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Research Article

Evolutionary Etiopathogenic Particularities of Peripartum Cardiomyopathy in a Disadvantaged Environment in Brazzaville

Solange Flore Mongo Ngamani[1], Kivie Mou-moue Ngolo Letomo[2, *], Christian Michel Kouala Landa[1], Eric Gibrel Kimbally Kaky[1], Rog Paterne Bakekolo[2], Synthiche Okoko[2], Gankama Thibault Naibe[2], Jospin Karel Makani Bassoukouahou[2], Fikahem Ellenga Mbolla[1]

[1]Health Sciences Faculty, Marien Ngouabi University, Brazzaville, Republic of Congo
[2]Cardiology B Department, Hospital Teaching of Brazzaville, Brazzaville, Republic of Congo

Abstract

It was carried out a study on peripartum cardiomyopathy (PPCM) in the cardiology and internal medicine department of the University Hospital Center of Brazzaville. This work takes place from May 1, 2019 to April 31, 2023. It had aimed at identifying the profile of peripartum cardiomyopathy in women disadvantaged in cardiovascular diseases. Fifty-two files were selected on the basis of predefined inclusion criteria. The frequency of myocardiopathy peripartum was estimated at 1.4% of admissions and 9.7% of women of childbearing age, or 13 cases per year. The average age of the patients was 30.78.02 (range: 16 to 44 years), the most frequently found risk factors were respectively high low level socio-economic (77%), multiparity (36%); pregnancy-induced hypertension (32%) and anemia (31%). clinical picture was stereotypical and included signs of heart failure. This one was global in thirty-nine (39) cases (75%), left in thirteen (13) cases (25%), cardiomegaly was noted in all cases with mean cardiothoracic ratio at 0.614 (range 0.52 to 0.83). Sinus tachycardia was observed in fifty-one 51 cases (98%). Left atrial dilatation was noted in twenty-three 23 cases (44.2%), left atrial dilatation was noted in thirteen (13) cases (25%). Diffuse disorders of the repolarization were noted in forty-three cases (83%). Echocardiography revealed: left cavitary dilatation in 100% of cases; thrombosis left ventricular intravenous was noted in two cases (4%).

Keywords

Cardiomyopathy, Peripartum, Brazzaville

1. Introduction

Peripartum cardiomyopathy (PPCM), or Meadows syndrome, is a condition rare in Europe [1] but quite common in underdeveloped countries and in particular Black Africa [2, 3]. The first observations of PPCM were reported by Ritchie at Oldenburg in 1849 [1, 4], and Virchow in 1870. The first precise description is due to Hull [1].

*Corresponding author: ngololetom@gmail.com (Kivie Mou-moue Ngolo Letomo)

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PPCM is rare in the United States, Canada, and Europe. In the US, PPCM is diagnosed in 1 in every 1,000 to 1 in every 4,000 deliveries. The number of patients diagnosed with PPCM appears to be increasing over time. PPCM may be more common in other countries such as Haiti, Nigeria, and South Africa. In 1937 (27 cases) and 1938 (80 cases). But the reference description is that made by Meadows [5] who defines the disease as "heart failure that occurs in the peripartum". Since then, the author's name has been given to the disease.

In 2010, the European Society of Cardiology described PPCM as an idiopathic cardiomyopathy with the following characteristics:

1. Development of heart failure toward the end of pregnancy or in the postpartum period.
2. Absence of another identifiable cause of the heart failure.
3. Left ventricular systolic dysfunction with a left ventricle (LV) ejection fraction nearly always less than 45 percent. The LV may or may not be dilated.

PPCM is of great scientific interest because of its prevalence in our regions and cause of its etiopathogenesis which remains unknown until now. Our study aims at the following objectives:

1) Determine the place of PPCM within cardiovascular diseases and heart failure.
2) Document the socio-economic characteristics of patients presenting this affection.
3) Document the risk factors for the disease.
4) Identify the epidemiological, ethiopathogenic and paraclinical characteristics therapeutic and evolving PPCMs.

2. Materials and Methods

2.1. Type, Setting and Period of Study

This study was carried out in the cardiology department B and internal medicine of the center Brazzaville University Hospital (CHUB). This was a retrospective study going from May 1, 2019 to April 31, 2023, a period 4 years old. We proceeded to read the entry registers longitudinally. We thus inventoried the admitted patients and selected the PPCM files.

During the reference period we listed 82 women with PPCM. Only 52 files were selected based on the criteria defined above.

A. Socio-economic categorization of patients

According to the standards used by the Congolese administration classifying patients into four socio-economic categories (CSE): we only retained the last two classes representing disadvantaged patients.

