Original Article

Clinical Outcome of Dilated Cardiomyopathy in an African Tertiary Hospital

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ABSTRACT

Background: Dilated cardiomyopathy (DCM) is a primary myocardial disease of unknown cause characterized by left ventricular or biventricular dilatation and impaired myocardial contractility. Morbidity and mortality in patients with DCM remain high. Data on DCM outcomes in Africa are lacking. This study aimed to determine the clinical characteristics and outcome of DCM at University College Hospital, Ibadan, Nigeria.

Methods: Analysis of data collected over 6 years (September 1, 2016, through August 31, 2022). Information included sociodemographics, clinical features, echocardiographic diagnosis, morbidity, and mortality.

Results: During this period, 127 cases of DCM were seen: 91 males (71.7%) and 36 females (28.3%) aged 52.0 (SD, 16.0) years (range, 17–86 years). More than 90% had formal education, and 108 (85%) were married. A history of current or past alcohol consumption was documented in 62 (48.5%). At 1-year follow-up, 27 (21.3%) of the 127 patients died. Those who died were older and had higher body mass index and white blood cell count but lower estimated glomerular filtration rate, systolic blood pressure, and sodium level. Decedents were more likely to be male ($P = 0.018$) and to consume alcohol ($P = 0.029$).

Conclusions: Our data show that male sex and alcohol use are predictors of mortality in patients with DCM seen at UCH Ibadan. ***(Iranian Heart Journal 2026; 27(1): 26-36)***

KEYWORDS: dilated cardiomyopathy; outcome; heart failure; mortality

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Dilated cardiomyopathy (DCM) is a chronic and mostly irreversible heart muscle disease characterized by dilatation of the left or both ventricles and systolic dysfunction, with normal wall thickness.[1] Historically, the term DCM was coined by Bridgen[2] in 1957, and the clinical characteristics were first described by Goodwin[3] in 1961. DCM can be genetic or acquired, inherited or non-inherited.[4] Inherited forms occur in 25% to 50% of cases.[1] It is clinically divided into primary or secondary forms.[1] In approximately 20% to 30% of cases, the cause involves mutation of genes encoding cardiac proteins such as troponin, myosin, and desmin.[4] A broad spectrum of aetiologic risk factors has been identified, including postmyocardial ischemia, cytotoxic medications, malnutrition, viral infections, drug addiction, trace-element deficiency, autoimmune diseases, endocrinopathies, and hereditary muscle diseases such as Duchenne and Becker muscular dystrophies.[4]

In Africa, DCM accounts for about 21.4% (95% CI, 16.0 to 27.2) of heart diseases.[5] In Nigeria, it ranks second or third among causes of heart disease, depending on location.[6] Previous studies in Ibadan, Nigeria, have described clinical features,[7] etiologic risk factors,[8] and echocardiographic characteristics.[9] DCM outcomes have not been studied in the country. The present study describes the 1-year outcome of DCM in Ibadan using data from the Ibadan Heart Failure Project.

METHODS

Study Location

The study was conducted at the Cardiology Unit, Department of Medicine, University College Hospital (UCH), Ibadan, Oyo State, Nigeria. UCH is the largest and oldest tertiary health institution in Nigeria and the West African subregion, with more than 1000 bed spaces and more than 60% bed occupancy. It receives referrals from all clinics and hospitals in the city, the state, and throughout Nigeria.

Study Population

Adult patients aged 18 years and older were recruited into the study, part of the wider Ibadan Acute and Chronic Heart Failure Project that commenced in September 2016 (ClinicalTrials.gov identifier: NCT05936957). Informed consent was obtained from all participants.

Sample Size Calculation

A sample proportion of 5.5% for 1-year all-cause mortality in patients with DCM was used to estimate a minimum sample size of 80 participants based on a study by one author.[10] A 95% confidence interval (CI) with a margin of error of 5% was set.

Diagnosis of DCM

The World Health Organization (WHO) criteria[11] and the European Society of Cardiology position statement on classification of cardiomyopathies[12] were drawn upon for diagnosis. A standardized, detailed case-report form captured history, physical examination findings, blood parameters, 12-lead ECG, echocardiogram, medications, and follow-up data for all participants.

Patients had left ventricular ejection fraction of 40% or less.[11] The fraction was determined by the biplane Simpson method. Heart failure attributable to hypertensive heart disease, valvular heart disease, coronary artery disease, peripartum cardiomyopathy, myocarditis, HIV infection, or thyroid disease was excluded. Coronary angiography was not performed because of unavailability. Patients with typical symptoms of coronary artery disease, ECG features of ischemic heart disease, and regional wall motion abnormalities on echocardiography were excluded. Cardiac magnetic resonance imaging (MRI) was not included because of nonavailability.

