pagepress

GLOBAL CARDIOLOGY
Global Cardiology 2024; **3**
Published online 5 November 2024 | **DOI**: 10.4081/cardio.2024.42

ARTICLE

Clinical profiles and short-term outcomes of women with peripartum and dilated cardiomyopathies

Babalawan Muhammad,[1] Naser A. Ishaq,[2] Kamilu M. Karaye[2,3]

[1]Department of Medicine, Bauchi State Specialist Hospital, Bauchi State; [2]Department of Medicine, Aminu Kano Teaching Hospital, Kano; 3Department of Medicine, Bayero University, Kano, Nigeria

Abstract

Background: We aimed to compare the clinical and socio-demographic characteristics, rate of left ventricular reverse remodeling (LVRR), heart failure hospitalization and all-cause mortality of women with peripartum and dilated cardiomyopathies (PPCM and DCM respectively) in Nigeria.
Methods: This was a prospective longitudinal study and a total of 130 patients (65 for each group) were recruited consecutively and followed up for 6 months.
Results: PPCM patients were younger, but the DCM patients had higher frequency of atrial fibrillation and complete left bundle branch block, higher mean left atrial and LV dimensions, higher LV filling pressures, and worse renal dysfunction, respectively, at baseline. At the end of the 6-month follow-up, 15 female DCM vs 21 PPCM patients (p=0.684) had achieved LVRR, 13 DCM vs 11 PPCM patients (p=0.098) were hospitalized for heart failure, and 10 DCM *vs* 5 PPCM patients (p=0.098) had died. The odds for achieving LVRR was independently increased by systolic blood pressure (SBP) >100 mmHg and tricuspid annular plane systolic excursion >16 mm at baseline in PPCM patients, and by use of angiotensin converting enzyme inhibitors at baseline in female DCM patients. The odds for mortality were increased by tachycardia and pericardial effusion and reduced by the use of loop diuretics at baseline in DCM patients, and SBP <90 mmHg at baseline increased it by 9-fold in PPCM patients.
Conclusions: Our results suggest that women with DCM and PPCM differ significantly in their demographic and clinical characteristics, and predictors of clinical outcomes.

Key words: peripartum cardiomyopathy, dilated cardiomyopathy, women, outcomes.

Received: 25 February 2024; Accepted: 16 May 2024.
**Correspondence to: Kamilu M. Karaye, Department of Medicine, Bayero University and Aminu Kano Teaching Hospital, PO Box 4445, Kano, Nigeria.*
E-mail: kkaraye@yahoo.co.uk

Introduction

Peripartum cardiomyopathy (PPCM) was most recently defined by the Heart Failure (HF) Association of European Society of Cardiology (ESC) Working Group on PPCM *as an idiopathic cardiomyopathy presenting with HF secondary to left ventricular (LV) systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of HF is found. The LV may not be dilated but the LV ejection fraction (LVEF) is reduced below 45%*.[1] The exact cause is still unknown, but several hypotheses have been put forward to explain it, including the 16KD prolactin, autoimmunity, myocarditis and selenium deficiency hypotheses.[1,2] PPCM is commoner in sub-Saharan Africa and among women of African ancestry, perhaps as a result of genetic and nutritional factors, as well poor standard of healthcare.[1] Investigators of the Peripartum Cardiomyopathy in Nigeria (PEACE) Registry reported the highest ever incidence of PPCM in the world of 1 in 96 live births in Kano, Nigeria.[3]
Dilated cardiomyopathy (DCM) is a primary heart muscle disease defined by the presence of LV dilatation and systolic dysfunction in the absence of abnormal loading conditions or coronary artery disease and congenital heart disease, sufficient to cause the LV systolic dysfunction.[4] DCM is one of the leading causes of HF in Africa.[5] It has an annual incidence rate of 5 to 8 cases per 100,000 population in the USA.[6] In some parts of Africa, about 17-48% of patients admitted for HF had DCM.[5,7] It affects all ages, but is commoner in the third and fourth decades of life.[5-7]

Although many studies have shown similarities between PPCM and DCM in terms of clinical presentations, their risk factors seem to differ.[4-7] Recent reports suggest that only history of eclampsia, underweight, lack of formal education, unemployment status, selenium deficiency and rural residency were shown to be significantly associated with PPCM in Nigeria.[3] On the other hand, excess alcohol consumption, tobacco use, HIV infection, advanced age and history of diabetes are associated with DCM.[8]

Few studies had compared the sociodemographic and clinical profiles and outcomes of PPCM and female DCM patients.[9,10] Lu *et al*. in a retrospective cohort study in Taiwan showed that at the end of 1- and 3-year follow-up periods, patients with PPCM had significantly more favorable outcomes for all-cause mortality, cardiac death and major adverse cardiovascular events, than those with DCM, except for HF re-admissions at the second and third years of follow-up.[9] The rate of left ventricular reverse remodeling (LVRR) was however not described. In the present study, we therefore aimed to prospectively determine and compare the clinical features, socio-demographic characteristics, LVRR, HF hospitalization and all-cause mortality of female DCM with PPCM patients in a Teaching Hospital in Kano, Nigeria. We hypothesized that Nigerian women with DCM would share morphologic characteristics with PPCM patients, but would differ significantly in their sociodemographic characteristics and clinical outcomes.