1) Categories I: salary with pay index varying between 830 and 2000 of the Congolese civil service: these are senior executives
2) Category II: employees with a salary index varying between: 530 and 820: these are middle managers
3) Category III: monthly income less than 530: these are workers and small employees
4) Category below IV: these are the destitute, often without social assistance.

B. Definition of concepts

Peripartum cardiomyopathy (PPCM) is a serious condition and is defined as a dilated cardiomyopathy which meets three necessary criteria: occurring during the period peripartum, no history of cardiovascular disease, no cause obvious. Symptoms appear between the last trimester of pregnancy and the fifth first months following childbirth [1, 6, 7]. In addition, it meets the criteria characteristics of primary dilated cardiomyopathy defined by the Organization World Health [1] namely:

1) An unknown origin (idiopathic non-obstructive, non-ischemic).
2) A more or less significant dilation of one of the two ventricles.
3) Impairment of systolic function.
4) A progression towards heart failure.
5) A risk of death at all stages of the disease

2.2. Inclusion Criteria

The selected files met the following criteria:

1) Existence of signs of heart failure in the last trimester of pregnancy, during childbirth or during the five months following childbirth in disadvantaged patients.
2) Absence of known heart disease prior to pregnancy
3) Absence of a classic etiology that could lead to cardiac decompensation during the peripartum period
4) A medical file including: obstetric clinical data from the gynecology department of the Brazzaville University Hospital and that of cardiology and internal medicine. Biological examinations, chest x-ray, electrocardiogram and echocardiography/Doppler

2.3. Echocardiography Criteria

Dilation of at least the left ventricle (DTDLV> 32mm/m 2 of body surface area) associated with left ventricular systolic dysfunction, i.e. a lower left ventricular ejection fraction (LVEF) of 0.45 and/or or a shortening fraction < 30%.

2.4. Inclusion and Exclusion Criteria

It has included in these case series all patients with a low socio-economic level who presented the PPCM criteria. Those excluded from the study are patients with a high socio-economic level, patients who did not have an echocardiogram. Patients with a known cardiac condition prior to pregnancy.

2.5. Parameters Analyzed

This study looked for parameters likely to identify the profile of patients with PPCM, particularly in the following

aspects: epidemiological (age, socio-economic, consultation time, obstetric history, course of pregnancy); Radiological, electrocardiographic, echo cardiographic, therapeutic and progressive clinics.

2.6. Statistical Analysis

The study used the mean of the standard deviation, the variance for the calculation of the mean age and measurements, and the chi-square test in the Epi-info 6.04 software for the comparisons. The significance threshold retained was 0.05.

3. Results

3.1. Epidemiological Aspects

3.1.1. Frequency

Out of a total of 1856 patients admitted to the department during the study period for cardiovascular disease, 52 cases of PPCM occurring in disadvantaged women were listed, i.e. a frequency of 2.8% of cardiovascular diseases and a frequency of 9.7% of 534 women of childbearing age.

This frequency of PPCM in disadvantaged areas is estimated at 13 cases per year.

3.1.2. Age

The patients were distributed by age groups as shown in Table 2.

Table 1. *Different age groups.*

Age (year)	N	%
11-20	8	15,3
21-30	12	23
31-40	29	55,7
≥ 40	3	6
TOTAL	52	100%

The average age of the patients was 30.7 ±8.2 (range 16 to 44 years).

The age group of 31 to 40 years was the most affected (n = 32 cases or 61.7%) with a peak in the age group of 31 to 40 years (n = 29 or 55.7%). 15-30 years versus 31-40 years: Chi2=5.54; P = 0.18.

3.1.3. Socio-Economic Status

Socio-economic category IV is dominant (n =40 or 77%); category III (n=12; 23%), All her patients were unemployed.

3.1.4. Parity

The parity of the patients was distributed as follows: multiparous (n=36; 69%); primiparous women (n=16; 31%). The average parity was 4 with extremes of 1 to 11. There is a statistically significant difference (p<10-5) between the group of disadvantaged primiparous women and that of multiparous women.

3.1.5. Risk Factors

The following risk factors have been listed: Low socio-economic level (n=52; 100%); age>30 years (n=27; 52%); multiparity (n=36; 69%); hypoproteinemia (n=32; 61.5%); Pregnancy hypertension (n=17; 32.7%); anemia (n=16; 31%); twinning (n=4; 7.7%).

3.2. Clinical Aspects

3.2.1. Time to Onset of PPCM

The patients were distributed according to the period of occurrence of clinical manifestations of PPCM such as: last trimester of pregnancy (n=17; 32.7%); 5 months after delivery (n=35; 67.3%).

The majority of patients showed clinical signs of PPCM in the postpartum period with a statistically significant difference (chi = 12.46; P = 0.0004).

