

Peripartum Cardiomyopathy: Evolutionary and Prognostic Aspect in the Cardiology Department of the Ignace Deen National Hospital

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Abstract

Introduction: Peripartum cardiomyopathy is encountered in practice in Guinean cardiology, although it is difficult to have reliable statistics.

Objectives: The aim was to describe the clinical and echocardiographic course and the prognosis for peripartum cardiomyopathy.

Methodology: This was a one-year prospective cohort study from March 1, 2018 to February 28, 2019.

Results: We collected 40 cases of peripartum cardiomyopathy. The hospital frequency was 7.31%. The average age was 28.40 ± 8.22 years with extremes of 16 and 44 years, the most represented group was that of 26 - 35 years. Primiparas were the most represented 32.5%. Clinical signs appeared in the postpartum in 85% of our patients, mean time to onset of symptoms was 52.75 ± 50.1 days. The mode of occurrence was that of global heart failure in the majority of patients in 90%. Echocardiography had shown dilation of the heart chambers associated with left ventricle dysfunction in all patients. Complications were dominated by thromboembolic events (12%). The treatment was that of heart failure.

We reassessed 24 patients, Six (6) cases of death were recorded and 10 patients were lost to follow-up. The evolution was marked by 58.3% of complete recovery. The mortality rate was 25%.

Conclusion: Peripartum cardiomyopathy is common in Guinean cardiology. The obstetrical prognosis is often reserved, good coverage in terms of family planning could be beneficial for the management of recurrences.

Keywords: Peripartum Cardiomyopathy; Heart Failure; Guinea

Introduction

Peripartum cardiomyopathy (PPCM) is "a dilated cardiomyopathy manifesting itself in the peripartum period in a previously healthy patient" [1]. It consists of a systolic dysfunction of the left ventricle with a decrease in the ejection fraction of the left ventricle (LVEF) attested by echocardiography (LVEF < 45%), manifested in the month preceding or the 5 months following childbirth without a etiology. found and excluding any pre-existing heart disease [1,2]. In Guinea, the hospital prevalence is not known. On the other hand, in Africa, there are several studies devoted to this disease [3-6]. Its incidence is variable; it is more common in black women living in Africa [3]. It is potentially serious because it puts the vital prognosis of both mother and child at risk [7]. When the diagnosis is made early and adequate treatment is initiated, half of the patients recover with recovery of a normal ejection fraction in 50% of cases [8-11].

Objective of the Study

The objective of this study was to describe the clinical, echocardiographic and prognosis course of peripartum cardiomyopathy in our department.

Materials and Methods

The Cardiology Department at Ignace Deen National Hospital served as the framework for this study. This was a one-year prospective cohort study from March 1, 2018 to February 28, 2019 in women diagnosed with confirmed peripartum cardiomyopathy. Were included in this study patients hospitalized or followed on an outpatient basis for left heart failure (HF), or global failure occurring in the month preceding or the 5 months following childbirth, without a history of known cardiovascular diseases, nor of etiologies found, having performed at least one cardiac ultrasound showing an alteration of LVEF < at 45%. They agreed to participate voluntarily.

The following echocardiographic parameters were analyzed at inclusion of patients and during follow-up under medical treatment: ventricular dilation, segmental kinetics, ejection fraction measured by the Simpson biplane method, filling pressures, presence or not intracardiac thrombi, the degree of pulmonary arterial hypertension.

Conduct of the investigation

It took place in two distinct phases:

- **A first phase:** For a period of 6 months (the first 6 months), from March 1 to August 31, 2018. During this phase, for all patients, the clinical examination data, the echocardiography data as well as the treatment were noted.
- **A second phase:** Lasting 6 months (the last 6 months of the study), it took place from September 1, 2018 to February 28, 2019. During this phase, we recalled and reassessed all the patients followed during the first phase, except for deceased patients and those who were lost to follow-up.

