Journal of Case Reports and Medical History

(ISSN: 2831-7416) *Open Access*

Case Report Volume 5 – Issue 3

Pregnancy with a History of Meadows Cardiomyopathy

Sanaa Benrahhal*, Yasmine Elyafi, Houdaigui Najlaa, Ehlali Driss, Bensouda M, Lamrissi A and Samouh N

Department of Maternity, Centre Hospitalo Universitaire Elharouchi Casablanca Morocco, Gynecologic Obstetric, Casablanca, Morocco

***Corresponding author:** Sanaa Benrahhal, Department of Maternity, Centre Hospitalo Universitaire Elharouchi Casablanca Morocco, Gynecologic Obstetric, Casablanca, Morocco

Received date: 10 Feb, 2025 | **Accepted date:** 05 March, 2025 | **Published date:** 14 April, 2025

Citation: Benrahhal S, Elyafi Y, Najlaa H, Driss E, Bensouda M, et al. (2025) Pregnancy with a History of Meadows Cardiomyopathy. J Case Rep Med Hist 5(3): doi https://doi.org/10.54289/JCRMH2500111

Abstract

The Meadows syndrome is characterized by systolic heart failure occurring in the last month of pregnancy or within the first five months postpartum **[1]**. The presence of severe cardiomyopathy is considered a contraindication for pregnancy. We report the case of a patient with a history of Meadows cardiomyopathy during her last pregnancy, with an impaired ejection fraction, who became pregnant again **[2]**. She presented with an infectious syndrome, severe anemia, and global heart failure, but had a good cardiac outcome, giving birth to a full-term newborn, and the pregnancy was without complications. The condition presents as heart failure associated with dyspnea as the main symptom. Treatment of peripartum cardiomyopathy (PPCM) follows that of dilated cardiomyopathy and systolic heart failure **[3]**. Early management is crucial to offer these patients appropriate treatment. Future pregnancies can only be allowed for patients who recover normal heart function.

Abbreviations: PPCM: Peripartum Cardiomyopathy

Observation

Peripartum cardiomyopathy (PPCM), also known as Meadows syndrome, is characterized by systolic heart failure occurring during the last month of pregnancy or within the first five months postpartum, in the absence of any known etiology or pre-existing heart disease. Severe cardiomyopathy is considered a contraindication for pregnancy **[4]**. Transthoracic echocardiography is the key examination, having triple diagnostic, prognostic, and monitoring interest. The degree of recovery of heart function mainly depends on the initial extent of the damage to this function **[5]**.

We report the case of a 28-year-old patient with a history of two living children delivered by cesarean section, currently pregnant with a 6-month intergenetic interval. During her second pregnancy, the patient developed meadows cardiomyopathy with a 15% ejection fraction. Now, at 8 weeks of pregnancy, she presented with an infectious syndrome, global heart failure, and severe anemia for which she was transfused. Her ejection fraction improved to 50%, with Pro BNP at 85 ng/l. She was placed on Aldactone, Cardensiel, and Tecpril. The patient was considered for a medical abortion, but after a collegial decision, the pregnancy continued as it was deemed a theoretical risk and the patient maintained a good ejection fraction (FEVG at 60%, 50%, 55%, 45%). She was complicated by anemia in the third trimester, requiring injectable iron transfusion. The patient

underwent cesarean delivery at term with cardiac and gynecological preparation and resuscitation. A tubal ligation was performed. The postoperative course was without anomalies, with a 50% ejection fraction, and she was discharged on Cardensiel and HBPM, with good cardiac evaluation.

Peripartum cardiomyopathy (PPCM) is a rare congestive cardiomyopathy. In 1971, Demakis et al. defined peripartum cardiomyopathy as heart failure appearing in the last month of pregnancy or the first five months postpartum in the absence of pre-existing heart disease **[6]**. In 1999, Judith et al. proposed incorporating echocardiographic criteria into this definition. It is more common in Africa than in Europe, occurring in 1 in 100 births in Nigeria, 1 in 299 births in Haiti, and 1 in 1000 births in South Africa, compared to 1 in 4000 births in the USA. This difference could be explained by the predisposition to risk factors for PPCM, such as age >30 or <20 years, multiparity, twin pregnancies, maternal obesity, hypertension, pre-eclampsia, and prolonged tocolysis. In the United States, Black women are three times more affected than White women, and their mortality rate is five times higher **[7]**. The etiopathogenesis of PPCM remains poorly understood. Many pathophysiological hypotheses have been proposed. However, there is growing evidence supporting the inflammatory theory, as observed by Melvin on myocardial biopsy showing characteristic myocarditis lesions in 60 to 75% of cases **[8]**. The condition presents as heart failure associated with prominent dyspnea (posing a differential diagnosis problem with pulmonary embolism), orthopnea, tachycardia, cough, OAP (acute pulmonary edema), and OMI (peripheral edema)—symptoms of dilated cardiomyopathy in global heart failure—and, less frequently, chest pain **[9]**. Cardiogenic shock may develop in hours, with hypotension, signs of peripheral hypoperfusion, and oliguria. Chest radiography is nonspecific but typically shows cardiomegaly **[10]**. The electrocardiogram may show sinus tachycardia, supraventricular (atrial fibrillation) or ventricular arrhythmias, left ventricular hypertrophy in twothirds of cases, and repolarization abnormalities in almost all patients. Conduction disorders are rare. Transthoracic echocardiography is the key examination, with triple diagnostic, prognostic, and monitoring benefits. It shows left ventricular dilation and decreased ejection fraction. It also helps identify complications such as intracardiac thrombus (found in almost 30% of cases), mitral and tricuspid valve insufficiency, and pericardial effusion (a sign of severity) **[11]**. Cardiac MRI may be performed for prognostic assessment of PPCM. In everyday practice, the treatment of PPCM is the same as for dilated cardiomyopathy and systolic heart failure. The anesthesia principles are to reduce afterload and stimulate contractility. Epidural anesthesia is suitable for this purpose, while spinal anesthesia is contraindicated due to its excessive sympathetic block. Mortality rates vary widely across studies, ranging from 2-3.3% in the most recent studies to 28% in older studies. A study evaluating the outcomes of 55 patients with PPCM found that around 62% of women showed improvement in their ventricular function, and 45% recovered normal ventricular function **[12]**. The degree of recovery of heart function mainly depends on the extent of initial damage to this function (left ventricular ejection fraction, end-diastolic diameter, fractional shortening) **[13]**.