Of the seventeen women who presented clinical manifestations of PPCM in the last trimester of pregnancy, the delivery method was cesarean section. We listed as fetal complications: acute fetal distress (n=7; 41.2%); neonatal death (n=4; 23.5%).

We divided the patients according to the period of occurrence of clinical manifestations of PPCM in the postpartum period such as: ≤3 months (n=28; 80%); >3 months (n=7; 20%). Chi=25.2; p = 10-6.

In total, 35 patients presented clinical manifestations in the 5 months following vaginal delivery, with a high peak in the trimester following delivery (n = 28 or 80%). The average interval between Delivery and first clinical signs was 600 ± 42 days (range: 2 to 180 days).

3.2.2. Functional Cardiac Signs

Table 2. *Distribution according to functional signs of PPCM.*

FS	N	%
Class III and IV dyspnea	52	100
Cough	20	38,4
Palpitation	7	13,5
Hémoptyssis	2	3,8
Hépatalgia	30	57,7

3.2.3. Main Examination Signs

Table 3. Distribution of patients according to main symptoms.

Symptôms	N	%
Heart failure	52	100
left ventricular failure	39	75,0
global heart failure	13	25,0
Auscultation:		
Tachycardia	52	100,0
Galloping noise	51	98,0
Mitral regurgitation murmur (MI)	30	57,7
Tricuspid insufficiency (TI) murmur	16	31,0
Compilation		
Acute pulmonary edema	6	11,5
Pulmonary embolism	3	6,0

3.2.4. Main Examination Signs

Table 4. Distribution of patients according to main symptoms.

RCT	N	%
0,51-0,54	3	5,8
0,55-0,60	26	50
0,61-0,64	8	15,4
0,65-0,70	10	19,2
> 0,70	5	9,6
Total	52	100

3.3. Paraclinical Signs

3.3.1. Ray-X

Cardiomegaly (CMG) was noted in all cases. The mean cardiothoracic ratio was 0.61 ± 4 with extremes of 0.52 to 0.83; 23 patients had an RCT greater than 0.60 or 44%

Table 5. Distribution of patients according to radiological signs.

Radiological signs	N	%
Venocapillary stasis	44	85
Images of broncho pneumonia	8	15,4
Frank edema of the lungs	6	11,5
Single or bilateral pleural effusion	5	9,5

3.3.2. Electrocardiographic Data

Table 6. Distribution of patients according to electrical anomalies.

Electrical anomalies	N	%
Sinus rhythm	51	98
Atrial fibrillation	1	2%
Cavitary hypertrophy	23	44,2
Left ventricular hypertrophy	13	25
Left atrial hypertrophy	3	5,8
Diffuse repolarization disorders	43	82,7
Q wave of pseudo necrosis	1	2
Arhythmia	3	5,8
Ventriculair		
Ventricular extrasystoles	1	2
Complete left bundle branch block	2	3,8
Supraventricular		
Supraventricular extrasystoles	1	2

3.3.3. Doppler Echocardiographic Data

LV dilation (n=52; 100%); RV dilatation (n=40; 77%). LV ejection fraction ≤45 (n=43; 82.7%), LV shortening fraction < (n=39; 75%) IVC evaluated in 29 patients had a mean of 20±5.5 mm (range: 13 to 28 mm).

Pericardial effusion of minimal abundance was noted in six cases, i.e. 135 cases of mitral regurgitation by annular dilatation. 20 cases of Tricuspid regurgitation by annular dilatation. The average systolic pulmonary arterial pressure was evaluated in 24 patients and was 40.5 ± 20 (extreme): 28 to 60 mm Hg). Left intraventricular thrombus was observed in 2 cases or 4%.

Table 7. Distribution of patients according to the average of the anatomical measurements obtained.

Measurements	Average	Standard deviations	Extremes
LV (mm) diastole	60	± 19,1	50-69,6
systole	45,3	± 7	31- 56,2

Measurements	Average	Standard deviations	Extremes
LA	40	± 5,1	31- 50, 1
LA/OA	1,4	± 0,04	1,3 -1,9
RV (mm) diastole	30,5	± 7	17,2-46

3.4. Biological Datas

The blood count revealed normocytic normochromic anemia in twelve patients (31%) (tx Hb < 11g/dl), four patients had hypochromic microcytic anemia (8%), i.e. a total of 39% of patients who presented anemia in peripartum among these patients. In four (23.5%), delivery hemorrhage was noted. The average hemoglobin level was 7.5±2.2 (range: 3.3 to 10.7 g/dl). An inflammatory syndrome was noted in almost all patients with a sedimentation rate greater than 45 mm at the first hour and an average of 70 ± 13.5 (range: 45 and 102). The BNPs were not carried out due to lack of financial means on the part of the patients.

