Clinical Investigations

Clinical Profile and Predictors of Complications in Peripartum Cardiomyopathy

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ABSTRACT

Background: Clinical profile and predictors of major adverse events (MAE) associated with peripartum cardiomyopathy (PPCM) have not been characterized.

Methods and Results: A retrospective review and analysis of clinical data of 182 patients with PPCM. Forty-six patients had ≥1 MAE, including death (13), heart transplantation (11), temporary circulatory support (4), cardiopulmonary arrest (6), fulminant pulmonary edema (17), thromboembolic complications (4), and defibrillator or pacemaker implantation (10). Diagnosis of PPCM was delayed ≥1 week in 48% of patients with MAE that preceded the diagnosis in 50% of these patients. Seven (32%) of the surviving patients who had MAE and did not undergo heart transplantation had residual brain damage. Significant predictors of MAE were: left ventricular ejection fraction ≤25% (HR 4.20, CI 2.04−8.64) and non-Caucasian background(HR 2.16, CI 1.17− 3.97). These predictors in addition to diagnosis delay (HR 5.51, CI 1.21−25.04) were also associated with death or heart transplantation.

Conclusions: 1. PPCM may be associated with mortality or severe and lasting morbidity. 2. Incidence of MAE is higher in non-Caucasians and in women with left ventricular ejection fraction ≤25%. 3. Diagnosis of PPCM is often delayed and preceded by MAE. 4. Increased awareness of PPCM is required for early diagnosis and aggressive therapy in an attempt to prevent complications. (*J Cardiac Fail 2009;15:645−650*)

Key Words: Peripartum cardiomyopathy, complications, predictors, outcomes.

Peripartum cardiomyopathy (PPCM) is a cardiomyopathy of unknown cause that occurs during or after pregnancy.[1] Although this disease is relatively uncommon, there has been an increase in its incidence over time.[2] Reports on outcome of patients with PPCM have been conflicting[2−5] with relatively favorable outcome described by Amos et al,[4] who reported no mortality in 55 patients with PPCM followed in 1 institution and by Brar et al,[5] who found a mortality rate of only 3.3% over 4.7 years of follow-up in the Southern California Kaiser health care system. At the same time, however, severe complications have often been reported, and this condition accounts for an increasing proportion of reported pregnancy-related death.[1,6−9] In addition, a recent report by Berg et al suggested that a substantial number of reported deaths from pregnancy-associated cardiomyopathy could be prevented.[10,11]

Because of the relatively low incidence of PPCM in the United States, which has been reported as 1 of 4075 deliveries in a recent study,[5] the clinical profile of potential complications and their risk factors have not been well described. The purpose of this study was, therefore, to define the clinical characteristics of major adverse events (MAE) and attempt to identify risk factors for such events in patients with PPCM.

Methods and Patients

Medical records of 182 patients diagnosed with PPCM were reviewed for the purpose of this study. Relevant information was collected through review of records as well as interviews of the patients

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Manuscript received December 25, 2008; revised manuscript received March 24, 2009; revised manuscript accepted March 26, 2009.

The authors have no conflicts of interest.

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1071-9164/$ - see front matter

doi:10.1016/j.cardfail.2009.03.008

and/or their referring physicians. The diagnosis of PPCM was based on the following criteria: 1. Development of congestive heart failure during pregnancy or the first 5 months after delivery; 2. Absence of an identifiable cause for cardiac failure; 3. Absence of recognizable heart disease before pregnancy; and 4. Left ventricular systolic dysfunction with left ventricular ejection fraction (LVEF) $< 45\%$.[1]

Major adverse events were defined as either death or complications that were life threatening or resulted in long-term morbidity. These included heart transplantation, temporary circulatory support, cardiopulmonary arrest, or pulmonary edema at presentation that required intensive care, thromboembolic complications, ventricular arrhythmias leading to implantation of implantable cardioverter defibrillator (ICD), or bradyarrhythmias leading to implantation of pacemakers.