Outcomes

The primary outcome of interest was all-cause mortality. The secondary outcomes of interest were rehospitalization and mortality. Follow-up was through hospital visits, telephone calls, or review of patients' medical records.

Statistical Analysis

Data were tested for normality. Quantitative variables with normal distribution were expressed as mean (SD). Median (interquartile range [IQR]) summarized continuous variables that were not normally distributed. Categorical variables were summarized as proportions. The Pearson chi-square test or Fisher exact test, where appropriate, was employed to compare categorical variables. Continuous variables with normal distribution were compared using the Student *t* test; the Wilcoxon rank sum (Mann-Whitney) test compared medians for data that were not normally distributed. Univariable and multivariable logistic regression analyses were utilized to determine the association between the primary outcome and other variables after adjusting for confounders. Odds ratios (ORs) were calculated at 95% CI levels. The criterion for inclusion in the multiple logistic regression analysis was a *P* value less than 0.15 in the univariable analysis. Kaplan-Meier analysis was used to estimate survival over time. A 2-tailed *P* value less than 0.05 was considered statistically significant. Data management and statistical analysis were performed with SPSS statistical software (SPSS Inc, IBM, New York, USA). Survival analysis was performed with Stata statistical software.

Patient and Public Involvement

Patients or the public were not involved in the design, conduct, reporting, or dissemination plans of this research.

RESULTS

A total of 127 cases of DCM were encountered between September 1, 2016, and August 31, 2022. Figure 1 illustrates the selection process. The cohort included 91 males (71.7%) and 36 females (28.3%). Mean (SD) age was 52.0 (16.0) years (range, 17–86 years). Mean (SD) age was 53.0 (16.0) years for males (range, 17–86 years) and 47.0 (15.0) years for females (range, 23–80 years). Table 1 demonstrates sociodemographic characteristics. Thirty-four (26.5%) were younger than 40 years, six had no formal education, and more than 60% earned less than 50,000 Nigerian naira (US $33.28 as of March 1, 2025) per month. Twenty-nine participants (22.8%) were previous or current cigarette smokers, and 62 (48.5%) were previous or current alcohol users. Only 4 (3.1%) had a family history of heart failure (Table 2). Table 3 shows the pattern of symptoms and signs in the cohort. Dyspnea on exertion, paroxysmal nocturnal dyspnea, and bilateral ankle edema were the most common clinical features. There were no statistical differences between males and females. Sixty percent of the cohort had no previous history of acute heart failure admissions. About 41 (32%), 9 (7.15%), and 1 (0.8%) had 1, 2, and 3 hospital admissions, respectively. Seventy-four (58.4%) were in New York Heart Association (NYHA) class I/II, and 53 (41.6%) were in NYHA class III/IV.

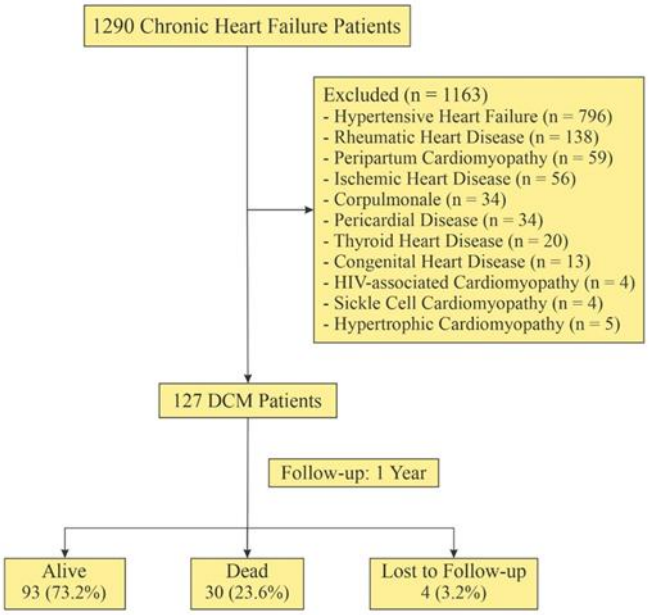

Figure 1. Flow chart used in the selection of the 127 studied patients with dilated cardiomyopathy (DCM)

Table 1. Baseline sociodemographic characteristics of the 127 studied patients with dilated cardiomyopathy