Renal failure (IR) with serum creatinine of 37.1g/l and creatinine clearance of 18.4ml/l in a 22-year-old primiparous woman with pregnancy-related hypertension (high BP 170/100 mm Hg). HIV serology was unfortunately only carried out in 21 patients. She was positive in 1 case or 2%. Hypoproteinemia < 60g/mg was only observed in thirty-two (32) patients, or 61.5%. The rest of the biological tests T4, TSH, proteinuria, viral serology could not be obtained.

3.5. Therapeutic Aspects

The treatment of PPCM is above all medical, different therapeutic protocols had been used associated with the low-sodium diet depending on the economic situation:

Protocol 1: Diuretics, ACE inhibitor/ARA2 and anticoagulants, Beta blockers

Protocol 2: Nitrogen derivatives, diuretics and beta-blockers

Protocol 3: Digitalis, Diuretic, ACE inhibitor, Anti aldosterone and Anticoagulants.

Table 8. *Distribution of diseases according to different protocols used.*

Protocols	N	%
Protocol N °1	5	9,6
Protocol N°2	17	32,7
Protocol N°3	30	57,7
Total	52	100,0

Protocol No. 2 was indicated in women presenting with PPCM in the last trimester of pregnancy. No. 3 in serious patients.

3.6. Evolutionary Aspects

The evolving aspects were considered from a clinical and radiological perspective. Indeed, echocardiographic checks of patients could not be obtained systematically.

Hospital development

The average length of hospitalization was 19 ± 12 days (range 5 to 102 days). Under well-monitored treatment, the evolution was favorable from the outset in 27 patients, or 52%.

Hospital morbidity: During hospitalization, we noted as complications: Repeated pulmonary embolisms in 3 patients (6%), Arterial embolism (acute ischemia of both lower limbs in one patient, i.e. 2%), Persistence atrial fibrillation in one patient, i.e. 2%And the delivery hemorrhage in 4 patients or 7.8. Ultimately, resolution of the initial heart failure affected 86.5% of patients, the cardio-thoracic ratio went from 0.61±4 to 0.50±1 (extreme 0.41 to 0.70).

Mortality: Two cases were noted, i.e. 3.8% deaths, one from pulmonary embolism, and the other from cardiogenic shock linked to arterial embolism.

The progressive modalities obtained for all of our patients: initial resolution (n=45; 86.5%); recurrences (n=5; 9.5%).

Favorable evolution according to the time of occurrence of PPCM: last trimester of pregnancy (n=15; 33.3%), after delivery (n=30; 66.5%). Chi = 10.0; P = 0.0015.

Medium-term evolution:

Hospital follow-up in outpatient cardiology consultation allowed us (on average 18 months):

14 women were lost to follow-up (27%) • 31 women remained stable (60%) • 4 cases of recurrent heart failure (9.6%) • 1 case of recurrence during pregnancy (2%).

4. Discussion

4.1. Epidemiological Aspects

4.1.1. Frequency

This series collected over 4 years shows a frequency of PPCM of 2.8% of total entries, it remained the same in our department (2.7%) more than 10 years ago [8]. However, it is comparable to that of other African authors [9].

This frequency is 9.7% compared to women of childbearing age. This rate is higher than that of Nkoua [10], Congo-Brazzaville (3.2%). This difference in frequency seems to be due to the fact that during the reference period most of the births admitted to the cardiology department had benefited from an echocardiogram with the diagnosis of PPCM. In addition, this frequency is 13 cases per year. A. NIAKARA [11] and NKOUA [10] find respectively (6.4 and 8 cases) per year.

4.1.2. Risk Factors

Age and parity

Peripartum cardiomyopathy mainly affects women after the age of 30 [12]. In our series the average age was 30.7±8.2 years. This figure is consistent with the results of the literature [12-14].

Elderly multiparous women represent 69% in our series with an average parity equal to 4. It is sensitive to those of A. NIAKARA [11] who found 3.88 and Machihude Pio et al who found 82.93% of multiparous women with a average parity of 3.07 [14].

In our series, all patients were black. Black race constitutes a risk factor recognized in the literature [15].

Socio-economic status

In the literature, the vast majority of cases of PPCM are observed in disadvantaged areas [16, 17, 29]. This observation is confirmed by our study with 100% cases.

Low socio-economic level is strongly implicated in the occurrence of PPCM. Indeed, social precariousness is responsible for malnutrition and therefore hypoproteinemia, vitamin deficiencies, and iron and trace element deficiency which can in turn lead to hemodynamic disorders responsible for PPCM [1]. However, it is also described to a lesser extent in patients with a high social level.