Materials and Methods

The study was a prospective longitudinal comparative study conducted in Aminu Kano Teaching Hospital (AKTH), Kano, Nigeria between June 2019 and October 2022. The study protocol was approved by the AKTH Health Research Ethics Committee (reference: AKTH/MAC/SUB/12A/P-3/VI/2891). Subjects were enrolled into the study after obtaining written informed consent. The study conformed to the ethical guidelines of the Declaration of Helsinki on the principles for medical research involving human subjects.[11] All data and supporting materials have been provided in the results section, but further request could be sent to the authors.

Patients were included if they had satisfied the following criteria: i) women with confirmed diagnosis of either DCM or PPCM, ii) written informed consent, and iii) valid telephone number and willingness to attend follow-up reviews. However, patients were excluded if: i) they had any other structural heart disease, and ii) longstanding history of hypertension.

The minimum sample size was estimated while taking into consideration the prevalence of LVRR among PPCM patients in Kano (47% by Karaye *et al*.) and among DCM patients (38% by Masci *et al*.) and adding an attrition rate of 10%.[12,13] Ultimately, a total of 65 patients were recruited for each group and followed up for six months. Detailed information on sociodemographic and clinical characteristics was obtained from all the participants using a pretested questionnaire. Echocardiography and electrocardiography were performed according to standard recommendations at recruitment for all patients and repeated at the end of the six months follow-up for the survivors.[14,15]

Subjects enrolled into the study were reviewed at the outpatient clinic every 4 weeks or followed-up via telephone interviews for those that could not attend, to enquire about symptoms of HF and clinical outcomes. For those that died we interviewed their next of kin.

For the purpose of this study, PPCM was defined according to the recommendations of the PPCM Working Group of the ESC, and DCM was defined according to the recommendations of the ESC Working Group on Myocardial and Pericardial Diseases.[1,4] Left ventricular reverse remodeling (LVRR) was defined as an absolute increase in LV ejection fraction (LVEF) by ≥10% and a decrease in LV end diastolic dimension indexed to body surface area (LVEDDi) by at least 10% or decrease in LVEDDi to ≤33 mm/m^2.[14]

Data analysis

Quantitative variables were explored for skewness and expressed as mean with standard deviation (SD) or as median with 25th-75th percentiles, as appropriate. Qualitative variables were expressed as frequencies and percentages. Chi-square, Fisher's exact, Student's t and Mann-Whitney tests were used to compare categorical and continuous variables, as appropriate. Kaplan-Meier method was used to compare survival rates of the 2 groups at six months. Logistic regression models were developed to generate the crude and adjusted odds ratios (OR) with 95% confidence intervals (CI) for predictors of LVRR. Cox Proportional Hazard models were developed to explore the relationship between mortality in PPCM and female DCM patients and variables of interest, and corresponding hazard ratios (HR) with 95% CI obtained. $p<0.05$ was considered as statistically significant. The statistical analysis was carried out using SPSS version 26.0 software.

Results

A total of 65 PPCM and 65 female DCM patients were consecutively recruited and compared for sociodemographic and clinical characteristics and clinical outcomes.

The socio-demographic and clinical characteristics of the study population are shown in Table 1. It shows that PPCM patients were significantly younger and had higher frequency of rural residency and formal education. Majority of the patients in both groups presented in New York Heart Association (NYHA) Functional Classes III or IV. DCM patients had higher frequency of atrial fibrillation (AF) and complete left bundle branch block, higher mean left atrial and LV dimensions, and higher LV filling pressures, respectively, at baseline. Angiotensin converting enzyme inhibitors (ACE-I) were more frequently prescribed for female DCM than PPCM patients, but prescriptions of the other HF medications were not significantly different between the two groups.

Table 1. Baseline socio-demographic and clinical characteristics of female dilated and peripartum cardiomyopathy patients.