Characteristic	Overall (No. =127)	Male (No = 91)	Female (No = 36)	*P*
Age, mean (SD), (y)	52.0 (16.0)	53.0 (16.0)	47.0 (15.0)	0.049
Age Group, No. (%)				0.502
<30 y	7 (5.5%)	4 (4.4%)	3 (8.3%)	
30–39 y	27 (21%)	16 (18%)	11 (31%)	
40–49 y	26 (20%)	19 (21%)	7 (19%)	
50–59 y	26 (20%)	19 (21%)	7 (19%)	
60–69 y	22 (17%)	18 (20%)	4 (11%)	
≥70 y	19 (15%)	15 (16%)	4 (11%)	
Marital Status, No. (%)				0.228
Single	10 (7.9%)	9 (9.9%)	1 (2.8%)	
Married	108 (85%)	76 (84%)	32 (89%)	
Divorced or separated	3 (2.4%)	1 (1.1%)	2 (5.6%)	
Widowed	6 (4.7%)	5 (5.5%)	1 (2.8%)	
Educational Attainment, No. (%)				0.464
No formal education	6 (4.7%)	3 (3.3%)	3 (8.3%)	
Primary education	23 (18.1%)	15 (16.5%)	8 (22.2%)	
Secondary education	46 (36.2%)	34 (37.4%)	12 (33.3%)	
Postsecondary or university	48 (37.8%)	37 (41%)	11 (30.6%)	
Postgraduate education	4(3.1%)	2(2.2%)	2 (5.6%)	
Employment History, No. (%)				0.311
Unemployed	39 (31%)	30 (33%)	9 (25%)	
Employed	71 (56%)	47 (52%)	24 (67%)	
Pensioner	17 (13%)	14 (15%)	3 (8.3%)	
Place Lived Most of Life				0.999
Rural	3 (2.4%)	2 (2.2%)	1 (2.8%)	
Urban	121 (95%)	87 (96%)	34 (94%)	
Mixed	3 (2.4%)	2 (2.2%)	1 (2.8%)	
Estimated Monthly Income (Naira)				0.020
<50000	79 (62%)	49 (54%)	30 (83%)	
≥50 000	48 (38%)	42 (46%)	6 (17%)	

Table 2. Cardiovascular risk factors and comorbidities in the 127 studied patients with dilated cardiomyopathy

Risk Factor/ Comorbidities (No./%)	Overall (No. =127)	Male (No. = 91)	Female (No. = 36)	*P*
Cigarette Smoking				<0.001
No	98 (77%)	62 (68%)	36 (100%)	
Previous	28 (22%)	28 (31%)	0 (0%)	
Current	1 (0.8%)	1 (1.1%)	0 (0%)	
Alcohol Consumption				<0.001
No	65 (51%)	32 (35%)	33 (92%)	
Previous	55 (43%)	52 (57%)	3 (8.3%)	
Current	7 (5.5%)	7 (7.7%)	0 (0%)	
Diabetes mellitus	12 (9.4%)	10 (11%)	2 (5.6%)	0.507
Other comorbidities	58 (45.7%)	41 (45.1%)	17 (%)	0.846
Family history of heart failure	4 (3.1%)	4 (4.4%)	0 (0%)	0.577

Table 3. Previous heart failure admissions, symptoms, signs, and New York Heart Association functional class in the 127 studied patients with dilated cardiomyopathy

Variable (No. /%)	Overall (No. =127)	Male (No. = 91)	Female (No. = 36)	*P*
Number of Previous Acute Heart Failure Admissions				0.736
0	76 (60%)	53 (58%)	23 (64%)	
1	41 (32%)	29 (32%)	12 (33%)	
2	9 (7.1%)	8 (8.8%)	1 (2.8%)	
3	1 (0.8%)	1 (1.1%)	0 (0%)	
Paroxysmal nocturnal dyspnea	77 (61%)	58 (64%)	19 (53%)	0.255
Neck vein distension	60 (47%)	41 (45%)	19 (53%)	0.432
Elevated Jugular venous pressure	45 (35%)	30 (33%)	15 (42%)	0.356
Basal crepitation	15 (12%)	10 (11%)	5 (14%)	0.761
Cardiomegaly on chest X-ray	72 (57%)	49 (54%)	23 (64%)	0.303
Acute pulmonary edema	5 (3.9%)	4 (4.4%)	1 (2.8%)	>0.999
S3 gallop rhythm	46 (36%)	37 (41%)	9 (25%)	0.098
Hepatojugular reflux	14 (11%)	9 (9.9%)	5 (14%)	0.537
Bilateral ankle oedema	77 (61%)	58 (64%)	19 (53%)	0.255
Nocturnal cough	68 (54%)	50 (55%)	18 (50%)	0.615
Dyspnea on ordinary exertion	88 (69%)	62 (68%)	26 (72%)	0.652
Hepatomegaly	23 (18%)	16 (18%)	7 (19%)	0.806
Pleural effusion	16 (13%)	10 (11%)	6 (17%)	0.386
New York Heart Association Classification				0.161
I	3 (2.4%)	2 (2.2%)	1 (2.8%)	
II	71 (56%)	53 (58%)	18 (50%)	
III	51 (40%)	36 (40%)	15 (42%)	
IV	2 (1.6%)	0 (0%)	2 (5.6%)	

Table 4 depicts the biophysical and laboratory profiles of the subjects. Mean body mass index was 25.0 (4.5) kg/m^2, mean pulse rate was 87.0 (17.0) beats per minute, systolic blood pressure was 107.0 (18.0) mm Hg, and mean estimated glomerular filtration rate was 53.0 (21.0) mL/min/1.73 m^2.