Pre-eclampsia

Pregnancy-related hypertension represents 32% of cases in our study, similar figures were found in the literature. Indeed, high blood pressure and pre-eclampsia are powerful risk factors. A meta-analysis published in 2013 brought together 22 studies including 979 patients diagnosed with PPCM. The prevalence of preeclampsia was 22%, more than 4 times the 5% of the general population [18].

These results prove that there is a close link between pregnancy-related hypertension and PPCM. Hypertension represents a risk factor that is all the more important and must be taken into consideration, especially as its prevalence is increasing in Africa. A very salty diet as required by African custom is likely to create the conditions for a hypertensive surge in the postpartum period [7].

Anemia

It represents 31% of cases in our series with an average hemoglobin level of 7.5±2.2 A. NIAKARA [11] found a high level of 10.7±1.7g/dI). Our results show that anemia is sometimes the cause of cardiac decompensation in the peripartum [20]: hence the need, according to certain authors, to provide iron supplementation to all pregnant women, given that the capital ferric is revised downward during this period.

Twinship

Twinning is poorly represented in our series (7.7%). Some authors admit that it is also one of the risk factors for this condition.

PPCM is more common in multiple pregnancies. In the previously cited meta-analysis, the prevalence of multiple pregnancies was 9%, compared to 3% in the general population [18].

Infections

HIV is not widely represented in our series. In the literature it is commonly accepted that there is a correlation between PPCM and HIV infection [19]. In these cases, we can discuss a concomitant cardiac attack with AIDS.

4.1.3. Etiopathogenesis

Taking into account the various risk factors mentioned above, it is clear that in the literature it is commonly accepted that PPCM is a primary dilated cardiomyopathy of unknown cause affecting white women in small proportions and black African women in large proportions. The latter most often have a precarious socio-economic level and therefore difficulties in accessing good living conditions and medical care could largely make this difference. We can also say that its etiology in our region is multifactorial and that only hypertension, anemia, malnutrition and infections seem to constitute serious areas of etiological research.

In these mice, cardiac cathepsin D (CD) expression and activity is enhanced and associated with the generation of a cleaved antiangiogenic and proapoptotic 16 kDa form of the nursing hormone prolactin [21].

However, recent data also support the probable hormonal hypothesis [18]: The first hypothesis is that of a vascular disease, linked to the strong hormonal influence at the end of pregnancy. The second hypothesis argues in favor of a genetic component after several observations [22, 23]. Other inflammatory, viral and autoimmune hypotheses have also been mentioned in the literature [25].

4.2. Diagnosis

4.2.1. Clinical Aspects

Time to onset of PPCM

The predominant period for the occurrence of clinical manifestations of PPCM is postpartum [26]. In the majority of cases (78%), symptoms appear during the first 4 months after delivery. In only 9% of cases, symptoms appear in the last month of pregnancy. 13% of women become symptomatic before the ninth month of pregnancy or beyond the first 4 months postpartum [27].

In our series, 67% of cases of PPCM occurred postpartum. However, the first three months following childbirth represent the predilection period for the onset of the disease; the literature is almost unanimous on this subject [14, 28].

Cardiac functional and physical signs

In our series, congestive heart failure dominates the clinical picture with 75% of cases. The same findings are described in the literature [14, 29-31]. It is important to point out here the preponderant place of late consultations linked to financial difficulties.

Inherent to this socio-economic category of patients. In addition, a certain number of symptoms, such as sloping edema of the lower limbs, exertional dyspnea, and orthopnea

may be wrongly attributed to pregnancy.

4.2.2. Paraclinical Aspects

Radiological data

In our series, thoracic cardiomegaly (CMG) was noted in 100% of cases. The average cardiothoracic ratio was 0.61±4. Our results are consistent with literature data [32, 33].

Electrical anomalies

They vary in the literature. Our series did not record any serious arrhythmias. However, in certain situations and dramatically, it is sometimes ventricular arrhythmias or sudden death that can lead to the diagnosis [34]. Also an American study which included 9841 adult women for a PPCM: among them, 18.7% presented ventricular arrhythmias, responsible for greater morbidity and mortality, longer lengths of stay, higher costs of hospitalization with more additional examinations carried out, compared to women who did not present arrhythmias [38].

Echocardiography

It showed dilatation of the LV and an alteration of ventricular function in all cases. This heart failure observed corroborates the data in the literature [10, 24]. Indeed, echocardiography is a tool of choice, making it possible to confirm left ventricular systolic dysfunction, to assess the degree of left ventricular dilatation, to quantify possible valvulopathies as well as possible associated right ventricular dysfunction and to search for a left intraventricular thrombus. It is of prognostic interest in cases of significant left ventricular dilatation (end-diastolic diameter greater than 60 mm) or severe systolic dysfunction of the left ventricle (ejection fraction less than 30%) [35].