Statistical Analysis

Summary statistics for numerical variables were presented as mean ± SD. Summary statistics for categorical variables were presented as count (percent). Two-group differences on continuous outcome measures were assessed by the *t*-test or the Wilcoxon 2-sample test, as appropriate. Two-group differences on categorical outcomes were assessed by the Fisher exact test.

Survival curves were estimated by the Kaplan-Meier method. Because event times often were known only to the nearest week, the time-to-event survival models were discrete time models. Variables related to the hazard (risk) of an event were assessed using stepwise multivariable Cox proportional hazards models.

As a predictor of risk, LVEF was used both as a continuous and a dichotomous variable. A cutoff of 25% was used to aid in clinical interpretation and also provided good balance in the group sample sizes. Hazard ratios with 95% confidence intervals were presented for significant predictors from the Cox models. A P value $< .05$ was regarded as statistically significant. Statistical calculations were performed using the statistical software package SAS, version 9.1 (SAS Institute, Cary, NC).

Results

Clinical Characteristics of all PPCM Patients

Mean age was 29 ± 7 years; 56% were white, 29% black, 10% Hispanic, 3% Asian, and in 2%, ethnic background was unknown. Mean follow-up was 19 ± 25 months (range, 0 to 168). Diagnosis was established antepartum in 23% and postpartum in 77%. Mean parity was 2.1 ± 1 and gravidity 2.5 ± 2.1. History of hypertension during pregnancy was obtained in 43% of the patients. Twin or triplet pregnancies were reported in 15%. Mean LVEF was 29 ± 11%. Recovery of LV function (LVEF ≥50%) at ≥6 months follow-up was observed in 49% of 145 patients. Information on time of onset of symptoms was available in 122 patients and was ≥1 week (mean, 2.3 ± 4.4 weeks) before diagnosis in 59 patients (48%).

Major Adverse Events

Forty-six patients (25%) had ≥1 MAE (Table 1). All patients with MAE had severe left ventricular dysfunction with a mean LVEF of 24 ± 10%. Last LVEF in 22 survivors without transplantation who sustained MAE was 38 ± 18% at mean follow-up of 14 ± 18 months (range, 1-108 months); 6 (27%) had complete recovery (LVEF ≥50%).

Table 1. Comparison between clinical characteristics of PPCM patients with and without major adverse events

	No MAE n=136	MAE n=46	p-value
Age (years)	30±6	27±8	0.03
Age > 30 years	53%	42%	0.3
Non-Caucasian	37%	61%	0.005
Parity	2.0±1.6	2.0±2.1	0.3
Multipara	53%	41%	0.3
Twin or triplet pregnancy	19%	4%	0.02
Gestation hypertension	46%	32%	0.2
Tocolytic therapy	18%	17%	1.0
Index pregnancy			0.3
1st	43%	56%	
2nd	16%	14%	
3rd and more	41%	30%	
Caesarian delivery	21%	15%	0.7
Occurrence of symptoms antepartum	30%	41%	0.2
Occurrence of symptoms postpartum	64%	54%	0.3
Diagnosis delay (weeks)	1.7±3.0	3.8±6.1	0.02
Diagnosis delay (≥ 1 week)	48%	60%	0.3
LVEF (%) baseline	31±11	24±10	<0.001
LVEF ≤ 25%	31%	63%	0.001
LVEF (%) at ≥ 6month	47±13	32±14	<0.0001
LV Recovery (LVEF ≥ 50%)	45%	18%	<0.001

PPCM-peripartum cardiomyopathy, MAE-major adverse events, LVEF-left ventricular ejection fraction.
Data are presented as mean ± SD or percentage (%).
P-value for differences between the two groups.

Thirteen patients died, all between day of delivery and 8 years postpartum: 5 (38%) died suddenly, 6 (46%) of progressive heart failure, and 2 of unknown causes. Eleven patients underwent heart transplantation (HTx): within the 6 months after diagnosis in 9 patients, and between 6 and 24 months in 2 patients.