Variables	All patients N=130	DCM N=65	PPCM N=65	p
Age, years	41.4±17.9	54.6±15.6	28.2±7.2	<0.001*
Age <20 years	41 (31.5)	4 (6.2)	37 (56.9)	<0.001*
Age ≥35 years	69 (53.1)	57 (87.7)	12 (18.5)	<0.001*
Rural residence	61 (46.9)	22 (33.8)	39 (60.0)	0.005*
Educational status				<0.001*
None	50 (38.5)	39 (60.0)	11 (16.9)	
Primary	27 (20.8)	12 (18.5)	15 (23.1)	
Secondary	41 (31.5)	7 (10.8)	34 (52.3)	
Tertiary	12 (9.2)	7 (10.8)	5 (7.7)	
Unemployed	108 (83.1)	57 (87.7)	51 (78.4)	0.447
NYHA functional classes				0.702
II	53 (40.8)	27 (33.8)	26 (40.0)	
III	51 (39.2)	24 (36.9)	27 (41.5)	
IV	26 (20.0)	14 (21.5)	12 (18.5)	
HR	102±19	104±18	100±20	0.720
SBP, mmHg	109±20	111±20	106±19	0.126
DBP, mmHg	73±20	73±18	73±21	0.790
BMI, kg/m^2	23.5±5.2	23.9±5.8	23.1±5.3	0.401
Underweight	16 (12.3)	6 (9.2)	10 (15.4)	0.672
Obesity	15 (16.9)	9 (13.8)	6 (9.2)	0.672
Electrocardiogram				
AF	4 (3.8)	4 (8.3)	0 (0.0)	0.007*
QRS, ms	95.9±27.0	108.9±33.8	85.3±12.6	<0.001*
Complete LBBB	16 (15.2)	14 (29.8)	2 (3.4)	<0.001*
Echocardiogram				
LA (mm)	45.2±6.0	46.2±6.4	44.1±5.5	0.048*
LVEDD (mm)	66.2±7.4	67.9±6.6	64.5±7.8	0.007*
LVEF (%)	30.8±7.9	30.4±7.6	31.3±8.2	0.552
Mitral E/e'	15.0±7.5	16.6±8.5	13.4±6.0	0.013*
RV basal (mm)	43.2±7.8	42.4±7.6	44.1±8.0	0.203
TAPSE (mm)	14.3±3.4	14.5±3.4	14.5±3.7	0.941
Pulmonary HTN	91 (70.0)	46 (70.8)	45 (69.2)	1.000
Intracardiac thrombus	13 (10.0)	8 (12.3)	5 (7.7)	0.397
Laboratory results				
Serum sodium (mmol/L)	135.18±9.92	135.82±5.08	134.44±13.10	0.479
Hyponatraemia	48 (36.9)	22 (33.8)	26 (40.0)	0.281
Serum creatinine (µmol/L)	106.31±63.86	106.31±63.86	93.11±52.16	0.199
eGFR (mL/min)	76.66±32.92	65.71±30.86	87.62±31.44	<0.001*
eGFR <60 mL/min	52 (40.0)	38 (58.5)	14 (21.5)	<0.001*
Hb (g/dL)	10.9±2.02	10.30±2.09	9.87±1.92	0.225
Anemia	25 (19.2)	15 (23.1)	10 (15.4)	0.374
Heart failure drugs at baseline				
ACE-I	38 (29.2)	25 (38.5)	13 (20.0)	0.033*
ARB	14 (10.8)	9 (13.8)	5 (7.7)	0.258
B-blockers	78 (60.0)	38 (58.5)	40 (61.5)	0.720
MRA	125 (96.2)	63 (96.9)	62 (95.4)	1.000
Loop diuretic	129 (99.2)	64 (98.5)	65 (100.0)	1.000
Digoxin	101 (77.7)	48 (73.8)	53 (81.5)	0.400
Anticoagulant	28 (21.5)	18 (27.7)	10 (15.4)	0.134
Sacubutril/valsartan	3 (2.3)	2 (3.1)	1 (1.5)	1.000
Ivabradine	4 (3.1)	3 (4.6)	1 (1.5)	0.619

N, number of subjects; DCM, dilated cardiomyopathy; PPCM, peripartum cardiomyopathy; SOB, shortness of breaths; NYHA, New York Heart Association; MR, mitral regurgitation; PR, pulse rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; JVP, jugular venous pressure; eGFR, estimated glomerular filtration rate; Hb, hemoglobin; ACE-I, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blockers; MRA, mineralocorticoid receptor antagonists. Values are expressed as numbers with percentages in parentheses or as means± standard deviation. *Statistically significant.

During a maximal follow-up of six months, a total of 15 (11.5%) patients died, of whom 10 (15.4%) had DCM while the remaining 5 (7.7%) had PPCM (p=0.272) (Figure 1). In addition, a total of 9 patients were lost to follow-up (7 in the DCM group and 2 in the PPCM group; p=0.164). Thus, a total of 48 female DCM patients and 58 PPCM patients had an echocardiogram and ECG at the last follow up. Overall, 15/48 (31.3%) female DCM and 21/58 (36.2%) PPCM patients (p=0.597) achieved LVRR in the study (Table 2). The determinants of LVRR among PPCM and female DCM patients are presented in Table 3. In the multivariate analysis, systolic blood pressure (SBP) >100 mmHg and tricuspid annular plane systolic excursion (TAPSE) >16 mm independently increased the odds for LVRR by 8.6 and 6.6-fold respectively among PPCM patients, while the odds for LVRR was reduced by 90% by the presence of complete left bundle branch block (cLBBB) and increased by 6.3-fold by the use of ACE-I among the women with DCM.

The Kaplan Meier survival curves presented in Figure 2 compared the survival patterns of female DCM patients with PPCM patients over a 24-week follow up period.