Left intracavitary thrombosis was found in our series in 4% of cases. It should be noted in passing that in the literature PPCM is considered an emboligenic disease [35, 36]. Indeed, the combination of a hypercoagulable pregnancy state, intracardiac blood stagnation, endothelial dysfunction and possible immobilization would favor this thrombogenic state [39]. Curative anticoagulation therefore seems reasonable in cases of severe systolic dysfunction of the left ventricle (ejection fraction less than 35%) during the end of pregnancy and the first two months postpartum. Anti-Vitamins K are contraindicated during pregnancy [25].

The average pulmonary arterial systolic pressure (PAPS) was 40.5±20, which indicates pulmonary arterial hypertension observed in PPCM [26].

4.3. Processing of PPCM

The treatment of PPCM follows the conventional treatment of heart failure with low systolic ejection fraction; it is necessary to point out the role of prolonged rest from a low-salt diet which is one of the essential elements of the treatment.

The PPCM management should focus on three elements: reduction in pre-load, reduction in afterload, and increase in inotropy [40].

In the postpartum period, ACE inhibitors/ARA2 and mineralocorticoid receptor antagonists are also prescribed in European and other countries. On the other hand, beta-blockers and ivabradine were more often prescribed in European countries and diuretics, bromocriptine and digoxin were less so [37].

Beta-blockers can be used during pregnancy; selective beta-blockers of beta-1 adrenergic receptors are then recommended, in order to avoid the risk of uterine stimulation.

Diuretics and nitrates can be used during pregnancy, while paying attention to the risk of uterine hypoperfusion in the event of arterial hypotension [41]. Furosemide and hydrochlorothiazide are commonly used. Anti-aldosterones should be avoided during pregnancy, due to their fetal anti-androgenic effect. ACE inhibitors and ARBs2 are contraindicated during pregnancy, and only certain ACE inhibitors are authorized during breastfeeding [42]. Effective contraception is essential in these young women following a first episode of PPCM. In cases of persistent dysfunction, further pregnancy should be discouraged, and contraindicated due to the high risk of death.

In the event of cardiogenic shock, inotropes should be considered as a first resort.

In the absence of recovery of ventricular function, cardiac resynchronization may be offered, left ventricular assistance and, as a last resort, heart transplantation [43].

Prognostic and evolutionary modalities

PPCM is generally sensitive to conventional treatment of congestive heart failure, clinical signs disappear within a few weeks, cardiomegaly within a few months or even a year [7, 43].

The particularity of PPCM compared to secondary CMD is its possibility of definitive cure [43]. It was noted in 31% of our patients.

In our series, incomplete remission represents 9.6%; other authors find higher figures. A. NIAKA [11], finds 26.9%. This difference in rates is explained by the fact that 27% of our patients were lost to follow-up.

We recorded 2 cases of death (3; 8%), one linked to pulmonary embolism and the other to arterial embolism (acute ischemia of two lower limbs) and this during the hospital period. Mortality at 1 year was 4% in a large prospective North American study which followed 100 women treated for PPCM for 12 months [43].

This fatal outcome observed in our series precipitated by thromboembolic accidents confirms the data in the literature [19, 43].

In our series we recorded one case of recurrence during pregnancy. This seems to be explained by the fact that the rate of patients lost to follow-up is high, around 27%. And also by the fact that most of our patients in complete remission at 6 months had a stable recovery over time. These same observations were made by A. NIAKARA [19] in his series of 32 cases of PPCM, no recurrence of heart failure was noted.

In the event of pregnancy, strict clinical, echocardiographic and biological (BNP) monitoring is recommended. Furthermore, an in-depth discussion around the different possible contraceptive methods is essential.

Certain parameters are considered poor prognosis. These are African origin, age greater than 30 years, a time to onset of symptoms greater than 3 months, persistence of clinical signs 6 months after the onset of the disease, an ICT greater than 0.6 and the characteristics of the left ventricle: slight dilation (DTDLV < 55-60 mm), ejection fraction < 30%, shortening fraction less than 20% at the time of diagnosis [43].

5. Conclusion

This study stands out some evolutionary etiopathogenic particularities of Peripartum Cardiomyopathy. it is clear that in the literature it is commonly accepted that PPCM is a primary dilated cardiomyopathy of unknown cause affecting white women in small proportions and black African women in large proportions. The majority of patients showed clinical signs of PPCM in the postpartum period with a statistically significant difference.

Its etiologies in our region is multifactorial and that only hypertension, anemia, malnutrition and infections seem to constitute serious areas of etiological research.