Figure 2 demonstrates the 12-lead ECG findings in the cohort, while Supplementary Table 1 presents ECG and echocardiographic findings. Left atrial enlargement was documented in 32 (34%) overall, 27 (40%) in males, and 5 (19%) in females. Atrial fibrillation and widened QRS complex were seen in 13 (13%) and 42 (43%) of subjects, respectively. There was no sex difference in mean corrected QT interval or QRS duration. Mean left atrial dimension was 4.66 (0.85) cm, and mean left ventricular internal dimensions in diastole and systole were 6.47 (0.95) cm and 5.49 (0.97) cm, respectively. Mean left ventricular fractional shortening

and ejection fraction were 15.3% (6.5%) and 38.0% (13.0%), respectively. Mean tricuspid annular plane systolic excursion was 1.7 (0.43) cm.

Table 4. Biophysical profile and laboratory findings in the 127 studied patients with dilated cardiomyopathy

Variable	Overall (No. =127)	Male (No = 91)	Female (No = 36)	*P*
Weight (kg)	68.0 (13.0)	71.0 (13.0)	62.0 (12.0)	<0.001
Height (cm)	165.0 (8.0)	167 (8)	160.0 (5.0)	<0.001
Body mass index	25.0 (4.5)	25.2 (4.1)	24.4 (5.4)	0.176
Pulse (bpm)	87.0 (17.0)	87.0 (17.0)	89.0 (18.0)	0.881
Respiratory rate (cycles/min)	24.6 (4.1)	24.7 (4.3)	24.1 (3.6)	0.483
Systolic blood pressure (mm Hg)	107.0 (18.0)	107.0(16.0)	109.0 (21.0	0.874
Diastolic blood pressure (mm Hg)	73.0 (13.0)	74.0 (13.0)	71 (13)	0.251
Packed cell volume (%)	39.1 (5.1)	39.5 (5.5)	38.3 (4.0)	0.356
White cell count	7.4 (3.4)	7.2 (3.3)	7.7 (3.7)	0.574
Platelets	206.0 (70.0)	200.0 (68.0)	220.0 (74.0	0.224
Serum sodium (mmol/l)	136.2(5.0)	136.1(5.1)	136.2 (4.7)	0.925
Serum potassium (mmol/l)	3.85 (0.56)	3.87 (0.59)	3.79 (0.48)	0.418
Serum urea (mg/dL)	49.0 (32.0)	51.0 (32.0)	44.0 (32.0)	0.073
Serum creatinine (mg/dL)	1.45 (1.90)	1.56 (2.21)	1.14 (0.35)	0.013
Estimated glomerular filtration rate	53.0 (21.0)	49.0 (18.0)	63.0 (26.0)	0.005
Blood glucose (mg/dL)	106.0 (33.0)	105.0 (31.0)	108.0 (37.0)	0.500
Total serum cholesterol (mg/dL)	153.0 (33.0)	147.0 (27.0)	171.0 (42.0)	0.009
Low-density lipoprotein cholesterol (mg/dL)	94.0 (22.0)	91.0 (20.0)	102 .0(25.0)	0.094
High-density lipoprotein cholesterol (mg/dL)	41.0 (17.0)	39.0 (11.0)	47.0 (26.0)	0.373
Triglyceride (mg/dL)	95.0 (27.0)	92.0 (28.0)	102.0 (22.0)	0.012