A total of 24 (18.5%) patients were hospitalized during the study period, of whom 13 (20.0%) had DCM and 11 (16.9%) had PPCM (p=0.098).

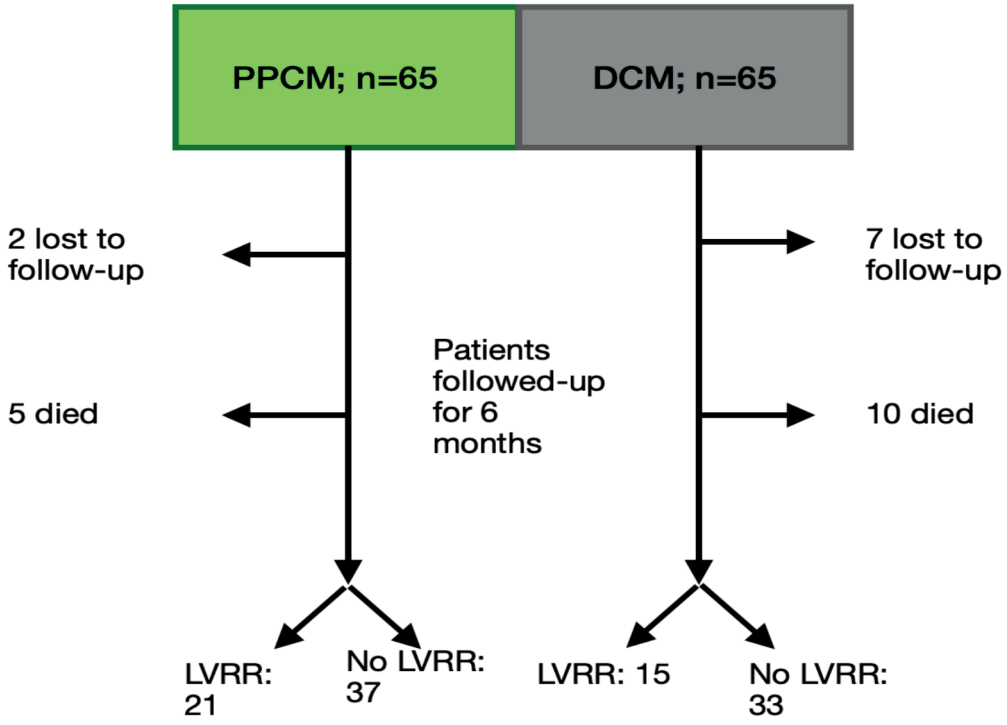
The Cox proportional hazard regression models for predictors of mortality among the study population are presented in Tables 4 and 5. In the multivariate analysis for independent determinants of mortality among female DCM patients, increase in heart rate above the mean of 102 beats/minute at recruitment increased the risk by 4%, presence of pericardial effusion at recruitment increased the risk by 6-fold, while prescribed loop diuretics at recruitment reduced the risk by 96%. However, SBP <90 mmHg was the only independent determinant of mortality among PPCM patients.

Discussion

To the best of our knowledge, this is the first study that has prospectively determined and compared the socio-demographic and clinical characteristics, and rates of LVRR, hospitalization and survival among PPCM and female DCM patients. We studied females with DCM to exclude any influence of male gender on the results.

Sociodemographic and clinical characteristics

Firstly, the results showed that PPCM patients were significantly younger, had better education and majority lived in rural areas, when compared with the female DCM patients. These findings corroborate what Karaye *et al*., Lu *et al*. and



Flow chart showing the number of patients at recruitment and follow-up, and clinical outcomes in the 2 groups. PPCM, peripartum cardiomyopathy; DCM, dilated cardiomyopathy; LVRR, left ventricular reverse remodeling.

Figure 1. Study flow chart.

Petryka-Mazurkiewicz *et al*. previously reported among PPCM and male and female DCM patients in Nigeria, Taiwan and Poland respectively.[7,9,10,16] Rural residency was previously found to significantly correlate with selenium deficiency, which is strongly associated with PPCM in northwestern Nigeria.[2,17] Poor socioeconomic indices have been associated with PPCM and other non-ischemic cardiomyopathies, and to have negative impact on clinical outcomes, although the exact mechanisms of effects are not yet clear.[2,3,18]

Our study has also shown that the symptoms and signs of PPCM and DCM in women were similar, and majority of the patients presented with moderate-severe HF symptoms. This clearly indicates that the two diseases cannot be distinguished on the basis of symptoms and physical signs alone, and so the historical relationship between onset of symptoms and childbirth is critically important. However, female DCM patients had larger left heart chambers, higher LV filling pressures and higher burden of co-morbidities of atrial fibrillation (AF), LV desynchrony and renal dysfunction, in keeping with a more chronic disease process as compared with PPCM, which develops rapidly within a few days to weeks.[1,4] It is important to note that Cardiac Magnetic Resonance imaging on a small sample of women with DCM and PPCM, had previously revealed similar sizes of left heart chambers, as well as the frequency and the extent of late gadolinium enhancement, among the two group of patients.[10] Interestingly, Strain analysis showed that the PPCM patients had less impaired LV global and circumferential strain in comparison with women with DCM, in spite of the above-mentioned similarities.[10]

Table 2. Echocardiographic variables at baseline and 6-month follow-up in female dilated and peripartum cardiomyopathy patients.