Abbreviations

PPCM: Peripartum Cardiomyopathy
DTDLV: Left Ventricular Telediastolic Diameter
LVEF: Left Ventricular Ejection Farction
CMD: Cardiomyopathy Dilated
CMG: Cardiomegaly
AIDS: Acquiered Immunodeficiency Syndrome
HIV: Human Immunodeficiency Virus
IVC: Inferior Vein Cave

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Ferrière M, Sacrez A, Bouhour JB et al – Peripartum cardiomyopathy current aspects. Multicenter study: eleven observations. Arch mal Cœur Vaiss 1990; 83: 1563–9.

[2] Cenac A, Djibo A. Post-partum cardia failure in Sudanese – Sahelian Africa: Clinical prevalence in Western Niger. Am J. Trop. Med. Hyg. 1998; 58: 319-23.

[3] David NM, Parry EH, Peri-partum cardiac failure QJ Méd. 1978, 47: 431-61.

[4] Ritchie C. Clinical Contributions to the pathology, diagnostic and treatment of certain chronic diseases of the Heart Edinburgh M. and SJ 1849; 12: 333.

[5] Meadows WR. Idiopathic myocardial failure in the last minister of pregnancy and the puerperium. Circulation, 1957, 15: 903-73.

[6] Oakley C, Child A, Lung B et al. Task Force on the Management of Cardiovascular Diseases During Pregnancy of the European Society of Cardiology: expert consensus document on management of cardiovascular diseases during pregnancy. Eur Heart J. 2003; 24 (8): 761-81.

[7] Abdelmajid Bouzerda et al. Peripartum cardiomyopathy: about an observation and review of the literature. Pan African Medical Journal. Volume 25, Article 21, 26 Sep 2016.

[8] Solange F. Mongo Ngamami, Bertrand F. Ellenga Mbolla, Siama Nzaka-Sikou et al. Peripartum cardiomyopathy: epidemiological, clinical and prognostic aspects in the cardiology and internal medicine department of the CHU de Brazzaville (Congo). CAMES SANTE Vol. 2, N° 1, Juillet 2014; 69-73.

[9] Kane Ad, Mbaye M, Ndiaye MB, et al. Evolution and thromboembolic complications of peripartum idiopathic cardiomyopathy at Dakar University Hospital: a prospective study of 33 cases. J Gynecol Obstet Biol Reprod 2010; 39: 484-9.

[10] Nkoua JL, Kimbaly-Kaky G, Onkani AH, Kandosi S, Bouramoue C. Postpartum cardiomyopathy: about 24 cases. Cardiol Trop 1991; 17: 105–9.

[11] A. NIAKARA, BELENWIRE, et coll. Postpartum cardiomyopathy in black African women: epidemiological, clinical and evolutionary aspects of 32 cases. Cardiologie tropicale 2000. 26: 69-73.

[12] Kolte, D. et al. Temporal trends in incidence and outcomes of peripartum cardiomyopathy in the United States: a nationwide population-based study. J. Am. Heart Assoc. 3, e001056 (2014).

[13] Duran N, Günes H, Duran I, Biteker M, Özkan M. Predictors of prognosis in patients with peripartum cardiomyopathy. Int J Gynecol Obstet 2008; 101: 137–40.

[14] Machihude Pio, Yaovi Afassinou, Soodougoua Baragou et al. Particularities of peripartum cardiomyopathy in Africa: the case of Togo on a prospective study of 41 cases at the Sylvanus Olympio University Hospital in Lomé. Pan Afr Med J. 2014; 17: 245.

[15] Mielniczuk, L. M. et al. Frequency of peripartum cardiomyopathy. Am. J. Cardiol. 97, 1765–1768 (2006).

[16] Modi KA, Illum S, Jariatul K, Caldito G, Reddy PC. Poor outcome of indigent patients with peripartum cardiomyopathy in the United States. Am J Obstet Gynecol 2009; 201: 171. e1-5.

[17] Bahloul M, Ahmed MN, Laaroussi L et al. Myocardiopathie du péripartum: incidence, physiopathologie, manifestations cliniques prise en charge et pronostic. Ann Fr Anesth Reanim 2009; 28: 44–60.

[18] Bello, N., Rendon, I. S. H. & Arany, Z. The relationship between pre-eclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. J. Am. Coll. Cardiol. 62, 1715–1723 (2013).

[19] Arnould N, Diemunsch P, Raiga J, Brettes JP Gynecol obstét Fertil 2002; 30: 59-63.

[20] CENAC A., GAULTIER Y., SOUMANA I., et coll. Postpartum cardiomyopathy: clinical and echographic evaluation of response to treatment. Thirty cases from the Sudano-Sahelian region – Presse Méd. 1988; 17: 940–944.

