1.34−3.57)	.0017	2.34 (1.23−4.46)	.0098	2.45 (1.39−4.32)	.0020
c-Statistic				0.7729 (0.7198−0.8260)		0.7683 (0.7163−0.8204)	

MI, myocardial infarction.

et al,[25] in a study including 90 Nigerian patients with chronic HF, found QTc to be prolonged in 63% of patients. However, in the present study of patients with acute HF, we found that few patients had QTc prolongation: 7 out of 254 male patients (2.8%) had a borderline to abnormal QTc (>430 ms) and 6 out of 267 female patients (2.2%) had a borderline to abnormal QTc (>450 ms).

Taking into consideration that a significant proportion of the patients in our study were hypertensive, we expected to find a substantial prevalence of ECG signs of LVH. However, only 158 patients (30.6%) met the criteria for LVH. Moreover, in disagreement with earlier subanalysis of the CHARM program,[26] where ECG LVH was reported as an independent predictor of worse outcome, in the present study patients with LVH tended to be at decreased risk for both all-cause mortality and readmission through day 60 as well as all-cause mortality through day 180. This finding is unexpected and requires confirmation in further studies; however, it is possible that hypertensive HF has a relatively benign prognosis in Africa.

There are several weaknesses of the present study. First, ECGs were available in only 523 of the 1,006 patients in the THESUS-HF cohort. However, patients with available ECGs had characteristics similar to the rest of the cohort. The second issue is the lack of standardization of the manner in which the ECGs were acquired. Inconsistent placement of ECG electrodes is known to have an effect on ECG voltages which are important in the assessment of cardiac hypertrophy.[27] Third, missing data for other variables may have diluted the associations that were sought in this study. Fourth, owing to no access to cardiac catheterization in a number of centers, we might have missed some cases of HF due to ischemic origin. And finally, we can not rule out the possibility that by recording ECGs at ±15 days and not absolutely on admission, our study missed some transient ECG abnormalities. These limitations, however, have to be considered in the context of the study setting, where major constraints to high-quality clinical research are still commonplace. The study was conducted in selected specialized centers, and only patients who consented to the study were enrolled; thus, not all patients admitted with AHF are represented and the study's generalizability is somewhat limited. However, we have increased our understanding of the growing importance of chronic cardiovascular disease in this population, who are often thought to suffer much from infectious diseases.

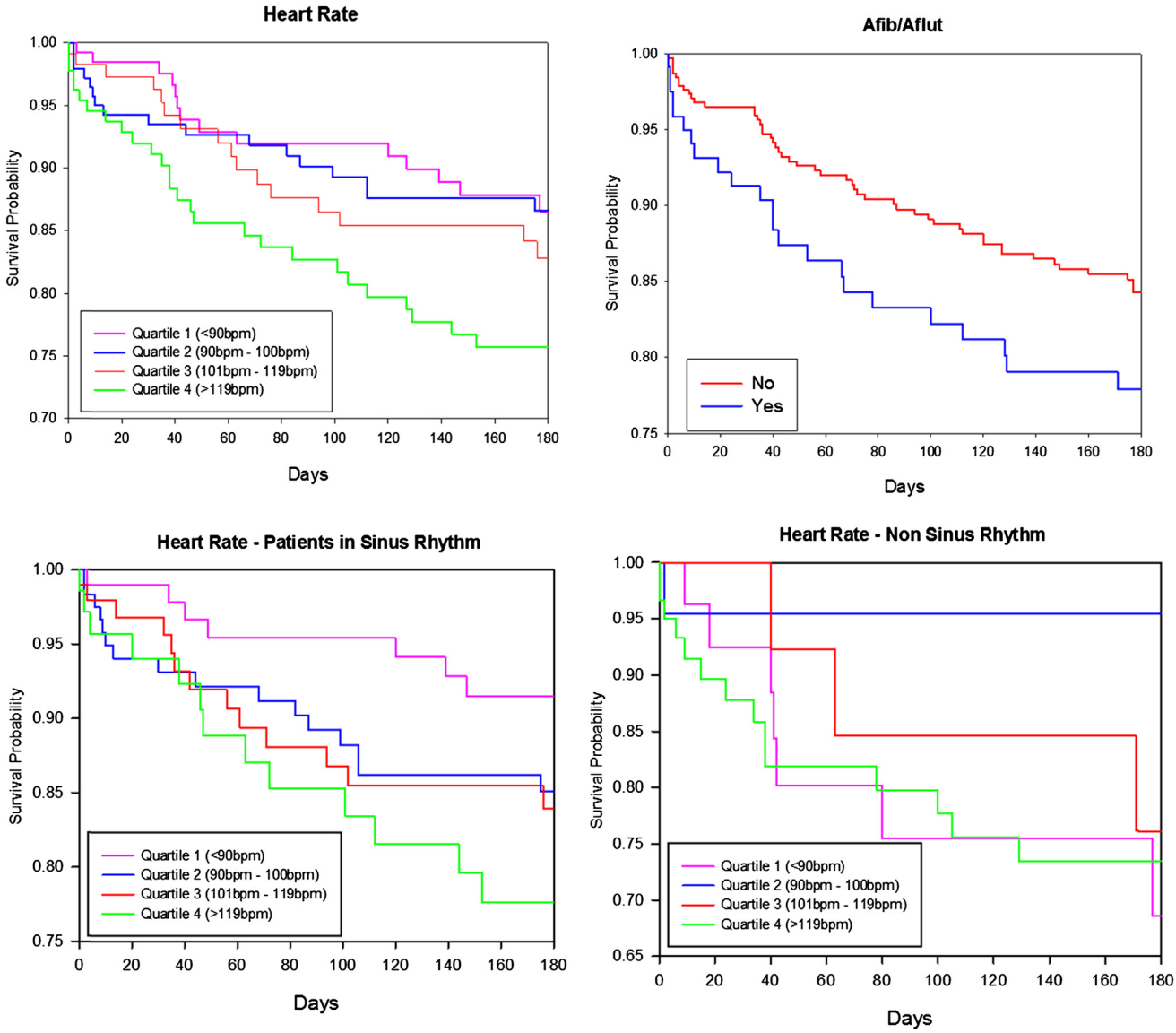

Fig. 3. Kaplan-Meier estimates of 180-day survival according to various electrocardiographic findings within 15 days of admission. Afib/Aflut, atrial fibrillation/flutter.

Conclusion

In African patients admitted for acute HF, ECG abnormalities are present in 98% of cases, and some of these abnormalities (eg, higher heart rate, nonsinus rhythm, presence of Q waves) are associated with higher risk for adverse outcomes. Although ECG should be routinely obtained in patients admitted for acute HF to confirm presence of heart disease and rule out acute ischemia and severe rhythm disorders, for the most part ECG abnormalities in African patients with acute HF are nonspecific and add little to the risk stratification of those patients.

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Disclosures

None

Supplementary Data

Supplementary data related to this article can be found online at http://dx.doi.org/10.1016/j.cardfail.2013.11.005.

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