Variable	Baseline	Follow-up	p
Female DCM patients			
Left atrium, mm	46.5±6.0	42.2±5.6	<0.001*
LVEDDi, mm/m^2	43.6±7.2	39.6±8.2	<0.001*
LVEF, %	30.9±7.2	40.8±12.5	0.069
RV basal, mm	42.0±6.7	37.3±5.4	<0.001*
TAPSE, mm	14.5±3.1	16.3±2.3	0.263
PPCM patients			
Left atrium, mm	43.6±5.2	37.9±7.0	<0.001*
LVEDDi, mm/m^2	41.7±6.5	37.1±7.8	<0.001*
LVEF, %	31.9±8.3	43.4±13.9	0.083
RV basal, mm	44.1±8.3	37.6±8.5	<0.001*
TAPSE, mm	14.8±3.8	16.9±4.2	<0.001*

DCM, dilated cardiomyopathy; LVEDDi, left ventricular end diastolic dimension index; LVEF, left ventricular ejection fraction; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion; PPCM, peripartum cardiomyopathy. *Statistically significant.

Table 3. Determinants of left ventricular reverse remodeling among peripartum cardiomyopathy and female patients with dilated cardiomyopathy.

Variables	PPCM only				Female DCM only			
	Univariate analysis		Multivariate analysis		Univariate analysis		Multivariate analysis	
	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p
Age, years	1.00 (0.93-1.08)	0.934	-	-	0.99 (0.96-1.03)	0.840	-	-
Heart rate, bpm	0.99 (0.96-1.02)	0.526	-	-	0.93 (0.95-1.04)	0.750	-	-
SBP >100 mmHg	6.22 (1.75-22.23)	0.005*	8.58 (1.90-38.77)	0.005*	0.91 (0.25-3.36)	0.886	-	-
eGFR >60 mL/min/1.32m^2	8.45 (1.01-71.09)	0.049*	6.20 (0.58-65.74)	0.130	1.67 (0.49-5.75)	0.416	-	-
cLBBB	1.80 (0.11-30.36	0.683	-	-	0.10 (0.01-0.89)	0.039*	0.10 (0.01-0.93)	0.043*
LA (mm)	1.07 (0.96-1.20)	0.213	-	-	1.09 (0.97-1.23)	0.142	-	-
LVEF <25%	0.49 (0.14-1.78)	0.278	-	-	1.08 (0.27-4.28)	0.917	-	-
Mitral E/e'	1.05 (0.95-1.16)	0.332	-	-	1.04 (0.95-1.14	0.374	-	-
TAPSE >16 mm	4.71 (1.44-15.46)	0.010*	6.63 (1.39-31.61)	0.018*	4.67 (0.53-41.32)	0.166	-	-
Beta-blockers	1.08 (0.35-3.36)	0.890	-	-	0.83 (0.23-3.03)	0.782	-	-
ACE-I	2.07 (0.57-7.50)	0.270	-	-	6.05 (1.54-23.74)	0.010*	6.29 (1.46-27.04)	0.014*
ARB	1.84 (0.24-14.14)	0.557	-	-	0.55 (0.10-3.03)	0.492	-	-
Digoxin	0.82 (0.20-3.32)	0.784	-	-	0.56 (0.14-2.19)	0.404	-	-
BMI, kg/m^2	0.86 (0.76-0.98)	0.019*	0.86 (0.73-1.01)	0.064	0.96 (0.87-1.07)	0.475	-	-

PPCM, peripartum cardiomyopathy; DCM, dilated cardiomyopathy; OR, odds ratio; CI, confidence interval; SBP, systolic blood pressure; eGFR, estimated glomerular filtration rate; cLBBB, complete left bundle branch block; LA, left atrium; LVEF, left ventricular ejection fraction; E/e', LV filling pressure; TAPSE, tricuspid annular plane systolic excursion; ACE-I, angiotensin converting enzyme inhibitors; ARB, angiotensin receptor blockers; BMI, body mass index. *Statistically significant.