The progress of our patients was assessed in two ways:

- **Good evolution:** Asymptomatic patients with complete recovery of LVEF greater than or equal to 55% or between 50 - 54% on cardiac ultrasound during reassessment.
- **Bad evolution:** Symptomatic patients with persistence of an altered LVEF less than 50%, with refractory heart failure or death. The epidemiological, clinical, and paraclinical data collected during the survey were classified into two variables: qualitative and quantitative. The data was collected on a pre-established survey form. Our precoded data was collected in Epi-Data version 3.1, then we exported it to version 21 of the SPSS (Social Science Statistical Package) software for processing and analysis. For statistical calculations we used Fisher's test and it was considered significant for a P value less than 0.05.

Results

We collected 40 cases of CMPP during this study, including 22 cases followed on an out patient basis, and 18 cases of hospitalization out of a total of 246 hospitalized patients, or a hospital frequency of 7.31%. The mean age of our patients was 28.40 ± 8.22 years with extremes of 16 and 44 years. Housewives were the most represented at 62.5%. The first-time mothers were the most represented at 32.5%, followed by the pauciparas 27.5%. Twinning was observed in 17.5% of cases. The medical history was represented by high blood pressure during pregnancy (10%). Clinical signs appeared postpartum in 85% of cases, with a mean time to onset of 52.75 ± 50.1 days. Heart failure was the circumstance of discovery 90% of the time. The functional signs at the initial phase were dominated by the dyspnea

which was present in all our patients with 65% of cases of stage 4 dyspnea, on the other hand at the re-evaluation a clear regression of the functional signs was observed. Persistence of dyspnea was noted in only 29.1% of patients of which 20.8% were stage 2, no case of stage 4 dyspnea was observed. Cough and abdominal pain also decreased from 95% to 92.2% respectively at initial examination, to 8.3% and 4.2% at reassessment. The table 1 below indicates the epidemiological elements and clinical signs.

Epidemiological elements and clinical signs Initial assessment		Average or % values	
		Revaluation	
Middle age		28,40 ± 8,22 years	
Twinning		17,5%	
Primiparas		32,5	
Presence of LV failure		10	
Presence of RV failure stage		90	
Dyspnea	2	2, 5	20,8
	3	32,5	8,3
	4	65	00
Stroke		5	
Embolism Pulmonary		7,5	
Atrial Fibrillation		5	

Table 1: Distribution of patients according to epidemiological elements and clinical signs.

LV: Left Ventricular; RV: Right Ventricular.

At the initial phase the cardiac echo-Doppler objectified a dilation of the cardiac chambers in 100% of our patients, the filling pressures were high in 52.5%, the kinetic disturbances were present in the majority of cases (the hypokinesia in 92.5% of cases, akinesia in 2.5%, and dyskinesia in 65%). Pulmonary arterial hypertension (PAH) was observed in 76.7% of our patients. Longitudinal systolic function of the right ventricle (RV) was impaired in 40% of cases. Pericardial effusion was present in 12.5% of patients. Twelve point five percent (12.5%) of our patients had intracavitary thrombus and 5% spontaneous intra-LV contrast. The ejection fraction was altered in all of our patients, with severe alteration in 47.5% and moderate in 52.5%. We reassessed 24 patients. Cardiac cavity dilation persisted in 41.7% of patients, filling pressures normalized in 87.5% of patients. Hypokinesia was noted in 41.7% of our patients. The PAH was normalized in 83.3% of cases and the longitudinal systolic function of the RV in 91.7% of our patients. No case of pericardial effusion was observed. The thrombi had persisted with a single case of spontaneous contrast. The table 2 below summarizes the echocardiographic parameters.