[21] Hilfi ker-Kleiner, D. et al. A cathepsin D-cleaved 16 kDa form of prolactin mediates postpartum cardiomyopathy. Cell 128, 589–600 (2007).

[22] Horne, B. D. et al. Genome-wide signifi cance and replication of the chromosome 12p11.22 locus near the PTHLH gene for peripartum cardiomyopathy. Circ. Cardiovasc. Genet. 4, 359–366 (2011).

[23] Haghikia, A. et al. Phenotyping and outcome on contemporary management in a German cohort of patients with peripartum cardiomyopathy. Basic Res. Cardiol. 108, 366 (2013).

[24] Ware, J. S., Seidman, J. G. et Arany, Z. Shared Genetic Predisposition in Peripartum and Dilated Cardiomyopathies. N. Engl. J. Med. 374, 2601–2602 (2016).

[25] Sliwa, K. 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. 12, 767–778 (2010).

[26] ZABSONRE P, BAMOUNI J, FR FALL, DAOB, DYEM-KOUMA-Fx. Epidemiology of peripartum heart failure: 116 cases in BOBO - DIOULASSO – Médecine d'Afrique Noire 2000, 47(4).

[27] Lampert, M. B. et Lang, R. M. Peripartum cardiomyopathy. Am. Heart J. 130, 860–870 (1995).

[28] CENAC A., GAULTIER Y., SOURNANA I., HA ROUNAY, DEVELOUX M. Postpartum cardiomyopathy in the Sudano-Saharan region: a clinical and epistemological study of 66 cases. Arch. mal. Cœur vaiss. 1989; 82: 553-558.

[29] Katibi I. Peripartum cardiomyopathy in Nigeria. Hesp Med. 2003; 64 (4): 249.

[30] MBOULLET KOTTO R., BOUELET B. A. Postpartum cardiomyopathy in Douala: 12 observations card. Trop. 1999: 25: 98.

[31] Ntusi N, Mayosi BM. Étiologie et facteurs de risque de cardiomyopathie péripartum: une revue systématique. Int J Cardiol. 2009; 131(2): 168-79.

[32] Mandji LJM, Mayi S, Sima A, et al. Postpartum cardiomyopathy: About five cases in Gabon. Clin Mother Child Health. 2009; 6: 1037–41.

[33] Moioli M, Valenzano Menada M, Bentivoglio G, Ferrero S. Peripartum cardiomyopathy. Arch Gynecol Obstet. 2010; 281(2): 183-8.

[34] Diao, M. et al. [Electrocardiographic recording of long duration (Holter) of 24 hours during idiopathic cardiomyopathy of the peripartum]. Arch. Mal. Coeur Vaiss. 97, 25–30 (2004).

[35] Chapa, J. B. et al. Prognostic value of echocardiography in peripartum cardiomyopathy. Obstet. Gynecol. 105, 1303–1308 (2005).

[36] Duran, N., Günes, H., Duran, I., Biteker, M. et Ozkan, M. Predictors of prognosis in patients with peripartum cardiomyopathy. Int. J. Gynaecol. Obstet. Off. Organ Int. Fed. Gynaecol. Obstet. 101, 137–140 (2008).

[37] Sliwa, K. et al. Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy (PPCM): EURObservational Research Programme in conjunction with the Heart Failure Association of the European Society of Cardiology Study Group on PPCM. Eur. J. Heart Fail. 19, 1131–1141 (2017).

[38] Mallikethi-Reddy, S. et al. Burden of arrhythmias in peripartum cardiomyopathy: Analysis of 9841 hospitalizations. Int. J. Cardiol. 235, 114–117 (2017).

[39] Elkayam, U. Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis, and management. J. Am. Coll. Cardiol. 58, 659–670 (2011).

[40] Egan, D. J., Bisanzo, M. C. et Hutson, H. R. Emergency department evaluation and management of peripartum cardiomyopathy. J. Emerg. Med. 36, 141–147 (2009).

[41] Ghuman, N., Rheiner, J., Tendler, B. E. et White, W. B. Hypertension in the postpartum woman: clinical update for the hypertension specialist. J. Clin. Hypertens. Greenwich Conn 11, 726–733 (2009).

[42] Rasmusson, K. et al. Peripartum cardiomyopathy: post-transplant outcomes from the United Network for Organ Sharing Database. J. Heart Lung Transplant. Off. Publ. Int. Soc. Heart Transplant. 31, 180–186 (2012).

[43] McNamara, D. M. et al. Clinical Outcomes for Peripartum Cardiomyopathy in North America: Results of the IPAC Study (Investigations of Pregnancy-Associated Cardiomyopathy). J. Am. Coll. Cardiol. 66, 905–914 (2015).