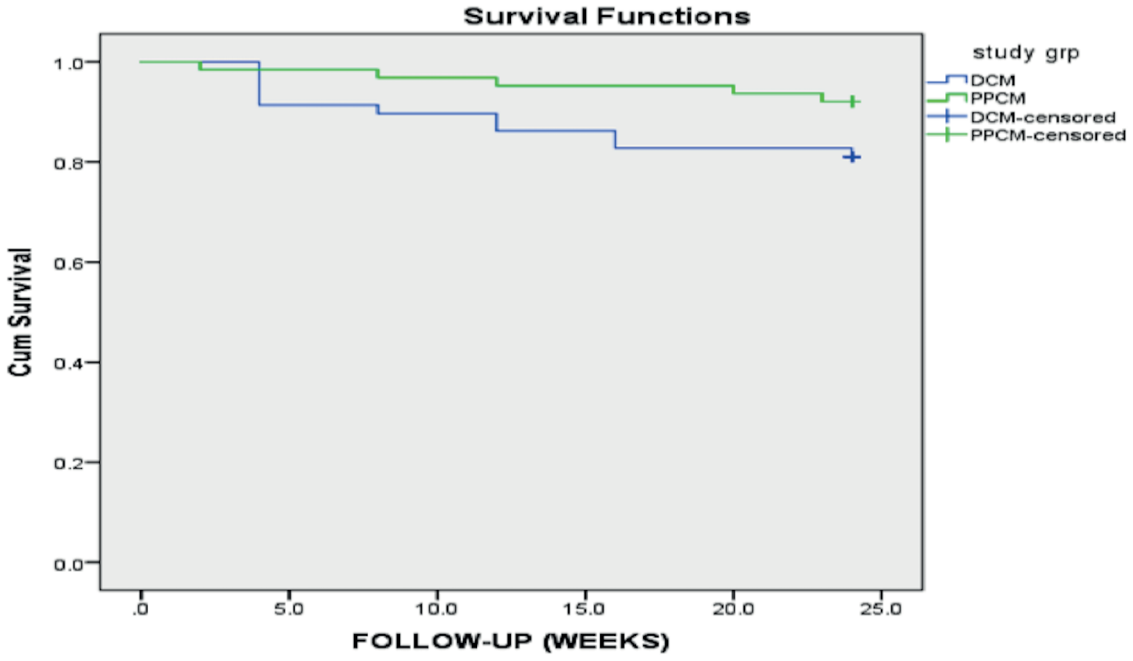
Clinical outcomes

This study also demonstrated that both diseases could achieve significant LVRR over time, although surprisingly, the differences between them were not statistically significant. Studies by Masci *et al*. and Merlo *et al*. similarly demonstrated the prevalence of LVRR among male and female DCM patients to be 37% and 38% respectively.[13,19] Recovery of LV function in PPCM patients was previously reported as 21% at 6 months of follow-up in South Africa, 28% at 2 years of follow-up in Haiti, 71% at 1 year of follow-up in the USA, and 95.5% at 5 years of follow-up in Germany.[20-23] In the present study, different variables predicted LVRR in the two groups; SBP >100mmHg and TAPSE >16mm for PPCM, and cLBBB and use of ACE-I for the women with DCM. In the PEACE registry, 24.1% of the PPCM patients achieved LVRR and 22.6% achieved LV full recovery over 18 months of follow-up.[24] Progressive reverse remodeling of all cardiac chambers was similarly observed, and LVEF <25% at baseline and regular use of beta-blockers independently predicted LV functional recovery.

At the end of our 6-month follow up, a total of 24 (18.5%) patients were hospitalized due to HF and 15 (11.5%) patients had died. The mortality rate among the women with DCM was twice (15.4% *vs* 7.7%) that of PPCM but the difference was not statistically significant. The factors associated with mortality differed between the two groups, which further supports the hypothesis that DCM and PPCM are distinct disease entities. In agreement with our findings, Lu et al similarly reported a mortality rate of 8.4% among PPCM and 14.1% among DCM patients over a 12-month period, and no difference was observed in the risk of HF readmission.[9]

Sliwa *et al*. reported NYHA functional class and baseline levels of FAS/Apo-1 as the independent predictors of mortality among PPCM patients.[25] The results of PEACE registry on survival of PPCM patients, differed in several ways with ours.[24] In summary, 18.7% of the patients died during follow-up, and the independent predictors of mortality were maternal age <20years, hypotension, tachycardia and LVEF <25% at baseline. Obesity and regular use of beta-blockers at 6 months follow-up independently associated with improved survival.

Our study has some limitations. Firstly, this was a single-center hospital-based study of modest sample size, which could limit the generalization of our findings. Secondly, natriuretic peptides, other biomarkers and genetic markers, which would have provided more information, were not assessed due to paucity of funding. Finally, the duration of follow-up was modest due to paucity of funding, and a longer duration could have provided more information. Still the study has provided important information from a population with the highest burden of PPCM in the world, which could be used to justify and guide future larger studies on the subject.



Weeks	4	8	12	16	24
PPCM	62	61	60	59	57
Female DCM	57	52	51	49	47

Figure 2. Kaplan-Meier survival curves showing the survival pattern of female patients with dilated cardiomyopathy (DCM) *versus* peripartum cardiomyopathy (PPCM) over a maximum of 24-weeks follow-up (p=0.073).

Table 4. Determinants of mortality in female dilated cardiomyopathy patients.