Peripartum cardiomyopathy is a common condition in our country. Young, multiparous women are most at risk, and it most frequently occurs postpartum. The follow-up of these patients is marked by a high proportion of patients lost to follow-up, making it difficult to fully assess the social impact of this disease. Cardiomyopathy often goes unrecognized, leading to high morbidity. Early intervention is crucial to provide these patients with appropriate treatment **[14]**.

Conflict of Interest: No Conflict of Interest

References

1. Elkayam U., Jalnapurka S., Kealey AJ. Pregnancy associated myocardial infarction:contemporary in 150 cases between 2005 and 2011. JACC. 2012;59:A1174. [PubMed]
2. Demakis JG., RahimtoolS LI., Sutton GC. Naural course of peripartum cardiomyopathy. Circulation. 1971;44: 1053–61. [PubMed]
3. Judith U., Hibbard., Marshall Z., Roberto M., Lang. A modified definition for peripartum cardiomyopathy and prognosis based on echocardiography. Obstet Gynecol. 1999;94(2):311– 6. [Ref]
4. Ord RF., Barton JR., O'Brien JM., Hollingsworth PW.

Demographics., management., and outcome of peripartum cardiomyopathy in a community hospital. Am J Obstet Gynecol. 2000;182(5):1036–8. [PubMed]

5. Moioli M., Valenzano Menada M., Bentivoglio G., Ferrero S. Peripartum cardiomyopathy. ArchGynecol Obstet. 2010; 281:183–8. [PubMed]
6. Bahloul M., Ben Ahmed MN., Laaroussi L., Chtara K., Kallel H., Dammak H., et al. Myocardiopathie du péripartum:inci- dence., physiopathologie., manifestations cliniques., prise en charge thérapeutique et pronostique. Ann Fr Anesth Reanim. 2009; 28:44–60. [Ref]
7. Melvin KR., Richardson PJ., Olsen EG., Daly K., Jackson G. Peripartum cardiomyopathy due to myocarditis. N Engl J Med. 1982; 307:731–4. [PubMed]
8. Sliwa K., Fett J., Elkayam U. Peripartum cardiomyopathy. Lancet. 2006; 368:687–93. [PubMed]
9. Pearson GD., Veille JC., Rahimtoala S., Hsia J., Oakley CM., Hosen- pud JD., et al. Peripartum cardiomyopathy:national heart., lung and blood institute and office of rare diseases (National Insti- tutes of Health) workshop recommendations and review. JAMA. 2000;283: 1183–8. [PubMed]
10. G Vanzetto. Peripartum cardiomyopathy: A review. le praticien en anesthésie réanimation. Pôle cardiovasculaire et thoracique, clinique universitaire de cardiologie, centre hospitalier universitaire de Grenoble, BP 217, 38043.
11. Mielniczuk LM., Williams K., Davis DR., Tang AS., Lemery R., Green MS., et al. Frequency of peripartum cardiomyopathy. Am J Cardiol. 2006;97(12):1765–8. [PubMed]
12. Amos AM., Jaber WA., Russell SD. Improved outcomes in peripartum cardiomyopathy with contemporary. Am Heart J. 2006;152(3):509–13. [PubMed]
13. Gerbaux A. La cardiomyopathie dilatée primitive. Médicorama. These Paris 1962.Contribution à l'étude idiopathiques. Demange J. 1992; 294:92.
14. McKenna WJ., Maron BJ., Thiène G. Classification, épidémiologie et fardeau mondial des cardiomyopathies. Circ Res. 2017; 121:722 – 30. [PubMed]