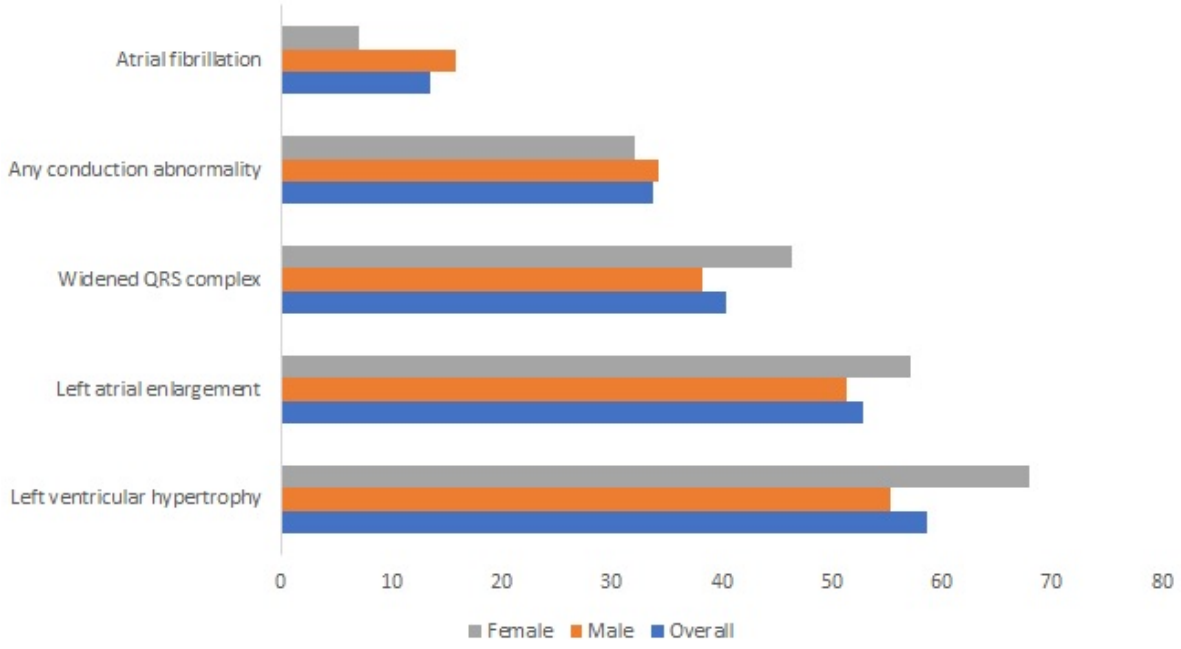

Figure 2. Twelve-lead ECG findings in the 127 studied patients with dilated cardiomyopathy

Supplementary Table 1. Some 12-lead ECG and echocardiographic findings in the 127 studied patients with dilated cardiomyopathy

Variable (n/%)	Overall (No. =127)	Male (No. = 91)	Female (No. = 36)	*P*
12-Lead ECG				
QT interval (ms)	402 (86)	403 (90)	400 (77)	0.635
QTc interval (ms)	489 (88)	486 (91)	497 (81)	0.397
QRS interval (ms)	128 (47)	128 (49)	126 (40)	0.784
Echocardiography				
Aortic root dimension (cm)	2.89 (0.52)	3.01 (0.55)	2.60 (0.29)	<0.001
Left atrial dimension (cm)	4.66 (0.85)	4.70 (0.89)	4.54 (0.75)	0.401
Interventricular septal thickness diastole (cm)	0.93 (0.29)	0.97 (0.29)	0.83 (0.24)	0.022
Interventricular septal thickness systole (cm)	1.02 (0.32)	1.06 (0.34)	0.92 (0.24)	0.071
Left ventricular posterior wall thickness diastole (cm)	0.99 (0.31)	1.03 (0.30)	0.88 (0.31)	0.026
Left ventricular posterior wall thickness systole (cm)	1.30 (0.34)	1.31 (0.36)	1.27 (0.30)	0.601
Left ventricular internal diameter diastole (cm)	6.47 (0.95)	6.45 (1.04)	6.51 (0.67)	0.872
Left ventricular internal diameter systole (cm)	5.49 (0.97)	5.45 (1.07)	5.60 (0.65)	0.660
Left ventricular ejection fraction. (%)	38 (13)	39 (14)	35 (10)	0.207
Left ventricular fractional shortening (%)	15.3 (6.5)	15.8 (7.0)	14.0 (4.9)	0.278
TAPSE (cm)	1.74 (0.43)	1.71 (0.45)	1.83 (0.37)	0.368
Mitral E-wave (m/s)	0.90 (0.27)	0.89 (0.27)	0.94 (0.27)	0.347
Mitral A-wave (m/s)	0.44 (0.21)	0.46 (0.23)	0.41 (0.13)	0.326
E/A ratio	2.43 (1.11)	2.23 (1.03)	2.61 (1.29)	0.321
Deceleration time of the E-wave (ms)	135 (51.3)	139.7 (54.8)	124.1(40.00)	0.183
Isovolumic relaxation time (ms)	124.6 (42.7)	124.0 (39.2)	126.2 (51.7)	0.847
Left atrial volume (mL)	115.1(57.2	114.9 (39.2)	115.9 (64.9)	0.972
Right atrial volume (mL)	87.6(45.1	87.7 (44.6)	87.2 (54.6)	0.985
Mitral regurgitation (No. /%) (n=90)	64 (71.1)	22 (81.5)	22 (81.5)	0.207
Tricuspid regurgitation (No. /%) (n=90)	63 (70)	4 (4.8)	22(81.5)	0.139
Spontaneous echo (No. /%) (n=90)	11 (12.2)	2 (7.4)	4 (14.8)	0.728
Intracardiac clot (No. /%) (n=90)	3 (3.3)	12	2(7.4)	0.213
Pericardial effusion (n/%) (n=89)	24 (27.0)	12 (13.5)	12 (13.5)	0.101

TAPSE: tricuspid annular plane systolic excursion

Figure 3 illustrates the pattern of medications prescribed to the 127 patients with DCM.