Circulatory support for hemodynamic instability in spite of inotropic therapy was required in 4 patients (intra-aortic balloon pump in 1, left ventricular assist device in 2 and biventricular assist device in 1).

Cardiopulmonary arrest was reported in 6 women, 1 due to ventricular tachycardia, and 5 due to acute respiratory failure. All events occurred early, either during the delivery (3 patients) or within the first 6 days postpartum (3 patients). Anoxic brain damage and slow recovery was reported in 5 patients.

Severe pulmonary edema requiring intensive care occurred in 17 patients: between 1 day before and 7 days after delivery in 16 and at 3 months postpartum in 1 patient.

Thromboembolic events were reported in 4 patients between 5 days and 3 months postpartum. All of them developed left ventricular thrombus, 3 presented with cerebrovascular accident (plus pulmonary embolism in 1), and 2 with leg ischemia requiring amputation in one.

Ventricular arrhythmias leading to ICD implantation were reported in 7 patients (3 of which received also cardiac resynchronization devices); bradyarrhythmias leading to pacemaker implantation were reported in 3.

Eighty-four percent of MAE occurred during the first year (80% at 6 months) after diagnosis of PPCM (Fig. 1A). Similarly, 74% of death or HTx occurred in

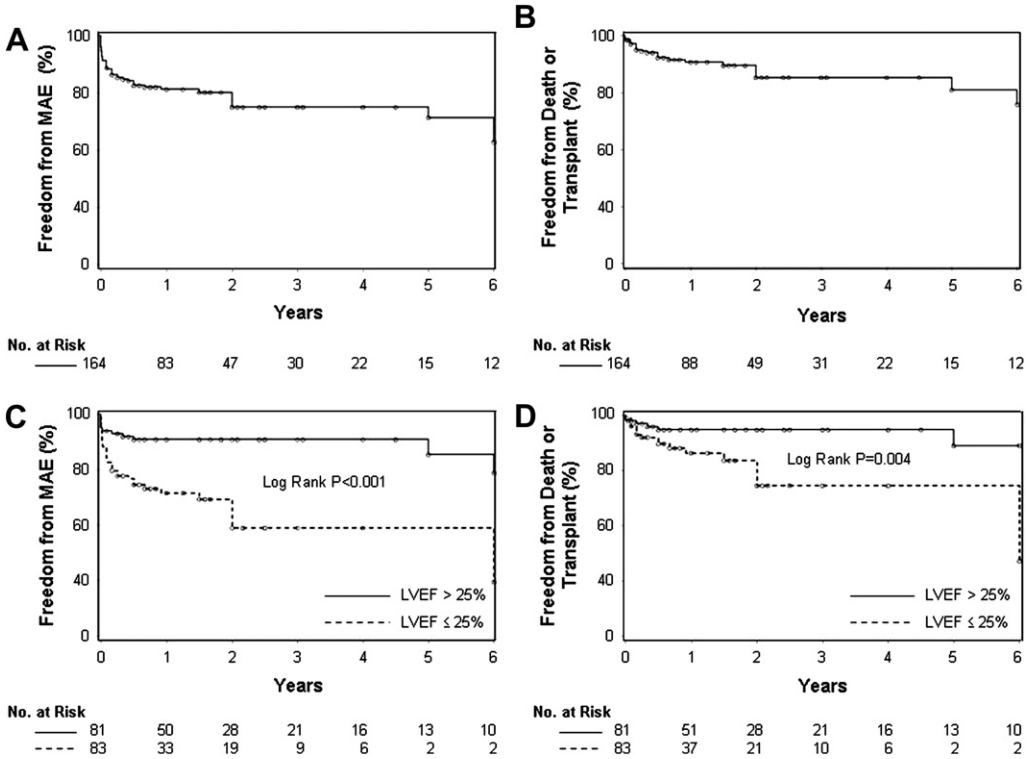

Fig. 1. (A) Kaplan-Meier survival curves demonstrating proportion of patients free from major adverse events (MAE). The numbers below the graph represent the number subjects at risk of the event. (B) Kaplan-Meier survival curves demonstrating proportion of patients free from death or heart transplantation. The numbers below the graph represent the number subjects at risk of the event. (C) Kaplan-Meier survival curves demonstrating comparison of proportion of patients free from major adverse events (MAE) between those with left ventricular ejection fraction (LVEF) ≤25% and >25%. *P* value: for the comparison between the 2 groups. (D) Kaplan-Meier survival curves demonstrating comparison of proportion of patients free from death or heart transplantation between those with left ventricular ejection fraction (LVEF) ≤25% and >25%. *P* value: for the comparison between the 2 groups.