Variables	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Heart rate, bpm (mean 102)	1.04 (1.01-1.07)	0.019*	1.04 (1.01-1.07)	0.013*
LVEF <25%	1.95 (0.59-6.38)	0.272	-	-
Complete LBBB	1.07 (0.28-4.01)	0.926	-	-
Anticoagulation	0.31 (0.09-1.01)	0.052	-	-
Pericardial effusion	6.22 (1.89-20.53)	0.003*	6.12 (1.60-23.45)	0.008*
Loop diuretics	0.70 (0.01-0.63)	0.017*	0.04 (0.00-0.46)	0.010*
Beta-blockers	0.17 (0.04-0.78)	0.023*	0.31 (0.06-1.50)	0.144
Digoxin	1.52 (0.44-5.19)	0.506	-	-
MRA	0.33 (0.04-2.66)	0.286	-	-
Obesity	1.60 (0.21-12.50)	0.654	-	-
SBP <90 mmHg	2.50 (0.73-8.54)	0.144	-	-
Serum creatinine, µmol/L	1.01 (1.00-1.01)	0.068	-	-
Hyponatremia	1.64 (0.50-5.37)	0.415	-	-

HR, hazard ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; LBBB, left bundle branch block; MRA, mineralocortocoid receptor antagonist; SBP, systolic blood pressure. *Satistically significant.

Table 5. Determinants of mortality in peripartum cardiomyopathy patients.

Variables	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Heart rate, bpm (mean 102)	0.98 (0.95-1.95)	0.116	-	-
LVEF <25%	1.94 (0.32-11.62)	0.468	-	-
Complete LBBB	21.12 (0.00-6.92)	0.785	-	-
Anticoagulation	0.12 (0.02-0.69)	0.018*	0.19 (0.03-1.25)	0.84
Pericardial effusion	1.35 (0.15-12.09)	0.788	-	-
Beta-blockers	0.14 (0.02-1.26)	0.079	-	-
Digoxin	0.82 (0.09-7.35)	0.861	-	-
BMI, kg/m^2	0.92 (0.76-1.11)	0.402	-	-
SBP <90 mmHg	13.84 (2.31-82.98)	0.004*	9.09 (1.40-59.05)	0.021*
Serum creatinine, µmol/L	1.01 (0.99-1.02)	0.372	-	-
Hyponatremia	1.04 (0.17-6.23)	0.965	-	-

HR, hazard ratio; CI, confidence interval; LVEF, left ventricular ejection fraction; LBBB, left bundle branch block; BMI, Body mass index; SBP, systolic blood pressure. *Satistically significant.

Conclusions

Our results suggest that PPCM and DCM differ in some important sociodemographic and clinical characteristics, and predictors of LVRR and mortality. Women with DCM were older, and had higher frequency of AF, renal dysfunction, LV dyssynchrony and larger cardiac chambers than PPCM patients. However, the rates of LVRR, HF hospitalization and survival were not significantly different between the 2 groups.

Clinical perspectives

PPCM and DCM are important etiologies of HF in Nigeria and many other regions of the world. Their morphologic features were considered similar, but details of the other differences including clinical outcomes were not clear. The present study has made a prospective head-to-head comparison between them and found that PPCM patients were younger, had better education and tended to live in rural areas. However, DCM patients had higher frequency of AF, renal dysfunction and LV dyssynchrony, and larger cardiac chambers. The rates of LVRR and mortality were not significantly different between the 2 groups, although the rates were numerically higher among the female DCM patients. Different factors predicted LV functional recovery and mortality in the 2 groups. This study further supports the argument that DCM and PPCM are distinctly different disease entities. A larger multisite study with a larger sample size is needed to further clarify the differences between them.

Contributions

BM, NAI and KMK have all made the following contributions in the preparation of the manuscript: substantial contributions to the conception and design of the work, and the acquisition, analysis and interpretation of data for the work; drafting the work and reviewing it critically for important intellectual content; final approval of the version sent; agreement to be ac-

countable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Conflict of interest

The authors declare no potential conflict of interest.

Ethical statement

The study protocol was approved by the Aminu Kano Teaching Hospital, Kano, Health Research Ethics Committee (reference: AKTH/MAC/SUB/12A/P-3/VI/2891). Subjects were enrolled into the study after obtaining written informed consent. The study conformed to the ethical guidelines of the Declaration of Helsinki on the principles for medical research involving human subjects. All data and supporting materials have been provided in the results section, but further request could be sent to the authors.