Ultrasound parameters		Average values or%	
		Initial assessment	Revaluation
Dilation of the heart chambers		100	41,7
End-diastole LV		61,33% 56 - 76 (Extreme)	56,63 43 - 76 (Extreme)
End-systole LV		55,80% 30 - 7 (Extreme)	51,27 29 - 76 (Extreme)
LA		42,15% 26 - 50 (Extreme)	37,94 23 - 53 (Extreme)
RV		32,20% 20 - 42 (Extreme)	27,70 19 - 4 (Extreme)
RA		21,76% 12 - 30 (Extreme)	19,50 10 - 46 (Extreme)
LV contractile function	Hypokinesia	92,5%	41,7
	Akinesia	2,5%	4,2
	dyskinesia	65%	00

Functional valve disease	Mitral insufficiency	100%	41,7
Tricuspid insufficiency	82,5%	62,5	
Impaired RV systolic function		40%	8,3
Intracavitary thrombus		12,5%	00
Spontaneous contrast		5%	4,2
Ejection fraction	Normal	00%	33,33
	> 45%	00%	25
	Between 30 - 44%	52,5%	29,2
	< 30%	47,5%	12,5
Normal Pulmonary arterial hypertension		00%	83,3
High filling pressures		52,5%	12,5
Pericardial effusion		12,5%	00

Table 2: Distribution of patients according to ultrasound parameters.
 LA: Left Atrial; RA: Right Atrial.

Thromboembolic events were dominated by pulmonary embolism in 7.5% of cases followed by stroke in 5% of cases. Atrial fibrillation (AF) was present in 5% of our patients and acute lung edema (OAP) in 10%. The treatment was mainly that of heart failure: sodium diet, loop diuretic, anti-aldosterone, ACE inhibitor/ARB II, and after the acute phase beta blockers. Patients with intracavitary thrombi or spontaneous contrasts had benefited from curative anticoagulation based on anti-vitamin K (AVK), and iron treatment in cases of anemia. The poor prognosis factor linked to death found in our study with a statistically significant test ($p = 0.001$) was the dilation of the heart chambers (Table 3).

Variables	Death (%)	P
Age groups		
16 - 25	50%	0,089
26 - 35	33,3%	
36 - 45	16,7%	
Parity		
Primiparas	33,3%	0,26
Pauciparas	16,7%	
Multiparas	33,3%	
Large multiparas	16,7%	
Twinning	00%	1,49
Dilation of the heart chambers	15%	0,001

Table 3: Distribution of patients according to prognostic factors.

The mean follow-up time was 6 to 12 months \pm 3 months. Only 24 patients were reassessed. Of the 40 patients initially, there were 6 deaths and 10 lost to follow-up. The course was marked by a cure rate of 58.3%, incomplete remission in 16.7% of cases. The death rate was 25% (Table 4).

Evolution	Workforce	Percentages
Healing	14	58,3
Death	6	25
Incomplete remission	4	16,7

Table 4: Distribution of patients according to evolution.

Discussion

Epidemiological data

We recorded a hospital frequency of peripartum cardiomyopathy of 7.31%. The average age of our patients was 28.40 ± 8.22 years with extremes of 16 and 44 years the most represented age group was 26 - 35 years. The risk of developing the disease increases with age [12]. Primiparas were the most represented in our study, i.e. 32.5%, followed by pauciparas and multiparas, respectively 27.5% and 17.5%. Kane Ad., *et al.* in Senegal, in their study, found a frequency of 48% of first-time mothers [4]. Although most often described in multiparas, CMPP is also found in first-time mothers.