the first year (65% at 6 months) (Fig. 1B). Seven (32%) of the surviving patients with MAE who did not undergo heart transplantation had residual brain damage.

Among all patients with MAE, 3 patients demonstrated deterioration of LV function (3, 8, and 60 months after diagnosis) after complete recovery in 2 and partial recovery (LVEF 45%) in 1.

Comparison between Patients with and without MAE

Patients with MAE were somewhat younger, more likely to be non-Caucasian, had longer delay of diagnosis, and lower incidence of twin pregnancy (Table 1). Women with MAE had lower LVEF and higher incidence of LVEF ≤25% at time of diagnosis. Moreover, a smaller number of patients in this group had complete recovery of LVEF (≥50%). Patients with MAE who survived ≥6 months after the diagnosis had significantly lower LVEF (Fig. 2). Those who died or underwent HTx were somewhat younger, more likely to be non-Caucasians, to have a delay of diagnosis ≥1 week after onset of symptoms, and had a lower LVEF at time of diagnosis (Table 2).

Prediction of MAE

The following variables were evaluated as potential predictors of MAE and of death or transplantation: age, age >30 years, baseline LVEF, baseline LVEF ≤25%, non-

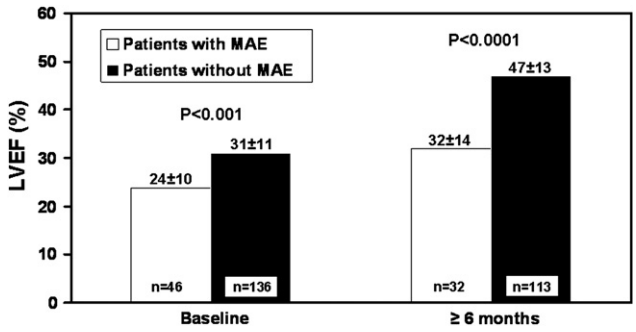

Fig. 2. Comparison of mean (± SD) left ventricular ejection fraction (LVEF) at the time of the diagnosis of peripartum cardiomyopathy between women with and without major adverse events (MAE); and at follow-up ≥6 months between survivors with MAE without heart transplantation and patients without MAE. *P* value: for the comparison between the 2 groups.

Table 2. Comparison between Clinical Characteristics of Patients who Died or Underwent Heart Transplantation and the Rest of PPCM Cohort

	PPCM (n = 158)	PPCM Death/Transplant (n = 24)	*P* Value
Age (y)	29 ± 7	27 ± 8	.03
Age >30 y	53%	34.8%	.2
Non-Caucasian	39%	76%	.001
Parity	2.0 ± 1.7	2.1 ± 2.4	.3
Twin or triplet pregnancy	17%	4%	.1
Gestation hypertension	45%	29%	.5
Tocolytic therapy	17%	13%	.9
Index pregnancy			.2
1st	42%	60%	
2nd	16%	16%	
3rd and more	42%	24%	
Caesarean delivery	23%	9%	.2
Occurrence of symptoms antepartum	27%	46%	.06
Occurrence of symptoms postpartum	64%	46%	.09
Diagnosis delay (weeks)	1.6 ± 3.0	6.6 ± 7.7	<.001
*Diagnosis delay ≥1 week	46%	81%	.004
LVEF (%) baseline	30 ± 11	24 ± 8	.006
LVEF ≤25%	36%	58%	.046

PPCM, peripartum cardiomyopathy; LVEF, left ventricular ejection fraction.