References

1. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail 2010;12:767-78.
2. Karaye KM, Yahaya IA, Lindmark K, Henein MY. Serum selenium and ceruloplasmin in Nigerians with peripartum cardiomyopathy. Int J Mol Sci 2015;16:7644-54.
3. Karaye KM, Ishaq NA, Saidu H, Balarabe SA, Talle MA, Isa MS. et al. Incidence, clinical character-istics, and risk factors of peripartum cardiomyopathy in Nigeria: results from the PEACE registry. ESC Heart Failure 2020;7:236-44.
4. Pinto YM, Elliott PM, Arbustini E, Adler Y, Anastasakis A, Böhm M, et al. Proposal for a revised definition of dilated cardiomyopathy, hypokinetic non-dilated cardiomyopathy, and its implications for clinical practice: a position statement of the ESC working group on myocardial and pericardial diseas-es. Eur Heart J 2016;37:1850-8.
5. Sliwa K, Damasceno A, Mayosi BM. Epidemiology and etiology of cardiomyopathy in Africa. Circulation 2005;112:3577-83.
6. Hershberger RE, Hedges DJ, Morales A. Dilated cardiomyopathy: the complexity of a diverse genetic architecture. Nat Rev Cardiol 2013;10:531-47.
7. Karaye KM, Saidu H, Habib AG. Peripartum and other cardiomyopathies in a Nigerian adult popula-tion. Int J Cardiol 2011;147:342-3.
8. Dec GW, Fuster V. Idiopathic dilated cardiomyopathy. N Engl J Med 1994;331:1564-75.
9. Lu C, Lee W, Wu M, Chen S, Yeh J, Cheng C, et al. Comparison of clinical outcomes in peripartum cardiomyopathy and age-matched dilated cardiomyopathy. Medicine 2017;96:19.
10. Petryka-Mazurkiewicz J, Kryczka K, Mazurkiewicz Ł, Miłosz-Wieczorek B, Śpiewak M, Marczak M, et al. Cardiovascular magnetic resonance in peripartum cardiomyopathy: comparison with idiopathic dilated cardiomyopathy. Diagnostics (Basel) 2021;11:1752.
11. World Medical Association Declaration of Helsinki. Ethical principles for medical research involving human subjects. J Postgrad Med 2002;48:206-8.
12. Karaye KM, Lindmark K, Henein MY. One-year survival in Nigerians with peripartum cardiomyopa-thy. Heart Views 2016;17:55-61.
13. Masci PG, Schuurman R, Andrea B, Ripoli A, Coceani M, Chiappino S, et al. Myocardial fibrosis as a key determinant of left ventricular remodeling in idiopathic dilated cardiomyopathy. Circ Cardiovasc Imaging 2013;6:790-9.
14. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1-39.
15. Kadish AH, Buxton AE, Kennedy HL, Knight BP, Mason JW, Schuger CD, et al. ACC/AHA clinical competence statement on electrocardiography and ambulatory electrocardiography: a report of the American College of Cardiology/American Heart Association/American College of Physicians Ameri-can Society of Internal Medicine Task Force on Clinical Competence (ACC/AHA Committee to de-velop a clinical competence statement on electrocardiography and ambulatory electrocardiography). J Am Coll Cardiol 2001;38:2091-100.
16. Karaye KM. Right ventricular systolic function in peripartum and dilated cardiomyopathies. Eur J Echocardiogr 2011;12:372-4.
17. Karaye KM, Shehu MN, Ngantcha M, Bonny A, Awad AM. Peripartum cardiomyopathy: a review article. West Afr J Med 2023;40:104-13.
18. Getz KD, Lewey J, Tam V, Irizarry OC, Levine LD, Aplenc R, et al. Neighborhood education status drives racial disparities in clinical outcomes in PPCM. Am Heart J 2021;238:27-32.
19. Merlo M, Pyxaras SA, Pinamonti B, Barbati G, Di Lenarda A, Sinagra G. Prevalence and prognostic significance of left ventricular reverse remodeling in dilated cardiomyopathy receiving tailored medical treatment. J Am Coll Cardiol 2011;57:1468-76.
20. Fett JD, Christie LG, Carraway RD, Murphy JG. Five-year prospective study of the incidence and prognosis of peripartum cardiomyopathy at a single institution. Mayo Clin Proc 2005;80:1602-6.
21. Blauwet LA, Libhaber E, Forster O, Tibazarwa K, Mebazaa A, HilfikerKleiner D, et al. Predictors of outcome in 176 South African patients with peripartum cardiomyopathy. Heart 2013;99:308-13.
22. McNamara DM, Elkayam U, Alharethi R, Damp J, Hsich E, Ewald G, et al. clinical outcomes for peripartum cardiomyopathy in North America: results of the IPAC study (Investigations of Pregnan-cy-Associated Cardiomyopathy). J Am Coll Cardiol 2015;66:905-14.
23. Moulig V, Pfeffer TJ, Ricke-Hoch M. Long-term follow-up in peripartum cardiomyopathy patients with contemporary treatment: low mortality, high cardiac recovery, but significant cardiovascular comorbidities. Eur J Heart Fail 2019;21:1534-42.
24. Karaye KM, Sa'idu H, Balarabe SA, Ishaq NA, Adamu UG, Mohammed IY, et al. Clinical features and outcomes of peripartum cardiomyopathy in Nigeria. J Am Coll Cardiol 2020;76:2352-64.
25. Sliwa K, Forster O, Libhaber E. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. Eur Heart J 2006;27:441-6.