Initial clinical and echocardiographic data

The clinical signs appeared in the postpartum period in 85% of the patients of which more than half 52.5% appeared within a few days following childbirth up to the 3rd month of the postpartum period, the average onset was 52.5%, 75 ± 50.1 days. Our results corroborate with data from the literature [4,12]. The mode of onset was that of global heart failure in the majority of cases, (90%). This is described by Garg J., *et al* [12]. Global heart failure was found in 69.2% of patients in the study by Kane A., *et al.* in 2001 [5]. All of our patients had dyspnea. It was class 4 dyspnea in 65% in this study. Dyspnea is constant in this condition. In the study by Pio M., *et al.* dyspnea was class 3 in 41.7% and class 4 in 27.1% [6]. Clinical symptoms appeared postpartum in 85% of cases, with a mean time to onset of 52.75 ± 50.1 days prior to diagnosis. This delay is longer than that of Pio M., *et al* [6]. Our patients are often seen in consultation at an advanced stage of the disease; they have a precarious economic situation and often come from the suburbs of the capital or from the interior of the country. However, the only cardiology centers are in Conakry, near the urban center. This explains why the discovery of the disease sometimes at the complication stage: thromboembolic events were dominated by pulmonary embolism in 7.5% of cases, followed by stroke in 5% of cases. Atrial fibrillation (AF) was present in 5% of our patients and acute pulmonary edema (APE) in 10%. Echocardiography was performed on all of our patients. Cardiac chamber dilation associated with LV systolic dysfunction was consistent in all patients. The size of the left ventricle was 61.33 mm in end-diastole and 55.8 mm in end-systole, in our study. The ejection fraction was altered in all of our patients, with several alteration in 47.5% and moderate in 52.5%. Systolic dysfunction is a fundamental part of peri-partum cardiomyopathy. Several studies have underlined it [4-6,13].

Treatment

The treatment was mainly that of IC: sodium diet, loop diuretic, anti-aldosterone, ACE inhibitor/ARB II and after the acute phase beta blockers. Patients with intracavitary thrombi or spontaneous contrasts had benefited from curative anticoagulation based on anti-vitamin K (AVK), and iron treatment in cases of anemia. Routine anti-coagulation is not recommended for all peri partum cardiomyopathies but should be considered when the left ventricular ejection fraction is less than or equal to 35% [13]. Treatment with bromocriptine was not available. Prevention was based on spacing births with the use of contraception. Subsequent pregnancies were strongly discouraged in patients with impaired left ventricular ejection fraction.

Evolutionary and prognostic aspects

The re-evaluation of the patients was made on average after a delay of 14 ± 3 months. The cure rate in our study was 58.3%. This follow-up time was 32.7 ± 16.1 months in the study by Pio M., *et al.* [6]; between 1 and 18 months for Kane A., *et al.* [5] with respectively 39.6% and 42.3% cure. On re-evaluation: we observed a marked improvement in clinical signs under treatment, with significant regression of congestive signs. The functional signs persisted in 20.3% of our patients with class 2 dyspnea; no case of class 4 dyspnea was found. The evolution was marked by the persistence of the dilation of the cardiac chambers in 10 patients or 41.7%, a slight alteration of LVEF in 25%. However, LV dilation regressed from 61.3% to 56.6% on average. This persistent LV dilation has been reported by other African authors (6, 14). Peri partum cardiomyopathy is curable; about 50% recover a normal EF of LV [12]. The complete cure rate was 58.3%. This rate is slightly higher than that of Kane A., *et al.* (42.3%) and that of Pio M., *et al.* (39.6%). This difference could be explained by those lost to follow-up in our study, which represented 25% (10 out of 40 patients). We noted 5 cases of intracavitary thrombi whose evolution was favorable under effective anticoagulation. The poor prognosis factors were: dilation of the heart chambers and a collapsed left ventricular ejection fraction ($\leq 35\%$) ($p = 0.001$). The mortality rate in our study was 25%. The diagnostic delay, the severity of the ventricular dysfunction, the therapeutic non-compliance by drug disruption due to the low economic level explain this high mortality rate. Pio M., *et al.* reports a mortality of 8.3% [12].

Conclusion

Peripartum cardiomyopathy is a common cardiomyopathy in our context. It frequently occurs postpartum, often in multiparas. Heart failure remains the most common circumstance of discovery. The condition is curable if the diagnosis is made early followed by optimal management. Mortality is high in our study (25%) due to late diagnosis with severe impairment of left ventricular function. Prevention is based on family planning, screening for the disease by gynecologists, cessation of reproduction for patients who remain symptomatic with an impaired left ventricular ejection fraction.

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