Outcome

Follow-up was censored at 12 months, and 127 patients had completed 12-month follow-up at the time of analysis. During the follow-up period, 93 patients (73.2%) were alive, 30 (23.6%) died, and 4 (3.1%) were lost to follow-up. The 30-, 90-, 180-, and 360-day mortality rates were 2.37% (95% CI, 0.77 to 7.16), 11.21% (95% CI, 6.80 to 18.20), 16.1% (95% CI, 10.7 to 28.9), and 24.2% (95% CI, 17.6 to 32.8), respectively (Figures 4A and 4B).

Predictors of All-Cause Mortality

In univariable regression analysis, sex, alcohol consumption, respiratory rate, and presence of left axis deviation on 12-lead ECG were associated with the composite outcome (readmission and mortality). Nonetheless, in multivariable analysis, only a history of alcohol consumption achieved statistical significance after controlling for other confounding variables (Supplementary Table 2).

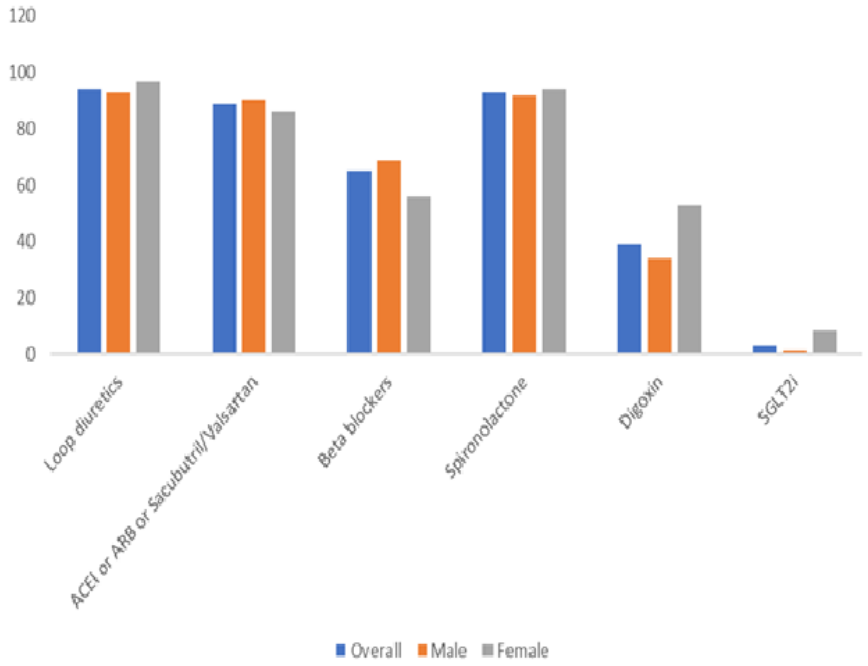

Figure 3. Pattern of heart failure-related medications prescribed for the 127 studied patients with dilated cardiomyopathy (DCM)

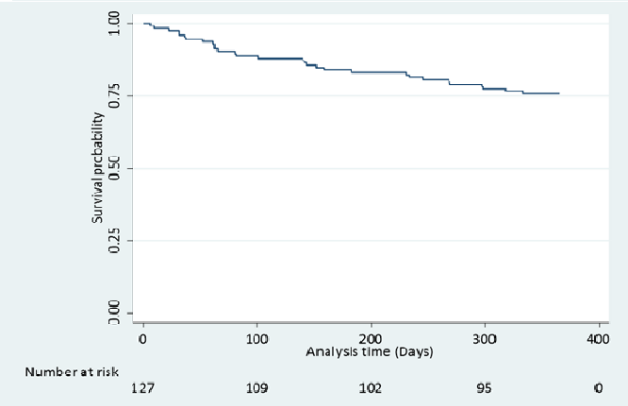

FIGURE4A

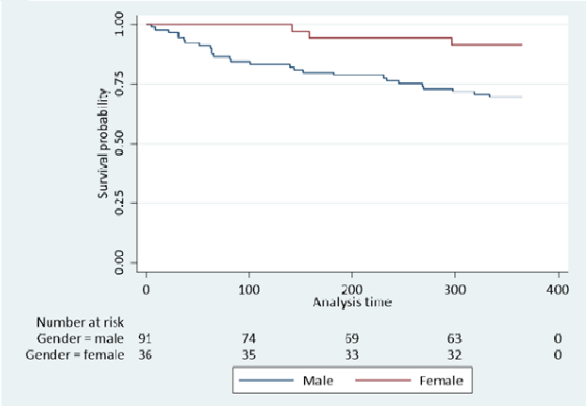

FIGURE4B

Figure 4A. One-year survival rate for the whole cohort
Figure 4B. One-year survival rate for the cohort and according to sex