Data are presented as mean ± SD or percentage (%).

P value for differences between the 2 groups.

*in Patients with available information.

Caucasian background, cesarean delivery, multiparity, antepartal onset of symptoms, and delay (≥1 week) of diagnosis from onset of symptoms. Left ventricular end-diastolic diameter was not available in all patients and therefore was not evaluated. The resulting significant predictors of MAE were baseline EF ≤25% (HR 4.20, CI 2.04−8.64 [Fig. 1C]) and non-Caucasian background (HR 2.16, CI 1.17−3.97). The same variables (LVEF ≤25%, HR 5.38, CI 1.87−15.50 (Fig. 1D), and non-Caucasian background, HR 4.78, CI 1.81−12.66), in addition to delay of diagnosis ≥1 week (HR 5.51, CI 1.21−25.04) predicted death or HTx.

Discussion

The present study provides a description of potential MAE associated with PPCM and defines predictors of such complications. Forty-six (25%) of the 182 patients included in the study had ≥1 MAE, which resulted in either mortality, HTx, or significant morbid events that were either life-threatening or had an important and long-lasting effect on quality of life, including cardiopulmonary arrest, thromboembolic events, and the need for a temporary or permanent use of devices. Almost a third of the patients with transplantation-free survival had residual brain damage as a result of cerebrovascular accident or cardiopulmonary arrest.

The results of the present study also show a frequent delay of diagnosis resulting in occurrence of MAE before recognition of PPCM in almost half of the patients. An important reason for delay of diagnosis is probably related to the similarities between clinical manifestations of PPCM and signs and symptoms often accompanying normal pregnancy.[13] At the same time, however, a low level of awareness and failure to routinely think of a cardiac cause for the presenting symptoms has clearly been related to delayed diagnosis in many of the cases. Although it is not possible to infer direct causality, it is possible that some of the complications could have been related to delay of diagnosis. These findings suggest the need for increased awareness of PPCM among health professionals caring for women during and following gestation, to assure timely diagnosis and early therapy that could prevent serious complications. Because the level of blood B-type natriuretic peptide does not show a clinically significant increase during normal pregnancy or the postpartum period,[14] an early measurement of this marker in women who present with worsening dyspnea during pregnancy or the postpartum period should be encouraged for early diagnosis of PPCM.

The likelihood of MAE was found to be higher among non-Caucasian women. This finding supports previous reports indicating a higher relative risk of adverse pregnancy outcome in African-American women as well as Hispanic and Asian women compared with non-Hispanic white women.[10] Higher incidence of complications in non-Caucasian women could be due to genetic or environmental reasons, but could also reflect disparity in access to health care.[15] The most powerful predictor of MAE, however, was the degree of initial myocardial insult manifested by depression of left ventricular function at time of diagnosis. These findings suggest the need for even a closer surveillance and aggressive therapy in patients with PPCM and the previously mentioned risk factors.

Death was reported in 13 patients who did not undergo HTx and in 4 additional patients who died after HTx. Because of a strong possibility of referral bias, these data may not reflect the true incidence of mortality associated with PPCM[2,5]; however, they provide important clinical information regarding timing and mode of death. Mortality often occurred within the first 6 months postpartum and it was sudden in more than one third of the patients. Similar findings were published by Sliwa et al,[8] who reported a mortality rate of 15% in 100 consecutive patients with PPCM diagnosed in South Africa. All death occurred during the first 3 months after the delivery, and 27% of them experienced sudden death. Similar data were also reported by Fett et al,[7] who described a mortality rate of 15% in 98 patients with PPCM diagnosed in Haiti. More then half of the patients died within 60 days after diagnosis and all death was due to cardiac causes, including arrhythmias, progressive heart failure, or thromboembolism. Whitehead et al[6] reported mortality within 6 months after the delivery in 85% of 120 patients who died after developing PPCM.