Supplementary Table 2. Cox regression analysis of predictors of death in the 127 studied patients with dilated cardiomyopathy

Variable	Hazard Ratio	*P*	95% CI
Male sex	2.19	0.348	0.426–11.252
Ever drank alcohol	1.21	0.716	0.431–3.400
Ever smoked a cigarette	1.54	0.446	0.507–4.683
Serum creatinine	1.08	0.163	0.969–1.205
LDL-cholesterol	0.98	0.084	0.958–1.002
Left axis deviation on 12-lead ECG	1.99	0.132	0.811–4.891
Left atrial enlargement on 12-lead ECG	1.11	0.800	0.479–2.593

DISCUSSION

This report describes the sociodemographic and clinical profile and 1-year outcome of DCM in a Nigerian tertiary hospital. The cohort is relatively young, with mean (SD) age of 52.0 (16.0) years (53.0 [16.0] years for males and 47.0 [15.0] years for females). DCM is predominantly a male disease, with more than 70% of cases in males and more than 80% older than 30 years. Mortality is high, and an independent predictor appears to be previous alcohol consumption. Mean age in this cohort is similar but older than that of a recent cohort in Johannesburg, South Africa, where mean age was 44.0 years.[13] Our data are comparable to mean age of patients with DCM in many parts of Africa, 52.5 (6.2) years, as reported by Fundikira et al.[14]

This study showed DCM is more common in adult males. Other workers in Africa have also reported predominance of male sex in their cohorts.[13] In the report by Tsabedze et al[13] from South Africa, 64.5% were males. In a meta-analysis of DCM cases in Africa, median proportion of males was 44.2% (IQR, 33.2–49.5).[14]

Previous or current cigarette use was observed in 17% and 1.9% of patients, respectively. Previous or current alcohol use was noted in 35% and 6.7%, respectively. Diabetes mellitus and a family history of heart failure were noted in 8.7% and 2.9% of patients, respectively. In a systematic review of DCM in Africa, tobacco use, excessive alcohol intake, diabetes mellitus, and family history of heart failure were shown to occur in 6.6%, 10%, 11%, and 4% of cases, respectively,[14] similar to the findings in this report. Unlike many other reports that included hypertension as a risk factor for DCM, we excluded cases with hypertension, as we consider hypertensive heart failure a different disease entity.[14]

This report also shows that hospital admission lags behind mortality in this setting. Only two cases were reported to have died in the hospital. Reasons may include a lack of health insurance, high out-of-pocket expenses, and a lack of awareness. More often, when heart failure symptoms recur, patients prefer to remain at home (where many die) or resort to alternative medicines or spiritual care.

The 1-year mortality rate is high. It is higher than the median mortality rate of 13.4% reported from South Africa or the 4% and 5% rates reported from other parts of the world.[13, 10, 15]

Tyminska et al[16] reported a mortality of 10% in a multicenter study in Europe. In a South African study, [17] a 5-year mortality rate of 40% was documented. Our finding is similar to the 26.4% mortality rate reported by the INTER-CHF study group.[18] Reasons for higher mortality in this setting include late presentation, severe New York Heart Association functional class at presentation, and poverty. Few patients can afford newer disease-modifying agents such as sacubitril/valsartan or sodium-glucose cotransporter 2 inhibitors.[19, 20] Furthermore, uptake of implantable cardioverter-defibrillators or cardiac resynchronization therapy is very low, and transplantation services are unavailable.

CONCLUSIONS

DCM in Ibadan, Nigeria, is a disease of young and middle-aged males. Patients often present late with severe ventricular dysfunction. Follow-up suggests all-cause mortality is high, and hospitalization lags behind mortality. A national or continental registry is needed to explore the clinical profile, risk factors, pathogenesis, and natural history of this disease in the region.

Strengths and Limitations of the Study

Coronary angiography was not performed to exclude ischemic cardiomyopathy, but none of the cases showed 12-lead ECG evidence of

previous myocardial infarction or echocardiographic evidence of regional wall motion abnormalities. Cardiac biomarkers and advanced cardiac imaging, such as CT or MRI, were not done routinely, as patients paid out of pocket. Autopsies were not performed for cases that died, although more than 95% of deaths occurred out of the hospital.

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Authors' Contributions:
O. S. O., A. A., A. M. A., A. A., O. O., and O. M. A. conceived the topic; O. S. O., O. A. A., A. T. A., O. Z. M., O. G. A., F. E. O., M. M. O., O. C. I., D. A. O., O. S. A., J. L. I., I. N., K. O., V. O. F., and C. I. U. searched the literature and gathered all data; J. O. A. and A. A. performed the statistical analysis; O. S. O., F. E. O., and D. O. designed the figures; O. S. O., M. N. O., O. C. I., F. E. O., C. U., and O. A. O. made the tables; O. S. O., F. E. O., M. N. O., O. C. I., F. E. O., and O. M. A. participated in the draft and review phases of the manuscript. All authors reviewed the manuscript.

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Data Availability
The data sets generated and/or analyzed during the current study are available.

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REFERENCES

1. Maron BJ, Towbin JA, Thiene G, et al. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. Circulation 2006; 113: 1807-1816.

2. Brigden W. Uncommon myocardial diseases: the non-coronary cardiomyopathies. Lancet 1957; 273: 1243-1249.

3. Goodwin JF, Gordon H, Hollman A, et al. Clinical aspects of cardiomyopathy. Br Med J 1961; 1: 69-79.

4. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J 2023; 44: 3503-3626.

5. Agbor VN, Essouma M, Ntusi NAB, et al. Heart failure in sub-Saharan Africa: A contemporaneous systematic review and meta-analysis. Int J Cardiol 2018; 257: 207-215.

6. Ogah OS, Stewart S, Falase AO, et al. Contemporary profile of acute heart failure in Southern Nigeria: data from the Abeokuta Heart Failure Clinical Registry. JACC Heart Fail 2014; 2: 250-259.

7. Ogah os, adebiyi a, aje a, et al. Gender differences in the clinical profile and sociodemographic characteristics of dilated cardiomyopathy in ibadan, nigeria. Ann ib postgrad med 2022; 20: 6-13.

8. Falase AO. Infections and dilated cardiomyopathy in Nigeria. Heart Vessels Suppl 1985; 1: 40-44.

9. Ogah O. Dilated Cardiomyopathy in Ibadan, Nigeria: an echocardiographic study. African Journal of Medicine and Medical Sciences 2020; 49: 181-188.

10. Dziewięcka E, Gliniak M, Winiarczyk M, et al. Mortality risk in dilated cardiomyopathy: the accuracy of heart failure prognostic models and dilated cardiomyopathy-tailored prognostic model. ESC Heart Fail 2020; 7: 2455-2467.

11. Richardson P, McKenna W, Bristow M, et al. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies. Circulation 1996; 93: 841-842.

12. Elliott P, Andersson B, Arbustini E, et al. Classification of the cardiomyopathies: a position statement from the European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J 2008; 29: 270-276.

13. Tsabedze N, Mpanya D, Bailly C, et al. Clinical characteristics and one-year all-cause mortality outcomes in Africans with dilated cardiomyopathy. Int J Cardiol 2023; 387: 131142.

14. Fundikira LS, Chillo P, Mutagaywa R, et al. Risk Factors and Prevalence of Dilated Cardiomyopathy in Sub-Saharan Africa: A Systematic Review. Glob Heart 2022; 17: 76.

15. Puntmann VO, Carr-White G, Jabbour A, et al. T1-Mapping and Outcome in Nonischemic Cardiomyopathy: All-Cause Mortality and Heart Failure. JACC Cardiovasc Imaging 2016; 9: 40-50.

16. Tymińska A, Ozierański K, Balsam P, et al. Ischemic Cardiomyopathy versus Non-Ischemic Dilated Cardiomyopathy in Patients with Reduced Ejection Fraction-Clinical Characteristics and Prognosis Depending on Heart Failure Etiology (Data from European Society of Cardiology Heart Failure Registries). Biology (Basel) 2022; 11

17. Ntusi NB, Badri M, Gumedze F, et al. Clinical characteristics and outcomes of familial and idiopathic dilated cardiomyopathy in Cape Town: a comparative study of 120 cases followed up over 14 years. S Afr Med J 2011; 101: 399-404.

18. Karaye KM, Dokainish H, ElSayed A, et al. Clinical Profiles and Outcomes of Heart Failure in Five African Countries: Results from INTER-CHF Study. Glob Heart 2021; 16: 50.

19. McMurray JJ, Packer M, Desai AS, et al. Baseline characteristics and treatment of patients in prospective comparison of ARNI with ACEI to determine impact on global mortality and morbidity in heart failure trial (PARADIGM-HF). Eur J Heart Fail 2014; 16: 817-825.

20. McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction. N Engl J Med 2019; 381: 1995-2008.