Risk of MAE was more common in women with lower LVEF at diagnosis as well as in non-Caucasian patients. Early use of effective therapy in these patients may prevent complications and improve outcome. This assumption is supported by a recently reported decrease in pregnancy-related death

due to bleeding, presumably secondary to improvement of care,[10] and by a reports by Amos et al,[4] who showed no mortality in 55 well-treated patients with PPCM over an average follow-up of 43 months in 1 medical center in the United States, and Brar et al, who reported only 3.3% mortality over 4.7 years of follow-up in the Southern California Kaiser health care system.[5] Although guidelines recommendations for the management of other populations of patients with heart failure have not been tested in patients with PPCM, the use of treatment shown to reduce mortality, including angiotensin-converting enzyme inhibitors, β-blockers, and aldosterone receptor antagonists makes good clinical sense except during pregnancy or lactation when drug therapy should be modified to prevent fetal or neonatal toxicity. The decision regarding the use of ICD in patients with PPCM may be more difficult. Because early sudden death is likely in high-risk patients, it is tempting to consider early implantation in such cases. At the same time, however, recent guidelines recommend considerations for ICD in patients with persistent left ventricular dysfunction on chronic optimal medical therapy.[16] These recommendations may be especially applicable to patients with PPCM in whom normalization of left ventricular function within 6 months has been commonly reported.[4,12,17] For this reason, and because recovery of LVEF usually occurs within the first 6 months after diagnosis, it may be advisable to consider the use of a wearable external defibrillator in high-risk patients with PPCM until recovery of left ventricular function occurs or as a bridge for an ICD in those with persistent left ventricular dysfunction in spite of optimal therapy.[18]

Thromboembolic events were reported in 4 patients who were found to have left ventricular thrombus, and 3 had severe embolic complications including cerebrovascular accident, pulmonary embolism, and leg ischemia requiring amputation. Other reports have described high incidence of left ventricular thrombus[4] and thromboembolic events leading to severe complications including mesenteric artery occlusion and myocardial infarction secondary to coronary embolism[19,20]; these cases emphasize the importance of early diagnosis in patients with PPCM and suggest a need for prophylactic anticoagulation in patients with PPCM and severe left ventricular dysfunction.

Very little information is available regarding long-term outcome of patients with PPCM after normalization of LV function. Three of our patients had deterioration of recovered left ventricular function unrelated to a subsequent pregnancy within several months or years after the initial diagnosis. These findings highlight the need for follow-up with annual echocardiographic assessment of left ventricular function in women with PPCM, even after normalization of cardiac function.

It is important to consider the limitations as well as the strengths of the study. The information may have been affected by the retrospective nature of our data collection in many of the patients and the dependency on reports of physicians and patients. These data can be influenced by selection as well as ascertainment bias and are likely to be incomplete. It is, therefore, possible that the outcome of the women included in this study may be worse than that occurring in the community[2,5] because a complication could have prompted the reporting. Additional limitation of the study is related to the fact that echocardiographic data of LVEF were based in many cases on the information of individual physicians as reported in the patients' records. In addition, complete information of pharmacological treatments the patients received was not available. The effect of therapy on the outcome could therefore not be assessed and will need to be evaluated in a prospective study. However, the strengths of the study include its large sample size, which represents the largest database available to date with a wide range of ages and different ethnic backgrounds. In addition, the study provides the most extensive and detailed information on a wide spectrum of potential and clinically important complications associated with PPCM and their predictors. A similar prospective study is presently not possible because of the relative rarity of this condition.

In summary, the present study clearly shows that PPCM can be associated with important complications, which may result in death or severe and long lasting morbidity. Adverse events are more likely to occur in non-Caucasian patients, but are mainly associated with low LVEF ($\leq$25%) at time of diagnosis. Diagnosis of PPCM is often delayed even in symptomatic patients, a fact that may allow the development of MAE. An effort should be made for increased awareness of PPCM among clinicians caring for women during and after pregnancies. Early diagnosis and aggressive therapy including treatment of heart failure, anticoagulation, and sudden death prevention could possibly reduce the incidence of complications in this condition.

Acknowledgment

The authors thank Michele De Robertis for help with preparation of figures.

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